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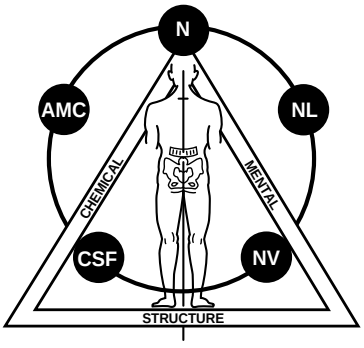

Message From the Chairman

Thomas Rogowskey, D.C., DIBAK

The members of the International College of Applied Kinesiology-U.S.A. are fortunate to share their insights, concepts and research through the papers presented in this issue of the Proceedings. The ICAK-U.S.A. continues to thrive as an "Arena of Ideas" through which members have the opportunity to present their observations and research results. These published works document the first steps toward the furtherance and development of the authors' hypotheses, concepts and procedural techniques. We invite all members to participate in this endeavor in the future.

Congratulations to all of our contributors. And a special thanks to Drs. Mark Duckwall, John Heidrich and Rebecca Hartle for all of their help during the review process. We look forward to seeing you at the Annual Meeting, June 17-20, 1999 in New Orleans.

Introduction

This forty-first collection of papers from members of the International College of Applied Kinesiology-U.S.A. contains 28 papers by 18 authors. The papers will be presented by the authors to the general membership at the Annual Meeting of ICAK-U.S.A. in New Orleans, June 17-20, 1999. 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 Publications Committee for originality and to determine that they follow the "Instructions to Authors" published by the ICAK-U.S.A. The primary purpose of the ICAK-U.S.A. in publishing the Proceedings is to provide an interchange of ideas to stimulate improved examination and therapeutic methods in applied kinesiology.

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

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

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

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

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

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

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

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

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

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

Following are the current requirements for papers submitted for publication.

1) 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.

2) Papers that do not include a clearly labeled Abstract, Introduction, Discussion, Conclusion and Reference List will be returned to the author for revision. Papers that discuss the outcome of a research study must also include separate sections labeled Materials/Methods and Results. Papers that describe clinical procedures or protocols should include a concise step-by-step outline or flow chart for each procedure described in the paper. The text of the paper, regardless of the subject material, should include numbered references. Note that the standard format for journal and textbook references is reviewed at the conclusion of this article.

3) Quotations must be short, usually no longer than three lines, and should be referenced, giving credit to the original author. All referenced articles, books, or persons other than the author must be properly referenced at the end of the paper. (See examples listed below.)

4) Any quotation of copyrighted material that is longer than that noted above must be accompanied by permission to print from the author and/or copyright holder. The permission must specifically note that the material is to be printed in The Proceedings of the ICAK-U.S.A., copyrighted by the International College of Applied Kinesiology-U.S.A.

5) Any material that is copyrighted by the author must include permission for the ICAK-U.S.A. to reproduce the paper and any accompanying graphs, illustrations, etc., at any time and in any manner that the ICAK-U.S.A. so chooses.

6) All artwork must be original, or permission to print must be obtained from the author or artist, referenced in the article, and a copy of the authorization sent along with the article at the time of submission for printing in The Proceedings of the ICAK-U.S.A. Photographs must be original black-and-white glossy prints. Do not scan photographs into your computer file.

7) Terminology or procedures that might be unfamiliar to some readers should be referenced at the end of the paper. Avoid using nontechnical terms such as, “blow-out,” “cleared,” “fixed,” or “TL’ed.” Papers that contain unsupported and unsubstantiated claims for efficacy of the therapy will be returned to the author.

8) The publication standards for the healthcare professions typically call for more details for the following types of papers:

   Research Studies - An investigation into the clinical efficacy of diagnostic and therapeutic procedures.

   Case Reports - An account of the diagnosis, treatment and outcome of an unusual or otherwise significant case.

   Case Studies - A comparative assessment of a series of related cases.

   Clinical Procedures - Informative papers that review the procedural aspects of diagnostic or therapeutic approach - clinical protocols.

   Hypothesis - A theory that explains a set of facts and presents a basis for further investigation.

   Clinical Observations - Unique observations that involve manual/mechanical muscle testing and related procedures.

   Commentary - Editorial-like, in-depth essays on matters relating to the clinical, professional, educational, and/or legal aspects of applied kinesiology.

   Critical Review - A critique or commentary on a paper that previously appeared in Division II of The Proceedings.

With the exception of a Commentary or a Critical Review, all papers must conform to the following format. Note that each section must be clearly labeled.

Title & Author's Name

Abstract: A brief description of the purpose of the study, basic procedures, main findings and principle conclusions.

Introduction: Summarize the rationale for the study or observation. Give background material when available and introduce the reader to what was done and why.

Materials and Methods: (for research studies) Describe the subjects, and identify the methods and procedures. Present sufficient detail to allow others to reproduce the procedures for comparison of results.

Results: (for research studies) Present results in a logical sequence and summarize the important observations. Include appropriate tables and illustrations.

Discussion: Discuss the implications of the findings and any limitations. Emphasize any new and important aspects of the findings. Discuss how the findings may relate to other relevant studies or observations.

Conclusions: Unqualified conclusions and statements not directly supported by data or observation must be avoided. Make any recommendations that are appropriate and relevant to the subject matter.

Summary of Procedures: Step-by-Step or Flow-Chart style description of diagnostic and therapeutic procedures described in the paper.

References: The numbered references that correspond to the text of the paper.

For journal articles: Author(s), Title in Quote " ," Name of Journal, Vol., No., (Month/Year).

e.g. Schmitt, Jr., Walter H., "Fundamentals of Fatty Acid Metabolism - Part II," The Digest of Chiropractic Economics, Vol. 28, No. 2, (Sept.-Oct./1985).

For textbooks: Authors(s), Title, (City of Publication, Name of Publisher, Copyright Date).

e.g. Walther, David S., Applied Kinesiology, Volume I - Basic Procedures and Muscle Testing (Pueblo, CO., Systems DC, 1981).

9) Authors are required to send articles to the Central Office on computer disk, IBM PC compatible format. (Articles not submitted on disk will be keyed at the author's expense of $5/page.) Disks should be sent to the Central Office in a padded envelope with the marking "Magnetic Computer Disk Enclosed" to ensure safe delivery. Disk labels must include type of software, author and document name. They must also provide a complete copy including all illustrations, flow charts and diagrams printed on 81/2 x 11 inch letter-sized paper. Papers without graphics, tables, and/or flowcharts may be attached as a word document on the official ICAK Email system.

10) Authors may only use text programs (i.e. Microsoft Word, WordStar, WordPerfect, MacWrite, etc.) to submit a paper on computer disk, but may use either IBM/DOS or Macintosh programs and diskettes. (Documents saved in a "page-layout" program are not acceptable.) The document for submission must be saved in a "text-only" format. All headers, footers and page numbers should be removed as should all italics, underlining, bold-face and any other special font formatting. If special formatting is required to preserve the tone of the paper, it should be present in the paper copy sent with the disk and it will be re-applied after the paper has been incorporated into The Proceedings of the ICAK-U.S.A.

11) Any computer-generated artwork to be included in the paper must be removed from the document before it is saved in the “text only” format. Artwork must be saved separately in either IBM/DOS or Macintosh formats as EPS, EPSF, GIF, TIF, TIFF, PIC OR PICT file types. The paper copy of the submission must include a print of the complete artwork and its correct placement. The artwork will be re-inserted into the paper in approximately the same location in the document, where space allows.

The articles to be published should be sent in duplicate (the original and one copy), to ICAK-U.S.A., 6405 Metcalf Ave., Ste. 503, Shawnee Mission, KS 66202-3929, ph: (913)384-5336, fax: (913)384-5112.

Applied Kinesiology Status Statement

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

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

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

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

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

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

- Myofascial dysfunction (micro avulsion and proprioceptive dysfunction)
- Peripheral nerve entrapment
- Spinal segmental facilitation and deafferentation

- Neurologic disorganization
- Viscerosomatic relationships (aberrant autonomic reflexes)
- Nutritional inadequacy
- Toxic chemical influences
- Dysfunction in the production and circulation of cerebrospinal fluid
- Adverse mechanical tension in the meningeal membranes
- Meridian system imbalance
- Lymphatic and vascular impairment

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

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

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

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

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

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

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

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

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

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

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

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

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

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Division I

Informative

Theraputic Regimen For a Patient Who Became HIV Negative

Dean B. McGee, D.C.

Abstract

The purpose of this report is to share procedures that were utilized in the resolution of a patient with Type A hemophilia who was HIV positive for over 7 years. The patient, who is now 17 years old, has been HIV negative for almost two years. Over this 9-year period, the patient's therapeutic regimen included soft tissue therapies, micro-current, nutrition, medications, diet, environment, prayer, faith, and family support. Each of these is felt to have played a significant role in his recovery. The author wishes to thank the following doctors for their contributions, in chronological order, to his understanding in providing care for this patient: Drs. Roy Swank, Ishak Shahied, Milton Dowty, George Goodheart, Walter Schmitt, David Walther, Michael Lebowitz, Rene Espy, Nancy McBride, Hulda Clark, and Joe Zollinger.

Introduction

AIDS was first reported in 1981, although there were cases in the United States as early as 1978. By 1985, over 7,250 cases had been reported. As of 1987, AIDS had surpassed hemorrhage as the leading cause of mortality among people with hemophilia. Although screening procedures have improved since 1981, the risk of contamination still remains. This is especially true for those, such as hemophiliacs, who are dependent on blood and/or plasma transfusions.[1] Since AIDS has been considered incurable to this point in time, the author believes that there is value in evaluating a case where a patient who previously had AIDS is now HIV negative.

The author does not in any way purport to be an expert in the management of HIV-related illnesses. Rather, his intention is to explore the spectrum of therapies that were used, both medical and chiropractic, with a three-fold purpose:

1) to document the complete resolution of a reportedly incurable disease
2) to present possible options to those who are managing similar cases
3) to provide hope for those in similar circumstances and faith in the ability of the human body to restore complete health

Materials/Methods

The patient's medical doctor, who is the HIV specialist at Kansas University Medical Center, utilized several lab tests at frequent intervals. The following tests were especially helpful in quantifying the patient's current status:

1) **CD4 level -** measures the number of Helper T-Cells per mm3 of blood. Normal range is 540 to 1600. If this value drops below 300, AZT is medically indicated. If it is below 200, the case is reportable to the Center for Disease Control as AIDS.[2]

2) **CD4:CD8 ratio -** measures the ratio of Helper T-Cells to Suppressor T-Cells. Normal range is 1.00 to 2.90.

3) **P24 Ag tests** - electrophoresis testing that consist of two tests:

   a) an investigational acid-wash test - breaks the bonding between the antigen and the immune complex so that it can be detected [2]

   b) a Coulter test - a straight antigen test - mainly used for early infections [2]

4) **HIV-1 RNA Viral Load count -** tests for the actual virus itself [2]

Discussion

1989

"Jerry" presented to our office in October of 1989 as an eight-year-old boy. For his chief complaint, his mother said, "He has hemophilia - I am hoping for help in keeping his immune system strong and possible exercises in strengthening his joints." Because of his hemophilia, Jerry had received plasma concentrates twice a week since he was five months old to raise his clotting factor VIII concentrations. At the time of his original visit, his mother may have known that he was HIV positive, but I was not informed of this for another five years. The hemophiliac concerns seemed legitimate enough by themselves, as hemophilia can cause disabling pain from multiple musculoskeletal complications.[1]

On his first visit, he was given Lacto+Plus (an intestinal flora product containing acidophilus, bifidus, and bulgaris), a homeopathic for candida (500X), and pyridoxal-5-phosphate to facilitate the citric acid cycle.[3]

1990-1991

Over the next two years, he was treated for a variety of musculoskeletal pains. The treatment was mostly on a symptomatic basis since he lived over 3 hours from our office.

In November 1990, he was first seen by the HIV specialist at KU Medical Center. He had been on AZT prior to that time. He had no thrush, but his tonsils were enlarged, and he had lymphadenopathy. His immune lab values were mildly depressed - his CD4 was 291, and his CD4:CD8 ratio (hereafter referred to as "his ratio") was 0.9. His weight was 72.5 lbs., which was above the 50 percentile mark for his age.

1992

In October 1992, I began to see Jerry on a more frequent basis. His weight was normal at 88 lbs., and his CD4 was virtually unchanged, but his CD4:CD8 ratio was down to 0.67. His mother wanted me to "check his whole immune system". His chief complaint was "bad headaches" which were worse when taking antibiotics. These were used on a frequent basis due to his compromised immune system. I prescribed a re-inoculation of the intestinal flora product, a combination homeopathic for candida (6X, 12X, 30X, 60X, 100X, 500X), and molybdenum.[4] The intestinal flora product was to be taken at a time of day other than when he was taking his antibiotics.

1993

Over the next few months, he had several unusual "injuries", including hurting his left elbow while sleeping, and pulling his left groin getting up out of a chair. In addition to soft tissue corrections, he was given a calcium supplement, Penta-Cal-Plus, for musculoskeletal support.

As of October 1993, he was still on AZT, and his weight dropped below the 50-percentile mark. It would stay in the mid-80 lb. range for five years. His CD4 was down to 218, and his CD4:CD8 ratio was down to 0.49.

1994

In February 1994, I started him on an immune support formula, and in March, he was again given the 500X candida homeopathic.

In June, I attended the ICAK 30th Anniversary Meeting in Chicago and was introduced to the work of Drs. Rene Espy and Nancy McBride, who were using micro-current to modify specific body responses.[5]

In July, he was given Lacto+Plus and Core Level Iron [4] to offset the negative effects of antibiotics he was taking, and Amino All to help with weight gain.

In October, I went to Cabo San Lucas for a weeklong seminar to learn the micro-current procedures from Dr. Espy and Dr. McBride. In that same month, Jerry's CD4 tested below 200 for the third consecutive time. As that was the Center for Disease Control's standard for AIDS, his diagnosis was changed from HIV positive to AIDS. That month was the first evidence in our records that we knew he was HIV positive. His CD4 was now down to 53, and his CD4:CD8 ratio was 0.33.

When I saw him in October, his chief complaint was his liver. He had a recent allergic reaction to some medication, and had a neuropathy that lasted four hours. His lab work indicated a macrocytic anemia (RBC count was 3.65, and MCV was 99.2). I prescribed Core Level B12, Core Level Thymus, Thymo-Lyph, Monolauric Acid, and Gentian (a Bach flower remedy). I also began the micro-current treatment for specific muscles associated with the thyroid, and then the thymus.

In November, the records from his medical doctor indicate that he was having allergic reactions to multiple drugs. Also, there was no more reference to the use of AZT beyond this point.

That month, I used micro-current for muscles associated with the stomach and the thymus. I added black current seed oil, and continued his intestinal flora product.

In December, Jerry and his mother went to Dr. Hulda Clark's clinic in Mexico. During that time, Jerry was put on her strict protocol,[6] and returned to my office with a large number of nutrients. Non-gustatory kinesiological testing procedures similar to those discussed by Lebowitz[7] were used to narrow down the nutrients to the following: Core Level Thymus, black current seed oil, Core Level B12, RNA/DNA, coenzyme Q-10, and black walnut (from a green hull source). He was to continue to avoid all products that produced parasitic proliferation per Dr. Clark's recommendations.[4] At that time, micro-current was also applied to the anterior and posterior abdomen to decrease parasitic activity.

Prior to Jerry's trip to Mexico, his mother had received a telephone call from the highest-ranking medical doctor in the state of Kansas. He told her that the clinic in Mexico would kill Jerry, and tried every possible argument to persuade her not to go. Several weeks after they had returned from Mexico, the doctor called her again to confirm that they had not gone. When she informed him that they had gone to Mexico, he hesitantly asked how Jerry was doing. When she told him that Jerry was doing great, he asked if he could call her back at another time. That weekend, he called her back and asked her for the name of the clinic - so that he could give it to a close friend who had a serious illness.

1995

In January, Jerry's CD4 count was down to 37 and his ratio was down to 0.28. This was his fifth consecutive CD4 below 200, and an AIDS report was filed at this time with the CDC. While I was on vacation, he saw another doctor who discontinued all of his previous nutrition and put him on an adrenal support product for fatigue that he was experiencing. When he returned one week later, his fatigue was worse, and he had run a fever all night at 102.8. I put him on monolauric acid to inhibit viral activity, and arginine to inhibit parasitic activity.[6] I told him to discontinue all red meats and use cold-pressed oils, for cellular support and for healthy myelin production,[8] and I advised him to eat a turkey sandwich each evening for his insomnia.

Over the next few months, Jerry felt better, but in April his weight was back down to 88 lbs., his CD4 was down to 8, and his ratio was down to 0.08. That month, we resumed his micro-current therapy, and started him on a 15C potency of a homeopathic called Aqua Pura, which is used for severe degenerative conditions.[9]

By May, his weight was up to 92.5 lbs., and his CD4 was up to 16. That month, a new lab test was used - P24 Ag. His acid wash was 64.0, and his Coulter was 32.0.

In June, his weight was down to 90 lbs., but he was doing better and playing 27 holes of golf each day.

By July, his weight was up to 93 lbs. His acid wash was down to 42.0, and his Coulter was negative.

In August, his CD4 and his ratio were unchanged from his May values, but his weight was up to 96.5 lbs., his acid wash was down to 27.0, and his Coulter was still negative.

In September, I saw him for a fall in Phys. Ed. class. He had landed on his right knee, and it was swollen and bleeding. I did soft tissue procedures for his right sartorius and right medial colateral ligament, and changed him to a LM/1 potency of Aqua Pura.

In November, he presented with extreme frontal headaches, which he had been having for the past two to three weeks. I did some cranial work and returned his Aqua Pura potency to 15C for one week, then discontinued it.

By December, his weight was up to 100 lbs., even though his CD4 was down to 9 and his ratio was still at 0.08. That month, his mother cancelled his appointments. Our notes don't indicate the reason ("he was feeling better at the time", weather complications, etc.).

1996

In January, his P24 Ag results were virtually unchanged.

Then in February, after canceling more appointments, he returned for ten visits. He had lost 12 lbs., and was suffering from loss of appetite, and stomach "burning" and "churning". He had been diagnosed with sinusitis following cranial X-rays.

The medical doctor had some knowledge that Jerry had been seeing me throughout this time, but this was the first time I was mentioned in her notes. She wrote, "weight 87 lbs. - has had a bad month. He has been seeing a kinesiologist, but is still on no medications . . . terrible fatigue and high fever . . . Hemophilia and AIDS worsening . . . His family still does not wish medications, and I see no sign of infection that I can document."

I ran micro-current for muscles associated with the hypothalamus to increase his appetite, and ran micro-current for conception vessel, stomach and gall bladder acupoints for his epigastric pain. Then I prescribed

Core Level Vitamin E and a LM/1 potency of Ferrum Phos (a homeopathic for inflammatory, febrile, emaciating, wasting conditions; “transparent skin type with the hemoglobin shining through”; sanguine constitutions; nervous, sensitive, anemic persons[9] - All of these described Jerry).

In March, his CD4 was down to 7, and his ratio was down to 0.07.

In April, another new lab test was performed: the HIV-1 RNA count - also referred to as a viral load test. Normal for this test is 400 units or less per 1 ml of plasma. His count was 600,000. At this time, we began a liquid mineral product called Sea Silver to supply enzymatic co-factors for his general metabolism.

In June, the medical doctor noted that she planned to discuss Retrovir and Crixivan after the next viral burden studies results. When the numbers came back, he was down to 400,000.

But by July, he was doing worse. His hair was falling out, which he and his mother associated with the high fever that he was running much of the time. The medical doctor’s notes said, “will give him another month to ‘get better’ and then repeat viral burden studies”. That month, I ran micro-current for spleen acupoints and for the chemical pathways involving the role of Vitamin A in immune function. I put him on Spleeno-Lyph, colloidal shark cartilage, the intestinal flora rebuilder, and an herbal / homeopathic immune formula that included echinacea, goldenseal, baptisia, phytolacca, watercress, fenugreek, propolis, and myrrh.

In August, his weight was up to 94 lbs., but now his viral burden load was up to 900,000, and his medical doctor prescribed Crixivan and Epivir. That month, he received micro-current for muscles associated with his thymus and lungs, and for his prostaglandin metabolism. He was also given Pit-Lyph-Whole, a pituitary glandular product.

By September, his weight was down to 89 lbs., and his WBC count was 1100. But he was feeling much better, and resisted being hospitalized at that time. I treated him with a timed micro-current therapy with the pads placed at the alarm point and the neurolymphatic point for the liver, with alternating frequencies of 0.9 Hz and 1 Hz. I use this procedure to “pump” the liver, and have found it especially useful in treating drug toxicities, hepatitis, and tonic labyrinthine reflex defects.

In October, his weight was up to 93 lbs., and he was feeling better enough to discontinue the steroids he had been on. I treated him with micro-current for his pituitary muscles. His medical doctor advised him to start Crixivan and Epivir. (It is not clear from the medical records if these were never taken when prescribed in August, or if they had been temporarily discontinued.)

On November 18th, his mother called me at 6:30 AM. Jerry was experiencing severe right kidney pain. Because of their distance from our office (over 3 hours), I referred him to the local emergency room. The X-rays taken showed blockage, and he was sent to a larger hospital in Wichita for observation and further evaluation. He was still in intense pain when they chose to leave the hospital to come to my office. What followed was one of the most dramatic responses I have experienced from localized soft tissue therapy. Digital pressure was applied just below the 12th rib. He went from screaming with pain, having an extended area of muscle hypertonicity, and an elevated core temperature, to a completely normal state within two minutes. To support his urinary system, I gave him cranberry concentrate, alfalfa, and Ness enzyme #5.

In December, he returned after a week of being in the hospital on antibiotics. He was running a constant fever, and had fallen in the hospital bathroom with an IV and hit his head. He had not eaten in a week, and he was not able to keep down his nutrition from his last treatment. I prescribed two liquid nutrients, Belladonna 30C for his fever, and Willow, a Bach flower remedy for the following: despondency and despair - resentment and bitterness with one’s fate - caused by over-compliance and suppression of emo-

tions - "the strong one."[9] (Throughout Jerry's illness, he had kept a strong front to protect those around him.) When he visited his medical doctor that same month, his right flank was asymptomatic, but he was now having severe left flank pain. The medical doctor discontinued the Epivir, Famvir, and Crixivan due to the nausea, vomiting, loss of appetite, and kidney stones.[10] His weight was back down to 90 lbs. However, his CD4 was up to 27, his ratio was up to 0.23, and his viral load was down to 850.

1997

In February, Jerry returned with severe back pain, which once again responded dramatically with localized soft tissue therapy. His weight was up to 97 lbs., he was out of the hospital, and he was not on any medications. He was doing better, but his viral load was back up to 80,827, so his medical doctor prescribed Epivir, Zerit, and Virocept. These were referred to as a "cocktail," and were to be taken together. Virocept was a replacement for Crixivan, which was believed to have caused the kidney stones. At the time, Virocept was not yet approved for children, but his medical doctor was able to get special permission to use it.

Because of their bad experiences with the previous "cocktail," his mother asked me to test the new medications. On previous visits, I had carefully explained to her that I was not licensed to prescribe medications, and that telling her to discontinue a medication was also outside my scope of practice. However, with her understanding of the situation, I had tested him before using non-olfactory procedures,[7] and when adverse responses were found I cross-referenced his symptoms with the Physician's Desk Reference. I then had her report back to his medical doctor what the symptoms were, and how they coincided with the timing of when he had taken his medicine. This was what we had done with the Crixivan. When I tested him with the new products, they tested individually as being toxic and having no potential benefit. However, when I tested them together, there was no evidence of toxicity and there was indication of significant benefit.

By March, he was having more severe back pain. Kinesiological testing[7] indicated that it was due to a new medication he had been given - Neupogen. His WBC count had dipped to 800, and the Neupogen was given to stimulate the bone marrow. The medical doctor felt the pain was probably due to the effects of the Neupogen on the bone marrow, and decreased it to once a week. She mentioned in her notes that month that he was "going to see his kinesiologist for ankle pain". That month, I prescribed Melanocentric Tyrosine and Alpha-Keto Glutaric Acid for his citric acid cycle,[3,11] and Core Level Magnesium for his back spasms.

At the time of his April visit, Jerry had spent the previous week in the hospital. He had a chronic sinus infection, he was running a 104-degree fever, and foods didn't taste right to him. His medical doctor had started him on Prednisone 20 mg QD the previous month, which stimulates the bone marrow, but also suppresses the immune system. On the positive side, his viral load was back down to 1393. My primary focus that month was on cranial work for the sinuses.

In April, his family attended a healing service at their local church. There they were given a message by a prophet that Jerry had been healed. Prior to that time, Jerry and his mother had faith that he would be healed, but from the time that the message was given to them, they had no doubt that he had been healed.

In May, he was running a fever of 104 degrees again, but his CD4 was up to 297, and his ratio was up to 2.09. Kinesiological testing indicated over-active thyroid function. Now that the immune system had ammunition to fight with, the thyroid was in overdrive trying to keep up with the demand. It seems that excess demand on the thyroid created a deficit in other areas of the triple warmer circuit, including a weakness of the right sartorius that produced a right Category II pelvic distortion. I adjusted his Category II and his cranials, and ran micro-current for localized musculoskeletal concerns.

That month, Jerry's medical doctor called them at home on a Sunday with the news that his viral load test had come back at less than 400, which meant the virus was non-detectable in the blood. She said that she had never seen anyone respond that fast to the medications.

By June, he was significantly improved. He was belching and gassy with bloating above the diaphragm, but his main concern is knee pain from a Jet Ski injury. My treatment that month involved peroneal reactivation procedures and PRYT corrections.

In July, his weight was up to 113 lbs. after taking Megace, a weight-gain medication. He and his mother made another trip to Mexico with no problems. His CD4 was down to 138, and his ratio was at 1.47. I made popliteus corrections for residual knee pain, and did some localized treatment for right ankle pain. He was also given Ness #8 for the joint pains.

In September, Jerry's viral load is once again non-detectable in the blood, but his CD4:CD8 ratio was down to 0.83. He had a poor appetite associated with food intolerance to Yellow #6, which was treated with micro-current.[5] He left the office with a good appetite and ate a large lunch. In the afternoon, I used micro-current to correct a spleen acupoint affecting his gait patterns.

Over the next few months, I worked on several soft-tissue extremity pains.

1998

In January, his viral load was non-detectable in the blood for the third consecutive time. His WBC count was much improved at 3400, his CD4 was 312, and his ratio was 1.79. Other lab values at that time included elevation of his BUN, uric acid, phosphorus, SGOT, GGT, and alkaline phosphatase.

In April, his weight was 121 lbs. He had left neck and shoulder pain the morning after playing golf, for which I treated his left clavicle. That same month, he weighed 127 lbs. when he visited his medical doctor. He was no longer taking Megace at that time, and her notes stated, "He looks better than I have ever seen him in his entire life".

A variety of neuromuscular corrections were made throughout the course of Jerry's care. I believe these were valuable in allowing the nervous system to focus on immune function instead of being preoccupied with nociception. I also believe they were valuable in helping Jerry and his mother to maintain a positive outlook on his health potential. However, I have only listed a few of these corrections because they were too numerous, and because I was not able to recognize a unique pattern to them that would be significant in helping others with HIV cases.

April 13, 1998 was the last time I saw Jerry, although I have continued to provide care for other members of his family, and have received updates on his condition.

As of December 28, 1998, his HIV-1 RNA was still negative, his CD4 was 512, and his WBC count was 5000. His current weight is 140 lbs. He still receives plasma transfusions, mainly if he feels he has injured himself (e.g. has twisted his hip playing golf), and in precautionary situations (e.g. before roller-skating). He still takes his Virocept-Epivir-Zerit "cocktail," and it is not known when he will be able to discontinue it. But he feels good, and is more active now than he has ever been.

His hemophilia specialist at Children's Mercy in Kansas City is amazed at how well his joints are performing. He told Jerry's mother that most young people with his condition have had joint replacements by this time.

Summary of Procedures

Nutrition

Anti-yeast products - homeopathics, flora rebuilders, molybdenum, & iron

Anti-viral products - monolauric acid, vitamin C

Anti-parasitic products - black walnut (green hulls), clove, wormwood, Arginine

General immune support - ISB complex, Core Level Thymus, Thymo-Lyph, Spleeno-Lyph, Echinaplex, Pit-Lyph-Whole, Colloidal shark cartilage

Citric acid cycle products - P-5-P, Melanocentric Tyrosine, Alpha-Keto

Musculoskeletal support - Penta-cal Plus, Amino All, Ness #8, Core Level Magnesium

Blood support - Core Level B12

Urinary support - cranberry concentrate, alfalfa, Ness #5

Cellular & neurological support - black currant seed oil, RNA/DNA, Co-Q-10, Core Level Vitamin E

General nutritional support - Core Level Health Reserve, Sea Silver

Bach flower remedies - Gentian, Willow

General homeopathics - Aqua Pura (15C & LM/1), Ferrum Phos LM/1, Belladonna 30C

Diet – No red meats or parasite proliferating ingredients

Increased cold-pressed oil intake

Turkey sandwiches at bedtime as needed

Environment – No parasite proliferating products (in shampoos, deodorants, etc.)

Neuromuscular corrections - as indicated

Microcurrent

For muscles associated with specific organs[5] - thyroid, thymus, stomach, hypothalamus, lungs, and pituitary

For frequencies associated with specific acupoints[5] - CV 8; ST 41, 3, 7;GB 20

For frequencies associated with specific metabolic pathways[5] - to enhance vitamin A & prostaglandin metabolism

For anti-parasitic effects - applied over the abdomen

For liver support - at the liver alarm & neurolymphatic points

For localized musculoskeletal corrections

Medications

Retrovir (a.k.a. zidovudine; previous known as AZT) - inhibits the enzyme reverse transcriptase to prevent the replication of HIV

Crixivan (a.k.a. Indinavir) - an HIV protease inhibitor adverse reactions: kidney stones, hematuria

Famvir - used to treat shingles (herpes zoster virus) adverse reactions: headaches & nausea

Epivir (a.k.a. Lamivudine) - initially used in conjunction with Retrovir; no results from controlled trials to date; currently used with Virocept & Zerit on an experimental basis

Virocept (a.k.a. Nelfinavir Mefylate) - an HIV protease inhibitor -replacement for Crixivan

Zerit (a.k.a. Stavudine) - inhibits HIV reverse transcriptase and inhibits viral DNA synthesis - replacement for Retrovir

Neupogen (a.k.a. GCSF) - stimulates bone marrow

Prednisone - an anti-inflammatory - inhibits bone marrow

Megace - an anti-neoplastic, progestational drug with a side effect of increasing body weight by increasing the appetite (not necessarily associated with fluid retention)

Personal faith and prayers

The faith, prayers, and support of others

Conclusion

Because this is a case report, instead of a controlled, double blind study, it would be impossible to delineate the efficacy of each specific procedure. Each of the above categories of procedures has reported cures associated with their usage. But in monitoring the chronology of the patient's progress, and the procedures associated with that time frame, there are four primary complications:

1) The number of variables at any given time - often new procedures were introduced simultaneously
2) The timing of the results - the positive and/or negative effects of each procedure may not have been immediately evident
3) The synergist effect of the procedures - the results may have been different if the procedures were used individually
4) The biochemical, structural, and electrical individuality of each person - the treatment that is effective for one person, both in the procedures used and in the sequence in which they are applied, may not be effective for another person

While this case report does not provide a formula for success for every HIV patient, it does provide viable options which may have value to others who are providing care in a similar situation.

Dr. Jack Kessinger makes extensive use of lab work, spirometry, and EKGs in his office to document the changes in his patients, but in his lectures he says, "You all are getting the same results in your offices - I'm just documenting it." This case report is simply a documentation of one of the many miracles of which we as health care providers have been privileged to be a part.

For anyone who saw Jerry during the time that he was coming into our office - frail, emaciated, wearing a ball cap to hide his thinning hair, groaning in pain and sometimes screaming - the transformation was nothing short of miraculous.

We practice in a litigious society that reminds us of the dangers of making claims about what we can do for our patients, especially our "terminally ill" patients. But we can be so overly concerned about not offering "false hope" that instead we offer something even worse: "no hope". I hope that someday the phrase "There's nothing more that can be done for you" will be grounds for malpractice. When treating patients, we may reach a point where we come to the end of what we are able to do for them at that time, but that doesn't mean that there is not a solution for their problem. As long as there is life, there is always hope!

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A Case Report of Neurological Disorganization

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Abstract

A male patient presented to my office complaining of low test scores and slow reading times due to diagnosed dyslexia and other learning disorders. This patient is currently a chiropractic student and is learning Applied Kinesiology (AK) in Los Angeles. He was interested to see if there could be anything done to help his problem by using AK methods and treatments. A complete and detailed history was taken and an AK exam was performed using muscle testing, cross crawl evaluation, and Neurolymphatic evaluation. The proposed treatment was for the patient to wear an 800 gauss, non-polar magnet on both Adrenal Neurolymphatic reflex points continuously (24 hours a day) for one month. The patient will be re-evaluated twice per week for the positive exam findings to see if there are any changes. The patient will monitor his reading time and his written examination time (to see if he needs less time to complete reading assignments and written exams) to determine if there are any decreases in them, as well as a decrease in the frequency of letter switching.

After the four weeks, patient showed considerable improvement in his reading time and his ability to retain the information that was read. In addition, the patient was able to read longer without becoming fatigued (reading 10 or more pages compared to just three). However, his written examination time was not reduced. Cross Crawl patterns and Homolateral Crawl patterns did not cause general muscle weakening.

In conclusion, wearing magnets may be a viable alternative to aid in the treatment of Neurological Disorganization. This case report suggests that using this type of treatment, in addition to traditional methods of Applied Kinesiology, can improve a person's learning ability and attention span. However, more research needs to be performed in this area.

Introduction

Dyslexia, as defined by Stedman's Medical Dictionary, is a level of reading ability markedly below that expected on the basis of the individual's level of over-all intelligence or ability in skills. Many children and adults are affected with dyslexia and other learning disabilities because of other problems that can be traced as far back as birth. Trauma, at birth, can lead to problems with proper cranial development and subsequent brain organization. Walther states that dysfunction in the cranial-sacral primary respiratory mechanism is a leading cause in learning diabilities.[1]

There are many causes of dyslexia and learning disabilities but there seem to be two main ones: functional hypoadrenia and neurological disorganization. Functional hypoadrenia has been detected as early as birth or can develop throughout life. There are many factors that initiate depletion of the adrenal glands, of which stress and poor diets lead the field.

According to Selye, there is a progression of how stress influences the adrenal glands and thus leads to dysfunction. He called this progression the "General Adaptation Syndrome (GAS)," in which there are

three stages of depletion.[1, 2, 3,] The alarm reaction is the first stage in this process, in which the body is in its "fight or flight" response. This is where the normal adrenals function to the point of hormone depletion. I find most people are past this stage. Stage two, or the Resistance Stage, is where the body goes into a continual state of fighting stress. The body is working to maintain a certain level of adrenal function in order to fight the stress but hypertrophy of the glands can be seen starting here. The third stage is the Exhaustion stage. Here, the prolonged stress placed upon the body eventually outruns the effective function of the adrenal glands. In today's world, we are exposed to stress many times per day. Therefore, the body's reaction to these stresses is to keep the adrenals in a constant resistance stage or even a constant exhaustion stage. Take this problem, add on a poor diet, and now the body can barely maintain any level of health.

Diet plays an important role in one's health, but in functional hypoadrenia diet is key. An example of a poor diet is one that consists mainly of carbohydrates and refined sugars. The average American consumes 150 pounds of sugar per year, which now places huge stresses on the body's blood sugar handling mechanisms. As a result of this increase, the body calls on its reserves within the adrenal glands, liver, pancreas, thyroid and pituitary gland. The increase in blood sugar causes a "panic" alert and an over stimulation of the blood sugar mechanisms, which can lead to a retarded system and the development of other problems.

Neurological disorganization is a decrease in the body's ability to communicate well with itself. It can be described that the right and left sides of the body are not communicating well with each other. This is seen between right and left brain activity where that patient can "switch" anything, from information to commands. Forcing an infant into using a particular "dominant" hand or not allowing an infant to explore outside of a crib or contained area can start this process as the infant develops. Other common factors that can lead to neurological disorganization are always being breast fed on one side, or always being carried on one side, or being taught to walk early. Walking early (if forced on by the parents) does not give the brain time to compose vital connections within itself. The connections are developed as the child crawls and when the body is ready, the child will start to walk.

Another area that can lead to neurological dysfunction is that of cranial trauma. Natural birth is very traumatic to the infant's cranium as evidenced by a "cone head" appearance of a newborn's skull. If these cranial faults are not corrected, then the primary cranial-sacral respiratory system does not fully develop. The brain relies on respiration and the movement of the cranial bones in conjunction with the sacrum and coccyx to move cerebral spinal fluid throughout the entire brain and spinal cord. Without this movement, proper nutrition to these tissues cannot be accomplished and the brain does not have a chance to operate at 100%.

Michael, a 31-year-old man, presented to my office with dyslexia and other learning disorders diagnosed by a psychologist in 1995. His initial complaints were dyslexia, trouble reading, and trouble taking exams.

The patient reported the following history. Michael was first tested for dyslexia, slow reading and basic phonics in 1987 while a student at California State University Long Beach (CSULB). He had originally noticed the problem starting as far back as age 11 where he showed trouble in his reading and English classes. Michael was always placed in less advanced reading and English classes that started in Junior High School and continued into college. CSULB was conducting a study of their own on these problems by using colored transparencies to cover reading material in an effort to decrease the glare off of white paper. The focus of that study was to see if the transparencies would reduce the glare that was thought to make reading more difficult. Michael stated that he continued using the transparencies until he graduated in 1990. However, he felt that they did not help much. Finally, in 1995, Michael was diagnosed again with dyslexia and other learning disabilities while studying for the Graduate Records Exam (GRE).

After Michael graduated, he worked as a financial analyst where he stated he switched numbers on his

reports of his first drafts. He then realized that numbers was not what he wanted to do, so Michael decided to become a physical therapist. While working as a physical therapist aid and sitting for the GRE, he continued having reading problems. In 1997, Michael decided to become a Doctor of Chiropractic and started school later that year. Currently, Michael is given extra time for his written exams and he still reports switching letters. He also says that his reading is very slow and his grades are not stellar.

Michael's past medical history is unremarkable except in the following areas: He currently wears reading glasses to help decrease the incidence of letter switching; he has had motion sickness since the age of 16; and has asthma. His asthma has been present since birth. Michael was born eight weeks early and had a collapsed lung, which led to pneumonia. He currently uses inhalers (Albuterol) to help with his breathing. He is presently a chiropractic student, he exercises 2-3 times per week, denies smoking or using elicit drugs, and drinks occasionally.

Michael is a 31-year-old male, who is 69" tall and weighs 160 pounds. His blood pressure was 134/84-mmHg seated and 150/90-mmHg standing. His pulse was 54 beats per minute and his respiration was 16 breaths per minute. His orthopedic examination was within normal limits with no decrease in lumbar ranges of motion and only decreases in cervical lateral flexion to the right and cervical rotation bilaterally. Michael's neurological examination was within normal limits with no evidence of cerebellar dysfunction or deep tendon reflexes. He was an alert, articulate individual.

Michael's Applied Kinesiology examination showed the following items: Posture evaluation revealed forward rounded shoulders accompanied by a slight anterior head carriage, a high right shoulder and low right mastoid process, a high right hip and a slight anterior lean of the entire body. He also showed the following weak muscles (as described by AK evaluation): The sartorious muscles bilaterally, the latissimus dorsi muscle on the right, the upper trapezius muscle on the right, the pectoralis major clavicular portion bilaterally, and the sternocleidomastoid muscles bilaterally.

Michael showed a hypertonic left psoas muscle as shown by a bilateral leg turn in test. Cross Crawl created general muscle weakness that was negated by a full breath in as well as a half breath in. Therapy localization to the adrenal Neurolymphatic reflexes bilaterally and the adrenal Neurovascular reflex negated the weakness, as did the Homolateral crawl pattern.

Michael's treatment plan consisted of using two 800 gauss, non-polar magnets (2mm in diameter). He placed these magnets, one on each side of the body, directly over the adrenal Neurolymphatic reflex point. He was instructed to wear them for 24 hours a day for a period of four weeks. In that time, Michael was instructed to watch for the following changes: First, did he notice a decrease in his reading time. In other words, was he able to read text, similar in length, in less time. Second, did he notice a decrease in the amount of letter switching while he was reading. Third, was there a decrease in his written examination time (i.e.: decrease in time for the same length test).

In addition, Michael was re-evaluated, in the office, twice a week during the four weeks. His cross crawl pattern and Homolateral Crawl pattern were evaluated on each visit to see if either produced general muscle weakness.

Discussion

Although four weeks is a very short time, there were some noticeable improvements with Michael's condition. Michael noticed that he was able to retain what he read after reading it once, thus decreasing his reading time approximately 10-15 minutes on average out of a three hour time period. This is a significant change since he has spent additional time, in the past, trying to learn a similar amount of material. Michael also noticed that he does not fall asleep after reading three pages, as he once did. In fact, he can now read over 10 pages without getting fatigued. Additionally, Michael has noticed that his class notes do not contain as many switched letters as they once did. However, his test taking time had not changed significantly. In relation to his Cross Crawl and Homolateral Crawl patterns, there was negation of general muscle weaknesses when performing these tests throughout the entire study.

Since this study was performed over a short period of time there has been discussion of continuing the treatment of magnetic therapy over the appropriate areas in addition to adding other treatments. Michael's improvement in his studies and neurological disorganization is very promising and should be continued further. It would be very interesting to see if magnetic therapy, as well as other interventions, would continue to improve his dyslexia and learning disabilities as time progressed. By using other treatment methods with in Applied Kinesiology, as well as magnetic therapy, one would hope to see further improvements with Michael's dyslexia and learning disabilities.

Conclusion

Neurological disorganization is a very prevalent problem that most people are unaware of. It is important to thoroughly evaluate each patient for this condition and make the necessary corrections. The positive improvements in this case report hopefully sheds some light into a different treatment arena that seems to be gaining a wider acceptance. Patient compliance seems to be a struggle in most practices. The hope behind this report is that it may help patient compliance in that wearing a magnet may be an easier treatment for some people. More research in different areas of magnetism should be performed to determine if there really are any long term benefits.

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The Effect of Omega Three Fatty Acids on the Prevention or Prolongation of Acute Episodes of Leg Pain in a 23-Year-Old Female Patient Who Has Been Diagnosed With Sacroiliitis Secondary to Ulcerative Colitis: A time series case report.

John G. Sherman, D.C.

Abstract

This study was conducted on a 23-year-old female who had been diagnosed with sacroiliitis secondary to ulcerative colitis. An AB time series design study was performed that lasted a total of 14 weeks. During the A phase, no intervention was done, and the subject's condition was tracked by using the Visual Analogue Scale (VAS) and repeated muscle testing (aerobic muscle testing) of the right psoas. The A phase lasted nine weeks. The B phase consisted of a five week intervention period where the subject supplemented her diet with omega three fatty acids once per day, as determined by standard nutritional testing methods in Applied Kinesiology. One tablet of the omega three fatty acids supplement contained the following: Marine Lipid Concentrate consisting of 60 mg of Eicosapentoaenoic Acid (EPA), 40 mg of Docosaheaenoic Acid (DHA); Borage Oil consisting of 15 mg of Gamma-Linolenic Acid (GLA); 107 mg of Olive Oil and 10 IU of Vitamin E. It was concluded that the use of omega three fatty acids did not aid in the reduction of acute episodes of leg pain.

Introduction

Ulcerative Colitis' (UC) etiology is unknown, but shares the same etiology as Crohn's disease. There have been many hypotheses tested (immunologic factors, infectious agents, viruses, chlamydiae and dietary factors), but none have been proven.[1] Epidemiologically, UC is seen in Western and Northern European areas as well as Anglo-Saxon ethnicities. Blacks, Hispanics and third world populations are at risk as well. There is no gender differentiation but it is more common among Ashkenazic Jews.[2] There is also a familial tendency that often overlaps with Crohn's Disease, which begins before the age of 30. However, the peak incidence of UC is between the ages of 15-30 with a possible smaller peak between the ages of 50-70.[1]

Sacroiliitis is a complication that arises from an inflammation within the body. It has been linked to Ankylosing Spondylitis (AS) and peripheral arthritis.[1, 3] Sacroiliitis is noted to cause fusion of the SI joints over time especially when linked to AS.[4]

A literature search was conducted in order to find any previous studies on the effects of omega three fatty acids on leg pain as a result of sacroiliitis. After an exhaustive search, no such studies were found either on Med Line or Chirolars. Sperling[5] concluded that dietary supplementation with omega three fatty acids may (partially) inhibit three pathways of the synthesis of lipid mediators of inflammation in patients with RA. Dekker-Saeys, et al.[6] established that inflammatory bowel disease is related to sacroiliitis and AS, as well as peripheral arthritis. It was shown that five out of 58 subjects who had UC were radiographically diagnosed with sacroiliitis. UC is a disease of unknown origin.

A 23-year-old female undergraduate college student, with a complaint of intermittent leg pain that was caused by sacroiliitis secondary to UC, was the subject of this study. The onset of the Ulcerative Colitis was 2/92, when she was hospitalized. Steroid therapy was initiated at that time, but a complete colonectomy was performed in 3/92 because of further complications. This was part one of a two part procedure. As recovery began, the subject had emergency surgery, in 4/92, to repair a perforated ulcer caused by steroid use. Finally, in 11/92, the second stage of the surgery was performed by a procedure called "endorectal illeal pullthrough," which reconstructs the distal end of the small intestine and re-attaches it to the external sphincter for normal bowel habits.

In 9/95, the subject was diagnosed with sacroiliitis secondary to UC. It was 5/95 when the subject started to complain of left-sided sacroiliac and posterior leg pain. The pain was "deep and achy" with a diffuse nature. The patient stated that the pain was intermittent but had episodes where the pain was sharp and stabbing to the point of "incapacitation." The pain increased when she sat most of the day but decreased with movement. There was no radiation of pain past the knee. Before the study, the pain was occurring, on average, twice per month.

With the onset of leg pain, the subject was placed on Azulfidine and Disalcid. The Azulfidine was for protection against antibacterial invasion of the intestinal pouch and arthritic pain control, and the Disalcid was for arthritic pain control. Informed consent was obtained for participating in this study.

An AB time series single subject study was conducted. The study began on 5/29/98 and ended on 9/3/98. The outcome measure used was the Visual Analogue Scale (VAS). The Visual Analogue Scale was a simple line that measured 10 cm long with NO PAIN on the left and SEVERE PAIN on the right. The patient was then asked to mark the line where she was feeling perceived. The VAS was then divided into 10 equal segments numbered 1-10 starting with one on the left and ten on the right. According to Hodgkins, et al., "The Visual Analogue Scale is considered one of the most reliable and sensitive pain rating methods currently available."[7, 8]

The first nine weeks of the study (A Phase) was our baseline, where the subject did not take any supplements. The patient marked a new VAS in the middle of the afternoon (2-3 PM) on Tuesdays. During the last five weeks of the study (B Phase), the subject took one tablet of omega three fatty acids just before bedtime. The tablets were taken for a total of 30 days, just before going to sleep. The omega three fatty acids supplement contained the following: Marine Lipid Concentrate consisting of 60 mg of Eicosapentoaenoic Acid (EPA), 40 mg of Docosaheaenoic Acid (DHA); Borage Oil consisting of 15 mg of Gamma-Linolenic Acid (GLA); 107 mg of Olive Oil and 10 IU of Vitamin E. The subject continued marking the VAS during the B Phase as before.

Results

The trends of the leg pain, both with and without supplementation, show no real effect. The patient's compliance of the supplementation and VAS charting was good. There were no reported missed weeks in charting her pain or missed nights with the supplements. There were also no side effects reported from the supplements.

Discussion

The patient showed no significant change in leg pain during the study. The VAS showed no significant change over the eight-week period, except for a flair-up during week two. Concurrent with this study, ESR was done during the base phase and it showed that no significant inflammation was present.

There were several factors that could have disrupted the internal validity of the study. Since the subject was medicated with Azulfidine and Disalcid for pain management, the medicines could have prevented leg pain from occurring. In addition, there was no inflammatory processes occurring at the time of the study, as shown by the ESR, which could render our supplementation useless. Furthermore, the subject's lifestyle factors, which could effect her condition, were uncontrolled. These included, but are not limited to, how much sitting/standing is done by the subject, how much or how little exercise is done by the subject, or how much driving is done or not. There are many other factors that could have caused the subject leg pain, but are not part of this study. The subject's compliance, ultimately, is unknown. It appeared that the subject was compliant in keeping accurate evaluations of the leg pain and taking the supplement, but the subject was not monitored for these activities. Finally, the study may not have been long enough or contain enough phases with different treatments to fully see if any other methods may seem helpful.

Ideas for future studies may include phases within which other treatments (Chiropractic Manipulative Therapy) are used. Using more than one subject for this condition may be helpful.

Conclusion

The use of omega three fatty acids to decrease acute episodes of leg pain in a female subject who has been diagnosed with sacroiliitis secondary to UC did not appear to be beneficial. More control should be used to increase the internal validity of the study by eliminating contributing factors to the leg pain. Further research is needed to see if other subjects with similar conditions could benefit from such treatment.

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Division II

Critical Review

Polarity, Meridians, and Consciousness

Bill Conder, D.C.

Abstract

A format is presented within the context of applied kinesiology techniques for evaluating the acupuncture meridians as a function of the polarity of the human body. Evidence is given to support the polarized current and field pattern as representing a governing principle which the nervous system and the meridians of traditional Chinese medicine abide. The way in which this polarity may relate to consciousness and sedation is discussed. Background information is presented.

Introduction

"Polarity,"[1] as with other words in our vocabulary, has many definitions many of which seem to have opposite meanings. The connotation of "polarity" that will be used in this article is the following: the direction in which a direct current flows. This definition is commonly used in the field of electricity.

Polarity, for the purposes of this article, signifies a one-way direction or gradient from higher to lower potential, not the idea of opposition. For example, we may think of a river that has its source in the melting snow of a mountain peak, and flows continuously to its end at the ocean. This river, as all rivers customarily do, flows always in the same direction, from mountain altitude to ocean sea-level. Moisture returns to the mountain as snow via a different route, that being evaporation of water over the oceans given certain atmospheric conditions, causing the formation of clouds which are carried by winds to the mountainous regions, where precipitation occurs, and so on. The mountain and the ocean are at opposite ends of the river but they do not oppose each other. In other words, the river flows out in one direction and in one form, and its source is replenished via a different route by different relationships. We can use this image as a metaphor to understand the polarized energy system observed in the human body-mind.

Robert O. Becker, M.D.'s work, especially as presented in his book The Body Electric[2] demonstrates a direct current in the perineural system of the human body. His measurement of this current and field pattern in/around the human body[3] makes it possible for us to interpret a kind of energy principle which is necessary for healing, consciousness - life. Furthermore, this principle retrieves aspects of certain traditional modalities, brings them up-to-date, and renews them.

Becker's research shows a polarized direct current in the perineural system and in other tissues. It shows, in frogs' lower extremity, a negative polarity in efferent motor nerves, a positive polarity in afferent sensory nerves, but a net negative polarity along the extremity as a whole.[4] Becker measured in humans some of the meridians and points of TCM acupuncture and found that they had electrical characteristics and that they seemed to have input into the central nervous system.[5]

Based on these observations of Becker, it is proposed here that the so-called acupuncture meridians are polarized in a specific pattern similar to the efferent and afferent polarization of the peripheral nervous system and that this specific polarity is necessary for healing and health.

Clinical Acupuncture Essentials[6] lists six different effects generally recognized as produced by acupuncture:

1. analgesia, or pain relief
2. sedation
3. regulation of homeostasis
4. immune enhancement
5. calming or tranquilizing (psychological)
6. motor recovery

Based on this list, it appears that the reduction of sensation, pain, or excitement is a primary effect of acupuncture and, presumably, other forms of meridian therapy.

Becker, in his discussion on awareness and anesthesia, reports the following:

1. passing a very small current through a salamander's head that cancelled its normal current caused unconsciousness;
2. chemical anesthesia dropped voltages in the head and could be cancelled by reinforcing normal currents;
3. currents in the human head varied with changes in consciousness just as in salamanders;
4. anesthesia and normal sleep reversed currents found in wakefulness in the human head;
5. the normal polarity of the head demonstrates a back-to-front current.[7]

From these observations, we might propose that a reversal of the normal polarity of the head tends to bring about unconsciousness and, in general, that a reversal of the normal polarity of an extremity tends to cause sedation or loss of pain sensation in the extremity. It might also be suggested that unconsciousness, sedation, and analgesia are analogous conditions.

TCM identifies 12 major bilateral and 2 midline meridians. There are three yin and three yang meridians in each of the four extremities. The yin meridians begin or end on the trunk; the yang meridians begin or end on the head. The upper extremity yin meridians are numbered from 1 starting on the trunk; the lower extremity yin meridians are numbered from 1 starting on the feet. Upper extremity yang meridians are numbered from 1 starting on the fingers; lower extremity yang meridians are numbered from 1 starting on the head. There exists a meridian-organ relationship; each meridian is named after an organ which it appears to affect. Chi/energy flows in a meridian only in one direction.

Applied kinesiology identifies a muscle-meridian relationship. Muscle weakness may be associated with imbalance in its associated meridian. In the standard applied kinesiology protocol, the meridian system is one of the 5 Factors of the IVF.

In addition to AK techniques, there are other methods that use systems of acupuncture points to derive information about the meridians and organs of the body. Electrical characteristics of the acu-points permit electrodermal diagnostic capabilities as demonstrated in Nakatani, Ryodoraku, and Voll technologies. Among the various electrodermal screening methods, different point systems are used for measurement.

"Five main groups of traditional points have been suggested for diagnosing visceral pathologies and energetic imbalances."[8] They are:

1. Jing-well/distal points on the fingers and toes,
2. yuan/source points on the wrists and ankles,
3. mu/alarm points on the chest and abdomen,
4. shu/associated points along the spine, and
5. Voll's points.

Dale lists several reasons for using the jing-well/distal points over the other point systems in electrodermal diagnosis. Some of these are appropriate to applied kinesiology methods including consistency of anatomical location, functional relationships with other meridians, and relationship of the points to the 24 hour cycle exchange.

For AK purposes, it might be pointed out that the mu/alarm points and the shu/associated points can be confused with other points and tissues that occupy the same anatomical area. Reference to another point system would be helpful in distinguishing energetic imbalances from, for example, active neurolymphatic points or vertebral subluxations.

The yin jing-well/distal points on the hand are LU11, HT9, PC9 and on the feet are SP1, KI1, LV1. The yang jing-well/distal points on the hand are are LI1, SI1, TW1 and on the feet are ST45, UB67, GB44.

Methods

The following has been observed by this researcher in normal, healthy patients who, for the most part, are free of muscle imbalance, subluxations, and nutritional deficiencies as would be determined by standard AK procedures:

1. Stimulation by patient or doctor TL, tapping, or soft laser of the yin jing-well/distal points always causes the positive neuro-musculo-physiological response* when the patient is awake, but never when the patient is asleep.
2. Stimulation by patient or doctor TL, tapping, or soft laser of the yang jing-well/distal points never causes the positive neuro-musculo-physiological response when the patient is awake, but always does when the patient is asleep.
3. Tracing an upper or lower extremity along its entire length distal to proximal (from finger or toe tips to trunk) always causes the positive neuro-musculo-physiological response.
4. Tracing upper or lower extremity along its entire length proximal to distal (from trunk to fingers or toes) never causes the positive neuro-musculo-physiological response, and often causes the strengthening of a weak muscle in that extremity.

(*This term, "positive neuro-musculo-physiological response," or positive n-m-p response, is used to designate either or both the weakening of an indicator muscle[9] and the lengthening of the right leg as a reaction to a specific stimulus. The leg length reaction is included here for the following reasons:

1. To demonstrate the function of the meridians while the subject is asleep, that is to detect a positive n-m-p response on the subject when it is not possible to perform a muscle test;

2. to show that the muscle test and the leg length reaction are like responses to the same stimulus and, therefore, have the same basis in neuromuscular physiology;
3. to elaborate its usefulness as a reference to the muscle test;
4. and to add to and promote the discussion of the leg length reaction in the AK context begun by Davis,[10] and others.

That meridian function is related to consciousness is shown by the following:

1. Positive n-m-p response with stimulation of yin jing-well/distal point and negative n-m-p response with stimulation of yang jing-well/distal point indicates proper polarity of the meridian within the overall net negative polarity of the extremity during wakefulness, and
2. negative n-m-p response with stimulation of yin jing-well/distal point and positive n-m-p response with stimulation of yang jing-well/distal point indicates proper polarity of the meridian within the overall net negative polarity of the extremity during sleep.

These observations may be interpreted in the following way: The meridian and nervous systems are polarized but independently of each other. Challenging the polarity of the extremity by tracing it from distal to proximal, which appears to challenge its net negativity, is a noxious stimulus that normally should evoke the n-m-p response. If it doesn't there is an imbalance that testing the muscles of the extremity (or shoulder or hip girdle) will reveal. Tapping the yin distal points normally causes the positive n-m-p response when the subject is awake, but not when he/she is asleep; tapping the yang distal points normally never causes the positive n-m-p response except when the subject is asleep; this suggests an association with the autonomic nervous system. Since tapping or indicating a point by TL may be a way of detecting positivity, one might say that, in wakefulness, the yin meridians are positively polarized like the sensory nerves.

Recommended Procedure

1. Challenge by tracing each extremity, one at a time, distal to proximal, and follow each challenge with an indicator muscle test. If the indicator muscle test is not positive - if it does not cause a weakening response - search by AK muscle testing and the Five Factors the cause of the polarity reversal; apply the appropriate treatment. Repeat the challenge until it evokes the positive response.
2. Challenge by doctor or patient TL, tapping, or soft laser the yin jing-well/distal points, one at a time, and monitor each by following with an indicator muscle test. If the indicator muscle test is not positive search the meridian's alarm, tonification, or sedation points for positive activity. Note that some jing-well/distal points double as tonification or sedation points for other meridians but, in any case, if it is a yin jing-well/distal point, it should cause the positive indicator muscle test. Failure to cause the positive response, in this case, indicates a need for treatment of the point or meridian. Tap out all positive tonification or sedation points.
3. Challenge by doctor or patient TL, tapping, or soft laser the yang jing-well/distal points, one at a time, and monitor each by following with an indicator muscle test. If the indicator muscle test is positive search the meridian's alarm, tonification, or sedation points for positive activity. The positive response in this case indicates a need for treatment of the point or meridian. Tap out all positive tonification or sedation points.
4. Rechallenge jing-well/distal points for proper polarity.

Discussion

The reversal of the normal cortical current in anesthesia and the manipulation of acupuncture meridians to induce analgesia and sedation seem to demonstrate a principle common to both, and that is polarity. This researcher has had the opportunity to evaluate subjects after traditional acupuncture treatment and observed a distinct pattern of what appears to be a re-configuration of the proper polarized arrangement of the meridian system. Also evaluated have been several subjects who have made use of so-called magnet therapy; these magnets appear to "work" by reversing the polarity of a meridian or meridians and inducing analgesia or sedation.

Analgesia, sedation, and increased pain threshold are the most remarkable effects of acupuncture. Acupuncture treatment, it should be noted, is applied according to signs and symptoms presented by the patient and observed by the doctor, and without the aid of feedback (as in the n-m-p response) from the subject of the treatment. It has been noted elsewhere[11] that increased pain threshold and decreased sensitivity are aspects of the body-mind's adaptive response to stress and less than optimal conditions, a necessary reaction to assure survival and continuity even as consciousness is dimmed.

As for acupuncture's reputed effect on the immune system, it must be pointed out that the absence of a potent immune reaction is not a sign of vitality but of immune suppression and low "energy;" distinguishing health as simply the absence of illness may be inaccurate. That immune enhancement can be confused with immune suppression is also found in the literature discussing the effects of dietary essential fatty acids: those promoting the use of such supplements say that they are absolutely necessary for health, while others say that they are toxic, immune suppressive, and have their effect by confounding oxidative respiration. Each side points to good, objective, scientific research to support its view.

It is proposed here that this so-called immune-enhancing effect of acupuncture is more an immune-modulating effect, that is it does not strengthen or energize the immune system but actually interferes with its full expression to make the immune reaction more tolerable, less painful. From this it may be hypothesized that acupuncture "works" to help cigarette smokers kick the habit, as it were, by sedating the physiological pain of frustration of withdrawal from addiction.

Conclusion

A general principle of how the body heals might be inferred from Becker's exploration of regeneration and bone fracture healing: in non-limb-regenerating higher vertebrates an injury produces a positive current in a negative field which positive current returns to negative with the healing of the injury. An aspect of the positive current is the relative shock or unconsciousness it provides the injured (in the same way traditional acupuncture creates analgesia), even as it extends the healing ordeal and limits it. Limb regeneration may be the price higher vertebrates pay for the shock/analgesia of injury, and the body's vitality/energy is the currency. It is proposed further, that the degree of shock is relative to the degree of injury and that many small or unnoticeable injuries work to limit immune function, healing, and consciousness via the same energy currency and may be cumulative.

This understanding may also shed light on the interdependence of the peripheral nervous system and the meridian system. Injury and pain confounds the polarity of the extremity, introducing a positive current, and causing the afferent/sensory component to override the efferent/motor component. Shock and analgesia mediated by local meridians access the survival mode of function from the autonomic nervous system and puts the injured on "automatic pilot." On a large scale, this adaptive response of the organism permits

flight from a life-threatening situation or the fight needed for defense. The toll is vitality/energy; one survives but healing remains to be completed.

The medium of this activity is the polarized electromagnetic current and field pattern of the human body. Restoration of the polarity pattern facilitates healing and consciousness. Sedation by reversal of the proper polarity pattern is a clinical decision that must be considered along with its side effects. Certainly, there are many occasions where it is appropriate to reverse or manipulate the body's energy system but this should be considered a temporary intervention.

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Evaluating and Treating Functional Hypothyroidism Utilizing Applied Kinesiology

Jeff Farkas, D.C., DIBAK

Absract

Although only a very small percentage of patients (approx. 3%) demonstrate thyroid hormone levels which deviate downward from the norm, much clinical evidence suggests that subtler forms of thyroid hypofunction are endemic. This paper reviews thyroid physiology, as well as standard methods for evaluating thyroid function. In addition, an argument will be made for the inclusion of functional thyroid evaluation, as well as the use of non-standard therapies, including those indicated by testing with Applied Kinesiology.

Discussion

Thyroid Physiology

The bi-lobular thyroid, located anterior to the trachea, is known to secrete 2 hormones, triiodothyronine (T3) and tetraiodothyronine, also called thyroxine (T4), significant for their roles in overall metabolic regulation, as well as calcitonin, a hormone involved in calcium and bone metabolism. Production of thyroid hormones reflect the only known role of iodine in human physiology, although the active concentration of iodine by cells of the gastric mucosa, salviary glands, choroid plexus and lactating mammary glands suggests other functions not yet determined. This may prove especially interesting with regard to central nervous system function, as the choroid plexus produces most of the plasma proteins bound for the cerebrospinal fluid, and where the T3 and T4 transport protein transthyretin is abundant.

The thyroid hormones increase the metabolic rates of all tissues of the body with the exception of the brain, lungs, spleen, testes and retina. In addition, growth rates in children, probably due to the effects on protein synthesis, are positively influenced by adequate thyroid hormone levels.

T3 and T4 are synthesized in the glandular cells inside the thyroid follicles. Within thyroglobulin molecules, each consisting of 140 tyrosine amino acids, oxidized iodine molecules will bind to tyrosine residues to form mono- and diiodotyrosine molecules. In a mechanism not fully understood but clearly regulated by thyrotropin (TSH), the thyroid-stimulating hormone secreted by the anterior pituitary, mono- and diiodotyrosine will combine to form T3 while, more often, diiodotyrosine molecules will couple to form thyroxine.

Most of the metabolic hormone secretion of the thyroid is in the form of thyroxine, with most of the remaining as T3. The functions of the tow hormones are thought to be qualitatively the same, the only differences being in rapidity and intensity of action. Triiodothyronine is about 4 times as potent as thyroxine, but exists in the blood in smaller quantities for shorter time intervals. There is a significant quantity of diiodotyrosine (DIT or T2) in circulation as well. Although current literature insists that it is inactive, DIT is the maturation hormone for frogs and was used in the 1940's as a standard therapy for hyperthyroidism in humans, suggesting that it might have some yet undetermined functions in humans as well.

Circulating thyroxine is deiodinized at regular intervals into “normal” T3 and “reverse” T3 (called reverse because of the removal of the iodine from a different location). Reverse T3 (rT3) is described as totally inactive. Interesting to note is the fact that non-diabetic, euthyroid individuals convert, via deiodinization, 36% of T4 into normal T3, while those same individuals, under fasting conditions, will convert only 18% of T4 into this active T3 form. This suggest a physiologic adjustment during fasting to accommodate the need to slow down metabolism and preserve resources. Significant is that euthyroid, diabetic patients (Type 1 Diabetes) demonstrate a T4 to active T3 conversion rate of only 12%. Other conditions under which rT3 is known to be produced in higher proportions include liver and renal disease as well as acute infections.

As standard blood thyroid hormone profiles do not differentiate between active T3 and the inactive reverse T3 form, the above would indicate that at least certain patient groups may have significantly less active circulation hormones, yet will still be classified as euthyroid. Wilson’s Syndrome (not the same as the copper metabolism disease) has already found it’s way into the literature to describe hypothyroidism mediated by improper ratios of T3 to rT3.

Also suggesting the existence of thyroid-related deficiencies in the absence of abnormal blood values is the documented existence of euthyroid hyperthyroxinemia, in which individuals have elevated thyroid hormone levels and normal thyroid function. While this conditions is certainly rare, one can apply a standard bell-shaped probability curve for predicting incidence. When rare, pronounced syndromes are found at the far end of the curve, more common, subtle variations of this syndrome are expected in the middle’ therefore, one would expect to find a greater number of patients with normal thyroid hormone levels with less than normal thyroid function.

As part of the standard blood testing for thyroid function, TSH or thyroid stimulating hormone (also known as thyrotropin) is also evaluated. This hormone increases well known activities of the thyroid glandular cells including: rate of trapping of iodide ions used in thyroid hormone metabolism, rate of iodination of tyrosine as well as the size and secreting activity of follicular cells.

A reverse-feedback mechanism exists to control the production of thyroid stimulating hormone and, therefore, thyroid hormone levels. As these levels rise approximately 75% above normal values, TSH levels are reduced to essentially zero. The feedback appears to work directly upon the hypothalamus which controls anterior pituitary production of TSH via thyroid releasing hormone (TRH) from the hypothalamus. However, animal studies demonstrate that the anterior pituitary is directly inhibited as well by elevated T3 and T4, probably by blocking the TRH receptor s from being stimulated to secrete TRH. It has been established that emotional stress can reduce the production of both TRH and TSH, both of which can result in diminished production of thyroid hormones.

Effects of Thyroid Hormone

Although it is understood that thyroid function is responsible for maintaining the rate of metabolic activity in most tissues of the bodies, the exact mechanism for this is not as clearly established. Some of the mechanisms of action which are under consideration make note that:

1. administration of T3 and T4 increase the size and number of mitochondria within most cells. The increases in mitochondrial cell surface area are proportional to increases in overall metabolic rates. One can deduce that the increases in mitochondrial number and activity influence the rate of ATP formation and therefore cellular function. On the other hand, it is also possible that the increases in mitochondrial energy production are actually mediated by a different effect of thyroid hormones, namely,

2. cells increase their rates of translation and transcription under the influence of thyroid hormone. This regulates the synthesis of cellular protein and ultimately the availability of enzymes which regulate cellular enzyme systems.

In any event, the up-regulation of ATP and cellular enzyme systems mediated by increased thyroid hormone levels stimulate some of the following body mechanisms:

1. Carbohydrate and fat metabolism-rates of absorption and utilization are increased by additional thyroid hormone. Glycolysis, gluconeogenesis and lipid mobilization are all enhanced by thyroid hormone levels.
2. Elevated basal metabolic rate-cellular activity and the production of heat are regulated by thyroid hormone levels.
3. Cardiac activity-thyroid hormone directly affects the excitability of the heart and therefore, heart rate.
4. Blood flow and volume-thyroid hormone produces vasodilatation which elevates blood flow and increases in circulatory blood volume.
5. Digestive function-thyroid hormone increases the rates of production and secretion of digestive acids and enzymes as well as the motility of the digestive organs.
6. Central nervous system function-CNS excitability is generally enhanced under the influence of thyroid hormone.
7. Muscle function-deficiencies of thyroid hormone cause muscles to become sluggish and slow to return to a state of contraction readiness.
8. Sexual function-the combination of effects of thyroid hormones on various organs, glands and their hormone production helps maintain the normal libido and sexual function in males, as well as normal libido, and menstrual cycles in women.

Increased Clinical Incidence of Thyroid Hypofunction

In his ground-breaking book Hypothyroidism: the Unsuspected Illness, author Broda Barnes alerts the reader to the growing clinical incidence of subtler forms of thyroid hypofunction. This theme has been repeated in other works from the same author, as well as various other researchers. The thesis which Barnes proposes is based upon the rising incidence of secondary manifestations of thyroid hypofunction, such as atherosclerosis and cardiovascular disorders, which were far less prevalent only a few years ago. Barnes attributes this development to a form of reverse Darwinism. In generations past, death by infection was the leading cause of premature death, with illnesses such a pheumonia, tuberculosis and influenza regularly striking in epidemic proportions. One of the known manifestations of thyroid hypofunction is susceptibility to viral and bacterial infections. Barnes proposed that, prior to the advent of antibiotic therapies, those individuals with less than optimal thyroid functions were those who succumbed to these illnesses. After the discovery of antibiotics, these individuals were able to survive often repeated bouts of infection, allowing the to

1. develop cardiovascular illness which, although it was virtually unknown previously, rapidly replaced infectious disease as the leading cause of death after the advent of antibiotic usage and

2. live longer and procreate, thereby perhaps passing on genetic thyroid weakness. This is supported by the known familial component observed regarding hypothyroid disease.

Other factors may also be involved in the increased observed clinical incidence of functional hypothyroidism. These include:

A. Competitive binding of halogens:

A look at the Periodic Table of the Elements shows us the iodine shares the same covalence (-1) as some other substances which are ubiquitous in our environment.

- fluorine is found in our water supplies as well as in the use as a poison
- chlorine is found in water in a vast array of chlorinated chemicals
- bromine is used in swimming pools and is used extensively as a food preservative

It certainly is not difficult to imagine that some of these halogens, in certain forms, might competitively bind with receptors which are designed to bind with iodine and iodine-containing hormones.

B. Hormone resistance

Insulin resistance is now a well documented medical condition in which hormone receptors to insulin are damaged and become non insulin-responsive. It should not stretch our imaginations to suppose that if a patient can have resistant cell receptors to one hormone, the same or another patient might have cells resistant to another hormone. Hormone resistance may be caused by auto-immune processes, oxidative damage, excessive incorporation of trans-or saturated fatty acids into cell structures or, simply, unfortunate genetics.

The overall increased presence of various environmental toxins and stresses including, but certainly not exclusive to, estrogen-mimicking chemicals, heavy metal poisoning, pollution, smoking, radiation and electro-smog has also been suggested as a source of irritation which may have a particular affinity for disrupting normal thyroid, as well as thyroid hormone receptor function. Recall that one of the previously proposed mechanisms for the action upon cells of thyroid hormone is via stimulation of the mitochondria. Medicine has, only recently, begun to understand the effect of oxidative stress on the ability of the mitochondria to function normally. Most of the above mentioned stress factors are considered oxidative stress factors which may suppress the mitochondrial capacity to produce energy for cellular function.

Signs and Symptoms of Hypothyroidism

Regardless if blood tests reveal a classic, pathologic form of thyroid hormone deficiency or not, presence of several of the following signs and symptoms strongly suggest the need to do a functional evaluation of thyroid function:

1. Cold Hands and Feet - in order to compensate for low core body temperature which is almost always present in hypothyroidism, blood will be shunted away from expendable extremities to the crucial vital organs. This loss of peripheral circulation has many consequences in including chronically cold extremities, and an overall sensitivity to the cold.
2. Fatigue-with cell enzyme systems operating at less than maximal efficiency, as mentioned earlier, the patient produces inadequate ATP and suffers from too little energy. Typically, these patients will have particular difficulty getting out of bed in the morning, when their metabolic rates at their at lowest levels, and report that many symptoms and general fatigue subside as their day progresses. They often

feel most improved after exercising, if they can motivate themselves to do it. In addition to fatigue, patients often complain of difficulty concentrating, as well as memory loss. They may also report morning headaches and dizziness which improve, as other symptoms do, during the course of the day.

3. Depression-although very often the patient will not admit to feeling depressed (lacking a traumatic life change, they lack a comparison reference to evaluate what depression is), appropriate therapy will often result in patients commenting on, in hindsight, how depressed they had been prior to therapy. A number of studies have documented that a significant (up to 50%) percent of psychotherapy-resistant, clinically depressed patients have been cured by intervention with thyroid hormone, even in euthyroid individuals.

4. Susceptibility to Infection-as mentioned previously, one of the known effects of thyroid hypofunction is susceptibility to repeated infections, particularly of the lung and airways.

5. Vit A Deficiency Symptoms-as the conversion of beta carotene to Vit A is thyroid hormone dependent, patients with subtle thryoid hypofunction tend to show signs and symptoms of subtle Vit A deficiency.

These include:
- night vision disturbances
- yellow-orange palms and soles (beta carotene deposition)
- follicular hyperkeratosis (small raised areas on the skin, usually not visible but easy to palpate, particularly on the skin of the posterior upper arm)

6. Irregular Menstrual Periods-particularly in the form of scanty bleeding with spotting between periods, often correlating with cervical dysplasia. This may also be due to Vit A deficiency, but correlates as well with insufficient progesterone, the production of which also requires adequate thyroid hormone. Low progesterone levels impede normal implantation and development of the fertilized ovum in the uterus, thereby suggesting the need to evaluate thyroid function in infertile women, particularly those who suffer from repeated miscarriages. Premenstrual syndrome also strongly correlates with incidence of hypothyroidism.

7. Hair Loss and/or Brittle Nails-be sure to differentiate between thyroid dysfunctions and other mineral deficiencies such as zinc and the iodine antagonist iron (if the hair loss is in a woman who also has heavy or long menstrual periods, then it is almost certainly due to iron deficiency). Very specific to thyroid hypofunction is loss of hair at the lateral eyebrow. In addition to brittle nails, patients often show nails with ridges and malformations, as well as repeated or stubborn fungal infections.

8. Edema-one of the classic signs of frank hypothyroidism is myxedema. The extreme forms which the practitioner is normally on the alert for are usually only found in endocrinology textbooks. More subtler forms of swelling around the eyes or the ankles, especially the non-pitting form, are also highly suggestive of the need to evaluate thyroid function.

9. Constipation-a sluggish bowel is a component of an overall hypofunction of the gastrointestinal tract, a function modulated to a great degree by thyroid hormone. This also includes inadequate production of stomach acids and pancreatic enzymes, which impedes optimal nutrient absorption, thereby exacerbating all other existing conditions.

10. Fibrocystic Breast Disease-this correlates with an increased tendency to produce fibrous or fibrotic tissue indicative of thyroid hypofunction. Clinically, the local use of saturated solution of potassium iodide (Lugol solution) is very helpful in this case, as well as in ovarian cyst production and DuPuytren contracture.

11. Skin Problems-probably due, at least in part, to the already mentioned decrease in peripheral vascular circulation, patients with decreased thyroid function tend toward skin problems of all varieties, including psoriasis and dermatitis. Often the clinical manifestation is as mild as dandruff or dry skin; however, often patients will experience severe cracking and bleeding of the skin on the hands and feet (usually the heels).
12. Cardiovascular Disease-elevated levels of cholesterol and triglycerides, both predisposing factors in the development of atherosclerosis and heart disease are clinical manifestations of hypothyroid function. Any abnormalities in carbohydrate of fat metabolism suggest the need to consider the thyroid as an etiologic factor.

Method

Diagnostic Parameters

In addition to the standard tests for thyroid hormones, evaluation of the TSH (anterior pituitary) and TRH (hypothalamus) can assist in determining if the thyroid dysfunction is rooted in the thyroid itself or results from insufficient stimulation from the pituitary or hypothalamus. As mentioned, the practitioner needs to consider that normal values may be completely inadequate to reveal subtle variations from optimal function in individual patients. Even seemingly innocuous findings such as T4 high in the normal range and T3 low in the normal range may be indicative of a subtle disturbance in T4 to T3 conversion, perhaps due to deficiencies of copper, zinc or selenium.

Seemingly euthyroid patients who nevertheless present with some or many, if not all, of the clinical signs and symptoms listed earlier need to be evaluated for thyroid hypofunction. For these patients, as mentioned previously, standard tests require supplementation in the form of functional analysis provided for reliably by

1. Basal Temperature Measurement and
2. Applied Kinesiology

Basal Temperature Measurement

Virtually all patients with depressed thyroid function will have a lower metabolic rate and, therefore, a lower core temperature. Although there are other means for establishing metabolic rates, they are either time consuming or expensive (hospital BMR evaluation or Achilles tendon reflex time test). Dr. Barnes considered temperature measurements to be a legitimate, reliable “poor man’s” version of these other tests.

Patients are instructed to prepare their digital thermometer (or shake down a mercury thermometer) the night before measuring, and measure the axillary temperature immediately upon waking the next morning. Axillary temperature has been shown to better correlate with core temperature than oral temperature. Rectal temperature, excepting cases of intestinal infection, also correlate well, but suggesting that patients begin their days in this fashion will not usually promote compliance. The measurement should be undertaken after the longest period of sleep and should be at approximately the same time every morning during the evaluation period. This period is a couple of consecutive days for women as well, being certain to wait until after the second day of menstruation. However, this type of test can be a reliable indicator for menstrual cycle hormone irregularities; therefore, it makes sense to put a possible thyroid dysfunction in the

context of an overall hormone evaluation by measuring throughout one entire cycle.

Normal basal axillary temperature is between 36.4 and 36.8 Centigrade (approx. 97.8 and 98.2 Fahrenheit). Properly measured temperatures which are below this level are very reliable indicators of depressed thyroid function, including in those patients who are already taking thyroid hormones. It is also worth mentioning at this juncture that this form of functional analysis is very useful, in conjunction with standard blood hormone level measurements, in determining the optimal dosage of thyrosuppressive medication in those patients with hyperthyroidism. These patients will often develop, as a result of the therapy, an array of hypothyroid symptoms if the dosage is too high, even if their hormone levels are in the normal range.

Applied Kinesiology

According to Goodheart, et al, use of standardized manual testing and associated diagnostic parameters as described in Applied Kinesiology texts, allow the examiner to draw reliable conclusions about associated organ function by properly ascertaining the functional integrity of specific muscles.

As an example, the Teres minor, an external rotator of the humerus, correlates specifically with the thyroid. When tested properly, a concept which incorporates specific

• patient position and stabilization

• examiner position

• timing and vector of eccentric elongation of tested muscle,

a hyporeactive (weak) Teres minor is a good indicator that the patient may be suffering from a disturbance in normal thyroid function. As any single muscle test can always be influenced by many variables, proper use of AK dictates that the examiner correlate the finding with muscle function of the contralateral Teres minor. Repeated hyporeactive Teres minor muscle finding as per AK, particularly if found in conjunction with the appropriate clinical signs ad symptoms, is highly indicative of thyroid dysfunctoin and strongly suggests the need to correlate blood thyroid hormone values with the basal temperature test described above in an effort to fully evaluate thyroid function.

In addition, by incorporating the concept of theapy localization (TL), it becomes possible for the experienced practitioner to quickly determine which glands are involved in a generalized endocrine imbalance, and the sequence in which they should be supported.

In this regard, of particular interest in evaluating the thyroid are:

1. Parotid Gland-one of the known long-term considerations in patients who have lost their parotid glands to surgery I s thyroid hormone deficiency. The salivary glands are one of the structures which also concentrate iodine. The deiodinations from food which is accomplished here apparently frees up iodine for use in other glands, specifically the thyroid.
2. Adrenal Glands-it is crucial to differentiate between those patients initially requiring adrenal support from those requiring thyroid stimulation. This is supported by one of the known clinical signs of frank adrenal insufficiency: sensitivity to thyroid hormone.

There is often an overlap of symptoms between functional adrenal weakness and functional hypothyroidism. In addition to the use of applied kinesiology muscle-organ-reflex procedures for differentiation, the clinician will observe some of the following hallmarks indicating the need to begin with therapy for adrenal insufficiency:

- fatigue with insomnia
- worsening of symptoms after exertion
- headache and dizziness precipitated by postural changes

As noted above, if patients respond with worsening of symptoms after introduction of therapy directed at the thyroid, it typically indicates the need to first provide nutritional, glandular and/or phytotherapeutic support for the adrenals. When this is appropriately done, thyroid support can be safely undertaken, and in some instances, found no longer to be necessary.

Therapy

Patients with frank deficiencies in thyroid hormone will require thyroid hormone, preferably in some combination of thyroxine with T3. Animal model studies have shown that thyroidectomized individuals will not attain pre-operation levels of T3 if supplemented with T4 alone, even in the presence of normalized TSH levels. This may have clinical implications for those patients receiving thyroxine hormone therapy, who often continue to demonstrate signs and symptoms of thyroid hypofunction.

Some patients are non-responsive to the synthesized T3 and T4 (either alone or in combination)prescribed for them. Often they will both test better (with applied kinesiology) and respond better clinically to desiccated thyroid tissue. This is commercially available from Armour (porcine) and Allergy Research (bovine). Wright and Gaby have suggested that the presence of DIT in these glandular preparations may be responsible for the approximately 30% of patients who respond better than to synthesized hormones. Although DIT is still considered to be inactive (in fact, even Armour does not list diiodotyrosine under the product's active ingredients), we should recall that other hormones which are now evaluated and utilized regualry, such as DHEA, were also considered biologically inactive and therapeutically useless for decades.

If patients are already taking synthetic hormones and the practitioner chooses to institute a trial with desiccated thyroid, the equivalent dosages should be considered when prescribing. For example, Armour thyroid is available in 1/2 grain and 1 grain tablets (each grain is the equivalent of approx. 61.4 mg of desiccated thyroid tissue). A 1 grain tablet is equivalent to approx. 0.1 mg of L-thyroxine (Synthroid) and 25 mcg. Of triiodothyronine (Cytomel).

Patients who have not previously used thyroid extract should begin with a trial dosage of 1/2 grain daily, which can, after 2 weeks be increased to 1 grain, if necessary. The practitioner should be cognizant of overdose symptoms such as shortness of breath, tachycardia and sleeplessness. A reliable way to screen patients for initial dosage is the "coffee test," as per Gaby. If patients report that they cannot sleep for many hours after a cup of coffee or espresso, they should begin with lower dosages of thyroid (1/4 grain). Those patients who can immediately sleep after caffeine ingestion can usually tolerate higher dosages without any side effects. Finding the proper dose will always negate any side effects which develop in response to too-high dosages of thyroid extract.

An effective therapy usually combines some combination of glandular extracts with nutritional and phytotherapeutic substances. The exact combination should be tailored individually, a process which is greatly facilitated by incorporating applied kinesiology.

The substances which should always be considered include:

1. Iodine-adequate iodine is required for hormone production. Although the minimum RDA is only .1 mg, Werbach suggests therapeutic dosages up to 1 mg/day. Iodine exhibits a bi-phasic function; therefore, excessive dosages will suppress thyroid hormone production and should be carefully avoided. Patients who present with a therapy resistant iodine deficiency should also consider avoiding foods which block iodine absorption (goitrogens). These include: peanuts, soy, millet, cabbage, mustard and turnips.
2. L-tyrosine-this amino acid combines with iodine to form the building blocks for T2, T3 and T4. As in all cases of amino acid deficiency, patients who require L-tyrosine should be evaluated for poor stomach acid production, a condition known to be particularly prevalent among elderly patients. Sometimes tyrosine deficiency is a manifestation of inadequate intake of the phenylalanine precursor.
3. Selenium, zinc and copper-these minerals are necessary for normal conversion of T4 to T3. Zinc, incidentally, is known to be one of those nutrients whose intestinal absorption ratio is reduced in the presence of hyperallergenic foods.
4. Essential fatty acids (EFA)-optimally functioning peripheral hormone receptor sites require healthy cell membranes. This is facilitated by the presence of high quality, pure essential fatty acids, particularly of the omega 3 variety. In addition, many of the skin conditions which plague chronic hypothyroid patients are responsive, in their own right, to EFA therapies.
5. Thyroid organ nosodes-these homeopathic organ preparations may simulate, via an unknown mechanism, activity of the corresponding organ.
6. Parotid glandular extract-as mentioned previously, insufficient parotid gland function predisposed the patient to thyroid hypofunction. In addition to suing glandular tissue, patients should be instructed to invigorate their parotid glands by chewing their food more thoroughly.
7. Fucus vesiculosis (Bladderwrack)-this form of seaweed is rich in fucose residues. There has been some speculation that certain hypothyroid cases are precipitated by infections caused by bacteria with cell markers similar to the fucus markers. By flooding the body with fucus, the plant residues bind to human cells with fucus markers, blocking the antigens of bacteria from attaching to the receptors. The theory is that, by this competitive inhibition, the bacteria and their antigens lose their attachment sites, and in effect, “slide” out of the body. This mechanism should function theoretically best for blood type O patients whose cell markers are richest in fucose residues and who statistically tend toward least efficient iodine utilization and thyroid hormone production.
8. High-potency multiple vitamin/mineral supplement-this is particularly important in patients receiving thyroid gland extracts. Thyroid hormone increased the rate of usage of various vitamins, particularly those involved in bone metabolism such as manganese, boron, Vit K, magnesium, zinc and copper.

The hyoreactive muscle can be repeatedly used as an indicator to determine which orthomolecular or phytoceutical substances will best promote optimal hormone production and conversion, as well as stability of peripheral hormone receptor sites. In a similar fashion, the tolerance of the various potential sources for thyroid hormone can be evaluated in those patients who may actually require hormone therapy.

By evaluating for a hyperreactive state in the Teres minor in response to the introduction of any potentially useful substance, the examiner is alerted to muscle reaction highly indicative of a hypersensitivity to that substance, ruling out it’s use in the therapy.

Determination of food allergies/sensitivities is almost always useful. As Breneman in "Basics of Food Allergy" points out, 60-70% of patients will have at least one symptom or manifestation of their problem disappear by eliminating foods which they do not tolerate. The combination of decreased overall stress and increased rates of absorption of various micronutrients, accomplished by 3-4 weeks of the appropriate elimination diet, often helps alleviate some of the signs and symptoms of thyroid hypofunction, particularly in the absence of abnormal hypothyroid levels. As it is often the case that patients, including but not exclusive to hypothyroid patients, suffer from allergies and sensitivities, it is always suggested that practitioners prescribe orthomolecular substances and phytoceuticals which are produced without the addition of unnecessary fillers, excipients, sugars and yeast which often prove hyperallergenic (see Addendum).

In addition, use of techniques incorporated and described in applied kinesiology literature, such as

- correction of vertebral lesions at thyroid function associated segments
- stimulation of thyroid-related reflex zones
- fascial flush of the Teres minor muscle
- pituitary drive technique
- set point technique for hypothalamic modulation

may all produce positive effects on thyroid function and status, when indicated. It is also recommended that patients incorporate an exercise program into their daily regimen. Care should be taken that the exercise be non-stressful and promote aerobic, fatty acid utilizing, biochemical pathways (Maffetone). This helps promote normal carbohydrate and fatty acid metabolism which tend to be disrupted in hypothyroid patients.

Addendum:

	Suggested Product	**Manufacturer**
Iodine	Algasan	Bioforce
	Iodine/tyrosine	Thorne Research
	L-tyrosine	Thorne Research
	Lugol solution (SSKI) *Lugol solution is only for topical application to fibrotic tissue	apothecary
L-tyrosine	L-tyrosine	Thorne Research
	D/L Phenylalanine	Thorne Research
Selenium	Selenium	Pure Encapsulations
Zinc	Zinc 30	Pure Encapsulations
Copper	Copper picolinate	Thorne Research

	Suggested Product	Manufacturer
Essential fatty	Super EPA	Thorne Research
Acids	Evening Primrose Oil	Pure Encapsulations
	Flax/Borage Oil	Pure Encapsulations
Organ Nosodes	Thyroidea Compositum	Heel
	Thyroidea D5	WALA
	Hypophysis G1	WALA
Glandular Extracts	TG 100 (thyroid)	Allergy Research
	Parotid	Allergy Research
	Thyroid	Armour
Fucus Vesiculosis	Fucus Similiaplexe	Pascoe
	Fucus Vesiculosis	Apothecary or herbalist
Multivitamins	Nutrient 950	Pure Encapsulations
	(with and w/o Fe and Cu)	
	+Cal+	Pure Encapsulations
Adrenal Support	Adrenal	Pure Encapsulations
	Cortrex	Thorne Research
	ADR Formula	Pure Encapsulations
	Licorice Extract	Apothecary or herbalist
	Pantothenic Acid (B5)	Nutri-West
	Ginseng	Apothecary or herbalist

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Lower Extremity Extraspinal Subluxation/Muscle-Syndrome Correlations

Timothy D. Francis, M.S., D.C., DIBAK, D.H.M.

Abstract

Various dysfunctions throughout the body may be caused from lower extremity extraspinal subluxations which can be correlated to specific muscle tests.

Introduction

Looking for the cause of a patient's symptom complex can lead a clinician to explore many possibilities. This paper discusses the structural component of the triad of health for the lower extremities on an extraspinal subluxation viewpoint.

Discussion

The following lower extremity extraspinal subluxations have been correlated over a twelve-year period of time to the following muscles and syndromes.

I. Hip

Subluxation	Muscle/Syndrome
A. Laterally Rotated Femur Head (Victor Frank)	1. Rectus Femoris (Victor Frank)

II. Knee

Subluxation	Muscle/Syndrome
A. Posterior Tibia (Proximal) (David Leaf)	1. Quadriceps (David Leaf) 2. Malabsorption
B. Medial Meniscus	1. Sartorius 2. Gracilus
C. Lateral Tibia	1. TFL 2. Biceps Femoris
D. Laterally Rotated Tibia	1. Popliteus
E. Inferior Patella	1. Genu Articularis

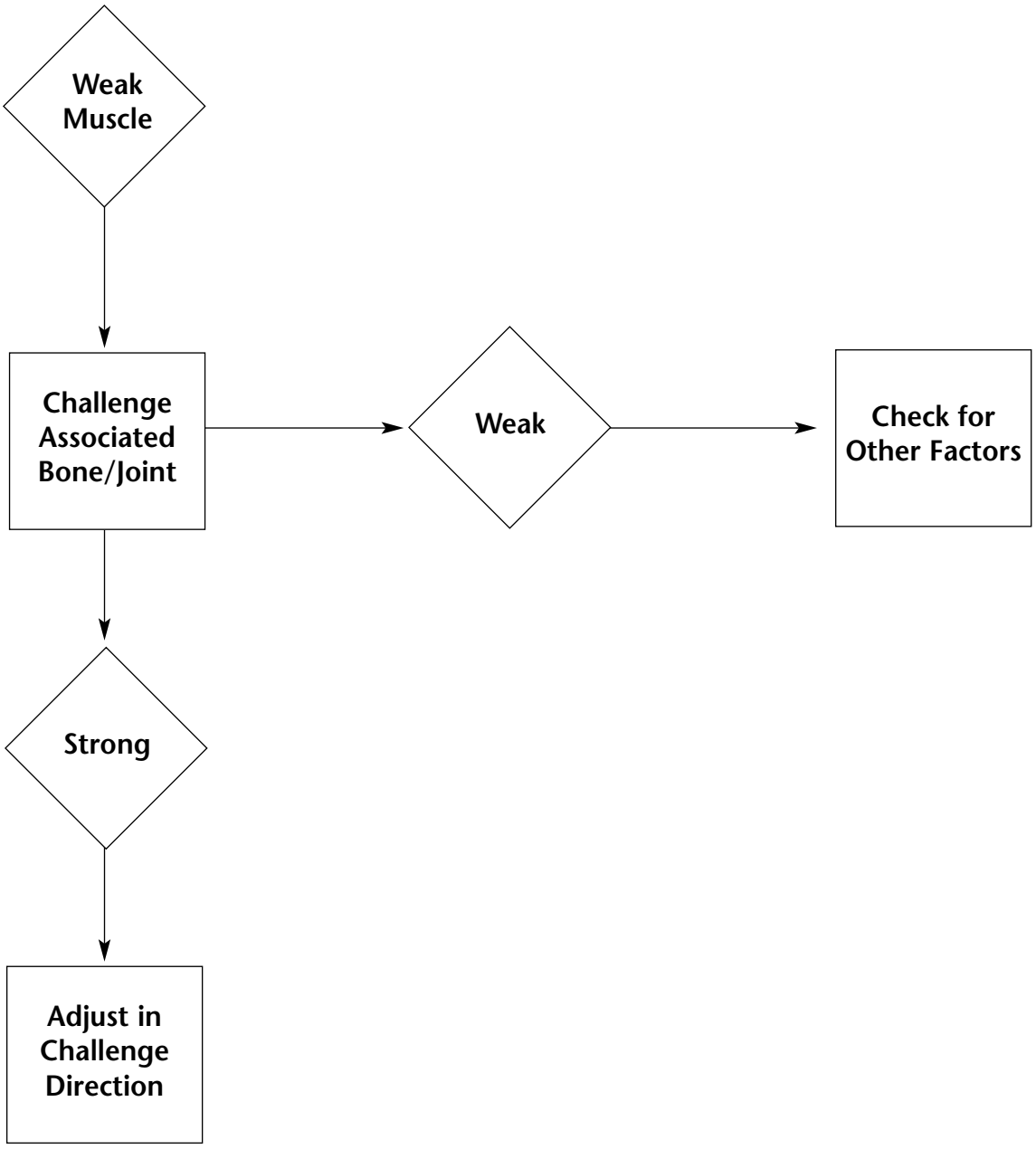
III. Foot

Subluxation	Muscle/Syndrome
A. Anterior Tibia (Distal)	1. Neck Flexors
B. Anterior Talus (David Leaf)	1. Neck Flexors (David Leaf)
C. Lateral Talus (George Goodheart)	1. Psoas (George Goodheart)
D. Lateral Cuboid (Edward Doss, Sr.)	1. TFL (Edward Doss, Sr.)
E. Medial Cuboid (Navicular) (George Goodheart)	1. Adductors (George Goodheart)
F. Posterior Calcaneus	1. Gastrocnemius
G. Inferior Navicular	1. Posterior Tibialis
H. Superior First Cuneiform	1. Anterior Tibialis/ Peroneus Longus
I. Superior Third Cuneiform	1. Supraspinatus
J. Rotated Fifth Metatarsal	1. Peroneus Tertius/Brevis
K. Dropped Metatarsal Head	1. Bilateral Gait Muscle Testing
L. Jammed Phalanges	1. Associated Meridian Involvement

Conclusion

These particular lower extremity extraspinal subluxation/muscle-syndrome correlations have been extremely useful clinically. Standard applied kinesiology therapy localization/challenge procedures may be utilized for diagnosis.

Lower Extremity Extraspinal Subluxation/Muscle-Syndrome Correlation Summary of Procedures



Lower Extremity Extraspinal Subluxation/ Muscle-Syndrome Correlations Summary

Timothy D. Francis, D.C., 1999

I. HIP	SUBLUXATION	MUSCLE/SYNDROME
	A. Laterally Rotated Femur Head (Victor Frank)	1. Rectus Femoris (Victor Frank)
I. KNEE	**SUBLUXATION**	**MUSCLE/SYNDROME**
	A. Posterior Tibia (Proximal) (David Leaf)	1. Quadriceps (David Leaf) 2. Malabsorption
	B. Medial Meniscus	1. Sartorius 2. Gracilus
	C. Lateral Tibia	1. TFL 2. Biceps Femoris
	D.Laterally Rotated Tibia	1. Popliteus
	E. Inferior Patella	1. Genu Articularis
I. FOOT	**SUBLUXATION**	**MUSCLE/SYNDROME**
	A. Anterior Tibia (Distal)	1. Neck Flexors
	B. Anterior Talus (David Leaf)	1. Neck Flexors (David Leaf)
	C. Lateral Talus (George Goodheart)	1. Psoas (George Goodheart)
	D. Lateral Cuboid (Edward Doss, Sr.)	1. TFL (Edward Doss, Sr.)
	E. Medial Cuboid (Navicular) (George Goodheart)	1. Adductors (George Goodheart)
	F. Posterior Calcaneus	1. Grastrocnemius
	G. Inferior Navicular	1. Posterior Tibialis

I. FOOT	SUBLUXATION	MUSCLE/SYNDROME
	H. Superior First Cuneiform	1. Anterior Tibialis/ Peroneus Longus
	I. Superior Third Cuneiform	1. Supraspinatus
	J. Rotated Fifth Metatarsal	1. Peroneus Tertius/Brevis
	K. Dropped Metatarsal Head	1. Bilateral Gait Muscle Testing
	L. Jammed Phalanges	1. Associated Meridian Involvement

References

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3. Walther, David, Applied Kinesiology: Synopsis. System DC, Pueblo, Colorado, (1988)

Upper Extremity Extraspinal Subluxation/Muscle-Syndrome Correlations

Timothy D. Francis, M.S., D.C., DIBAK, D.H.M.

Abstract

Various dysfunctions throughout the body may be caused from upper extremity extraspinal subluxations which can be correlated to specific muscle tests.

Introduction

Looking for the cause of a patient's symptom complex can lead a clinician to explore many possibilities. This paper discusses the structural component of the triad of health for the upper extremities on an extraspinal subluxation viewpoint.

Discussion

The following upper extremity extraspinal subluxations have been correlated over a twelve-year period of time to the following muscles and syndromes.

I. Shoulder

Subluxation	Muscle/Syndrome
A. Clavicle (Proximal Portion)	1. Subclavius 2. Neurological Disorganization 3. Frozen Shoulder
B. Scapula (Inferior)	1. Serratus Anterior 2. Frozen Shoulder 3. Lung Conditions
C. Gleno-Humeral Head (Anterior)	1. Rotator Cuff Muscles 2. Rotator Cuff
D. Gleno-Humeral Head (Inferior)	1. Subscapularis 2. Pre-Post Cordial Tap 3. Cardiac Lowback (TBM)
E. A/C Joint (David Leaf)	1. Mid-Deltoid and/or Upper Trapezius 2. Separated Shoulder (David Leaf)

II. Elbow

Subluxation	Muscle/Syndrome
A. Posterior Radial Head	1. Supraspinatus 2. Tennis Elbow
B. Medial Olercranon	1. Triceps - Anconeus 2. Golfers Elbow

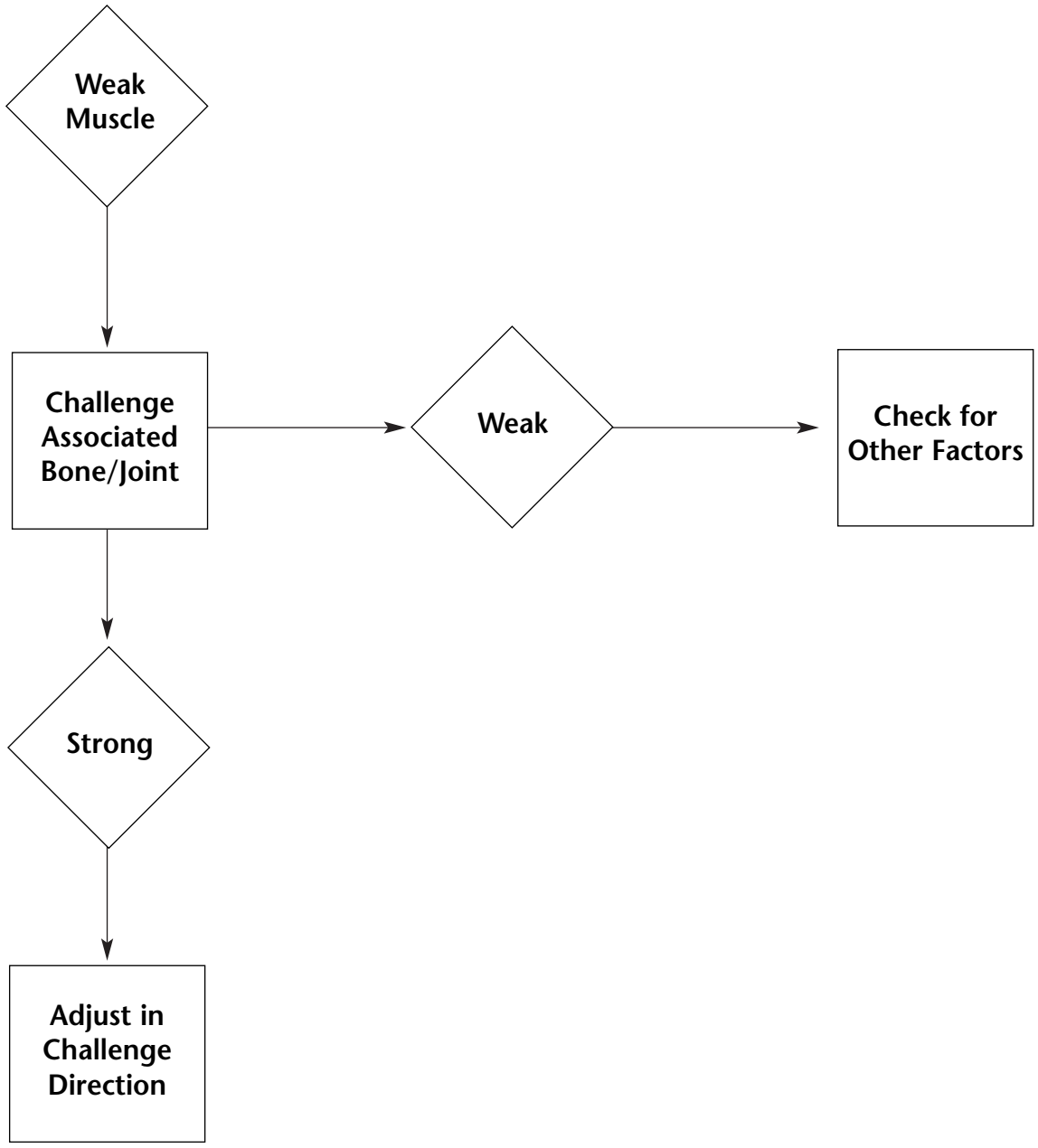
III. Wrist

Subluxation	Muscle/Syndrome
A. Scaphoid	1. Serratus Anterior 2. Frozen Shoulder 3. Lung Conditions 4. Stenosing Tenosynovitis
B. Jammed Carpals	1. Opponens Pollicus - Opponens Digiti Minimi 2. Carpal Tunnel

Conclusion

These particular upper extremity extraspinal subluxation/muscle-syndrome correlations have been extremely useful clinically. Standard applied kinesiology therapy localization/challenge procedures may be utilized for diagnosis.

Upper Extremity Extraspinal Subluxation/Muscle-Syndrome Correlations Summary of Procedures



Upper Extremity Extraspinal Subluxation/ Muscle-Syndrome Correlations Summary

Timothy D. Francis, D.C., 1999

I. Shoulder	SUBLUXATION	MUSCLE/SYNDROME
	A. Clavicle (Proximal Portion)	1. Subclavius 2. Neurological Disorganization 3. Frozen Shoulder
	B. Scapula (Inferior)	1. Serratus Anterior 2. Frozen Shoulder 3. Lung Conditions
	C. Gleno-Humeral Head (Anterior)	1. Rotator Cuff Muscles 2. Rotator Cuff
	D. Gleno-Humeral Head (Inferior)	1. Subscapularis 2. Pre-Post Cordial Tap 3. Cardiac Lowback (TBM)
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II. Elbow	**SUBLUXATION**	**MUSCLE/SYNDROME**
	A. Posterior Radial Head	1. Supraspinatus 2. Tennis Elbow
	B. Medial Olercranon	1. Triceps - Anconeus 2. Golfers Elbow**I.**
III. Writst	**SUBLUXATION**	**MUSCLE/SYNDROME**
	A. Scaphoid	1. Serratus Anterior 2. Frozen Shoulder 3. Lung Conditions 4. Stenosing Tenosynovitis
	B. Jammed Carpals	1. Opponens Pollicus/ Opponens Digiti Minimi 2. Carpal Tunnel

References

1. Frank, Victor, Dynamics of T.B.M. Workbook. Privately published. Sandy, Utah (1995).

2. Leaf, David, Applied Kinesiology Flowchart Manual, 2nd edition. Privately published, (1995).

3. Walther, David, Applied Kinesiology: Synopsis. System DC, Pueblo, Colorado, (1988)

Spinal-Rib Subluxation/ Muscle-Syndrome Correlations

Timothy D. Francis, M.S., D.C., DIBAK, D.H.M.

Abstract

Various dysfunctions throughout the body may be caused from spinal-rib subluxations which can be correlated to specific muscle tests.

Introduction

Looking for the cause of a patient's symptom complex can lead a clinician to explore many possibilities. This paper discusses the structural component of the triad of health from the spinal-ribs on a subluxation viewpoint.

Discussion

The following upper extremity extraspinal subluxations have been correlated over a twelve-year period of time to the following muscles and syndromes.

I. Spinal/Rib

Subluxation	Muscle/Syndrome
A. C6/7 Counterrotation	1. Sartorius/Gracilis 2. Adrenal Stress 3. Category II Pelvic Lesion
B. Lateral Occiput	1. Ocular Lock Pattern with the Eyes Held Up and Out to the Right, and/or Left 2. Hyoid Dysfunction
C. C2	1. Ocular Lock Pattern with the Eyes Held to the Right or Left 2. Hyoid Dysfunction
D. Inferior Occiput	1. Ocular Lock Pattern with Eyes Held in Straight Superior Direction 2. Neurological Disorganization 3. Hyoid Dysfuction 4. Hiatal Hernia 5. Psoas Imbalance 6. Gait Imbalance

Spinal/Rib continued

	7. Category II Pelvic Lesion 8. Short Leg 9. Rib Torque (Victor Frank) 10. Anterograde Lymphatic
E. C5/Anterior C5 (Victor Frank)	1. Bilateral Latissimus Dorsi (When tested together) 2. Blood Sugar Dysfunction (Victor Frank)
F. C3	1. Deltoid/Serratus Anterior/Coracobrachialis 2. Lung Conditions
G. Anterior T2	1. Subscapularis 2. Pre/Post Cordial Tap 3. Cardiac Lowback (Victor Frank) 4. Emotional Broken Heart Syndrome (Howie Cohn) 5. Blood Sugar Dysfunction (George Goodheart)
H. Anterior T5/ T5 Rib Head	1. Bilateral PMC 2. Temporal Bulge
I. Anterior T7/ T7 Rib Head	1. Gluteus Maximus/Medius/Minimus 2. Adductors 3. Piriformis 4. Gonadal Hormone Related Imbalances
J. Second/Third Rib Heads	1. Sartorius/Gracilis 2. Adrenal Stress
K. First Rib	1. Almost all Gonad Related Muscles Gluteus Maximus/Medius/Minimus/ Piriformis/Adductors 2. Gonadal Hormone Imbalances
L. Anterior T10/ T10 Rib Head	1. PMS (Pectoralis Major Sternal) 2. Liver Dysfunction
M. Anterior L1	1. Bilateral PMC 2. Temporal Bulge
N. Anterior L3	1. Quadriceps 2. Spastic ICV 3. Failure to Pass Deep Tendon Reflex Test 4. Allergies 5. Digestive Disturbance

Spinal/Rib Continued

O. Base Posterior Sacrum	1. Ocular Lock Pattern with Eyes Straight Inferior 2. Retrograde Lymphatic 3. Pectoralis Minor 4. Bilateral Piriformis (When Tested Together) (Victor Frank)
P. Apex Posterior	1. Ocular Lock Pattern with Eyes Held Straight Superior 2. Anterograde Lymphatic
Q. Posterior Sacrum	1. Ocular Lock Pattern with Eyes held Down and to the Right or Left 2. Piriformis
R. Pubes	1. Adductors (George Goodheart) 2. Incontinence
S. Coccyx	1. Coccygeus/Ileococcygeus/Pubococcygeus 2. Incontinence

In reference to the inferior occipur (letter D); this is usually corrected with an alpha-biotic alignment procedure.

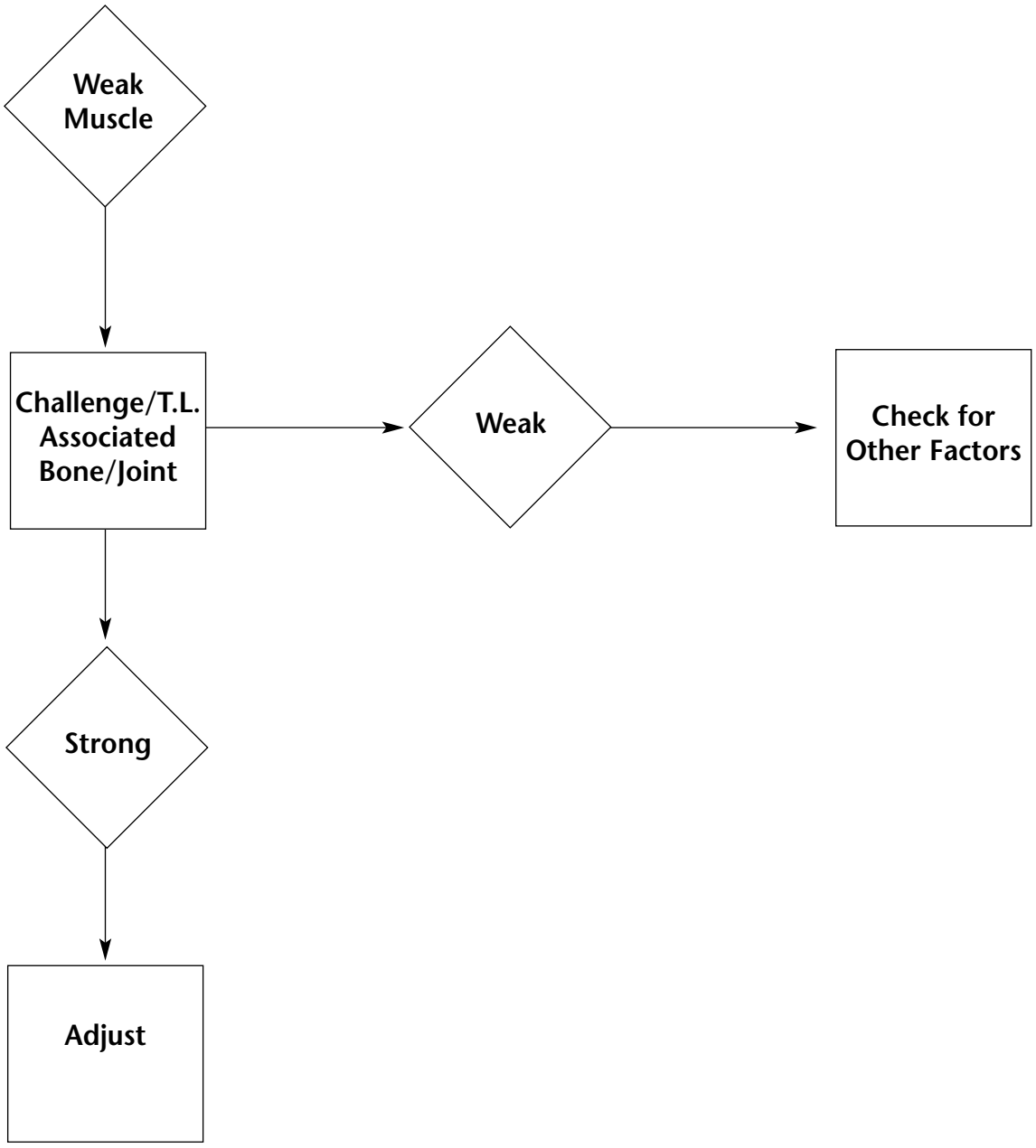
Regarding the anterior T2 (letter G); a screening procedure is to have the patient flex the head in a weight bearing position and check a strong indicator muscle. If it weakens, then there is a strong likelihood and anterior T2 subluxation exists. Challenge and adjust (often done in a standing position).

The first rib subluxation (letter K); this is adjusted with the patient in a seated position. The doctor takes a web-hand contact over the first rib. The patient's head is allowed to extend and rotate away from the contact with the thrust being delivered lateral to medial and superior to inferior.

Conclusion

These particular spinal/rib subluxation/muscle-syndrome correlations have been extremely useful clinically. Standard applied kinesiology therapy localization/challenge procedures may be utilized for diagnosis along with the additions noted above.

Spinal-Rib Subluxation/Muscle Syndrome Correlations Summary of Procedures



Spinal-Rib Subluxation/Muscle-Syndrome Correlations
Summary

Timothy D. Francis, D.C., 1999

I. Spinal/Rib	SUBLUXATION	MUSCLE/SYNDROME
	A. C6/7	1. Sartorius/Gracilis 2. Adrenal Stress 3. Category II Pelvic Lesion
	B. Lateral Occiput	1. Ocular Lock Pattern with the Eyes Held Up and Out to the Right, and/or Left 2. Hyoid Dysfunction
	C. C2	1. Ocular Lock Pattern with the Eyes Held to the Right or Left 2. Hyoid Dysfunction
	D. Inferior Occiput	1. Ocular Lock Pattern with Eyes Held in Straight Superior Direction 2. Neurological Disorganization 3. Hyoid Dysfuction 4. Hiatal Hernia 5. Psoas Imbalance 6. Gait Imbalance 7. Category II Pelvic Lesion 8. Short Leg 9. Rib Torque (Victor Frank) 10. Anterograde Lymphatic
	E. C5/Anterior C5 (Victor Frank)	1. Bilateral Latissimus Dorsi (When tested together) 2. Blood Sugar Dysfunction (Victor Frank)
	F. C3	1. Deltoid/Serratus Anterior/Coracobrachialis 2. Lung Conditions

I. Spinal/Rib	SUBLUXATION	MUSCLE/SYNDROME
	G. Anterior T2	1. Subscapularis 2. Pre/Post Cordial Tap 3. Cardiac Lowback (Victor Frank) 4. Emotional Broken Heart Syndrome (Howie Cohn) 5. Blood Sugar Dysfunction (George Goodheart)
	H. Anterior T5/ T5 Rib Head	1. Bilateral PMC 2. Temporal Bulge
	I. Anterior T7/ T7 Rib Head	1. Gluteus Maximus/ Medius/Minimus 2. Adductors 3. Piriformis 4. Gonadal Hormone Related Imbalances
	J. Second/Third Rib Heads	1. Sartorius/Gracilis 2. Adrenal Stress
	K. First Rib	1. Almost all Gonad Related Muscles - Gluteus Maximus/ Medius/Minimus/ Piriformis/Adductors 2. Gonadal Hormone Imbalances
	L. Anterior T10/ T10 Rib Head	1. PMS (Pectoralis Major Sternal) 2. Liver Dysfunction
	M. Anterior L1	1. Bilateral PMC 2. Temporal Bulge
	N. Anterior L3	1. Quadriceps 2. Spastic ICV 3. Failure to Pass Deep Tendon Reflex Test 4. Allergies 5. Digestive Disturbance

I. Spinal/Rib	SUBLUXATION	MUSCLE/SYNDROME
	O. Base Posterior Sacrum	1. Ocular Lock Pattern with Eyes Straight Inferior 2. Retrograde Lymphatic 3. Pectoralis Minor 4. Bilateral Piriformis (When Tested Together) (Victor Frank)
	P. Apex Posterior	1. Ocular Lock Pattern with Eyes Held Straight Superior 2. Anterograde Lymphatic
	Q. Posterior Sacrum	1. Ocular Lock Pattern with Eyes held Down and to the Right or Left 2. Piriformis
	R. Pubes	1. Adductors (George Goodheart) 2. Incontinence
	S. Coccyx	1. Coccygeus/Ileococcygeus/ Pubococcygeus 2. Incontinence

References

1. Chrane, Virgil, Alphabiotics. Privately published, Dallas, Texas, (1996).
2. Francis, Timothy D., "Structural Corrections for Eyes Into Distortion Patterns," Collected Papers of the Members of the I.C.A.K., Vol. I, Summer, (1990-91).
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A Screening Procedure For Determining Which Supplemental Sources A Patient Requires

Timothy D. Francis, M.S., D.C., DIBAK, D.H.M.

Abstract

Various dysfunctions throughout the body may be caused from and/or involve the chemical side of the triad of health. An efficacious procedure has been developed to determine what source (herbal, homeopathic, allopathic, and/or nutritional) the patient requires for return to a balanced homeostasis.

Introduction

Searching for the cause of a patient's symptom complex can lead a clinician to explore many possibilities. This paper explains the procedure to determine the supplemental sources (herbal, homeopathic, allopathic, and/or nutritional) on the chemical side of the triad of health that the patient requires for a prompt return to balance and harmony within the body.

Discussion

Due to the extremely large number of possibilities in determining which supplements a patient requires for expedient return to health, a screening procedure is required to narrow down the categories and possibilities. This paper is an expansion on the observation of Scott Walker, D.C. who originally proposed the concept that need for homeopathy could be determined by therapy localization (T.L.) to bladder one points with the index and middle finger on the same hand while cross therapy localizing to one of the five factors of intervertebral foramen (IVF), and/or an organ, and/or causing a muscle that manually tested weak in the clear to then test strong.

That one observation helped to narrow down whether a patient would obtain therapeutic benefit from a class of substances known as homeopathic medicines. However, there were still the other broad classifications of herbs (naturopathy), allopathic medicines, and/or nutrition (vitamins/minerals/glandulars).

"If the only tool you have is a hammer, you tend to see every problem as a nail." (Abraham Maslow)

The more tools you have and the better you know when and how to use them and when not to use them, the better your clinical results will be. Therefore, this author set about expanding on Walker's original observation. Herbs (naturopathic medicines) are required when positive T.L. to bladder one points (utilizing the same criteria as for homeopathic medicines) via the thumb and index fingers of the same hand. Allopathic medicines are required (or referral if your license does not permit) if there is positive T.L. to bladder one points (B.M.I. - body memory indicator) with the middle and ring finger of the same hand.

Nutritional supplements (vitamin/ mineral/ glandular) are necessary if there is positive T.L. with the ring and little fingers of the same hand to B.M.I. points. We then must determine thru individual testing of supplements the particulars in each case; however, this initial screening will determine the broad category required (herbal, homeopathic, allopathic, and/or nutritional) or if no supplement is required for the particular muscle/organ/reflex.

Conclusion

This procedure allows the clinician to have at his/her disposal a reliable rapid screen for all four categories (herbal, homeopathic, allopathic, and/or nutritional) or supplements which may be required for the most efficacious approach on the chemical side of the triad of health.

References

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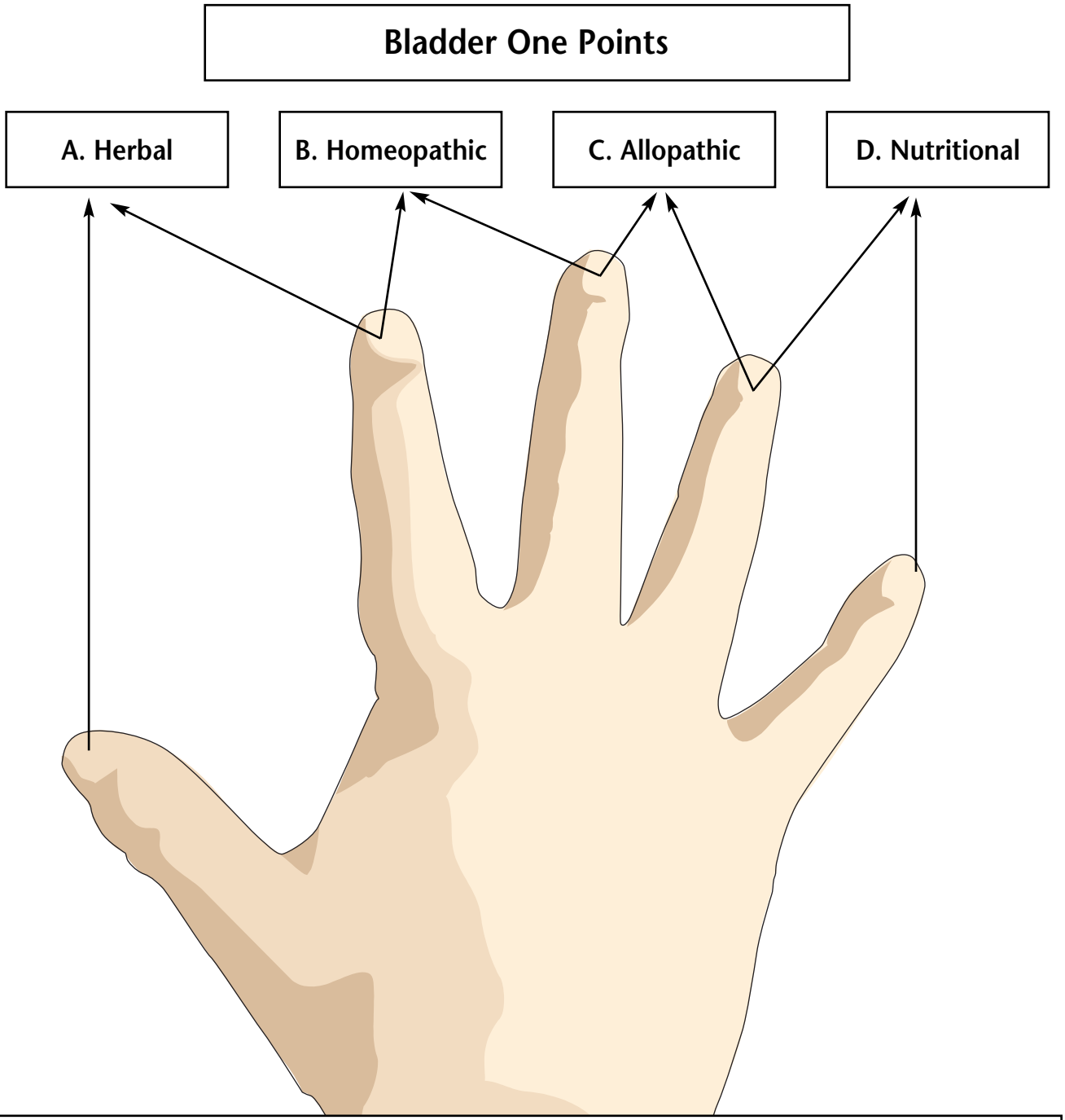
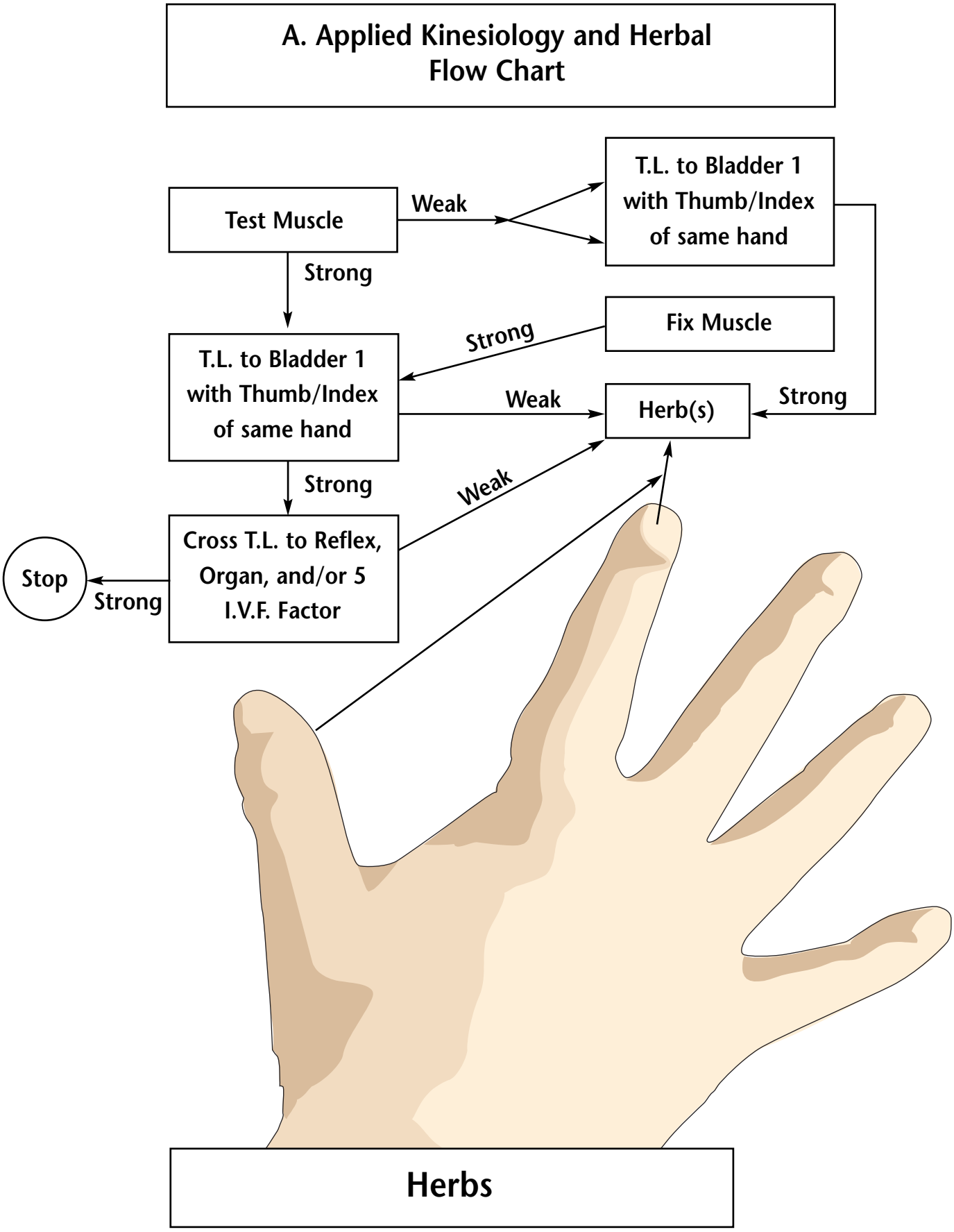
2. Ibid. "Total Integration of Muscles, "Proceedings of the Annual Meeting of the I.C.A.K. - U.S.A., (1996-1997).
3. Frank, Victor, Dynamics of T.B.M. Workbook. Privately published. Sandy, Utah (1995).
4. Walker, Scott. NET Remedies, Privately Published, Encinitas, CA (1994).
5. Ibid. Neuro-Emotional Extra Techniques, Privately Published, Encinitas, CA (1994).

Summary of Procedures

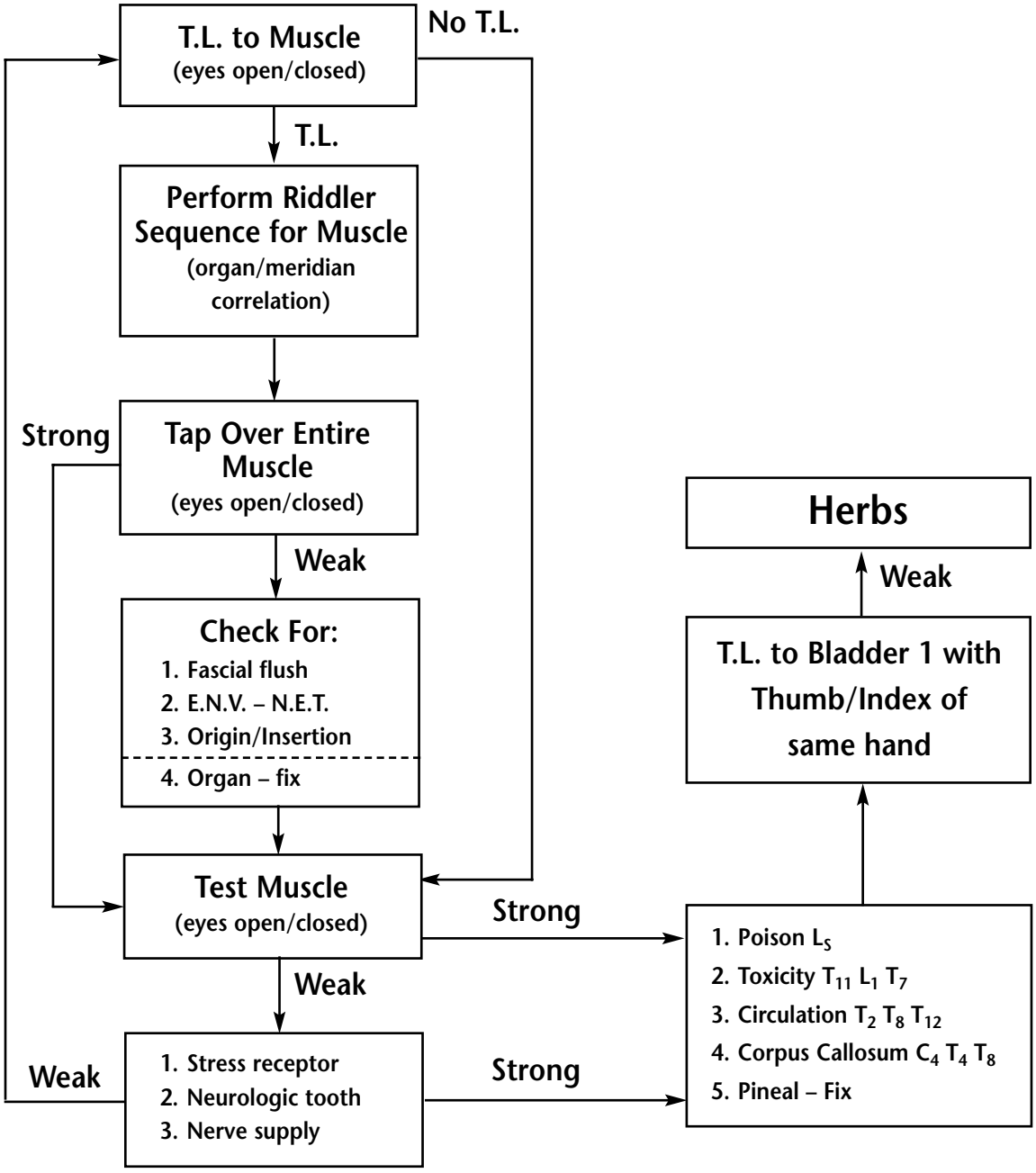


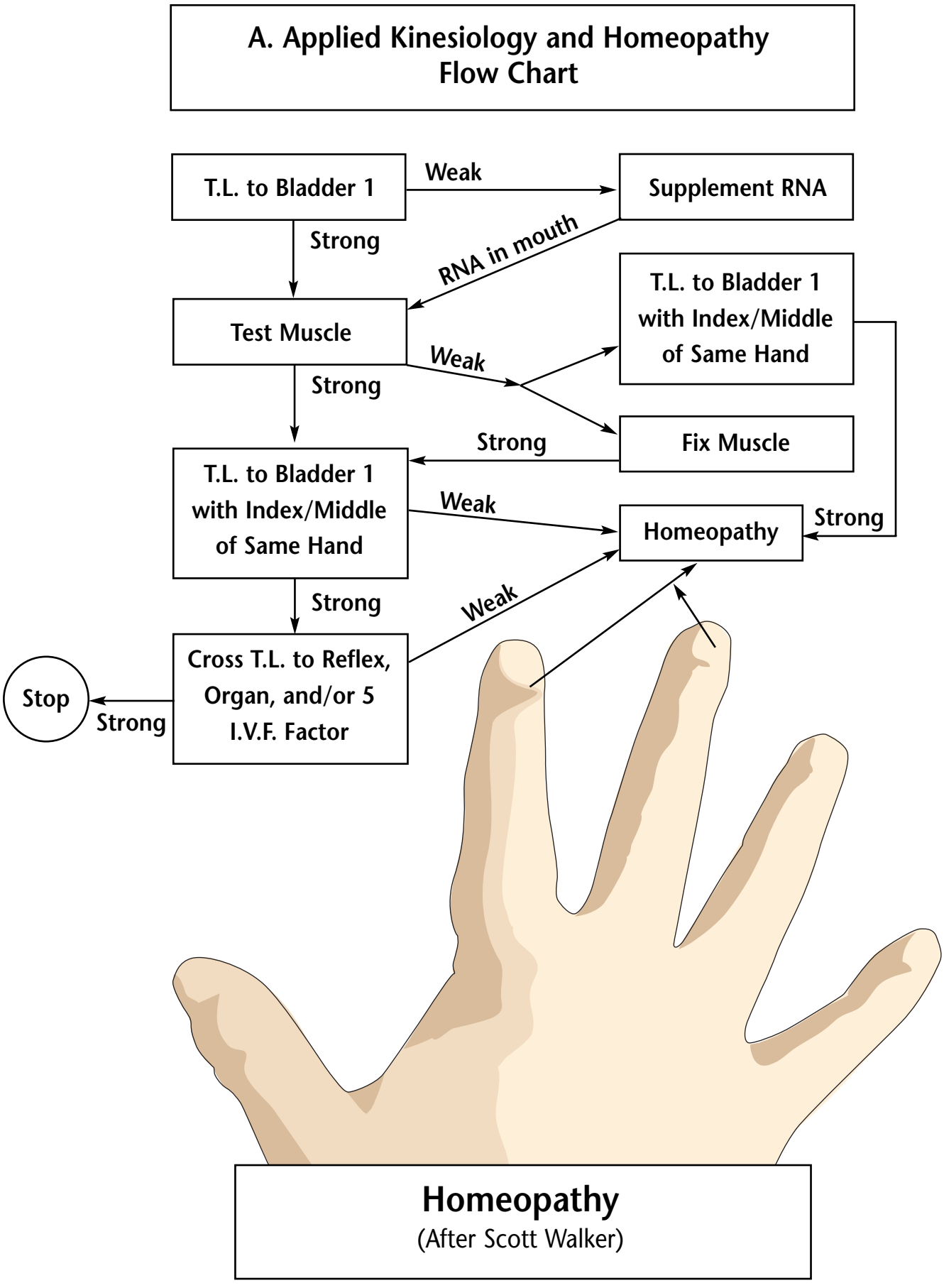
T.L. to Bladder One Points

1. Strengthens a weak muscle
2. Negates a positive T.L. to an organ, a reflex, and/or one of the five factors of the I.V.F.
3. Causes positive T.L. to an organ, a reflex, and/or one of the five factors of the I.V.F. (51% er)
4. Re-weakens a strong muscle previously corrected by another procedure (51% er)

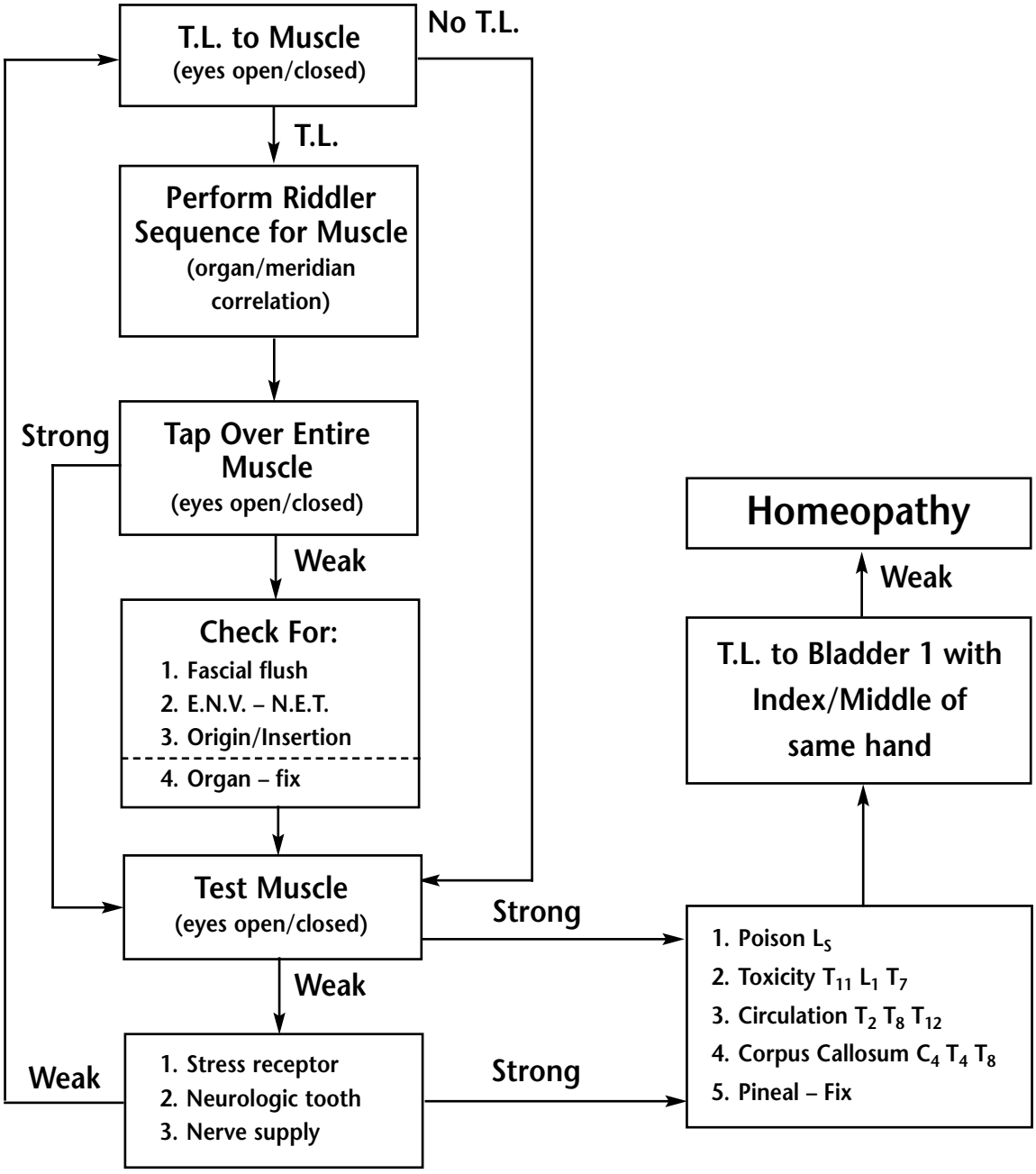

A. Applied Kinesiology and Herbal Flow Chart
Test Muscle
Weak
T.L. to Bladder 1 with Thumb/Index of same hand
Strong
Fix Muscle
Strong
T.L. to Bladder 1 with Thumb/Index of same hand
Weak
Herb(s)
Strong
Strong
Weak
Cross T.L. to Reflex, Organ, and/or 5 I.V.F. Factor
Stop
Strong
Herbs

Total Integration of Muscles Flow Chart

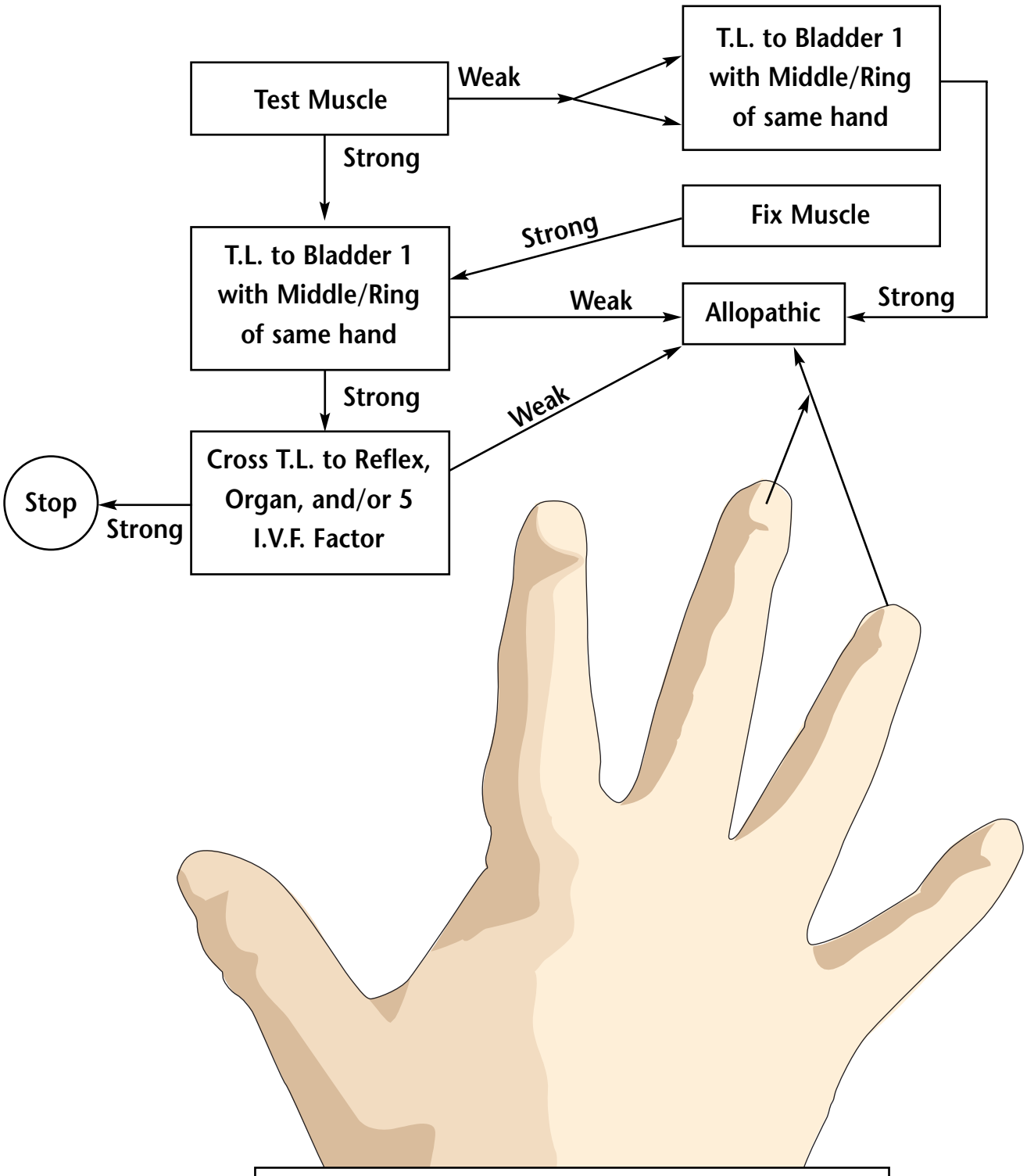



A. Applied Kinesiology and Homeopathy
Flow Chart
T.L. to Bladder 1
Weak
Supplement RNA
Strong
RNA in mouth
Test Muscle
T.L. to Bladder 1
with Index/Middle
of Same Hand
Weak
Strong
Fix Muscle
Strong
T.L. to Bladder 1
with Index/Middle
of Same Hand
Weak
Homeopathy
Strong
Strong
Weak
Cross T.L. to Reflex,
Organ, and/or 5
I.V.F. Factor
Stop
Strong
Homeopathy
(After Scott Walker)

Total Integration of Muscles Flow Chart

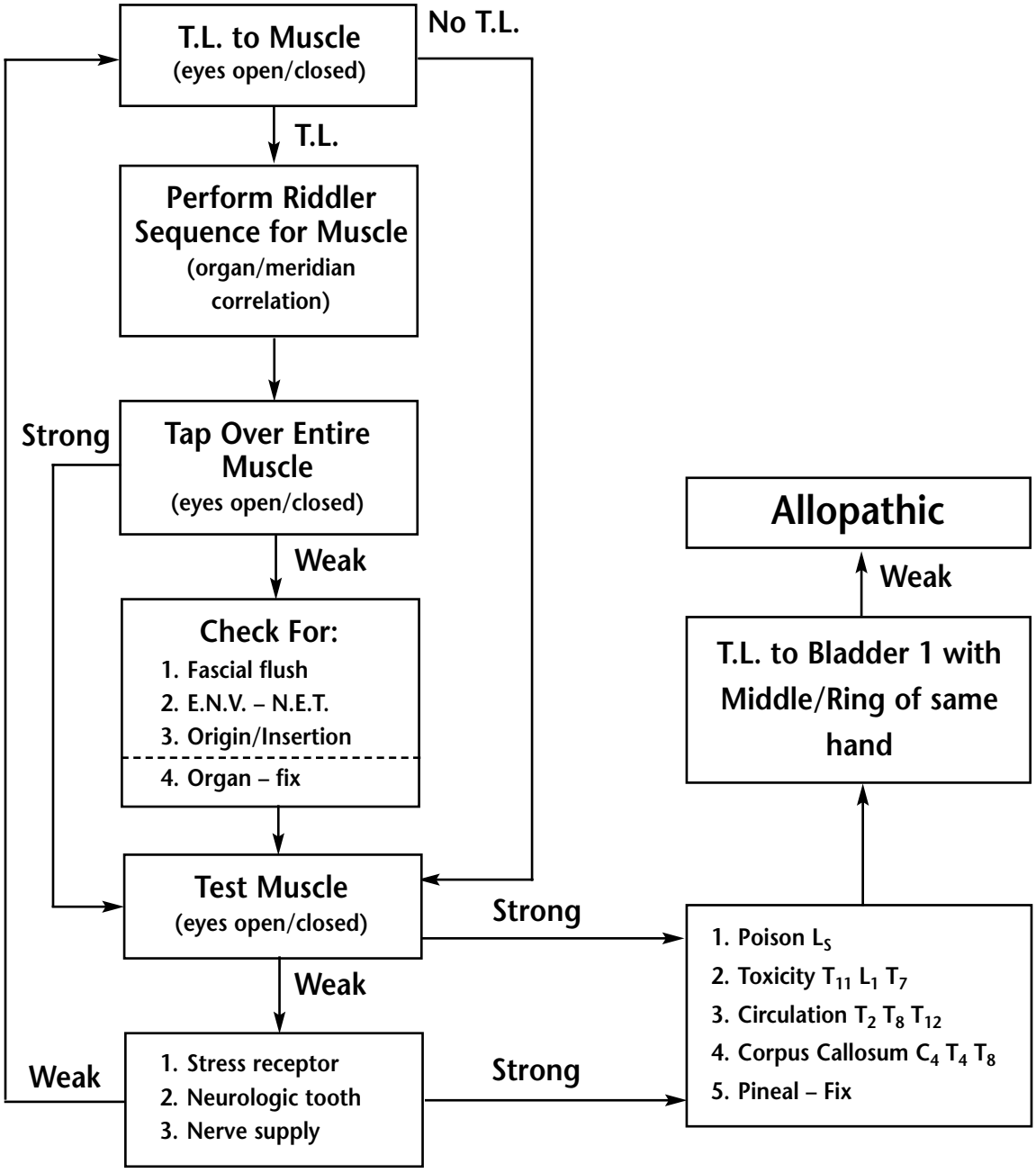


C. Applied Kinesiology and Allopathic Flow Chart

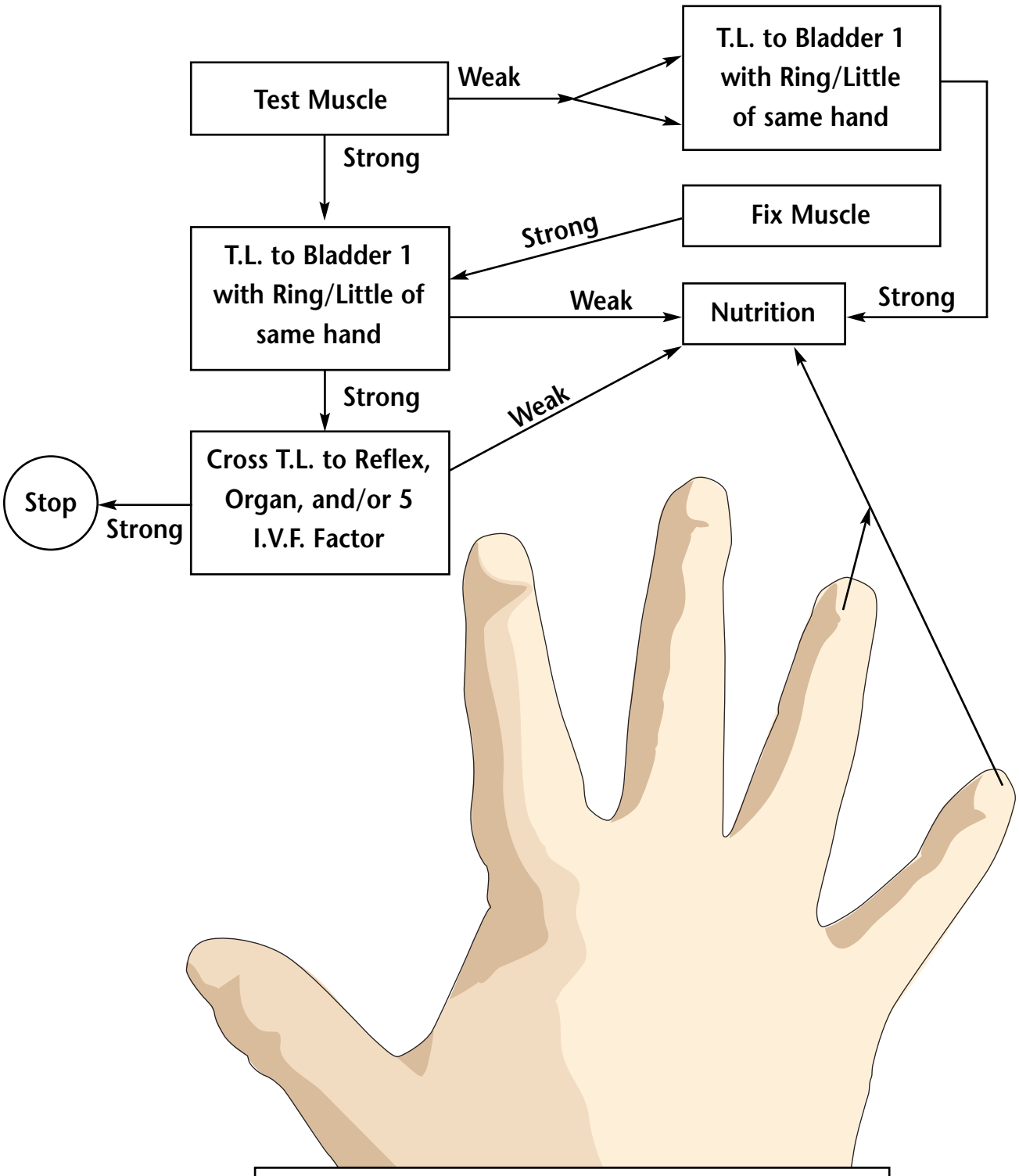


Allopathic

Total Integration of Muscles Flow Chart

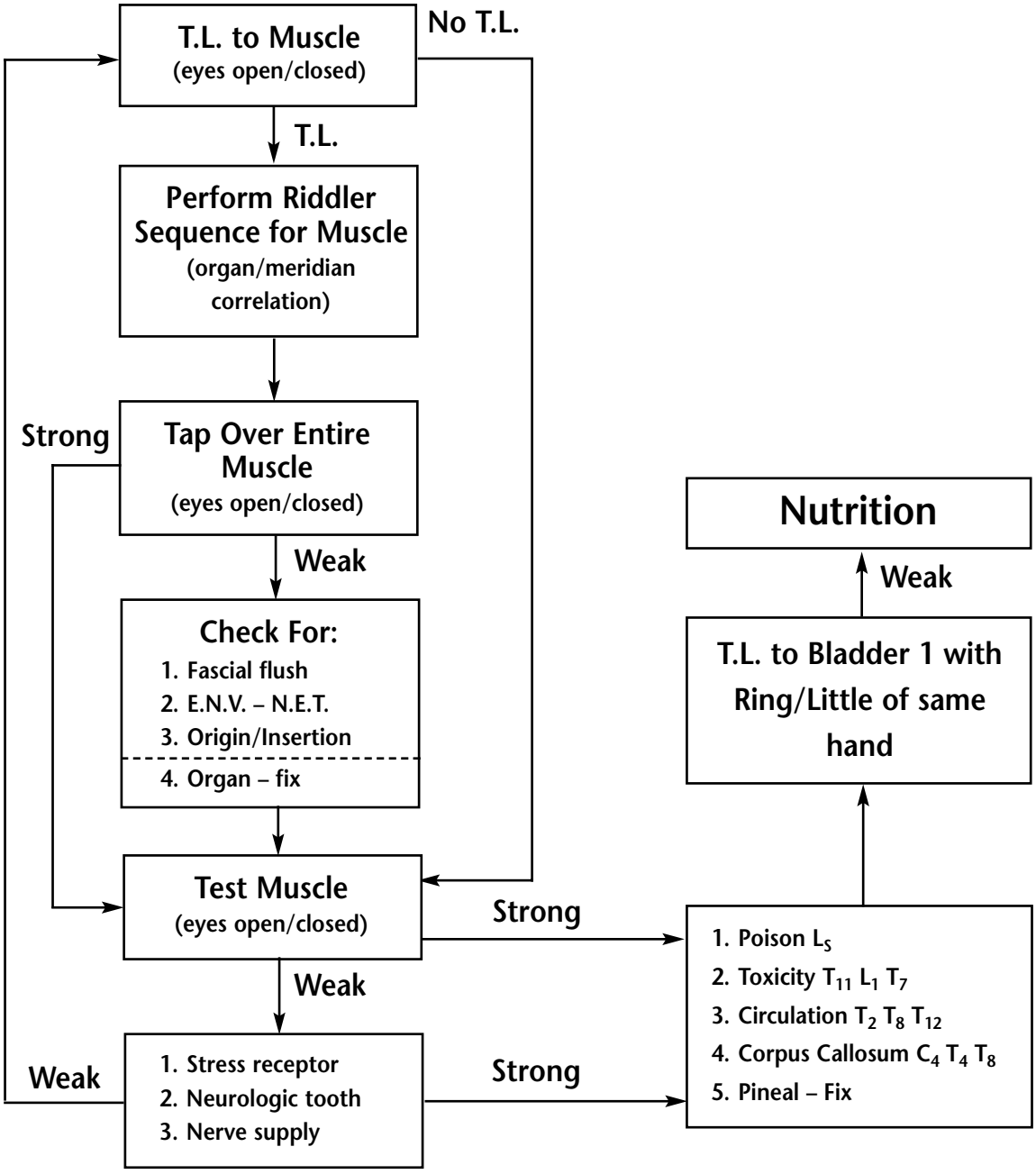


D. Applied Kinesiology and Nutritional Flow Chart



Nutritional

Total Integration of Muscles Flow Chart



Foci and Field Disturbances (Storfeld): Definition and Improved Testing with Applied Kinesiology

H. Garten, DIBAK

Translation: Jeff Farkas, DIBAK

Abstract

We owe our awareness about field disturbances or foci, as well as their significance, to neural therapy as per Huneke (DOSCH 1980, BADTKE 1994, et.al.). The empirical phenomena of treating foci with local anaesthetics has resulted in expanded research on the topic, particularly in Vienna (PISCHINGER 1975, KELLNER 1963, 1965, BERGSMANN 1994, et.al.)

The focal disturbance as causal factor is as important a finding in Applied Kinesiology as in other forms of energetic and cybernetic diagnostic and therapy systems:

• The focus must be recognized as a possible factor in the development of muscle disfunction

• Muscle testing can be used to discover foci as well as differentiate their nature

This offers a vast improvement, in terms of efficiency and exactness, over the current neural therapy standard of performing trial injections of scars and other possible disturbances.

Key words: field disturbance, focus, Applied Kinesiology, focal and disturbance testing

Definition of focus

Focus refers specifically to a local tissue change, with measurable pathomorphology, assumed to be caused by effects of bacteria, viral agents or toxins.

Definition of field disturbance

Field disturbance refers to a region with compromised integrity of the ground substance (PISCHINGER; HEINE), not conducive to measurement as per routine histopathomorphological parameters. Nevertheless; these areas demonstrate vegetative and energetic anomalies resulting in segmental or systemic disturbances.

Consequences of field disturbances

As a result of compromised ground regulation integrity, consequences of a field disturbance include altered predisposition to noxious effects of infective, allergic, toxic, mechanical, chemical, thermal, neurologic and emotional stresses; in short, all the stress factors which can compromise the sides of the Triad of Health. The field disturbance may cause symptomatology directly or can play a role in altered resistance, allowing an additional stress factor to produce symptoms. Examples: An intestinal field disturbance may remain asymptomatic; however, with the addition of an emotional stress, the patient may suffer from low back pain.

Level 1: local disturbance

Local regulatory degeneration (release of pain and inflammatory mediators such as cytokines (HEINE) cellular migration and reaction, cellular proliferation (scarring), local disintegration of tissue fibers, replacement of differentiated tissue with compromised ground regulation reflect a local phenomena which still remains clinically silent. They may communicate, via neural and extraneural information systems (ground substance, biophotons and Vascular interstitial closed electrical circuits, VICC, HEINE 1991, POPP 1987) with somato- and viscerosensitive structures related to the respective spinal segment. This latter mechanism tends to produce overlapping segmental changes which can be easily identified using bioenergetic test methods (i.e. Applied Kinesiology, EAV) even in the absence of clinical symptoms which might develop at a later stage (see level 2 thru 4).

Example: Liver dysfunction can result in dysreactivity of Pectoralis major sternalis, even before changes in laboratory values, hyperalgia or dysfunction in the 8th &/or 9th thoracic segments can be detected.

If this is not the case, the definition of a field disturbance does not apply; rather, the patient is presenting with a local tissue alteration (i.e. scar) with no regulatory disturbance.

Level 2: Segmental disturbance

The viscerosomatic (in the case of primary affect to inner organs), the somatovisceral (in cases of primary affect to somatic structures), as well as somatosomatic (muscle function, or rather, muscle lesion chains) reflexes can lead to typical tonic-algesic pain syndromes in respective segmental regulation complexes (BERGSMANN, 1988, PISCHINGER 1991): palpatory changes in skin, fascia and muscle tissue (“gelosis,” “tender points,” “trigger points”) appear.

Level 3: Ipsilateral symptomatology

Under the influence of prolonged regulatory degeneration and increasing irritation, ipsilateral signs and symptoms may result. This phenomena has been well corroborated by the Austrian researchers. It correlates with signs of hyporegulation as measured in the elbow crease when performing Regulation Thermography according to ROST.

Levels 2 and 3 can also be referred to as “projection syndromes.”

Level 4: General illness

With the addition of secondary and tertiary factors (referred to as 2nd and 3rd "shocks" in neural therapy), general illness can ensue, such as predisposition to infection, chronic fatigue syndrome, pain syndromes, etc. The classic case presented by Huneke of right shoulder "arthritis" actually resulting from an osteomyelitis scar of the left leg (DOSCH, 1980) would fall under this level category, although the symptoms were actually manifested very locally.

The best general description of the field disturbance phenomenon is found in the summation of HUNEKE:

"Every illness can be related to a field disturbance."

A reflection of the concept of stress as it is presented in applied kinesiology facilitates the understanding of foci and field disturbances. As long as the ground regulation system is still basically intact, utilization of regulation influencing therapies such as manual therapy, neural therapy, acupuncture and nosode therapy can yield positive results. A ground regulation system destabilized under the influence of one or more minimal chronic loads may, if exposed to an additional "2nd shock" (toxic substances, heavy metals, pesticide, toxic bowel syndrome) may collapse or produce an inappropriate reaction in the form of a global regulatory disturbance independent of the location of the source ("butterfly effect"). Even in these cases of regulatory disturbances, one can recognize the development of a typical adaptation syndrome; after initially reacting with excess acute symptomatology, the patient will show long-term signs of regulation blockage and fatigue (BERGSMANN 1989, HEINE 1991).

A blocked regulatory mechanism resulting from chronic inflammatory reactions leads to a complete dissociation of the ground regulation and immune systems. In such cases, marked overreactivity of the humoral immunity can be observed. PERGER (in HEINE, 1991, p.,108) reported an extreme case of primary chronic polyarthritis with IgG elevations, within 3 hour time frame, of 1466mg%.

Foci and field disturbances can predispose the organism, dependent upon condition, constitution and size (meaning duration and size of exposition) to suffer under the consequence of exposure to noxious stimuli of infective, allergic, toxic, mechanical, chemical and thermal nature. In other words to every form of stress.

Objective findings as evidence of the effects of field disturbances

Many reports regarding results of research into the effects of field disturbances can be found in BERGSMANN (1988), research conducted for the most part at the Ludwig-Boltzmann Institute in Vienna (Austria).

1. Assymetrical blood pressure regulation

The Shellong test (equivalent Ragland) revealed significant decrease in reduction of blood pressure amplitude on the side of the suspected disturbance upon standing up. Differences in oxygen exchange, leukocyte counts, sedimentation rates between the side of the body with a disturbance as opposed to the unaffected were measured, in addition to reaction to exposure to pyrogenic provocation and electric potential as per the Decoder Dermograph (see below).

2. Decoder dermograph

This procedure involves the placement of 3 pairs of electrodes (bilaterally to hands, feet and forehead), allowing for a baseline measurement of the interaction along 7 electrical pathways (4 vertical and 3 horizontal).

Along each of these pathways, after registering the initial electrical potential, a 1- Hertz impulse will be introduced and the storage capacity of the skin, as well as the change in electrical potential will be measured. Performing this baseline measurement along 7 various pathways allows for a good overview of the electrical condition of the organism.

As this measurement alters the conditions, a second measurement is required for comparative purposes and it is the type of changes noted between the baseline and second measurements, which may indicate the effects of a field disturbance or focus. One can well differentiate between normal regulation, hypo- or hyperregulation: hyporegulation indicates a degenerative process or field disturbance as opposed to hyperregulation, which is indicative of vegetative overactivity or a bacterial focus. After provocation of the focus or field (best performed with neural therapy), a 3rd measurement will be performed, where one can observe a normalization of regulation, assuming the provocation was performed in the correct area with a remedy addressing the nature of the focus.

3. Contact Thermography as per ROST

Substantially more time consuming is contact thermography, developed by ROST (1987, 1994) and SCHWAMM. The method according to Rost requires measurement of viscerotopic points on the skin. Subsequently, the patient disrobes and after 10 minutes a second measurement is performed. A 0.2 to o.3 degrees Celsius elevation of temperature in the head region and 0.5 to 1 degrees Celsius cooling of the trunk is normal. Hypo-and hyperregulation using this measurement also suggests degenerative or hyperreactive states. A focus results in hypo-or hyperregulation in a single measured value, in combination with a global disregulation as measurable by regulation changes measured at the elbow crease. After provocation, preferably with neural therapy injection, a third measurement will be performed. Successful localization of the focus will be demonstrable by a return to normal regulation.

More important than single values, judgements regarding thermography require an overview of the entire picture. Other factors such as heavy metal poisoning and electromagnetic stress can be detected in the form of general hyporegulation. A predisposition to malignancy reveals itself in the form of chaotic regulation, where hypo- and hyperregulation are simultaneously on display (ROST 1994).

It is critical that the two disadvantages to thermography are also noted here:

1. Disturbances will not always reflect in a local measurable change; without AK, the examiner is "fishing in the dark," using only the distinct elbow crease finding as proof of the existence of a focus.
2. Some field disturbances do not show up in the thermography at all. This is only apparent to the examiner who uses thermography in conjunction with other tools for focal testing.

The examiner should always utilize at least two methods (i.e.applied kinesiology and decoder, radiology) for evaluating the presence of foci and field disturbances, particularly if the consequences of the finding are significant (i.e. tooth extraction).

Improved AK diagnosis in the case of intraoral field disturbances

In comparison to the current method of using the finger for therapy of localization to the tooth and evaluating for nosodes, which negate the positive finding, an improved, more sensitive method is available:

1. For screening purposes the TL is first performed using a strong axially polarized (3000 Gauss) ring magnet placed over the front or side teeth. If an indicator muscle change occurs, this non-specific TL suggests the presence of a disturbance in the respective area, which must then be localized using single therapy localizations.

2. Without simultaneous placement of the magnet to the jaw, a TL should be performed to EAV jaw lymph point 2 describe where this is. If this point is positive "in the clear" it indicates that the effects of the field disturbance are distributed widely. Usually, the magnet TL will now negate the positive lymph 2 TL. Lesions to facial bones (maxillary, zygomatic, vomer, mandible) may also result in positive test to the magnet. These can be differentiated using mechanical challenge.

To increase efficiency, the following may be undertaken when performing dental therapy localization:

3. The patients thumb can be placed within the hole of the ring magnet (this is similar to electric irritation to the teeth as focal provocation, regularly performed in EAV).
4. Using a normoreactive indicator muscle, the patient should therapy localize to a suspected tooth. This is best performed by holding the tooth and supporting structures between finger and thumb, insuring contact with labial as well as lingual surfaces. This is particularly important in cases of teeth with multiple roots. In addition, using a dental prong, tweezer or other metal instrument can also allow for precise location of a single affected root.
5. In the absence of radiological findings, a mechanical evaluation for "neurologic tooth" should be performed.
6. In the event of positive TL, challenges with nosodes can be performed. An ampule with a low potency nosode in question (ostitis, granuloma, etc.) should be placed in the hand which has been used for the TL.
7. Should a nosode ampule negate the TL, a temporal tap (8) should be performed. If one ampule is insufficient, the muscle dysreactivity will subsequently resurface.
8. A second ampule of the same nosode should be placed in the patient's hand and the temporal tap performed again to see if the muscle dysreactivity remains negated. If the muscle agains become dysreactive, a third ampule can be used, etc.

As EAV physicians do regularly, the disturbance may be conservatively treated with a series of nosode injections. This should only be undertaken if 1 or 2 ampules were necessary to fully negate the finding. The necessity of 3 or more ampules is strongly suggestive of the need for surgical intervention.

Control: the effects of one field disturbance exclusively?

A tool which has had a root canal treatment may develop into a field disturbance due to decomposition (TL negated by gangrenous pulpa or root canal nosodes). In addition, it may be covered with a crown made of materials which are not tolerated. Therefore, after determining the correct number of ampules as described above, the following must also be performed:

A magnet TL is reperformed over the dental area and the ampules (ex: gangrenous pulpa) which functioned using the finger TL are now placed upon the belly. A temporal tap is performed. If the magnet TL does not remain negated, there is another disturbance which has yet to be diagnosed, either in a neighboring tooth (retest with finger TL and magnet on thumb) or another form of disturbance to the same tooth.

In the latter case, the entire procedure should be performed using nosodes of potentized toxic agents (i.e. palladium D12, etc.).

Case History

39 year old male

Chronic recidivous pharyngolaryngitis, susceptibility to infection, recurrent candidiasis, hightened chemical sensitivity, repeated problems of the motion apparatus such as tendonitis, diminished sense of well being and performance capacity.

Acute episodes of fever up to 39 degrees Celsius (102.2 F).

History includes post-resection extractions of molars 6 and 7 in the lower left quadrant (36 and 37) due to "canal supporation." Radiologic examination of the lower left "empty" mandibular region was pronounced as free of findings by many dentists and dental surgeons.

Focal testing with AK: TL with finger negative.

Magnet TL to lower left dental region positive. Ring magnet placed on lymph point 2 resulted in a massive positive TL to the empty dental region, negatable only by 4 ampules of sclerotic ostitis D4 nosode. A dental surgeon suggested trial therapy with Sobelin, an antibiotic absorbed by bone tissue, resulting in esophagitis and candidiasis. Treatment with Nystatin and anti-candida diet. Finally, a dental surgeon was recruited who was willing "against his better judgement" to do a revision of this "healthy" region. In contrast tot he surgeon's expectations, completely altered tissue was exposed with remnants of silver amalgam. A post-operative granulation healing, without antibiotic therapy, was undertaken. With the addition of therapy with Arthrokehlan A and U (Sanum Co.) healing time was short and full convalescence achieved.

Conclusion

The concept of field disturbance is more complete than the term focus and improves our ability to understand the nature of the disturbance, which may be independent of infection or toxicity.

With the assistance of new differential test methods using magnet challenges, field disturbances, which were previously not detectable, can now be identified as the causes of general disturbance and an appropriate therapy form chosen.

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Ayurvedic Body/Mind Typing Using Applied Kinesiology Pulse Testing and Standard Questionnaires

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Abstract

Building on an earlier article[1] by the author, a method is presented for classifying individuals by the ten Ayurvedic constitutional types. A pulse analysis method using muscle testing is presented as well as a questionnaire. Suggestions to improve accuracy of both methods are presented as well as use of the results in making diet and exercise recommendations for the different constitutions or "Doshas". Seasonal influences are also briefly addressed.

Introduction

Metabolic individuality is not only a long established fact, especially in "alternative" health care but is central to the applied kinesiology approach to patient evaluation and treatment. This being the case, however it is also true that everyone has certain basic similarities metabolically and that, beyond this, we can seek to find groups that have an even higher degree of similarity. Thus most of us have encountered and used various methods of metabolic "typing". There are popular metabolic typing systems based on endocrine dominance and blood type available in most bookstores which have enjoyed great popularity.

Ayurveda is a science of life and health care that dates back five thousand years in India. Constitutional typing is a key component in Ayurvedic care. This system allows the practitioner to make recommendations with regard to diet and lifestyle changes[2, 3, 4, 5] as well as the most appropriate types of exercise and sports to promote balance and health for the different constitutional types.[5] Using Ayurvedic principles, lifestyle recommendations can also be offered to allow better adaptation to seasonal and weather changes for each constitutional type.[2, 4, 5] Ayurveda offers a treasure trove of health science that has been formulated, tested, revamped and clinically proven on a vast scale. Recently Ayurvedic herbal remedies have migrated successfully to Europe and America where ancient applications are making a good showing under western scientific scrutiny. The National Library of Medicine contains hundreds of studies supporting the efficacy of Ayurvedic approaches to a wide variety of visceral problems. Like Chinese Medicine, Ayurveda has been somewhat inaccessible here in the West due to a combination of differing paradigms (for instance, in Ayurveda, the taste of a food is at least as important a consideration as the nutritional content) and complex analysis methods.

Pulse analysis is a major Ayurvedic analytical tool. Like Chinese pulse analysis, it can take considerable time to develop the accuracy needed to be reliable in a clinical setting. In 1994 I had the extreme good fortune to attend an Ayurvedic pulse analysis class from John Douillard, D.C. who is, according to Deepak Chopra, M.D, "...the leading expert in pulse diagnosis in America."[5] Dr. Douillard's clinical excellence is matched by his skill as a teacher and I left the class with a good "feel" for the basics of Ayurvedic pulse analysis. For the next several years I improved my skill in the palpatory art as well as developing a muscle

testing format which greatly reduced the learning curve (at least for those already proficient in manual muscle testing) and allows even beginners to enjoy a high degree of accuracy. I was delighted when Dr. Douillard produced a home study course in pulse analysis last year. I highly recommend it to anyone who wants to enrich their understanding and skill in this area.[5] Although I do not consider myself to be an authority on Ayurveda, it is my hope that this paper, along with my previous one,[1] will help open Ayurvedic science to investigation and use by our ingenious and creative membership.

Discussion

The Ayurvedic pulses are located along the radial artery of each wrist. There is some overlap with the Chinese pulse points commonly used in applied kinesiology.[6] The most distal or Vata pulse point is just proximal to the radial apophysis, the same as the most proximal point in the Chinese system. The index finger is considered to be the Vata finger and is always used to feel the Vata pulse point. The Pitta pulse point is just proximal to the Vata point and is palpated with the middle finger. The Kapha point is the most proximal and is palpated with the ring finger. If you keep all three fingers resting comfortably next to each other while palpating the pulse the spacing is about right. For males, the right radial pulse is used, for females, the left.

There are (at least) three levels at which the Ayurvedic pulses may be felt. The most superficial level, at which the pulse can just barely be felt, represents transitory Vikriti, indicating how the body is adapting, both normally and abnormally to ongoing environmental factors. The middle level represents more long standing Vikriti, changes in physiology of the type most commonly seen in practice. Please see my earlier paper on using this middle level clinically.[1] The deepest level, found by pressing hard enough to occlude the pulse and then letting up until it can just be felt returning, represents Prakriti, the unchanging mind/body constitutional type. The deeper the pulse, the more it represents long-standing conditions, with the deepest, the Prakriti being permanent.

It is sometimes difficult to distinguish between very deep or longstanding imbalances and the Prakriti. Because of this, Dr. Douillard suggests that the Prakriti be analyzed every morning for two weeks and consider the most frequent findings to indicate the Prakriti. This would ordinarily be accomplished by feeling for the relative strength of the pulse at the above three areas with the strongest one or two (or sometimes all three) indicating the Prakriti.

Determining the Prakriti with the addition of muscle testing is much easier but still may be complicated by deep level imbalances. It has been my experience that anything that may result in neurologic disorganization will be reflected in the pulse, sometimes deeply. I use Ayurvedic mind/body questionnaires in tandem with pulse analysis. In all but a handful of cases I have found a high degree of correlation. Although I consider both methods to be fallible, in 95% cases of disagreement so far I have found that some form of neurologic disorganization has been involved. After discovery and correction of the causative factors, I find the correlation between the two methods to be around 98%. With the above in mind, I have found the best time to test for Prakriti is at the end of an office visit, which will usually have improved any problems being reflected at a deep level as well as any neurologic disorganization.

I have found that the best way to determine Prakriti with muscle testing is to place three fingers on the Ayurvedic pulse points as above, right radial pulse for males, left for females. Leaving all three fingers on the radial pulse, with the index finger just proximal to the radial apophysis and the ring finger most proximal of the three, press on the index (Vata) finger deep enough to occlude the palpatory pulse. Maintaining pressure, test any previously strong muscle. Repeat this test with all three fingers. The points that do not

cause weakening with the pulse occluded correlate with the Prakriti. My feeling is that, if a particular Dosha is weaker in an individual, it cannot withstand full occlusion of the pulse without causing enough stress to weaken a strong muscle. The strongest Dosha(s) will be able to handle full compression of the pulse point. I suggest combining this pulse analysis with having the patient fill out an Ayurvedic body type questionnaire to provide additional accuracy. A questionnaire is provided at the end of this paper along with a diagram of the pulse points. To prevent preconceptions I suggest performing the pulse analysis first, then having the patient fill out the questionnaire without telling them the results. Alternately, have them fill out the questionnaire first but avoid looking at the results until after you do the pulse analysis. Patients should be instructed to answer questions based on habits, reactions and personality characteristics they've exhibited most of their life, not just the most recent part of it. Ask them to get input from family members if possible, especially if they're unsure of some answers.

Most people are a combination of the three Doshas with one or more commonly two being primary. If both Pitta and Vata points stay strong while occluded, the Prakriti is Pitta/Vata or Vata/Pitta. Usually only one Doshic area will stay strong with full compression. Repeat the above test by occluding the pulse at each point then releasing enough pressure to just barely feel the pulse. If one of the points that tested weak with occlusion now tests strong, this is the secondary Dosha. For example, if only the ring (Kapha) finger point stayed strong with full compression but with the pulse just barely felt, the middle (Pitta) finger point also now stays strong but the index (Vata) point still tests weak, the Prakriti is Kapha Pitta. If all three points stay strong with occlusion, the Prakriti is "Tridosha", the most stable type. If two stay strong with full compression, I have dubbed that a "Duodosha" and if only one stays strong with full compression but the other two stay strong with the pulse barely felt you have what I call a "Virtual Tridosha" who will react like a Tridosha in most circumstances but when stressed will be somewhat more likely to manifest the imbalance symptoms of the Dosha indicated with full occlusion.

The next section will contain a brief exploration of basic Ayurvedic principles and constitutional types as well as the effect of seasonal influences and simple dietary guidelines. Experienced Ayurvedic practitioners may wish to skip further down.

Ayurveda is based on five elements that make up the world. These five elements, Air, Fire, Water, Earth and Space or the Void are more similar to the Greek five elements than the Chinese. Each of the three main Doshas, Vata, Pitta and Kapha, are felt to be made of and to exhibit the properties of these elements. Thus Vata is composed of Air and Space, Pitta of Fire and Water and Kapha of Earth and Water.

Vata individuals tend toward the thin, sometimes wiry ectomorph body type. The influence of air makes them tend toward mental activity and creativity. Vatas are great "idea people" and love to have a lot of irons in the fire jumping from one to the other. When out of balance they can be flighty and seem like "air-heads", never completing much of anything. Vatas tend to have a lot of health problems, but usually not life threatening ones, somewhat like "thyroid" endocrine types. Their energy comes in bursts and when out of balance they may seem to be on an energy "rollercoaster". When stressed, Vatas tend toward worry, anxiety, constipation and insomnia. Vatas are usually fast oxidizers who may have trouble keeping their weight up when under stress but rarely become overweight.

Pitta individuals tend toward the mesomorph body type of medium, athletic build. They have a lot of "fire" or passion in their approach to life and tend to be hard-driving "A type" personalities. They are the people who "get things done", the managers who may be counted on to implement the creative ideas of the Vatas who they may regard as rather frivolous. When stressed or out of balance, Pittas tend to become angry, aggressive and critical of themselves and others who they may feel don't "measure up". They commonly overwork with the associated health and social problems. Physical indications of Pitta imbalance include

various types of inflammatory conditions, hypertension, rashes, acne and other types of skin eruptions and heartburn. Both Vatas and Pittas may have trouble with hypoglycemia but for different reasons. Vatas just tend to burn glucose faster while with Pittas I feel it may be more of a hyperinsulin situation. One difference between the two is that Vatas often don't realize they've missed a meal until they crash while Pittas tend to get very cranky if dinner is late!

Kapha individuals tend toward the endomorphic body type with large skeletal structure and a tendency to be heavy in general. They tend to be "earthy" or "salt of the earth" types who are easy going and slow to anger. They often have highly developed empathy and make good counselors. Kaphas may have a hard time getting started but once they do, they have more endurance and physical strength than the other Doshas. Mass is a central Kapha quality with resultant effects on both inertia and momentum. When stressed or out of balance, Kaphas tend toward inertia, lethargy and depression. Kaphas are good at accumulating things. On the up side this applies to savings and muscle as well as a strong memory although it may take them longer to learn something new than the quick Vatas (who also forget quickly). On the down side, the tendency toward accumulating also applies to fat and cholesterol. Kaphas have the most trouble keeping their weight down of the Doshas. They are more prone to the cardiovascular problems associated with atherosclerosis. They also tend toward watery type of sinus and other mucous membrane problems as well as joint problems when they go out of balance.

The elemental qualities of the three Doshas make them sensitive to the weather and the seasons. Cold, windy, dry weather typical of Winter aggravates Vata tendencies and Vatas have more problems and need more attention during this kind of weather. To help stay in balance in Winter three keys are Warm, Wet and Regular (WWR). They need to make sure they stay as warm as possible, avoiding unnecessary chills, stay hydrated especially well and keep their daily habits as regular as possible, something Vatas dislike but really need, especially in Winter.

The Fire in Pitta is accentuated by the heat of Summer. This is especially true of a hot, humid weather since Pitta is also part Water. Pittas, especially in Summer, need to stay Cool, Moderate and Mellow (CMM). Staying in the shade when practical, wearing shade hats and cool (but not icy) beverages help Pittas stay balanced in Summer. Pittas tend to push too hard so moderation and pacing is key, especially in hot humid weather. Pittas need to try to stay mellow to balance out their fiery tempers and avoid stimulants like caffeine and hot, spicy foods.

When you mix Earth and Water you get mud. Spring, with it's cool, wet, muddy weather is likely to bring out any lurking Kapha imbalances. Sinus problems, colds and the lethargy of "Spring fever" are among the possible manifestations of excess Kapha. Three things Kaphas need to remember in Spring is to stay Warm, Active and Dry (WAD). Kaphas, more than the other two Doshas have a strong need for exercise, often strenuous exercise. Weight control is also key to keeping Kapha in balance and Spring is a good time for some gentle fasting, especially if you've got a lot of Kapha in your constitution. I find the only time I can comfortably fast is in Spring or if I'm manifesting some Kapha imbalance like a cold. I have run across many references to cultures, such as the Hunzas, that fast in the Spring. No doubt this is, at least in part, because food is scarce after a long Winter, but in most cases these people consider the Spring fast to contribute to their good health.

Another method used to regain and maintain a balanced constitution in Ayurveda is through diet. The simple diet suggestions I'm offering in this paper would be appropriate for each Dosha if that is your primary Dosha, if you are having weather that accentuates that Dosha or if you are manifesting symptoms of imbalances in that Dosha.

To pacify/balance Vata eat food that is: sweet, sour, salty, warm, heavy, and oily. Reduce foods that are: spicy, bitter, astringent, cold dry and light.

To pacify/balance Pitta eat foods that are: sweet, bitter, astringent, cool and liquid. Reduce foods that are: salty, sour, spicy (hot spicy like cayenne) or hot in general.

To pacify/balance Kapha eat foods that are: spicy, bitter, astringent, light, dry and warm. Reduce foods that are: sweet, salty, sour, heavy, oily and cold.

In the above guidelines, spicy refers to foods that are spicy "hot" with a "bite" to them. Astringent foods have a sort of "dry" taste to them such as most legumes and some raw greens like spinach. Much more detailed dietary, exercise and lifestyle guidelines are available in the references at the end of this paper.

Procedure Outline

1. Have patient fill out Ayurvedic mind/body type questionnaire.
   a. Give questionnaire on visit before pulse analysis is to be performed
   b. Patient should be instructed to answer questions based on how they've been most of their life, not just the last few years.
   c. Input from family members can help improve accuracy
2. Process patient through regular office visit, including any needed cranial, spinal, allergy and neurologic disorganization corrections.
3. Locate the radial pulse
   a. Male, right side
   b. Female, left side
4. Contact the radial pulse immediately proximal to the radial apophysis with three fingers
   a. Index (Vata) finger most distal
   b. Ring (Kapha) finger most proximal
   c. Middle (Pitta) finger in between
5. Press the index finger until the pulse is no longer felt, leaving the other two fingers resting lightly on the radial pulse
   a. Test a previously strong muscle
      i. Stays strong = a primary Dosha
      ii. Weakening = not a primary Dosha
   b. Release pressure on index finger, leaving it resting lightly on the radial pulse
   c. Repeat with the remaining two fingers
6. Repeat step 5, with the points that showed weakness, occluding the pulse and releasing just enough pressure to let the pulse become palpable
   a. If indicator muscle now stays strong = secondary Dosha
   b. If indicator weakens = not a secondary Dosha

7. Put it all together, it spells “Prakriti”
   a. All three points strong with occlusion = Tridosha
      i. All three points strong with pulse barely returned but only one or two with occlusion = “Virtual Tridosha”
   b. Two points strong with occlusion, third weak with occlusion and pulse barely returned = Duodosha
   c. Only one point strong with both occlusion and pulse barely felt = “Monodosha”
   d. One point strong with occlusion, one other point strong with pulse barely felt tells primary and secondary, referred to a “Bidosha”
      i. This is the most common finding
8. Possible combinations include:
   a. Primary secondary combinations
      i. Vata pitta, Pitta vata, Vata kapha, Kapha vata, Pitta kapha, Kapha pitta, (capitals indicate primary dosha)
   b. Dudosha versions of the above
   c. Monodoshas, Vata, Pitta, Kapha
   d. Tridosha and Virtual Tridosha.
9. The terms Doudosha “Virtual Tridosha” are my own invention and not, to my knowledge, traditional Ayurvedic classifications.

Conclusion

Ayurvedic constitutional typing can be a valuable clinical tool, enabling the practitioner to guide their patients with diet, exercise and other lifestyle advice that is specific for them. It is part of a vast field of largely untapped healthcare knowledge from India that has stood the test of time. The translation of Ayurvedic pulse analysis procedures into muscle testing format allows the doctor who is proficient in applied kinesiology quick, easy and accurate access to this method of constitutional typing. A complete coverage of Ayurvedic principles and techniques is far beyond the scope of this paper, however the references below provide a good source of additional information on Ayurveda as well as lifestyle recommendations based on your patients’ Prakriti or constitutional type.

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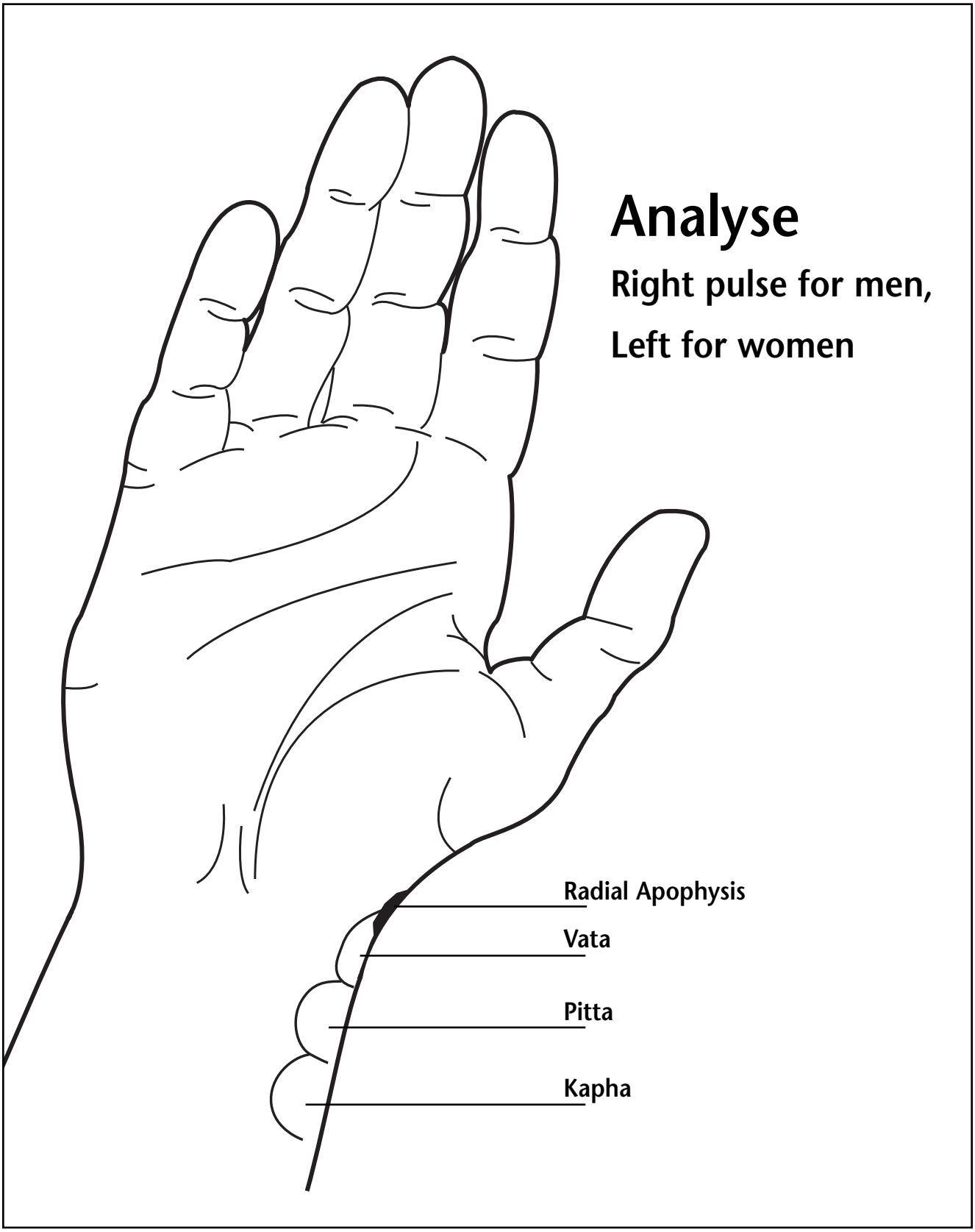
As mentioned earlier, it is useful to compare the pulse evaluation of Ayurvedic constitution with the results of a questionnaire. The following questionnaire is reprinted with minor adaptations from "Body Mind and Sport" with the kind permission of author John Douillard, D.C..

Give the answer that best describes how you've been most of your life. Try to select only one answer that best describes you. If two answers apply, check both. Total the columns at the end. The higher score(s) indicate your body type.

MENTAL PROFILE	**VATA**	**PITTA**	**KAPHA**
Activity	quick mind, restless	sharp intellect, aggressive	calm, steady, stable
Memory	short term is best	good in general	long term is best
Thoughts	constantly changing	fairly steady	steady
Concentration	short term focus best	better than average	good long term focus
Learning speed	quick to learn	medium time to learn	takes longer to learn
Dreams	fearful, flying, running	angry, fiery, violent	water, cloud, relationships
Sleep	interrupted, light	sound, medium	sound, heavy, long
Talk	fast sometimes, miss words	fast, sharp, clear cut	slow, clear, sweet
Voice	high pitch	medium pitch	low pitch
Sub Total			
BEHAVIOR PROFILE			
Eat	quickly	medium speed	slowly
Hunger	irregular	sharp, needs food	can miss meals easily
Food & Drink	prefer warm	prefer cold	prefer dry, warm
Mood	changes quickly	slowly changing	very steady
Sex drive	variable, low	moderate	strong
Weather	aversion to cold	aversion to hot	aversion to damp, cool
React to stress	excite quickly	medium	slow to get excited
Financial	doesn't save, spends quickly	saves, but big spender	saves regularly
Friendships	tends to short term friendships	tends to be a loner, or just work friends	lasting friendships
Sub Total			

PHYSICAL PROFILE	V	P	K
Amount of hair	average	thinning	thick
Type of hair	dry	medium	oily
Color of hair	light brown	red/auburn	dark/brown/black
Skin	dry/rough	soft	medium
Skin temperature	cold hands/feet	warm	cool
Complexion	darker or yellowish	pink-red	pale-white-creamy
Eyes	small	medium	large
Whites of eyes	bluish/brownish	yellowish or redish	white and glossy
Size of teeth	crooked, very large very small	small-medium	medium-large and or straight
Weight	thin, hard to gain	medium weight	heavy, easy to gain
Elimination	dry, hard, thin, constipation	many, soft to normal	heavy, slow, thick
Resting pulse rate			
Men	70-90	60-70	50-60
Women	80-100	70-80	60-70
Veins & Tendons	very prominent	fairly prominent	well-covered
Sub Total			

ATHLETIC PROFILE	V	P	K
Exercise tolerance	low	medium	high
Endurance	fair	good	excellent
Strength	fair	better than average	excellent
Speed	very good	good	not so fast
Competition	doesn't like competition	excellent (driven) competitor	easily deals w/competition
Gait speed	fast, quick	average	slow and steady
Muscle tone	lean, low body fat	medium with good definition	bulk w/higher fat percentage
Runs like	deer	tiger	bear
Body size	small frame, lean or long	medium frame	large frame, fleshy
Reaction time	quick	average	slow
Sub Total			
TOTALS			
MENTAL			
BEHAVIORAL			
PHYSICAL			
ATHLETIC			
YOUR MIND-BODY TYPE GRAND TOTAL			
	VATA	**PITTA**	**KAPHA**



A New Approach for the Reticular Formation

Datis Kharrazian, D.C.

Abstract

This paper discusses two simple procedures. One technique is for the ascending reticular system and the other for the descending reticular system. These techniques have been especially helpful in those patients that do not facilitate or inhibit clearly to normal applied kinesiology receptor challenges such as therapy localization, gustatory nutrient challenge and pinch and rub techniques. The importance of these two techniques and its relation to the hierarchy of the nervous system will be discussed.

Introduction

Dr. Walter Schmitt has stated, "It is our task to begin to employ the principles of muscle testing as functional neurology in unraveling as many areas of the nervous system as possible."[1] We now know that using three types of muscle testing (GI, GII. GII-S) we are able to differentiate which inhibitions are segmental and which are supra segmental in origin. Dr. Richard Belli has also demonstrated that individual muscle weakness is not possible unless it is purely structural as in origin insertion technique. He has revealed that different muscle inhibition patters (that demonstrate GI, GII, GII-S) are related to different areas of the nervous system.[2] For example, upper and lower body extensor inhibition patterns are related to the cerebellum and are corrected by the proprioceptive bombardment of a coupled adjustment. Bilateral muscle inhibition patterns tested simultaneously are related to the vagal motor nuclei and are treated with techniques that stimulate the autonomic nervous system.

After attending three amazingly informative seminars on "The Neurological Applications of Applied Kinesiology", by Dr. Belli and Dr. March, I realized the importance of treating patients suprasegmentally downward. I noted that the muscle inhibition patter related to the reticular formation was not emphasized. This is probably due to the fact that the reticular pattern of inhibition, which is demonstrated by weakness of the upper body extensor and lower body flexor tested bilaterally, is not very common.

I theorized, that just as we could stress out the adrenal neurolymphatics with the insalivation of caffeine, the reticular formation also can be stressed out with a loud noise such as clapping. The specifics of the techniques will be discussed later in this paper.

I found these techniques would sometimes clear all inhibition patterns, sometimes clear no muscle inhibitions, and sometimes facilitate a few. However, the most essential point I observed was with those patients that had slight or minimal facilitation to therapy localization and other receptor challenges revealed dramatic increases in facilitory and inhibitory muscle responses after performing these techniques.

For example, with one of my patients that accumulated mercury toxicity demonstrated by hair analysis had no inhibitory muscle response after having her insalivate a homeopathic mercury product (12X). I also had her therapy localize to the liver neurolymphatics with the mercury stress and observed no change in muscle response as well. I observed that after performing the techniques for the ascending and descending

reticular formation, she had profound inhibitory response to lingual mercury exposure. I believe that the initial mercury challenge did not cause an inhibitory muscle response because the nervous system was not optimally integrated to manifest such an inhibition.

Another patient that demonstrated functional hypothyroidism secondary to the anterior pituitary evidenced by low T3 and a TSH of 0.5 that did not respond to nutrients for the pituitary until these techniques were performed. This may explain why we may observe applied kinesiology muscle responses at times that do not coincide with other data such as lab tests. It may not be the nutrient that is inappropriate, but the nervous systems inability to demonstrate itself as a muscle response.

Discussion

The reticular formation is composed of many scattered groups of diffuse nuclei (formations) in the large portions of the tegmentum. Because the nuclei and their interconnecting chain have a diffuse and poorly defined appearance, they were given the name the reticular system (a reticule is the network of lines in a telescope sight). The reticular formation extends all the way from the superior end of the spinal cord to the diecncephlan, passing through the medulla oblongata, the pons, the mesencephlan, and even into the middle of the thalamus where it is represented by the intrlaminar nuclei.[3]

A generalized function of the reticular formation is to regulate the level of consciousness and arousal that are generated form the somatosensory, auditory, visual and visceral systems.[4] The reticular formation is one of the primary centers of the nervous system for controlling the brains overall level of activity. Guyton states, " The reticular formation though dispersed rather broadly in the brain stem is functionally one of the most important of all brain structures."[5]

The Ascendng Reticular Formation

The reticular formation, phyogenitically is one of the older systems in the nervous system. It is divided anatomically and functionally into two parts: an ascending and a descending formation.[3] We will first discuss the importance of the ascending reticular system. The reticular ascending system has also been called the reticular activating system and is concerned with various levels of conscious alertness. The lateral spinothalmic tract, the anterior spinothalamic tract, auditory and visual pathways all send collateral axons that end in the nuclei of the reticular activating system. These scattered nuclei formations then transmit sensory information from the aforementioned pathways to a collection of nuclei in the thalamus called the midline group. From the relay center of the thalamus, information is sent up to the cerebral cortex. In the cerebral cortex, the impulses influence the states of mental alertness and sleep.[3]

The functional integrity of these pathways are especially significant in applied kinesiology assessments. Many A.K. techniques use pinch (to stimulate a nociceptive or a sympathetic response), rub (to stimulate a mechanoreceptor input or parasympathetic response), and touch (therapy localization) in the assessment of patients.[13, 14] If the ascending reticular system is not functioning at an optimal level of integration we may not have the optimal sensory information reaching the cerebrl cortex. Therefore, the upper brain centers may not send correct or optimal information down the common neural pathway which transcends into muscle testing. This ascending pathway may not be as crucial for pinch and rub techniques that affect segmental spinal reflexes, but they are important in many ascending and descending supra segmetnal influences.

An approach to distress this system is suggested in this paper. It requires the doctor to begin with a facilitated indicator muscle. The doctor then claps next to the patients ears to stimulate the reticular activating system. If the ascending reticular system is not properly integrated the patient will demonstrate a supra segmental injury recall pattern. The muscle weakness pattern includes all three types of weakness that will not autogenically facilitate. The correction is made with clapping and then performing injury recall technique.[6]

Occasionally, clapping alone is not enough to distress the reticular activating system. In these cases, increased stimulus to the lateral spinothalamic tract is required. When increased stimulus is necessary, apply a cold stimulus to the body to stimulate the lateral spinal thalamic tract. After this tract has been activated you can then clap next to the patients ears to stimulate the reticular activating system, this increased the summation of influences reaching the reticular activating system, this will then cause muscle weakness. This pattern must be differentiated from weakness caused by a cold sensation as discussed by Dr. Michael Allen, which is a dysfunction of the spinothalmic tract alone.[15]

Rarely, the patient will demonstrate all three types of muscle weakness that does not autogenically facilitate. As we know IRT will not influence the nervous system when there is autogenic facilitation.[7] There must be a withdrawal response significant enough that affects the alpha motor neurons, by bypassing the gamma motor loop. In these cases, you must first correct one of the following: spinal centering, a coupled cervical adjustment, or the emotional neurovasculars.[8]

One or more of the above techniques will either correct the weakness induced by clapping or set up the nervous system for IRT by producing a weakness pattern that does not autogenically facilitate. When clapping causes a weakness pattern that autogenically facilitates it demonstrates a reticular system that is influenced by higher level inputs. This is most likely from the parabrachial nucleus located in the mesencephlan. Guyton states the inputs from the parabrachial nucleus and the mesencephalic reticular formation stimulate patterns of activity such as gait, flexion and extension patterns.[9] This is most likely the pathway normalized when spinal centering corrects or allows autogenic facilitation to not take place.

When a coupled cervical adjustment corrects the reticular pattern it is most likely due to the influence of coordinated cerebellar inputs sent to the pontocerebellar pathways and back into the parabrachail nucleus which influences the mesencephalic reticular formation.[9] The atlas also has a direct relationship with the ascending reticular system. The neuromere of the atlas, embryonically, migrates upwards and becomes the locus cereleus in the upper brain stem. The locus ceruleus is the neurological center of the ascending reticular system and can cause deafferentation to the reticular system when it is subluxated.[16]

If emotional neurovasculars correct the reticular pattern it is most likely the influence of the limbic lobe and the hypothalamus which sends neurological messages that impact the mesencephalic reticular formation.[9]

Summary for the Ascending Reticular System

1. start with a facilitated indicator muscle
2. clap next to the patient's ear
3. test muscle weakness (GI, GII,GII-S) with autogenic facilitation

4a. if there is no autogenic facilitation, correct with IRT

4b. if there is autogenic facilitation, correct with spinal centering, a coupled adjustment, or emotional neurovasculars

The Descending Reticular System

Submerged in the brainstem are a dispersed group of nuclei belonging to the descending reticular formation. In the midbrain they are called the deep and dorsal tegmental nuclei, in the pons they are called the central tegmental nuclei, and in the medulla they are called the central and inferior nuclei. These scattered nuclei obtain data from the mamillotegmental tract and the longitudinal fasiculus which are hypothalamic fiber tracts. Also, information from various parts of the nervous system (globis pallidus, substantia nigri, subthalmic nucleus and the extapyramidal system) go to the nuclei or formations of the descending reticular system. From these nuclei axons from the ponitine reticulospinal tracts and the medullary reticulospinal tract which are the medial and lateral reticulospinal tracts respectively. These are descending, crossed and uncrossed multisynaptic pathways that travel to all areas of the spinal cord. These tracts end in the common final pathway which terminates on the anterior horn or on the intermedial cell column.[3]

The descending reticular system performs two main functions. First, it relays involuntary motor impulses from the extrapyramidal system the voluntary muscles. Second, it relays impulses from the hypothalamus to pregangloinic neurons of the autonomic system.[3]

As we can see, there is a tremendous influence of the hypothalamus with the descending reticular formation. It has been theorized that the beginning and end points have a strong relationship with the hypothalamus.[10] A technique is suggested that incorporate the basic B and E points and the stimulus of a loud clap to stimulate the descending reticular system. In this technique the doctor must first make sure that the B and E points are negative in the clear. The doctor must also make sure that clapping does not cause an indicator muscle to weaken (if weakness occurs, correct using the protocol discussed earlier). At this point the doctor claps and has the patient therapy localize each of the B and E points to see if a weakness is induced. It is sufficient to just clap once and check all the B and E points. The weakness contains all three types of muscle weakness patterns and rarely autogenically facilitates. Having the doctor clap and tap the indicated B and E point for 5-10 seconds and then perform IRT makes the correction. If autogenic facilitation takes place correct in the same manner to allow autogenic facilitation to not take place as discussed previously.

Correcting the descending reticular system has been especially helpful with patients that demonstrate no or minimal facilitation to lingual stimulus testing. As Dr. Walter Schmitt has pointed out to us, based on the teachings of Dr. Fredrick Carrick, the descending reticular formation has a major role in this pathway. The proposed pathway, in sequence for the gustatory-motor response is the following: gustatory receptors-cranial nerves VII, IX and X-nucleus soltarious-hypothalamus-reticular formation-reticulospinal tracts-anterior horn-alpha motor neuron-muscle.[11] Clinically, consider performing the above protocol anytime you have a patient that shows all the signs for a certain supplement and there is no facilitory muscle response. The reason there might not be facilitation may not be due to an inappropriate substance, but rather an inability of the nervous system to facilitate the anterior horn. Also, it is recommended to correct this pathway before using protocols that require having the patient test offenders for IRT. These include techniques such as centering the spine and liver detoxification technique (Schmitt), on those difficult responding patients.

For all the above reasons, I recommend that doctors correct the ascending and descending pathways first before they use other A.K. challenges. It will make the afferent and efferent pathways much clearer and result in more accurate muscle testing.

Summary for the Descending Reticular System

1. make sure clapping and B and E points are negative in the clear
2. then clap and T.L. the B and E points to see if any of them causes a weakness (GI,GII,GII-S) that does not autogenically facilitate
3. if a B and E point is active after clapping, the doctor claps again and taps the B and E point for 5-10 seconds and performs IRT.

Conclusion

Dr. Walter Schmitt has written about adopting the neurological model and its importance to the hierarchy of treatment protocols.[12] I feel the aforementioned techniques should be placed on the top of the list. Not because they correct the most supreme centers of the nervous system, but due to the fact the reticular formation connects higher brain centers of supra segmental influences (GII, GII-S) to the anterior horn (GI). Since we use manual muscle testing as one of our prime methods of assessment, it makes sense to correct the bridge between the anterior horn and the rostral-organized systems first. I propose that not only should we work brain down, but also we should connect the higher brain centers to the local spinal centers as our initial procedure in those difficult patients.

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Static and Kinetic Visual Analysis of the Foot and Ankle

David W. Leaf, D.C., DIBAK

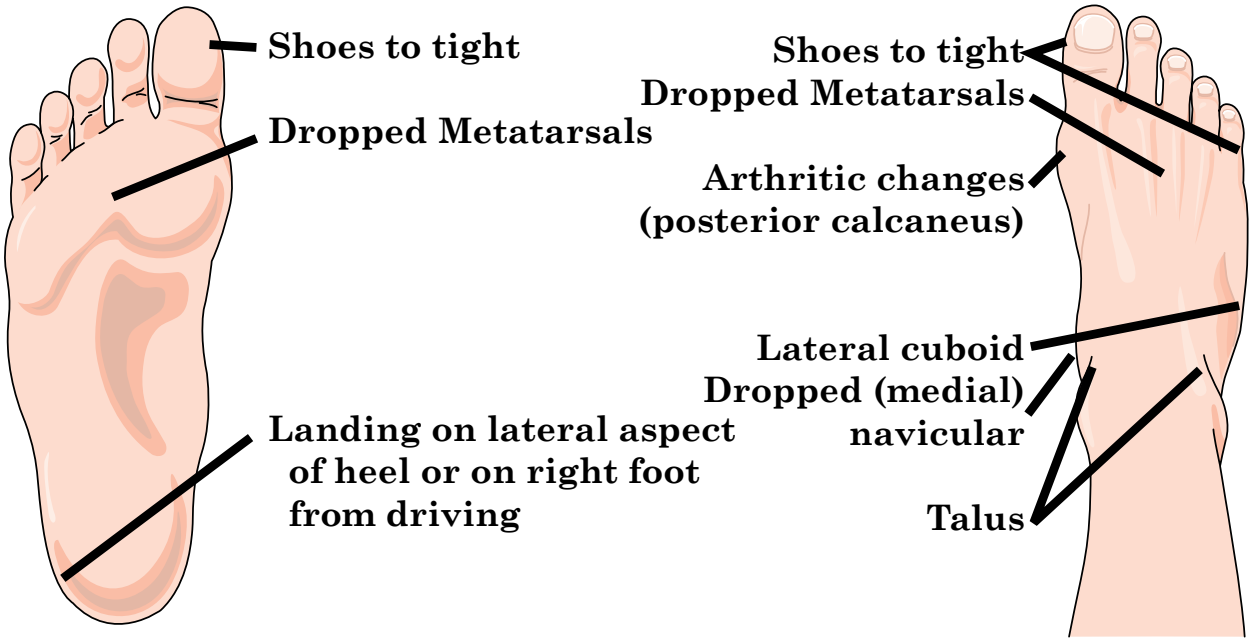
Examination begins with the visual analysis of the patient. Inspection is one of the key ingredients in helping you decide where to start in your examination process. However, inspection of the patient is usually an under utilized tool for the clinical practitioner. This is even more true of kinetic analysis of the patient. There are many charts of static findings. However, these usually take into account only the weakness or over contraction of a single muscle or group of muscles.

This paper will center on the visual analysis of the lower leg, especially the area from the tibia distally.

The visual examination should cover three distinct phases. These are the static examination of the foot weight bearing and non-weight bearing. Visualization while the patient is rising onto his/her toes, and then finally analysis during ambulation or running.

Static Examination

The patient should have their shoes and socks removed and the external surface of the foot and ankle should be examined for any areas of excess friction. These will show themselves by the development of blisters or calluses. You should also not the integrity of the skin, discolorations, conditions of the nails and any signs of circulatory problems. Pay special attention to the distal portion of the foot and observe the general alignment of the metatarsals and tarsal and the condition of the first metatarsophalangeal joint.



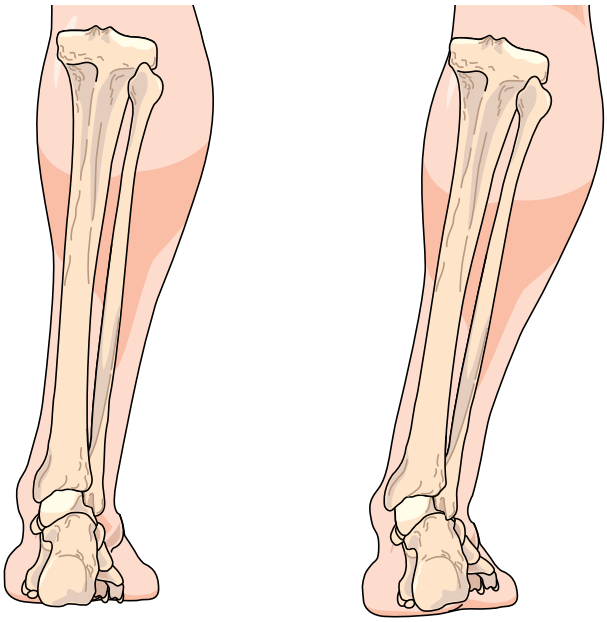
The patient should then be asked to stand on their feet. Have them walk in place for two to four steps and then stand normally. Starting from the posterior, observe the alignment of the Achilles' tendon. Then palpate and observe the condition of the longitudinal and metatarsal arches. Note any excessive dropping of the navicular. Have the patient lift the weight off of the foot slightly and observe the lateral expansion of the foot. On the lateral aspect of the foot, observe for any excessive lateral displacement of the cuboid. In addition, note the amount of toeing in or out that the foot makes from the midline. Stand behind the patient and place your fingers around the patella and note the degree of internal or external rotation of the femur as compared with the position of the feet.

The next step is to have the patient rise on to their toes slowly. Note the ability of the patient to maintain their balance. As the gastrocnemius contracts shifting the body weight forward, first the peroneus tertius then the tibialis posterior and then the peroneus longus and brevis stabilize the body weight. Finally the flexors of the toes take over and the end stage. Gauge at which stage of rising that the patient loses control of their balance and mark that muscle for special attention. Near the end phase of rising, you may see the great toe suddenly lift into the air due to contraction of the extensor hallucis muscles. This is a common finding when the metatarsal arch has collapsed

The final part of the examination is to have the patient walk. During this phase of the examination, you should observe the foot from the posterior, anterior and from the side.

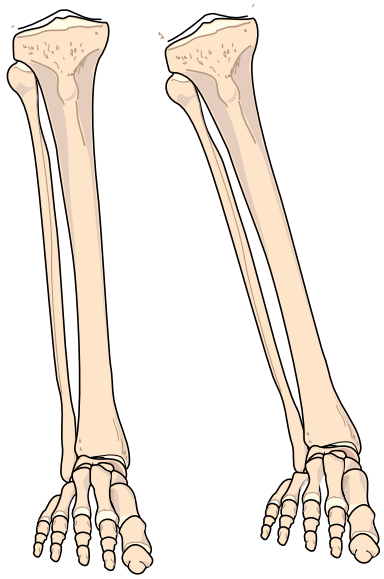
Posterior viewing

From the posterior, it is easy to watch the action of the foot from full loading after heel strike through the end of the stance phase of gait. During this transition, while the center of gravity of the body is being carried over the foot, observe for excessive dropping of the arch and medial shifting of the talus. Pay attention to the general stability of the pelvis at this point. Excessive pronation will result in tibial and femur torsion effecting the stability of the femur head in the acetabulum. This is indicative of a weak tibialis posterior. As the patient continues from full plantar contact into heel rise, observe the degree of elevation of the heel. Some patients walk with an extremely short stride and never get into toe off. Instead stopping the stride while the foot is still in almost complete contact with the floor. Others will toe off violently with excessive heel kick due to an overcontraction of the gastrocnemius. These patients will usually have extreme tenderness over the Achilles' tendon if it is palpated for. Observe the action of the foot and the lower leg during the swing phase of gait for a looping type action that swings the leg forward. This can be due to excessive pelvic rotation or an imbalance in the hamstrings.



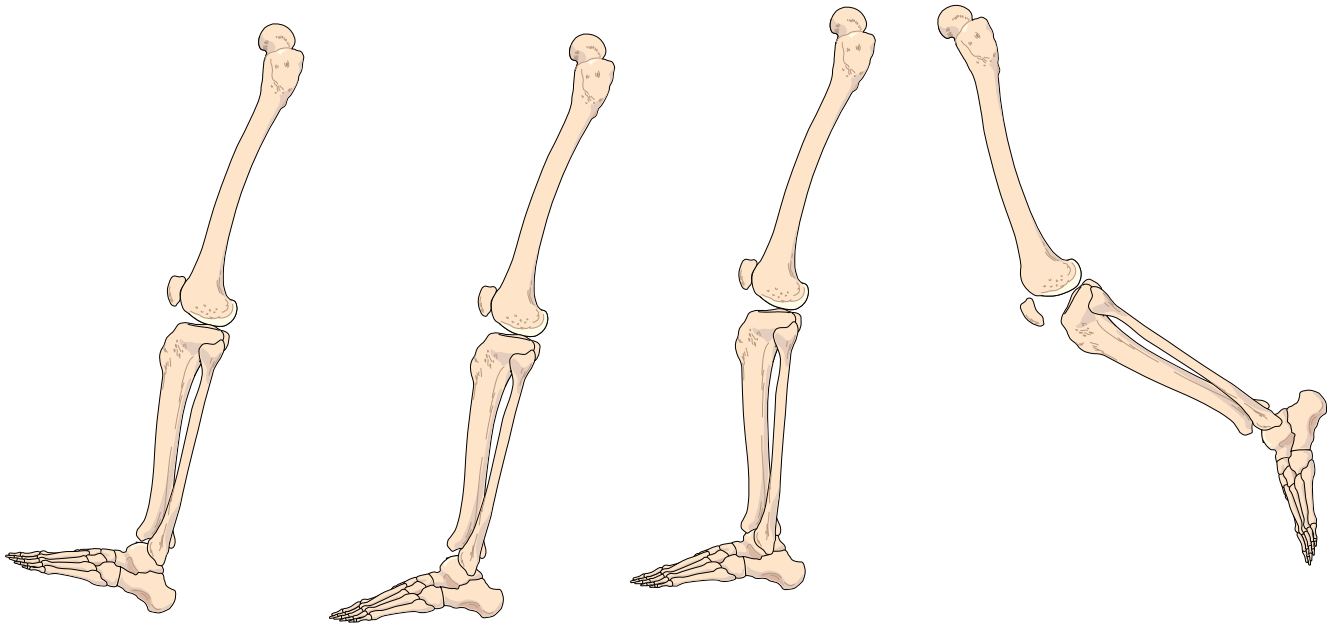
Anterior Viewing

When viewed from the anterior, observe the degree of internal or external rotation of the foot at heel strike. From this angle you first get an idea of the degree of contraction of the vastus muscles in kicking the lower leg forward. Excessive kicking will be found as an accommodation to a short stride on that side. Observe the angle that the foot makes with the ground at heel strike. Weakness of the tibialis anterior will result in a flat foot or almost horizontal angle at foot strike. Note the width of the stride. Excessive widening of the stance is usually due to adductor weakness or a neurological imbalance requiring the patient to widen their stance to gain stability. Crossing over the midline is usually a sign of a TMJ imbalance with inhibition of the abductors of the thigh. Observe the motion of the arms, the side of less motion will usually be opposite the side of the short stride. As the weight of the person is carried over the foot, observe the stability of the talus for any medial - lateral shifting.



Lateral viewing

From the side, it is easy to measure the length of the stride and to observe the angle of attack that the foot makes with the ground. Patients who attack the ground flat footed or with the distal portion of the foot are inclined to create anterior displacement of the talus. You should also observe the angle of the knee at the moment of heel strike. The knee should have a slight degree of flexion. This allows for shock absorption. An excessive degree of bend could indicate a weakness of the vastus muscles failing to kick out the tibia.



From left to right, the first diagram represents the normal knee bend on heel strike with the foot in a slightly flexed position to prevent tripping. The next picture shows landing with the foot in a non-flexed position. This results is easy tripping and anterior movement of the talus on the calcaneus. The third picture is an example of failure of the vastus muscles to extend the lower leg. This also results in a horizontal landing position of the foot at heel strike. The final picture represents excessive heel kick due to overcontraction of the gastrocnemius.

With a little practice, you can quickly evaluate the mechanics of motion and shorten the diagnostic time needed to find problems that your patient has.

This has been a short discussion on the various steps that should be made in the visual analysis of your patients. A video showing various weaknesses will be prepared for showing most of these patterns.

Releasing Emotional Patterns with Essential Oils

Carolyn L. Mein, D.C., DIBAK

Abstract

It takes more than talk therapy to change core issues. Changing a deeply ingrained response pattern requires releasing it from the cellular structure of the body. Acupuncture has proven that emotions are stored in specific glands and organs and can be accessed through the acupuncture alarm points. While helpful, there were still missing components. The seat of emotion is the limbic system of the brain and is accessed through smell. By identifying the emotion, its complementary other side, the lesson or the "way out" of the uncomfortable state, and applying essential oils to specific points, core issues can be accessed, released and transformed.

Introduction

Life experience is about experiencing, learning, growing and mastering. Since the body never lies, it is an incredible indicator as to how well a person is doing and what lessons still need to be learned. Other indicators are a person's emotional state and how their life is being reflected in their world.

The first indication that a person is out of balance is pain, physical, emotional or both. Many physical problems have an emotional component, especially when they are chronic. The presence of an emotional pattern needing attention can be identified by using an applied kinesiology (A.K.) muscle test and challenging the emotional points on the frontal eminences. The specific emotion can be identified by challenging the alarm points or focusing on the emotion and testing a strong indicator muscle.

A positive emotional response indicates an emotional blockage. Simply releasing the emotion helps relieve the pressure, but if the problem is deep seated and the "way out" is not clearly understood, the indicator and problem eventually return. The ultimate goal is to learn from life's experiences. Once a situation is understood and the decision has been made to change the pattern, the reprogramming begins to take place.

While the decision to change a pattern is the first step, it alone is not enough to shift a well ingrained response. Once the pattern or emotion is identified, the complementary other side needs to be explored to allow the energy to be expressed a different way. Now that the alternative response is understood the next question is, "How do I get there from where I am?" Using a statement that encapsulates what needs to be learned from the experience provides a "way out". For example, we know from traditional acupuncture that anger is stored in the liver. The opposite emotion, or the emotion that will shift and transmute anger, is laughter. Anger is a common response that flares up when a person's path has been blocked and they see no other way out, routinely seen expressed as "road rage". Shifting anger to laughter requires redirecting the energy. Using a statement that provides a "way out" such as "My direction is clear," changes a person's focus and redirects the energy so a solution can be seen.

Once the emotion has been identified, its other side understood, and the lesson or the way out known, it needs to be released from cellular memory. Caroline Myss, Ph.D.1 states that 70% of the body's cells need

to shift before a new direction becomes a reality. So, how do we get cells to shift? Cellular memory contains patterns held mentally, emotionally and physically. Emotions stored in the tissues of the body can be accessed through alarm points, and the emotional seat in the limbic system of the brain through smell.[2]

Clearing a deep pattern requires bringing the lesson to conscious awareness and fully understanding it, essentially learning the lesson. Clearing the emotional pattern out of the body requires feeling the emotion, and releasing it from the body's cellular memory. Emotions stored in the glands and organs can be accessed through the acupuncture alarm points and the limbic system through smell. Essential oils hold vibrational frequencies that can shift patterns. Using them on the acupuncture alarm points sends the frequency directly to the specific organ and smelling the oil releases the pattern from the limbic system of the brain, thus providing direct access to the body's cellular memory.

Case History

A twenty-six year old male whose dominant gland and body type is Thymus, core issues are judgement and control, with anger as the dominant emotional response. He began using the essential oil blend of Purification[3] on the liver alarm poins and the emotional points at the frontal eminences seven times a day along with feeling the emotions of anger, then laughter, and saying the statement, "My direction is clear."

In less than a week, he had an experience where his favorite guitar was knocked over and the case chipped while at band practice. His normal response to situations like this would be intense anger that permeated everyone in his environment and continued for at least a week before any solution could begin to surface. This time, he walked into the house and told his mother he was angry because his guitar had been chipped. Within the hour he was out in the garage, had found some black paint, and repaired the chipped spot to where is was almost undetectable. This incident has been followed by similar situations where he has been able to quickly move out of an anger response, and even by-pass the anger, to reach a viable solution that works for everyone.

Once he finished using the Purification oil for three weeks, the next emotion, frustration which is stored in the common bile duct,[4] surfaced. The "other side" of frustration is accomplishment, the "way out" is, "I move beyond my limitations." Having finished using lemon oil seven times a day for three weeks, he was ready to deal with the core issue for the Thymus body type, fear of failure.[5] The "other side" of fear of failure is unfoldment and the "way out" is, "I accept growth". The alarm point is the thymus and essential oil is peppermint.

Clearing the emotional patterns with the "way out" and essential oils has allowed him to transform core issues and change emotional responses he had been dealing with his entire life. He has been able to easily integrate the new patterns into his being and his life is easier and happier as a result.

Materials & Methods

An emotional pattern may be identified from the emotion itself or from the organ of complaint. The following chart lists the emotions found in the major organs. Due to space limitations, additional organs, points, emotions and point location charts can be found in Releasing Emotional Patterns with Essential Oils.[6]

It is important to use essential oils that are of therapeutic grade, as diluted and lesser grade oils do not carry the same vibrational frequency. Each company has its own blends, the ones listed are from Young

Living.[7] Single oils are available from any good supplier, I've referenced the companies I know to carry quality oils.[8 9]

The majority of the words listed under emotions are of the negative polarity. The ones that are positive relate to the fear of, such as the fear of love, not being loved or not being lovable.

EMOTIONS	OTHER SIDE	WAY OUT	OIL	ORGAN
Abandonment (Fear of)	At-one-ment	I learn from all of life's experiences	Lavender	S.Int.
Acceptance	Rejection	I can be accepted	Sara	Em. pt.
Addiction	Freedom	I am wanted & lovable	Peace & Calming	Brain
Anger	Laughter	My direction is clear	Purification	Liver
Betrayal	Faithfulness	I have the courage to accept the truth	Forgiveness	Pancreas
Control (Fear of losing)	Balance	I am content and blessed	Peace & Calming	Stomach
Criticism (Fear of)	Unconditional love and acceptance	I receive	Lavender	Skin
Disappointment (Feeling)	Encouragement	I express my vision	Joy	Bronchial
Facing the World (Fear of)	Embracing the world	I am safe	Myrrh	Adrenal
Failure (Fear of)	Unfoldment	I accept growth	Peppermint	Thymus
Frustration	Accomplishment	I move beyond my limitations	Lemon Bile Duct	Common
Guilt	Deserve (Get what you)	I learn from all of life's experiences	Clarity	Spleen
Identity	Purpose	I am in touch with my purpose	Release	Uterus/ Prostate
Injustice (Fear of)	Resolution	I accept the truth	Sacred Mtn.	Thyroid
Letting go	Happiness	Let go and let God	Sage	Bladder
Loneliness	Connectedness with all that is	I go to a loving space	White Angelica	Heart
Love	Detachment	I allow myself to be real	3 Wise Men	Kidney

Past (Fear of repeating)	Awareness	I learn from all of life's experience	Forgiveness	Gall-bladder
Rejection	Acceptance	I accept all that I am	Purification	Lung
Repression	Creativity	I change my perception	Clarity	Ovaries/ Testes
Success	Rejection	I accept awareness	Release	L.Int.
Unknown	Knowingness	Listen to your heart	Sacred Mtn.	Pineal
Used (Fear of being used)	Respected	I respect who I am	Jasmine	CX @ CV-5
Wisdom	Confined	Face the fear	Ylang Ylang	Pit.

To clear an emotional pattern, begin by identifying the feeling or emotion. This brings it into conscious awareness. Once it has been identified, the emotion and the thought pattern that created it needs to be understood. Next look at the "other side" or positive emotion, this allows the experience to be seen from a larger perspective. The "way out" is a statement or affirmation that provides a way to shift the energy from the negative to positive. It focuses on the essence of the lesson so it can be easily seen and understood. Once the "way out" of a negative feeling is known, it is easy to shift out of an undesirable emotional state. When the negative emotion has lost its hold, the lesson has been learned and the situation is free to shift. If the negative emotion or situation reappears, the tools are in place to quickly shift one's attention and focus. This allows a position of choice, and personal empowerment.

Clearing deep seated emotional patterns requires releasing held emotional patterns and replacing them with the desired response. The depth of the emotional pattern determines how often the release pattern needs to be done. Once the emotional pattern has been understood, it can be cleared by smelling the essential oil, applying it to the alarm point or points and the emotional points.

Typical application requirements have been three, seven, ten, or eighteen times per day for one, three, or seven weeks. The oils may be applied as close as 15 minutes apart, so patients can use them before and after work when they have a chance to focus on the emotions. If the patient is not able to use the oil as frequently as required or needs to take some time off to process the emotions, they can extend the length of time the oils are applied.

Some people experience the release of painful memories from the past with regrets about how they lived their lives and what could have been. While some become quite emotional, others can experience agitation or depression if they are unable to connect with the feelings from the past. It is helpful to encourage them to write, go for a walk, meditate, talk to a close friend, or exercise. Essentially, they need to do whatever works for them to release held emotions.

Adding sea salt to the bath, or using it like soap in the shower cleanses the emotional body. The two areas to emphasize are the solar plexus and chest. Using sea salt is particularly helpful when a person is going through their own emotional release or is around anyone else who is, and for many, this is generally most of the time.

Summary of Procedure

1. Therapy localize the emotional points on the frontal eminences. If a previously strong muscle goes weak, an emotional pattern is present.
2. Identify the emotion by asking the patient what issues or emotions they are currently struggling with, and testing the indicator muscle. Once a definite response has been found, confirm it by therapy localizing the emotional points and the corresponding organ alarm point for the identified emotion.

   If the patient is unable to identify an emotion, go to the area of complaint and therapy localize the organ alarm point. Confirm with the emotional points and the emotion.
3. Ask the patient to feel the emotion, then the emotion on the "other side", testing using the indicator muscle helps solidify the emotion into the patient's experience. Explain the emotions as necessary for the patient's understanding. State the "way out", test the indicator muscle and explain as indicated.
4. Have the patient smell the oil, place a drop in your non-dominant hand and rub the oil clockwise to activate. Place the oil on the alarm point, or points if bilateral, and the emotional points. While applying the oil, state the emotion, the "other side" and the "way out", encouraging the patient to connect with their feelings and allow them to surface.
5. Test the patient for frequency of use. Occasionally, one application is enough. Depending on how deep seated the pattern, patients usually need to apply the oil 3, 7, 10 or 18 times per day for 1, 3, or 7 weeks. The oils may be applied as close as 15 minutes apart, so patients can use them before and after work when they have a chance to focus on the emotions.

   If the patient is not able to use the oil as frequently as required or needs to interrupt usage, they can extend the time period. Frequency and duration of treatment is an indiction of the depth of the emotional pattern, not an absolute. Some patients may need to extend treatment, so recheck once they feel complete.
6. Review with the patient what they will be doing:

   a) feel the emotion and smell the oil,
   b) apply the oil to the alarm point and connect with the "other side" emotion,
   c) apply the oil to the emotional points and state the "way out".

Conclusion

Talk therapy is essential in understanding the situation, but it takes more than head knowledge to change a pattern. The pattern needs to be released from the body for a conditioned response to change. Once understood, the stored emotions can be released from the cellular memory through the alarm points. Aromatherapy is used to access the seat of the emotion in the limbic system of the brain. Identifying the (negative) emotion, its (positive) complementary "other side", and the lesson with the "way out" of the uncomfortable state brings the lesson to conscious awareness. This knowledge provides the awareness necessary to learn the lesson and change the behavior pattern. Smelling and applying specific essential oils to the areas where the emotions are stored allows the cellular pattern to be released from the body, so the emotional pattern can change.

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Lesion Accentuation Spinal Technique

Dean B. McGee, D.C.

Abstract

Throughout the healing arts, a common thread is found in the search for long-term improvements in the health of our patients. In the profession of chiropractic, perhaps the greatest concern historically has been the ability to make lasting adjustments to resolve spinal subluxations.

"Ideally, when a subluxation is corrected it does not recur."[1] But the difficulties in achieving this goal are evident in the concept of spinal "listings" - subluxation patterns that frequently require repeated attention. Admittedly, the spine can present a special challenge due to the complex interactions of the five extensive layers of musculature which cover it.[2]

This article proposes a spinal correction that does not directly reduce subluxations, but rather gently accentuates the lesions to allow the vertebrae to "rebound" into proper alignment. The author has found this form of stimulation to be effective in achieving lasting corrections by strengthening the weakened soft tissues, lengthening the shortened tissues, and restoring both to their proper function.

Introduction

Accentuating a defect to stimulate a physical response is not an new concept. This concept is prominent in homeopathy, which defines health as the ability of an organism to successfully adapt to its environment. When proper adaptation does not occur, an offending particle is introduced as a stimulus to cause the body to rebound to a corrective state.[3, 4]

The concept is also seen in trigger point therapy, where pressure is applied to an area of decreased vascularity to further constrict the blood vessels. When the pressure is released, the response is a "rebound vasodilation" that allows the tissues to return to normal vascularity, with a resulting decrease in ischemia, pain, and muscle hypertonicity.[5]

Applied Kinesiology has used similar procedures to improve the body's ability to achieve lasting corrections through restoring proper tension to the soft tissues, thus providing for proper alignment and function of the associated joints. In the soft tissues, the sensors that monitor and regulate tension are the muscle spindle cell, the Golgi apparatus, and the Golgi tendon organ. These sensors respond to accentuation of the tissue distortions by rebounding into correction. With static compression of ligaments, tendons, and muscle spindle cells, the approximated fibers respond with "rebound inhibition". Likewise, when these tissues are distracted, they respond with "rebound facilitation". In general, the principle in each of these procedures is the accentuation of a defect to stimulate the body to respond by rebounding into correction.[1, 6]

The correction does not occur instantaneously when a challenge is performed for the same reason that Bennett's reflexes are not treated by simply therapy localizing the points: the procedure requires a more prolonged stimulus. It involves minimal force at a sustained pressure as taught by Dr. I. N. Toftness.[7] The amount of stimulus to be provided should be the minimal amount required to cause recognition of the defect.

Discussion

This procedure produces compression of hypertonic muscles and tendons, and shortened ligaments, and distraction of weak muscles, flaccid tendons, and over-extended ligaments.

This principle can be demonstrated by analyzing a specific subluxation. For instance, if the atlas were subluxated with the right transverse process tilting superiorly, any of the following might be seen:

1) weakness of the left rectus capitis lateralis muscle
2) lengthening of the left rectus capitis lateralis tendon
3) lengthening of the left atlanto-occipital capsular ligament
4) hyperirritability of the right rectus capitis lateralis muscle
5) shortening of the right rectus capitis lateralis tendon
6) shortening of the right atlanto-occipital capsular ligament

The subluxation could be located either by rebound testing or by testing with direct pressure, as in standard AK testing. In this case, weakening of a strong indicator muscle would occur if tested after pressure was applied inferior-to-superior at the left transverse process of the atlas and then released. Weakening would also occur if tested as sustained pressure was applied inferior-to-superior at the right transverse process.

However, with this procedure, the correction is made in the direction and vector that produces maximal weakening with direct pressure, instead of with rebound testing. In the above case, sustained pressure would be applied inferior-to-superior at the right transverse process, producing the following stimuli:

1) distraction of:
    a) the left rectus capitis lateralis muscle
    b) the left rectus capitis lateralis tendon
    c) the left atlanto-occipital capsular ligament
2) compression of:
    a) the right rectus capitis lateralis muscle
    b) the right rectus capitis lateralis tendon
    c) the right atlanto-occipital capsular ligament

After the correction, in the majority of instances, the vertebral challenge will no longer be present. It has been my finding that, on future visits, the challenge will continue to be absent.

Conclusion

Low force corrections to the spine are not a new concept. However, this particular technique should be considered more of a soft tissue correction than an osseous adjustment, even though it has a direct impact on joint function.

The correction is thought to occur due to stimulation of the monitoring components of the soft tissues (muscle spindle cells, Golgi apparatus, and Golgi tendon organs), resulting in normalization of the tissues. With static compression of ligaments, tendons, and muscle spindle cells, the approximated fibers respond with “rebound inhibition”. Likewise, when these tissues are distracted, they respond with “rebound facilitation”.

Based on our understanding of homeostasis, the most effective and longest lasting corrections are ones that stimulate the body into active response to achieve health. It is only when the body fails to respond that we opt for corrections where the body is a passive recipient.

The author has no disagreement with dynamic adjusting in the direction and vector that produces maximal weakening with rebound testing, as the rationale in that case is to return the vertebra to its normal juxtaposition. However, it is recommended that the soft tissues are corrected with the above procedure, and the subluxation is re-examined prior to the adjustment to confirm that the subluxation still exists.

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Changes in Craniomandibular Neuromuscular Function and Biomechanics Following Applied Kinesiology Diagnostic Procedures and Manual Manipulative Therapy

Eric Kees Peet, D.C. and Karen Clister, D.D.S.

Abstract

This paper demonstrates the potential changes in temporomandibular (TMJ) function which are possible utilizing applied kinesiology diagnostic procedures, and subsequent manual therapies. A computer aided bioelectronic measuring device, a Myotronics K-6-I, was employed for the purpose of objective documentation of the craniomandibular and TMJ function. Pre and post measurements of both the electromyographic activity within the craniomandibular muscles, and the mechanical range of motion and movement patterns of the TMJ where taken. Manual manipulative therapy was performed according to Applied Kinesiology diagnostic and therapy procedures. Improvement in virtually every measured variable was demonstrated in this study.

Introduction

This case report is intended to demonstrate that applied kinesiology diagnostic procedures and subsequent manual therapy on the body affects the neuromuscular function and biomechanics of the temporomandibular joint (TMJ). Due to the common neurology shared in the brainstem between the dorsal columns of the spinal cord, and the trigeminal neurology of the cranium, analogous relationships in the neuromuscular balance between the two can be evaluated and modified. The musculoskeletal balance between the cranium and the body are dependent upon each other. Putting this physiological phenomenon to clinical use creates an ideal situation for co-management between functional dentistry and applied kinesiology. The skilled applied kinesiology physician can correct neuromusculoskeletal imbalance and optimize the person's metabolism required for healing, while the functional dentist can correct the proprioceptive and biomechanical imbalance in the occlusion. This complimentary relationship significantly improves the effective management of TMJ dysfunction. It can significantly reduce the treatment time, and improve the outcome of this often chronic and misunderstood disorder.

Bioelectronic analysis of TMJ and craniomandibular function as a diagnostic aide has been used in the field of functional dentistry for several years, and is well established. Its use for documenting changes from manual therapy however, is novel. With the increasing prevalence of applied kinesiology/ functional dentistry co-management, this type of documentation should become more common and undoubtedly yield further research.

Materials and Methods

Computer aided measurements utilizing a Myotronics K6-I device was used as an integer of TMJ function before and after manual manipulation to the body and cranium. A combination of functional assessment procedures and manual therapies consistent with applied kinesiology protocol was utilized. A staff technician of the dental office performed the K6-I computer evaluations in a separate exam room, and was blinded from the functional evaluation techniques, and manual therapy procedures.

Applied Kinesiology functional assessment procedures included: symptom survey forms, diet history, oral pH measurement, posture analysis, triplanar cranial analysis, manual TMJ measurements, circumferential cortical blind spot mapping, manual muscle testing, postural blood pressure response, and modified master two step heart rate response. As a brief side note, several of these assessment procedures including postural blood pressure response, and manual muscle testing to identify neurological derived muscle group weaknesses, are found within the American Medical Associations "Guides to the Evaluation of Permanent Impairment." A book of standards utilized by independent medical evaluators for legal and insurance purposes. Another excellent source of references for these procedures is a paper by (Maffetone).

Manual therapies included neuromuscular re-education, trigger point and fascia therapy, chiropractic manipulative therapy, osteopathic type cranial manipulation, and oral nutritional testing with subsequent supplementation and diet modification. There was no myofascial, or chiropractic, therapy applied directly to the TMJ.

The setting for the study was in a private clinic. An adolescent volunteer who was experiencing post-traumatic TMJ dysfunction and headaches, but had not yet received any manual, or dental, therapy for his condition was the test subject.

Discussion

According to (Kendal & Schwartz, and Kerr) the trigeminal neurology of the TMJ and cranium becomes continuous with the dorsal column neurology of the spinal cord in the brainstem nuclei. Because of this continuous relationship, any changes to the collective proprioceptic neurological balance of either the body, or the TMJ/cranium, will feed back though the central nervous system and affect the other. There is substantial documentation in the scientific literature describing this phenomenon. (Gelb, and Forgione) demonstrated that altering the posture of the mandible to the cranium influences muscle strength, posture, and performance of the human body. (Bran) also associated postural changes in patients experiencing craniofacial pain. In another study, (Esposito & Leisman) correlated TMJ dysfunction with significant loss of neurologic control of mechanical function affecting balance and coordination. (Raudino) associated it with what he describes as a cranial dystonia. (Gregory) associated TMJ dysfunction with the presence of sacroiliac sprain, demonstrating the reverse relationship, with the body mechanics affecting the TMJ. Changing the mechanics of the body, and subsequently influencing TMJ function as a therapy modality has been suggested by (Chinappi and Getzoff), as well as (Kraus), (Curl), and (Walther) in their respective articles and textbooks. Another significant advocate of postural and TMJ co-management has been (Walker) in his Chirodontic seminar series. (Cooper) writes on the significance of computer aided bioelectronic analysis as an integral part in the diagnosis and management of TMJ dysfunction. And finally, applied kinesiology manual muscle testing procedures where associated with central nervous system functioning by (Leisman, Shambaugh & Ferentz). While (Carrick) demonstrated that manual manipulative ther-

apy to the spine can alter central nervous system function. Two other significant papers within the applied kinesiology literature describing central nervous system derived patterns of muscle weakness are by (Allen), and (Peet).

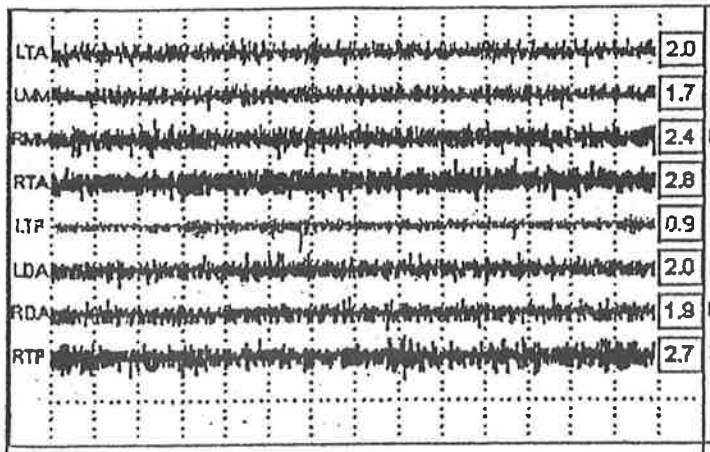
Results

Case study #1: Nathaniel, an eleven-year-old, male, patient had been experiencing symptoms of jaw pain, and headaches, for approximately six weeks following an automobile accident. He was evaluated and treated medically with anti-inflammatory and pain medication, but these provided only limited and temporary relief. Following assessment, he was diagnosed with a whiplash type cervical sprain/strain injury with musculoligamentous irritation of the craniomandibular, and upper cervical joints. Secondary muscle tension headaches with autonomic, and cortical, neurological dysfunction indicating a mild closed head injury was additionally diagnosed. The objective findings supporting the diagnosis are consistent with, deceleration type, mechanical trauma that would be experienced in a motor vehicle accident.

He displayed a pyramidal distribution of weakness of his left extensor musculature, which correlated with enlargement of the left physiological blind spot. This pattern also included the right iliopsoas muscle, which additionally displayed a chronic muscle tear at its femoral insertion. His blood pressure, and heart rate, responses suggested a hypersympathetic autonomic profile in an exhausted phase of compensation. This correlated with weakness of his subscapularis and quadriceps muscles bilaterally, and a sartorius muscle on the right side only. His oral Ph was 6.2, and his dietary habits included excessive high glycemic carbohydrates. There was spinal articular dysfunction present in the right sacroiliac joint, the right lower lumbar region, the right lower cervical region, and a mid thoracic fixation was present, again on the right. He additionally had articular dysfunction within both ankles. His cranium had a torque pattern involving both temporal bones, with the predominant suture restriction in a left internal frontal bone pattern.

Treatment for the above findings included; modifying his diet to include only low glycemic carbohydrate sources (Dr. Phillip Maffetone's two week diet test). Oral supplementation with betaine HCL, and flax seed oil. Walking as a low stress aerobic exercise to be done at least 45 minutes, a minimum of 4 times per week. Daily "Heartmath" relaxation exercises to stimulate parasympathetic neurology. The initial coaching of this technique was done using a heart monitor for biofeedback. Deep tendon cross friction message (origin/insertion technique), and neuromuscular reeducation (strain counter strain), was applied to his right iliopsoas. Chiropractic manipulative therapy to the indicated regions to stimulated proprioception, and restore normal articular biomechanics. And craniosacral manipulation for his left internal frontal fault to further stimulate parasympathetic neurology and proprioception.

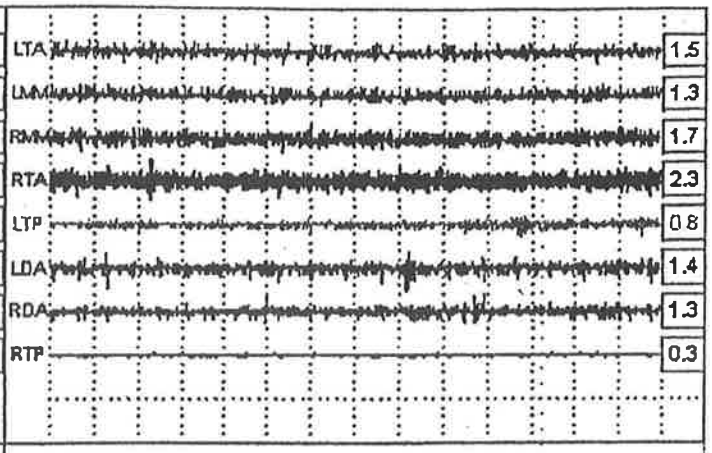
As evident from the graphs, (post manipulation graphs are on the right) the boy responded very well to treatment, demonstrating improvement in virtually every measured variable. The first graph shows the bioelectrical activity within the craniomandibular muscles. Any reading on these graphs above 2.0 is considered to be in a dysfunctional, or pathologic range. The remaining two graphs demonstrate marked improvement in the biomechanical range of motions, and movement patterns of the TMJ. Subsequent office visits revealed both subjective, and objective, improvement in his condition. However, due to difficulties in scheduling, follow up bioelectrical analysis could not be performed.



SAMPLE - 13.90 seconds

LTA	Peak = 11.0,	Average = 2.0 MicV
LMM	Peak = 10.3,	Average = 1.7 MicV
RMM	Peak = 14.5,	Average = 2.4 MicV
RTA	Peak = 15.0,	Average = 2.8 MicV
LTP	Peak = 18.0,	Average = 0.9 MicV
LDA	Peak = 12.8,	Average = 2.0 MicV
RDA	Peak = 13.3,	Average = 1.9 MicV
RTP	Peak = 15.8,	Average = 2.7 MicV

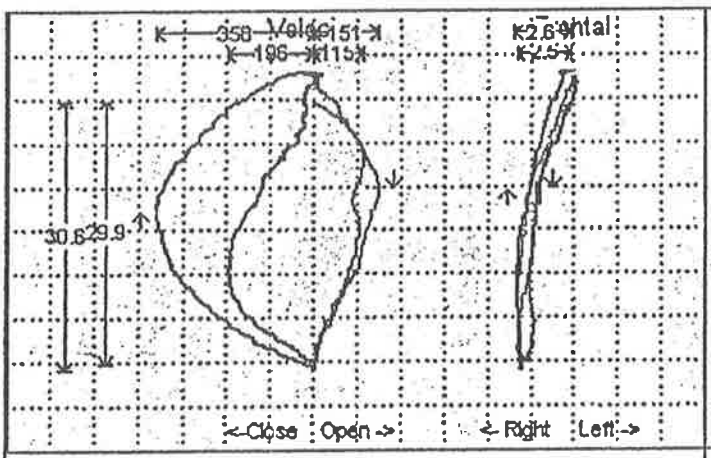
EMG= 30 | Set AB | Spd= 1.0



SAMPLE - 13.91 seconds

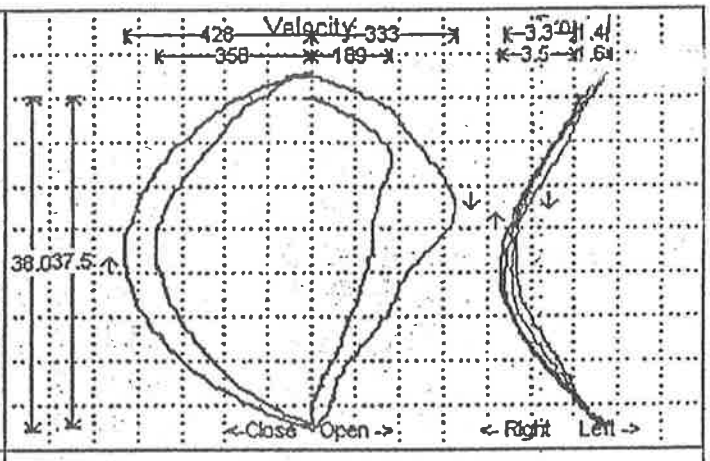
LTA	Peak = 8.5,	Average = 1.5 MicV
LMM	Peak = 7.5,	Average = 1.3 MicV
RMM	Peak = 10.3,	Average = 1.7 MicV
RTA	Peak = 15.0,	Average = 2.3 MicV
LTP	Peak = 9.0,	Average = 0.8 MicV
LDA	Peak = 15.0,	Average = 1.4 MicV
RDA	Peak = 13.0,	Average = 1.3 MicV
RTP	Peak = 2.5,	Average = 0.3 MicV

EMG= 30 | Set AB | Spd= 1.0



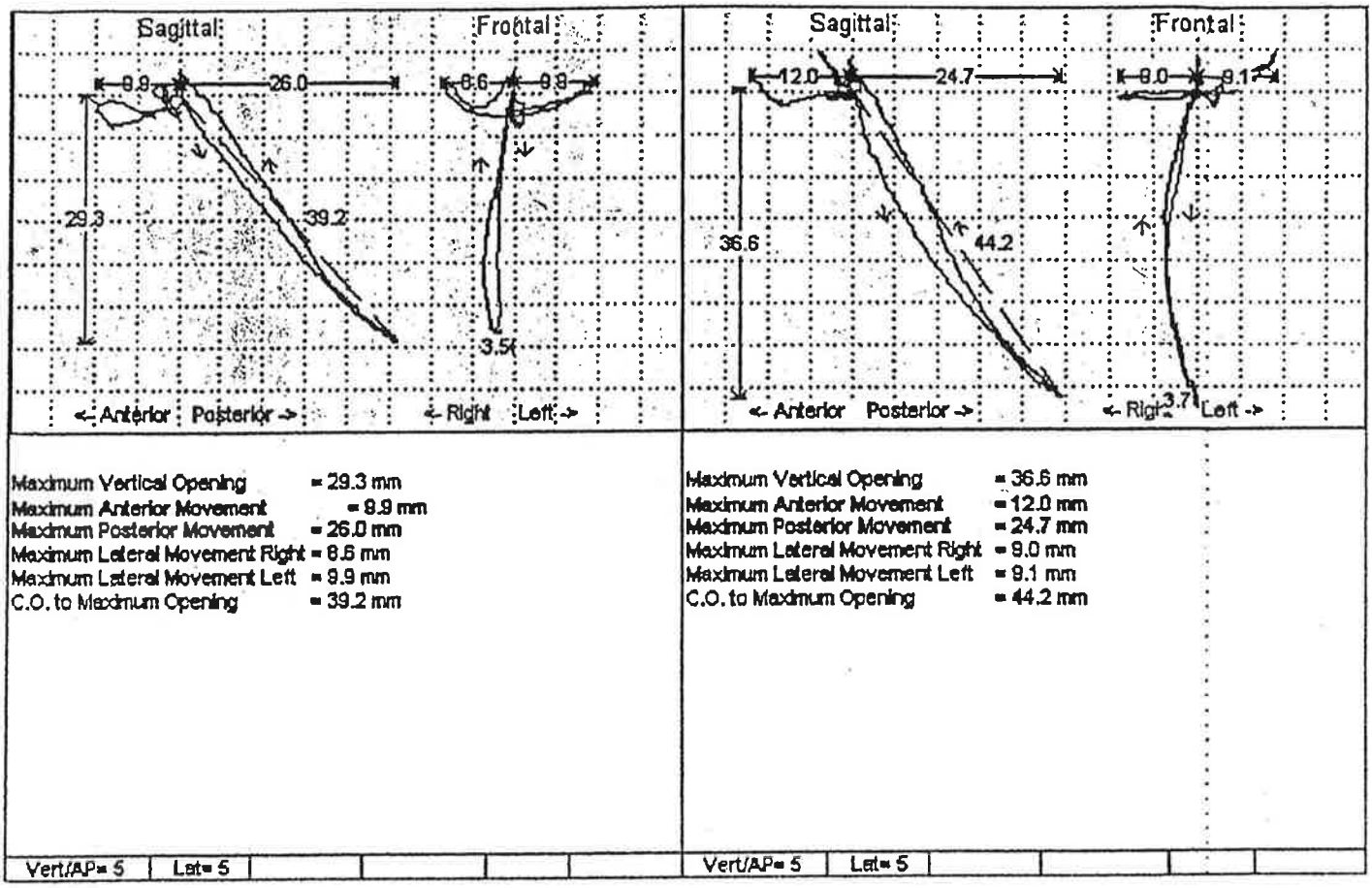
OPENING 1
Maximum Velocity = 115.0 mm/sec
Average Velocity = 75.6 mm/sec
CLOSING
Maximum Velocity = 196.3 mm/sec
Average Velocity = 113.3 mm/sec
Maximum Velocity of Terminal Tooth Contact = 18.6
OPENING 2
Maximum Velocity = 151.3 mm/sec
Average Velocity = 88.7 mm/sec
CLOSING
Maximum Velocity = 357.5 mm/sec
Average Velocity = 252.6 mm/sec
Maximum Velocity of Terminal Tooth Contact = 122.5

Vert/AP= 5 | Lat= 2 | Vel= 100



OPENING 1
Maximum Velocity = 188.9 mm/sec
Average Velocity = 105.4 mm/sec
CLOSING
Maximum Velocity = 357.5 mm/sec
Average Velocity = 248.6 mm/sec
Maximum Velocity of Terminal Tooth Contact = 132.5
OPENING 2
Maximum Velocity = 332.5 mm/sec
Average Velocity = 199.6 mm/sec
CLOSING
Maximum Velocity = 427.5 mm/sec
Average Velocity = 307.3 mm/sec
Maximum Velocity of Terminal Tooth Contact = 86.3

Vert/AP= 5 | Lat= 2 | Vel= 100



Conclusion

This study demonstrates that applied kinesiology diagnostic procedures and subsequent manual therapies can produce remarkable improvement in TMJ/ craniomandibular function. It is hoped that this brief case report will contribute to the scientific evidence for the efficacy of functional dentistry/applied kinesiology co-management, and potentially stimulate further research on this topic.

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A Neurological Basis For the Effects of Cranial Manipulation

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Abstract

A neurological model for the effects of cranial manipulation is presented. The model is based on afferent pathways from dural nociceptors and cranial mechanoreceptors which have their primary synapses in the cervical spinal cord. Secondary effects of cranial afferents impact motor activity to the cervical spine. The effects of cervical afferents on the cerebellum, in turn, alter efferents from the cerebellum which in turn explains all the diverse clinical effects observed by cranial techniques.

Introduction

In a 1991 paper by this author the relationship of dural nociceptors to cranial techniques was discussed.[1] The afferent messages generated by intracranial dural nociceptors travel along the trigeminal nerve, cranial nerve V. However, investigating the known connections of the afferent trigeminal terminations has made no sense in figuring out why so many widely different types of problems respond to cranial techniques.

In 1996 Richard Belli presented an overview of cranial manipulation and proposed a neurological explanation for the effects of cranial manipulation.[2] This paper is complementary to Belli's 1996 paper and offers some additional explanations and observations regarding cranial techniques.

Discussion

Dural innervation arises from nociceptor afferents originating in receptors whose axons travel in the trigeminal nerve (V) or the vagus nerve (X). Some intra-articular mechanoreceptor (MR) afferents will also travel with the trigeminal nerve. These afferent fibers which travel with the trigeminal nerve as well as those which travel with the vagus nerve have their primary synapse in the descending nucleus of V (which is also called the spinal trigeminal nucleus, the descending trigeminal nucleus, and the other names.) This termination of these trigeminal (and vagus) innervated structures stretches from the cervicomedullary junction down to as far as the C-5 spinal cord level in some patients, but certainly down as far as C-2 and C-3 spinal cord levels. The innervation of the dura is primarily, if not totally, nociceptive. Likewise, intracranial vascular structures, if they have any sensory innervation at all, are innervated by nociceptors.

Due to convergence of the cranial sensory receptors (nociceptors, and also MRs) travelling in cranial nerve V (and X) with cervical spine afferents (nociceptors, MRs, et al), the nervous system beyond the primary afferent synapses in the cervical cord cannot tell the difference between sensory activity arising from the areas innervated by cranial nerve V (or X) and the areas innervated by the cervical spinal nerves. Therefore, any abnormal afferentation from the intracranial cavity (such as that arising from dural nociceptors depolarizing from dural tension of cranial faults) will cause disturbances of transmission which will be indistinguishable from disturbances arising from cervical spinal afferent input. This includes flexor reflex

afferent effects at the ventral horn motorneuron in reaction to the nociceptive inputs at this level of the cord.

Picture cervical spinal sensory activity being synthesized in the spinal cord dorsal horn with whatever is happening in the cranial dura. Imagine the cervical spinal cord attempting to synthesize these two variant inputs. The resulting synthesis, including feedback from muscle spindle cell loops which has been altered by the intracranial nociception, can be a variety of aberrant sensory messages, totally inconsistent one from the other, to the CNS. Imagine the confusion that must be created by these two unsynchronized afferent inputs arriving in the upper cervical spine, especially when there is disruption caused by bombardment with intracranial nociceptors due to a cranial fault.

There are a variety of reflex pathways which would alter ventral horn activity (including flexor reflex afferent withdrawal reflexes - the basis for the bilateral neck flexor weakness seen in typical cranial faults.) Some of the primary afferents from the upper cervical area synapse directly into the cerebellar flocculonodular lobe, others enter the cerebellum via spinocerebellar pathways (the cuneocerebellar tract), and others impact the cerebellum via secondary afferents which arise from vestibular nuclei.

The cerebellum efferents modulate activity of vision, autonomic function (e.g., when you get dizzy you also get nauseous via these same pathways), somatic and autonomic motor activity of the reticulospinal tracts, vestibulospinal tracts, and feedback into cortical loops, and so on and so on. Putting this all together, it is not too difficult to see that cranial nociception arising from cranial bone lesions (cranial faults) are fully capable of disrupting cervical spinal MR activity via their flexor reflex afferent activity. The resultant changes in cervical muscle spindle and other MR feedback loops continuing on directly and indirectly to the cerebellum cause a potential of varied and bizarre inputs into these critical areas. These bizarre inputs cause cerebellar outputs which cause structural abnormalities (including off centering of the spine via the central pattern generators of the mesencephalic RF)[3] which might affect anywhere in the body.

The alterations of cervical muscle adaptations to the cranial nociception may cause a variety of local spinal problems, and since proper cervical afferentation activates cerebellar non-volitional activity of spinal intrinsic muscles, any problem in any spinal area may be affected by cranial faults, depending on the summation of other factors present in that individual.

These are far more plausible explanations of the widespread spinal and other structural changes seen with cranial technique than the traditional explanation of changes in spinal dural tension affecting the dural port at some specific segment or segments. (This is not to deny the importance of the spinal dura, because it too has a powerful nociceptive flexor reflex afferent effect. Note the opisthotonos of meningitis. But it is intended to downplay the importance of the purely mechanical effects of the spinal dura as a causative agent of any significant importance in most cases.)

The same aberrant cerebellar firing can and will result in visual changes in extraocular muscles through the medial longitudinal facsiculus to III, IV, and IV, as well as autonomic intraocular muscles through via the Edinger-Westphal nucleus of III. The visual changes following cranial techniques (in most cases) probably have nothing or very little to do with changes in orbit structure as is always suggested, but as has never made sense physiologically.

The visceral alterations which are often reported to improve from cranial technique are likely not due (or at best maybe only rarely due) to changes in cerebrospinal fluid flow, mechanical brain alterations from cranial bone displacement or dural port tension. Rather, autonomic changes can readily be explained from both spinal cord nociceptive activation of the autonomic intermediolateral cell column (IML) combined with abnormal cerebellar efferent activity bombarding the autonomic centers of the brainstem.

(Nociception from anywhere in the body, if it is not blocked prior to reaching the hypothalamus, will also create autonomic and endocrine effects via the hypothalamus and all of its connections.) To understand this better, take the nausea and vomiting of severe dizziness, dampen it a bit, and allow it to affect any visceral organ depending on other susceptibilities (prior central integrated states of the neurons), and the introduction of a cranial fault as easily explains autonomic symptoms anywhere in the body, as does its correction explain their eradication.

The activation of the cerebral cortex is dependent on afferentation from the entire body including cerebellothalamocortical pathways. Altered cervical muscle activity from the consequences of cranial fault-induced nociception results in altered cervical MR activity which may result in a variety of sources of functional deafferentation to the cortex, both directly through the thalamic relays and via the cerebellum as mentioned. The lack of afferent stimulation, or potentially worse, asymmetrical afferentation to the cerebral cortex may result in asymmetries of hemispheric functions with attendant alterations in emotion (right brain - left brain imbalances), consciousness, cognition, and reasoning (need for greater activation of cortical neurons to be able to reach threshold.)

Conclusions

This is the proposed neurological explanation for the far-reaching clinical effects of cranial technique. It is merely an anatomical coincidence (although there may be a number of plausible and intriguing energy medicine explanations for it) that cranial technique, directed to the cranial areas, has so many effects in the central nervous system which it overlies and structurally protects. The traditional explanation of cranial techniques and their effects was proposed originally by Sutherland, Dejarnette, and numerous other clinical pioneers. But their explanations are at least a half century old, based on outdated or never proven physiology, and are at best educated guesses. The modern neurological description herein is plausible, comprehensive, and based on principles of physiology which are accepted and can be found in the textbooks of our day. It is also an educated guess until it can be scientifically investigated, but there is a great deal of science upon which it is based.

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A Neurological Rationale For Injury Recall Technique

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Abstract

It is well known that muscle spindle mediated stretch reflexes are commonly modified by descending suprasegmental pathways arising from cerebellar and cerebral adaptations. These concepts are reviewed and a model is presented for understanding the effects of injury recall technique (IRT) in light of suprasegmental, primarily cerebellar, adaptation.

Introduction

The three major sensory inputs for postural control are the eyes, the inner ears (the vestibular mechanism), and mechanoreceptors from the ankle joint. Postural adaptation depends on cerebellar integration of all three areas and its efferent supply to brainstem centers for descending pathways to motor neuron pools. This paper proposes that the clinical findings of injury recall technique (IRT)[1, 2] are associated with cerebellar adaptation and habituation to areas of injury and trauma.

Discussion

The cerebellum receives primary inputs from the cerebral cortex (through the corticopontocerebellar tracts via the inferior olive,) the inner ear (both directly from the vestibular nerve and indirectly via vestibular nuclei,) and ipsilateral mechanoreceptors (MR) (via the dorsal and ventral spinocerebellar tracts and the cuneocerebellar tracts.) The most important MR inputs to the cerebellum arise from muscle spindle afferents, from both primary and secondary muscle spindle cell receptors.

The stretch reflex has local spinal cord effects to increase muscle tone proportionally to the amount of stretch. The stretch reflex afferent information is also carried to the cerebrum via the thalamus and to the cerebellum as previously mentioned. These higher levels allow for adaptive changes (plasticity) in the stretch reflex modulated by descending pathways.

These adaptive changes are most often explained in neurology texts by the example of stretch reflexes associated with dorsiflexion of the foot.[3, 4] When the gastrocnemius and soleus are stretched, this causes a reflexive contraction of both muscles to bring the foot back to its original position. If a standing person leans or sways forward, these muscles reflexively contract to bring the person back to original upright position. However, if the floor under the toes tilts upward causing the same amount of stretch to the gastrocnemius and soleus, the reflex contraction of these muscles causes the person to fall backward. If the floor is repeatedly tilted in this fashion causing dorsiflexion of the feet, there is an adaptive response where the stretch reflex response becomes decreased, that is, altered to meet the new environmental input.

A good example of this phenomenon is how a person's stretch reflexes adapt when on a rolling boat. After there is a new, habituated decrease in the dorsiflexion stretch reflexes, the person better tolerates the boat's

movement. However, when the person returns to dry land, there is a period of instability until the stretch reflexes adapt back to their original environment. As a result of this changing in adaptation, most people report that they feel like they are still on the boat for a period of time after they are on land.

Such adaptations depend on projections from cerebral cortex to the cerebellum. The cerebral cortex itself is activated in this regard by afferents arising from muscle spindle cells. People with cerebellar disorders do not have the ability to adapt to the rolling boat. In other words, there is no plasticity of the stretch reflex due to the cerebellum's inability to adapt.[3]

Following injury, there are various and numerous alterations in stretch reflexes as they adapt to flexor reflex afferent pathways arising from nociceptors. The secondary effects of nociception become the new normal as the person adapts to the trauma, just as the person adapts to the rolling boat. It is proposed that, if the trauma is significant enough, an adaptation of cerebellar modulation of stretch reflexes, which must take place, becomes the new norm as nociceptors continue to fire and change stretch reflex responses while the injury heals.

If the injury is one which will require IRT, the synthesis of local, cerebellar, and corticocerebellar adaptations creates an alteration in posture, part of which is reflected in ankle proprioceptive adaptations to the injury. It is interesting that most texts explain stretch reflex adaptation using the example of ankle dorsiflexion, and this is the same pattern we see associated with injury and IRT. (Note how a supine person's feet dorsiflex when they are exposed to nociceptive input such as a hard or painful manipulation.)

One can see that IRT is associated at least in part with cerebellar adaptation by applying functional neurological assessment of cerebellar function in parallel with IRT.

There are always inputs to the cerebellum from the vestibular complex in the inner ear. The three bilateral semicircular canals are arranged such that any head movement will be associated with activation of at least one, and usually two or three of them on each side. The semicircular canals cause reflex changes in postural muscle activity. Putting the head in a distinct position to activate primarily one of the canals will elicit reflex responses which are characteristic of that canal.

The six head positions which will activate specific semicircular canal activity are:

1) rotating the head to the right (right lateral canal)
2) rotating the head to the left (left lateral canal)
3) tilting the head anterior and left (left anterior canal)
4) tilting the head posterior and right (right posterior canal)
5) tilting the head anterior and right (right anterior canal)
6) tilting the head posterior and left (left posterior canal)

The positive challenge for IRT is when a conditionally facilitated (strong) muscle becomes conditionally inhibited (weak) when the area of previous injury is activated by patient touch or doctor stimulus (usually pinching) and the talus is challenged in a cephalward direction. To demonstrate the cerebellar relationship to a positive IRT pattern, simply activate the area of injury by patient touch or doctor pinching, but rather than add the talus challenge, place the head in one of the six positions to activate one of the semicircular

canals as mentioned above. This will result in a strong muscle weakening just the same as the cephalward talus challenge does.

Correction of IRT is by micro-manipulation of the talus in a distal direction while the area of injury is activated. Following this correction activation of the injury with the positive semicircular canal related head position is now negative. This suggests that the IRT problem was associated with plastic adaptation in the cerebellum which is no longer present following IRT correction.

Conclusions

A normalization of all of the accompanying concomitants to plasticity-altered (adapted) cerebellar and cerebral functions explains the far-reaching and dramatic effects often achieved by IRT. This includes changes in sensory and motor functions, autonomic concomitants, and improved cognitive function. The cerebellar adaptation theory of IRT also opens the door to understanding how to integrate IRT with other procedures; specifically why this author recommends that, when indicated, IRT be performed prior to any other therapies which may affect cerebellar functions. This includes, of course, all other manipulations.

The cerebellum is the integrator of all motor functions. If there is plasticity in the cerebellum causing adaptation away from optimal function, such as an adaptation to a major trauma, this must become the applied kinesiologist's first treatment step in the overall goal of muscle balancing and AK procedures.

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The Somatic Window on Neurological Function

Part 1 - Muscle Spindle Activation and Slow Stretch of Muscles

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Abstract

Muscle testing is a part of the neurological examination. When a weak muscle strengthens following a sensory receptor challenge, it suggests that a pathway between the sensory receptor and the muscle is inhibited somewhere along its course, and that the challenge creates enough activity in the pathway to temporarily overcome that inhibition. Slow stretch of a muscle activates its muscle spindle cells (MSCs). All MSC activity impacts several pathways including the ipsilateral cerebellum. A change in a muscle's strength following slow stretch of other distant muscles suggests involvement of a pathway related at least in part to cerebellar function. Various patterns of muscle testing responses to slow stretch of muscles are discussed including centering the spine patterns, tonic labyrinthine patterns, and others. A relationship has also been identified between the activity of meridian tonification points and responses to slow stretch of distal flexors and distal extensors.

Introduction

In the conscious, responsive patient the neurological examination may be greatly enhanced by observing for changes in manual muscle testing outcomes to various sensory receptor based diagnostic challenges. This form of clinical investigation is functional neurological assessment (FNA). FNA may be further expanded to include chemical challenges to gustatory and olfactory receptors which are familiar to AK doctors. This expanded process is called Neuro Metabolic Assessment (NMA) and plays an integral role in the overall neurological exam.

To perform FNA and NMA, the clinician must identify both a weak (conditionally inhibited) muscle and a strong (conditionally facilitated) muscle by manual muscle testing. Some muscles may be conditionally over facilitated as described below and should not be used to evaluate functions other than their own status.

The neurological exam compares responses to challenges of numerous pathways to each other and to normal expectations. A tenet of NMA is that if a weak muscle is strengthened by evoking sensory receptor activity, then there is a decrease in function (net source of inhibition) somewhere along the pathway that was evoked. The sensory receptor activation is adequate to temporarily overcome the inhibitory influence, and the weak muscle response changes to strength temporarily until the challenge wears off. It is the clinician's responsibility to identify the primary origin of that inhibition and to remove it by the most conservative therapy possible. This therapy may be structural, chemical, or mental, or combinations of these three approaches.

The procedures of NMA involve clearing of certain functional lesions which may represent adaptations and compensations. This is important as a stepping stone to make other functional and pathological lesions clearly and easily identifiable. But the clinician must be careful to be complete in exam and treatment. Optimal treatment depends on identifying the primary level of dysfunction since the body will have many adaptations and compensations to an original problem which may confuse the clinician. Treating an adaptation or a compensation may lead to short term improvement in the patient, but the long term consequences of treating an adaptation include recurrence, or at worst, an aggravation of the symptoms due to removing a compensation and leaving the underlying problem uncompensated for.

It is also important in this regard to consider the possibility of transneural degeneration of one or more neuron pools as a source of the patient's problems. Concepts of transneural degeneration are discussed in another paper in this Proceedings[1] (and in previous articles in the ICAK-USA News Update by this author and Dr. Kathy Power.[2] In this paper, however, we will only consider concepts dealing with conditional facilitation and conditional inhibition.

Discussion

Muscle spindle cell (MSC) activity bombards the central nervous system and the cerebellum in particular 24 hours a day. Cerebellar outputs affect various suprasegmental areas which project to motor neuron pools and affect muscular activity. When the cerebellum is involved in a functional neurological process, FNA using MSC activity is a tremendous guide to identifying the source(s) of the interference with normal muscular function.

There are several sources of neurological "noise" which interfere with MSC controls and must be cleared prior to the examination of motor pathways. These include patterns which affect autogenic facilitation and autogenic inhibition.

A weak muscle should strengthen when the MSCs in the belly are manually stretched apart from each other. This type of strengthening response is called autogenic facilitation by Richard Belli.[3] When autogenic facilitation does not strengthen a muscle, it is due to the body being in a response mode to an injury or trauma, either recent or ancient. In this case rubbing over the area of injury will cause a strengthening response. Correction of the problem is by injury recall technique (IRT) which is discussed in other papers by this author.[4, 5]

Richard Belli also describes autogenic inhibition as the normal weakening response which should take place when Golgi tendon organs (GTOs) are activated.[6] The same type of temporary weakening response is expected when MSCs are pushed together in the belly of a muscle. When autogenic inhibition does not weaken a strong muscle, this is an over facilitated muscle response. It may be due to the response to injury as part of a withdrawal reflex and is usually coupled with a problem of autogenic facilitation in antagonist muscles.

In a generalized over-facilitated muscular pattern throughout the body, there is a lack of response to autogenic inhibition. This is usually due to a systemic sympathetic "fight or flee" pattern. This systemic pattern must be corrected as soon as it is identified in order for NMA muscle testing procedures to be properly applied. Correction of this problem is related to centering the spine concepts[7] which are not be discussed in this paper.

Normal function of autogenic facilitation and autogenic inhibition must be present in order for the following clinical information to be applied. If muscles do not respond properly to autogenic facilitation and

autogenic inhibition, the causes of this improper response must be treated and eliminated as discussed a previous paper[8] prior to continuing the exam. The presence of abnormal MSC feedback due to either of these two problems represents a smokescreen which will prevent the clinician from applying the procedures in this paper, and which will alter other neurological findings, potentially misguiding the clinician.

Slow stretch of muscles can be used to evaluate MSC afferentation to the CNS and the many pathways that Ia and II afferents affect. Fast stretch activity can also be used. Fast stretch activates primarily GTO activity which tends to override the accompanying MSC activity. Slow and fast stretch modalities will certainly affect antagonist activity, activity of reciprocal muscles in all other limbs, IML activity, and cerebellar activity. It is the integration which takes place in the cerebellum, however, which appears to be the most important component being evaluated with muscle testing following MSC (and/or GTO) activation, especially when the muscles being stretched are distant to the muscle being tested.

Slow stretch and fast stretch have opposite reflex activities. Findings following slow stretch activity parallel manual stretching MSCs in the belly of the muscle as described in autogenic facilitation above. Slow stretch and MSC activity to strengthen may be used interchangeably.

Fast stretch activity is paralleled by MSC to weaken activity or GTO to weaken activity. MSC to weaken, GTO to weaken, and fast stretch modalities may all be used interchangeably. As mentioned above, it is clear that autogenic facilitation and autogenic inhibition must be normalized prior to investigation with the modalities of slow stretch or fast stretch.

Slow stretch (or autogenic facilitation procedure) or fast stretch (or autogenic inhibition procedure) may be used to evaluate centering the spine patterns including tonic labyrinthine reflex (TLR) patterns. We will focus on slow stretch patterns, but anything said of slow stretch should be applicable for fast stretch to the antagonist muscle.

Centering the Spine Patterns and Slow Stretch Challenging

It is hypothesized that centering the spine (CTS) patterns 67 are generated in the mesencephalic reticular formation (RF) by central pattern generator (CPG) neuron pools which reside there.[9] These areas such as the interstitial nucleus of Cajal, the prestitial nucleus, and others, are in the area of the mesencephalic RF which is impacted by cerebellar activity (which itself is impacted by MSC and GTO receptor afferents), hypothalamic activity, and basal ganglia (paleocortex) activity.

The effects of slow stretch (and fast stretch) of flexors and extensors may be evaluated by monitoring manual muscle testing outcomes immediately following application of these modalities. (In the unswitched patient, the effects of MSC slow stretch and GTO fast stretch activity around a given joint are always the opposite of each other.) In the unswitched patient slow stretch of distal extremities may follow several patterns which correlate with CTS patterns.

In the seven patterns listed below, strengthening responses of weak muscles (anywhere in the body) which result from slow stretch of the listed muscles are associated with six CTS patterns or a faulty TLR pattern which are listed in CAPITAL letters.

1. right flexors in both right limbs and left extensors in both left limbs are associated with SPINAL LATERAL FLEXION PATTERNS, CONVEX TO THE LEFT;
2. left flexors in both left limbs and right extensors in both right limbs are associated with SPINAL LATERAL FLEXION PATTERNS, CONVEX TO THE RIGHT;

3. right upper limb flexors, right lower limb extensors, left upper limb extensors, and left lower limb flexors are associated WITH THE SPINAL TORQUE OF A RIGHT FOOT FORWARD GAIT PATTERN;
4. right lower limb flexors, right upper limb extensors, left lower limb extensors, and left upper limb flexors are associated WITH THE SPINAL TORQUE OF A LEFT FOOT FORWARD GAIT PATTERN;
5. all upper limb and lower limb flexors are associated WITH SPINAL FLEXION-EXTENSION PATTERNS;
6. all upper limb extensors and all lower limb extensors are associated with SPINAL EXTENSION-FLEXION PATTERNS;
7. upper and lower flexors on one side only OR upper and lower extensors on one side only are associated with TONIC LABYRINTHINE REFLEX (TLR) PROBLEMS.

Any variation from these patterns suggests interference with MSC control systems from IRT patterns as mentioned above or switching patterns including small intestine dysfunction as will be described below.

Clearing of CTS problems (via procedures in a previous paper[8]) will negate the strengthening effects of slow stretch and fast stretch on weak muscles. The theory behind this is that when one of the seven slow stretch patterns temporarily strengthens a weak muscle, the strengthening response is due to the facilitation of a CTS (or TLR) pathway which is inhibited due to the CTS problem. The CTS problem may arise in the cerebellum or in any other area which projects to the CPG cells in the mesencephalic RF. Since the cerebellum connects to the CPG cells in the mesencephalic RF, the "jump start" achieved by the patterned slow stretch procedures through the cerebellum to the CPG cells causes a temporary facilitation of these cells, and a temporary strengthening of the CTS associated weak muscles. When the origin of the CTS problem is identified and corrected, slow stretch of muscles has no appreciable effect on weak muscles since the source of the weakness is not impacted by the pathways stimulated by slow stretch.

This suggests that muscle weaknesses which do not respond to stretch modalities are arising from sources other than cerebellar feedback loops. Or said in another way, when stretch modalities have no effect on weak muscles, cerebellar activity (including TLR activity) can receive less consideration as the source of the inhibition which is creating the problem. Recall that when activating a pathway results in a strengthening response of a muscle, it suggests that there is a problem along that pathway. When no change is seen in a weak muscle upon a sensory challenge, it suggests the source of weakness is not in that pathway. This is not to suggest that there is no cerebellar activation by the procedures being used, but only that there are no functional faults related to cerebellar activity under these circumstances.

Distal, Middle, and Proximal Muscle Stretches

The cerebellum has four major deep nuclei to which the cerebellar cortex projects, and which are the output side of the cerebellum: the fastigial nucleus, the globose nucleus, the emboliform nucleus, and the dentate nucleus. The MR inputs affecting the fastigial nucleus arise primarily from proximal midline structures, especially the spine. The dentate nucleus is associated with MR inputs from distal limbs, i.e., the wrists and hands and the ankles and feet. The globose and emboliform nuclei are often combined and referred to as the interpositus nucleus. The interpositus nucleus is associated with MR inputs from the shoulders and elbows and the hips and knees, in other words, the middle joints and muscles.

If the cerebellum is unfolded, it could be compared to a football field. The areas at midfield between the 40 yard lines are primarily concerned with proximal, midline inputs and output to the fastigial nucleus. The areas between the 20 yard lines and the 40 yard lines are primarily concerned with inputs from the middle extremities' joints and muscles (shoulders, hips, elbows, knees) and output to the interpositus nuclei. The areas from the 20 yard lines to the end of the end zones are associated with inputs from the distal extremities' joints and muscles (wrists, ankles, hands, feet) and output to the dentate nuclei.

Of course all parts of the cerebellum connect to all other parts of the cerebellum so that movements will be coordinated all over the body. Therefore, one would expect that if a flexor muscle, say a pectoralis major, sternal division (PMS) was weak, and the cerebellum was coordinating properly, that activating MSC activity in any flexor of that upper limb should spill over and affect the strength of the PMS. This is often the case, but it is also often not the case. Observation of these responses leads us to some very interesting and very useful clinical applications. Observations of proximal, middle, and distal inputs from flexors and extensors of all four limbs allow us to make important conclusions regarding the integration in the cerebellum.

The cerebellum must depend on its afferent inputs for its outputs. If it receives confusing or conflicting inputs, this will be reflected in the outputs and in muscle testing responses to MSC activities. There are several patterns which must be observed which are listed below in three general categories.

1. If MSC activation from all four limbs distal areas fits no common pattern, there is an injury response somewhere. A) If autogenic facilitation does not strengthen the muscle, then this is an indication of IRT as already discussed. B) If autogenic facilitation strengthens the muscle, but the pattern of MSC stretch in all four limbs fits no common CTS or TLR pattern (that is, the seven listed above), then this may also represents a response to injury. However, another technique must be employed, either nociceptor-stimulation blocking (NSB) technique or set point (SP) technique. Which technique must be used can be ascertained by rubbing (SP) and pinching (NSB) over the area of previous injury as described in a previous paper by this author.[10]

2. If there is no history of injury, and the MSC activation of all four limbs distal muscles fits none of the common patterns, this represents a common switching problem. Both K-27 points will therapy localize (TL) in one of the following ways: A) TL with the right hand to the right K-27 and the left hand to the left K-27; B) TL with the right hand to the left K-27 and the left hand to the right K-27 (crossed K-27 TL); or C) TL with the dorsal aspect of the right hand to the left K-27 and dorsal aspect of the left hand to the right K-27 (dorsal crossed K-27 TL.)

Correction of these different types of switching involves correction of cranial faults and tooth problems which create interference with normal cervical spinal cord reflex activities due to their trigeminal sensory barrage converging on cervical afferent pathways. Identification and correction of these switching problems may be done in a number of ways. However, we have learned that the most efficient way to correct and maintain correction of K-27 related switching is to identify and correct cranial and tooth problems. The specifics of this approach are outside the scope of this paper.

3. If there is a difference between distal flexor and extensor MSC response and middle flexor and extensor MSC response in the same limb, then the body is experiencing severe neurological disorganization or switching. An example of this is if the right PMS is strengthened by the following: autogenic facilitation, stretching the PMS, stretching the right wrist and finger flexors; but the PMS is also strengthened by stretching the right elbow extensors and NOT stretching the right elbow flexors. This means that the right upper limb is out of coordination with itself. If there are not already symptoms in this limb, this pattern leaves it in jeopardy of injury from even slight trauma.

This is the pattern we find to correlate with the brilliant work of Dr. Roger Callahan and his original observations of psychological reversal.[11] Dr. Callahan found that small intestine meridian involvement resulted in the psychological reversal pattern. We have learned that psychological reversal is a form of switching and is accompanied by a physiological reversal. The manifestations of this physiological reversal are seen as described above, when MSC responses are out of synch from muscles in one part of a limb to the other.

Correction of small intestine problems by visceral challenge technique (VCT)[12] will bring MSC challenges within the same limb back into expected, coordinated flexor or extensor pattern. VCT is indicated when an organ's Chapman's reflex (neurolymphatic reflex) TLs only in combination with another challenge such as an oral challenge. For example, in VCT of the small intestine, the Chapman's reflex for the quadriceps will not TL in the clear, but will only TL with simultaneous oral stimulation by an offender substance (e.g., an allergen, a bad fat, or fungal antigen.) Correction of VCT is by using IRT to the ipsilateral talus while the patient maintains simultaneous TL of the Chapman's reflex and oral activation of the substance.

Coordinated cerebellar output is dependent on cerebellar inputs. MSC challenging can help identify if the cerebellum is receiving simple or complicated inputs. When a weak muscle is present, stretching of the distal limb muscles of all four limbs is a huge aid in guiding the clinician to the next appropriate therapy. If one of the typical seven patterns described is present, then centering the spine techniques (or TLR techniques) are indicated. If an atypical pattern is present, the approach should first be as described above in the three categories of atypical patterns. The use of MSC challenges will be a guide until either no weak muscles can be found or the weak muscles which are present show both normal autogenic facilitation and no response to any MSC activity.

At this point, other sensory receptor challenges may be employed to evaluate for conditional facilitation and inhibition in other parts of the nervous system beyond the cerebellum. Muscle testing responses to MSC challenges can be correlated with other neurological challenges to identify the role that the cerebellum plays in more complicated CNS interactions. For example, activating emotional centers of the brain (via emotional recall technique or other challenges such as sniffing an odor in the right nostril which activates the right amygdalar - hippocampal emotional area) often results in weakness. MSC activity can be used to determine the extent of cerebellar involvement in such a problem. This type of FNA procedure is almost like determining which is more important, the chicken or the egg, The association of similar sensory challenges to brainstem and cortical areas with muscle testing responses will be the topic of a future paper, The Somatic Window on Neurological Function, Part 2.

Acupuncture Point Relationships

Slow stretch and fast stretch of distal joints are also correlated with acupuncture meridian tonification point activity. The tonification points are all located at or distal from the elbows and knees. Most are on the wrist and hand or the ankle and foot.

All Yang meridian tonification points on the limb of flexor slow stretch strengthening will yield strengthening responses on TL or tapping. Similarly, all Yin tonification points will yield positive strengthening responses when TLed or tapped on the limb on which slow stretch of extensors causes a strengthening response. The opposite effects are seen in limbs associated with fast stretch strengthening.

It is interesting to note that all the tonification points are located below the elbows and below the knees, in the same areas of the distal muscles being stretched. Just like the effects of slow and fast stretch disappearing following CTS correction, these generalized effects of tonification points will not be seen after CTS is cleared. (Sedation points have not yet been investigated.)

One can observe similar correlations when performing tonification point pain control activity. This three step pain control technique uses pulse point TL, alarm point TL, and finally tonification point to identify a point to tap for pain relief. When a tonification point is identified in this manner, it will TL. The positive TL will be negated by a slow stretch of the distal flexors or distal extensors on the same limb. Tapping the point will eventually negate its TL and the TL of its alarm point. However, if further tapping is required, the TL will recur on slow stretching in the opposite direction of that which previously negated the TL.

Note that we recommend using maximum pain relief as the clinical indicator of how long to tap an acupuncture point rather than stretching of the distal muscles of the limb just described.

Conclusions

When slow stretching of muscles causes a strengthening response to a weak muscle other than itself, the pathways associated with MSC afferent activity should be investigated as the source of the weak muscle. These can include a number of pathways and neuron pools, but cerebellar integration and cerebellar output patterns are most commonly involved, either directly or indirectly, as the source of inhibition creating the weak muscle. Common patterns can be identified by slow stretching distal flexors and distal extensors in all four limbs including centering the spine and tonic labyrinthine reflex patterns. Atypical patterns are suggestive of switching and small intestine problems, and should be corrected prior to other corrections. Otherwise other manipulative inputs will bombard a cerebellum which is already in adaptation to other inputs and hence must REACT to the afferent inputs (with further adaptation) rather than RESPOND to the afferent inputs with restorative activity.

FNA and NMA using muscle testing combined with specific sensory receptor challenges is an elegant form of examination. A knowledge of neurological relationships and pathways combined with observations of muscle testing outcomes guides the clinician to the most appropriate, efficient treatment of the patient.

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Transneural Degeneration and Links Between the Nervous System and the Body Chemistry

Walter H. Schmitt, Jr., D.C., DIBAK, D.A.B.C.N.

Abstract

Transneural degeneration (TND) is a common finding which has been clinically investigated by medical and chiropractic neurologists but so far overlooked by applied kinesiology (AK) doctors. This paper reviews some of the principles of TND and discusses some biochemical and nutritional factors which should be addressed in the TND patient, but which have, heretofore, not been discussed in the context of TND. These include such factors as macronutrients, micronutrients, antioxidants, essential fatty acid (EFA) metabolism, allergies, heavy metal toxicity, citric acid cycle metabolism.

Introduction

Transneural degeneration (TND) is the process of neuronal degeneration which ultimately can lead to neuron cell death. It is a process of Alzheimer's Disease, Parkinson's Disease, and other neurodegenerative disease processes, named and unnamed. It is a process associated with stroke, head trauma, and other neural injury. It is a process which is present in many of our patients, especially those with difficult or recurrent problems. Interestingly, the same process of TND is also an important part of the developing nervous system.

To fully understand the nervous system we are dealing with in AK, we must include an understanding of TND. Some of the basic concepts of TND were discussed in a recent ICAK-USA News Update article by this author. Ongoing investigations in our office are making some progress at correlating AK findings with neurology findings of TND. This paper explores the nutritional and chemical imbalances which can contribute to TND.

Discussion

Neurons are dependent on stimulation of their membrane receptors for their normal function and to maintain their health. The stimulation may be either excitatory or inhibitory just so long as the receptors are activated. Following activation, membrane receptors cause changes inside the cell by one of the two following mechanisms.[1] Ion channels can be opened or closed which changes the polarization of the cell membrane (closer to or farther away from its depolarization threshold) and alters intracellular free calcium which plays an important role in the functions of the cell.[2] Enzymes in the cell membrane are activated which affect second messenger systems (cAMP, cGMP, phosphatidylinositols, protein kinases, etc.) which in turn affect various intracellular chemical activities. See figures 1a and 1b[WHS1].

One of the important consequences of both types of membrane receptor stimulation is the impact via second messengers on genetic transcription. Second messengers turn on the process of our DNA being transcribed for the synthesis of intracellular proteins. These genetically directed protein molecules are essential for the life of the cell including such functions as growth and repair of the cell structural components, production of the enzymes which do the work of the cell, and, because intracellular proteins are anionic molecules, the maintenance of intracellular polarity and osmolarity.

Therefore, one could conclude that the normal function of a neuron, in fact its ability to exist, is dependent on second messenger activity which is dependent on membrane receptor stimulation which is primarily dependent on stimulation from neurotransmitters released by presynaptic nerves. When there is a deafferentation to a neuron or group of neurons (which is often due to lack of normal mechanoreceptor activity due to subluxations, muscle imbalances, immobility following injury, etc.) the normal function and health of the deafferentated neurons will be compromised.

TND occurs when a neuron's receptors are under stimulated which allows for decreased second messenger activity, hence decreased stimulation to the cell's nuclear DNA for genetic transcription (cellular immediate early gene response as well as delayed responses to the genetic machinery), hence decreased protein synthesis resulting in fewer intracellular anionic proteins (as well as alteration of other intracellular physiologies discussed above) hence lowered membrane potential bringing the cell closer to threshold. As the polarization of the membrane becomes more positive and closer to its threshold, the cell can be easily over stimulated (should there be a barrage of receptor activation) resulting in a rapid depletion of substrates, both DNA directed enzymes and nutrients.

When a neuron becomes depleted of enzyme and nutrient substrates, it has decreased ability to perform its normal functions including producing energy. Because these neurons are also closer to depolarization threshold, they will fire with less receptor activation, and sometimes spontaneously. The increase in frequency of firing in light of decreased receptor activation leads to a further depletion of substrates due to a decrease in the amount of genetic transcription accompanying the firing. The neuron becomes fatigued. In fact, the neuron may fire in spurts, even spontaneously, followed by periods of fatigue and inability to fire.

Since the under stimulated neuron already has a decreased level of metabolism and decreased enzyme activity due to decreased synthesis, (and probably increased membrane receptors from up regulation due to decreased stimulation, unless genetic transcription of receptor proteins has been ceased), this puts the cell in danger of metabolic overload and death by apoptosis (programmed cell death.) If these cells result in a muscle weakness pattern somewhere down the neural pathways, and we treat the patient to stimulate these pathways, we may be misled to thinking that we have achieved a desirable outcome. But the problem will recur and we will continue to treat the patient stimulating the same TND pathway with the possibility to achieving a short term gain for a long term loss. Our powerful AK therapies have the potential to worsen TND if it is present, which is of course very dangerous. I have observed this take place in my own practice.

In clinical practice, however, it becomes apparent that not all TND cells are highly fatigable. In fact, there is quite a range of possibilities. The one common feature of TND cell firing is that, due to the membrane polarization being closer to threshold for depolarization, it is easier to depolarize and fire the TND cell than the same cell in a more normal state. However, for a number of possible reasons (not to be discussed here) some TND cells may be extremely resistant to fatigue and therefore fire almost continuously. This creates a conundrum which is often at the core of the difficult and recurrent patient.

When looking at AK muscle testing responses in light of TND, a neuromuscular pathway which is facilitated will respond with strength to testing regardless of whether the pathways are facilitated from a presynaptic TND pathway or from a normally firing pathway. Presynaptic release of excitatory neurotransmit-

ters from normal neurons or from TND neurons results in the same postsynaptic facilitation, hence the same muscle strength response. The possibility of standard AK procedures identifying and correcting a TND pathway which is at the heart of a patient's problem is highly unlikely. We have never addressed this issue in AK until recently.

To assess the possibility of AK muscle testing responses being attributable to a TND neuron pool somewhere, other factors must be evaluated. These include autonomic evaluation of various neuronal pools (pupillary reflexes, heart rate, blood pressure, palate elevation, etc.,) repetitive muscle testing, response to oxygen, and other observations and challenges.[1] We are presently investigating the connections between AK testing and neurology findings of TND and these findings will be reported when they become conclusive.

What is clear at this time, however, is that restoration of neuron health will not occur without restoration of membrane receptor activation which is largely dependent on restoration of normal afferentation, including normal mechanoreceptor activity, throughout the body. This restoration is the main thrust of chiropractic therapies. However, neural afferentation is only one, albeit of critical importance, manner of stimulating the receptors in cell membranes.

Membrane receptors in nervous tissue (as well as other tissues throughout the body) are also activated by hormones and other neuromodulators including peptides. See figure 2[WHS2]. The presence of these other receptor activating substances can be associated with neural inputs, but they are also under other controls which operate regardless of neural stimulation. The balance of this paper concerns these other factors which are best assessed by AK doctors using a combination of laboratory and AK diagnostic skills.

Macronutrients

The macronutrient quality of the diet plays a major role in circulating levels of a number of hormones which have major roles in membrane receptor activation. For example, insulin is released following carbohydrate (CHO) ingestion. Cortisol (glucocorticoid) levels are increased or decreased depending on the levels of circulating glucose. Cortisol levels are also increased in emotional stress from either environmental or internal (corticocortical) sources. Although these are neurologically mediated, affecting the patient's outcome may depend on addressing these matters by either chiropractic treatment or other therapy. In the exhaustion stage of the general adaptation syndrome (GAS) of adrenal stress as described by Selye, lowered levels of cortisol will be available for the usual level of receptor activation. The decreased receptor activation would likely be aggravated after prolonged elevated cortisol levels in the resistance stage of the GAS which would likely have resulted in cortisol receptor down regulation throughout the body.

In other words, chronic stress leads first to increased cortisol levels, then decreased cortisol in the exhaustion stage. During the time of excess cortisol, the body decreases (down regulates) the number of cortisol receptors all over the body so cells will not become over stimulated by cortisol. When the final crash of exhaustion takes place, down regulated numbers of cortisol receptors in combination with lowered cortisol available leave the cells under stimulated. This can be thought of as a sort of chemical deafferentation. Unless neurons' genes are able to up regulate cortisol receptors, which may be difficult since the cell is now under stimulated, the cell could tend towards TND. Likewise, in lowered levels of insulin (with or without receptor down regulation from previous hyperinsulinism) there is a similar type of chemical loss of receptor activation.

Essential fatty acids in the diet play a major role in second messenger activity, acting like a rheostat to either increase or decrease second messenger activity. See figure 2[WHS3]. The proper EFAs in membranes also play a role in membrane and receptor stability. Poor EFAs in the diet can result in inhibition of

a number of second messenger signals, and hence, a dampening effect on neural afferentation which would otherwise promote genetic transcription in the nucleus.

Of course, adequate protein intake, digestion, and assimilation play a necessary role in the eventual availability of amino acids for protein replication which is signaled by genetic transcription. We will say more about amino acids immediately below.

Micronutrients

The micronutrient quality of the diet also plays a major role in proper receptor activation. Nearly all neurotransmitters (NTs) are amino acids or amino acid derivatives. Each amino acid requires between three and eight vitamins and minerals to activate it. Adequate NT receptor activation depends not only on available amino acids, but available micronutrients for conversion of the amino acid into its active NT form.

Other nutrient activity dramatically affects the second messenger systems. The second messenger cAMP is activated by magnesium. Without magnesium, cAMP will not turn on intracellular machinery including genetic transcription. Likewise, caffeine (i.e., from dietary sources) inhibits the enzyme phosphodiesterase which is necessary to turn off cAMP activity once it is turned on. See figure 1[WHS4]b. Caffeine then, turns off the cAMP turn off switch allowing continued activation by cAMP, hence its upper effect in some cells. Excess cAMP activity from caffeine is the same as excess cAMP activity from over stimulation of membrane receptors which activate adenyl cyclase. In other words, one can make a case that caffeine (and similar xanthine derivative drugs often used in the treatment of asthma) could contribute to TND in cells that are activated by cAMP. This might be beneficial if the cell needs more cAMP activity or it could be detrimental if increasing cAMP would further deplete an already exhausted cell.

Antioxidants and EFD

Inadequate antioxidant activity leads to lipid peroxidation with membrane damage contributing to transneural degeneration. This is aggravated by improper dietary EFA which themselves are essential to membrane integrity as mentioned above.

Some patients strengthen when they breathe oxygen and others weaken when the breathe oxygen. If patients demonstrate a weakening effect when they breathe oxygen, one or more of the following will usually be found: a need for antioxidants, a need for EFA balancing, or a need for improved adrenal function to raise the level of the reducing corticosteroids.

In patients with antioxidant, EFA, and/or corticosteroid problems, the use of oxygen could actually lead to cell damage and death by creating excessive oxidation and free radical destruction of the cell. This is one place where AK evaluation is an essential part of the TND patient's work up.

Hypervigilant Immune System and Citric Acid Cycle

A hypervigilant immune system (due to allergy, hypersensitivity, infection, toxicity, etc.) results in a release into the systemic circulation of interleukin-1 (IL-1), tumor necrosis factor - alpha (TNF-alpha) and other cytokines. Receptor activation of neurons (and other cells) by cytokines (IL-1, TNF-alpha, etc.) causes second messenger activation which stimulates the activity of nitric oxide synthetase (NOS) which increases the cellular levels of nitric oxide (NO). NO inhibits the citric acid cycle (CAC) by blocking

aconitase enzyme. Clinical experience using AK testing suggests that other free radical activity blocks other CAC enzymes as well.

The blocking of the CAC leads to intracellular acidosis due to build up of lactic acid and CAC intermediates (which are all keto acids.). It also leads to a deficiency of NADH and FADH2 entering the electron transport chain (ETC) to neutralize oxygen which is also entering the ETC for the production of ATP. This causes a net effect of the ETC producing little or no ATP and lots of free radicals. This free radical production can be interrupted by ascorbate, but the intracellular acidosis inhibits the cell from absorbing anything to make it more acid. The use of buffered ascorbates here is crucial.

In addition, the decreased ATP will cause cell membrane pumps (e.g., Na-K pump) to operate at a decreased level of activity. Neurologist David Perlmutter, M.D. says that the decreased ATP allows partial depolarization of the neuronal cell membrane which leads to persistent activation of the NMDA receptor by glutamate which allows an influx of free calcium into the cell which activates NOS to produce more NO free radicals.[2] The calcium influx along with the intracellular acidosis can contribute to the cell becoming more positive and moving closer to its threshold. Of course, this becomes an even greater danger if the cell is not receiving its normal level of stimulation via afferent neurons.

Heavy Metal Toxicity

Heavy metal toxicity in neurons contributes to the same unhealthy intracellular processes of TND arising from deafferentation and hypervigilant immune system activity. Heavy metals readily bind to sulfur groups including lipoic acid which is necessary for CAC activity. Mercury, for example, has also been shown to cause degeneration of tubulin[3] which is the protein which allows for axonal transport from the cell body to the axon terminal. Sulfhydryl-reactive heavy metals also promote lipid peroxidation which alter membrane structure and can interfere with mitochondrial function. These metals also induce hydroxyl free radical activity.

Sulfhydryl (-SH) groups are essential functional areas of most proteins in the cell. In fact, most enzyme functions are turned on or turned off by the creation or destruction of -S-S- and -SH bonds. Heavy metal binding to a protein's -SH groups inactivates the enzyme. This can even inhibit Na/K ATPase disrupting the membrane pumps and leading to intracellular swelling. This is known to take place in astrocytes.[4] These actions of heavy metals further contribute to transneural degenerative processes.

Heavy metal toxicity also interferes with CAC function by binding to the sulfur group on lipoic acid and truncating CAC activity. This leads to decreased ATP, increased free radicals, intracellular acidosis, in addition to the destruction of intracellular proteins (tubulin) and the other factors described above.

Lowered CAC activity results in decreased carbon dioxide production. Carbon dioxide is the most powerful vasodilator to the cerebral circulation. In addition, titers of CO_2 (pCO_2) stimulate respiratory activity in the brainstem. Lowered CO_2 results in decreased drive to the respiratory centers for costal and diaphragmatic ventilation. This results in decreased afferentation from respiratory muscles and from the costovertebral and other respiration associated joints, as well as decreased oxygenation of the tissues due to decreased tidal volume. Add to this the effect of decreased vasodilatation to the brain from the decreased CO_2 production, and it is easy to see the importance of the CAC in the healing process of neurons which are in a state of transneural degeneration.

Conclusions

The chemical problems associated with TND are best addressed by dietary and nutritional supplementation measures. One might make the case that the properly afferentated individual will make the correct choices, but in our society we are "trained," usually improperly, about what to eat. Giving a multiple vitamin and mineral is no more adequate than is general chiropractic manipulation, and in some critical patients, just as dangerous. Individual assessment of each patient may be performed using the procedures of Neuro Metabolic Assessment (NMA) developed by this author by combining principles of AK and chiropractic neurology. NMA employs manual muscle testing for assessing motor outcomes to a variety of sensory receptor (often gustatory or olfactory) challenges. NMA is an essential part of the neurological exam for determining the exact nutritional approach for each patient. It is as essential to the patients' outcomes as is the remainder of the thorough AK and chiropractic neurological exams.

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.

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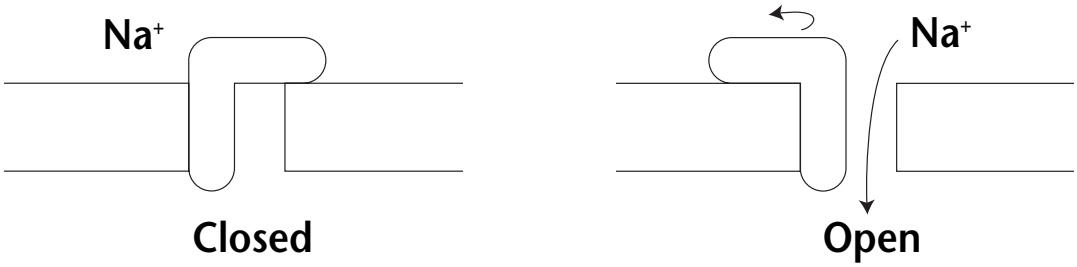
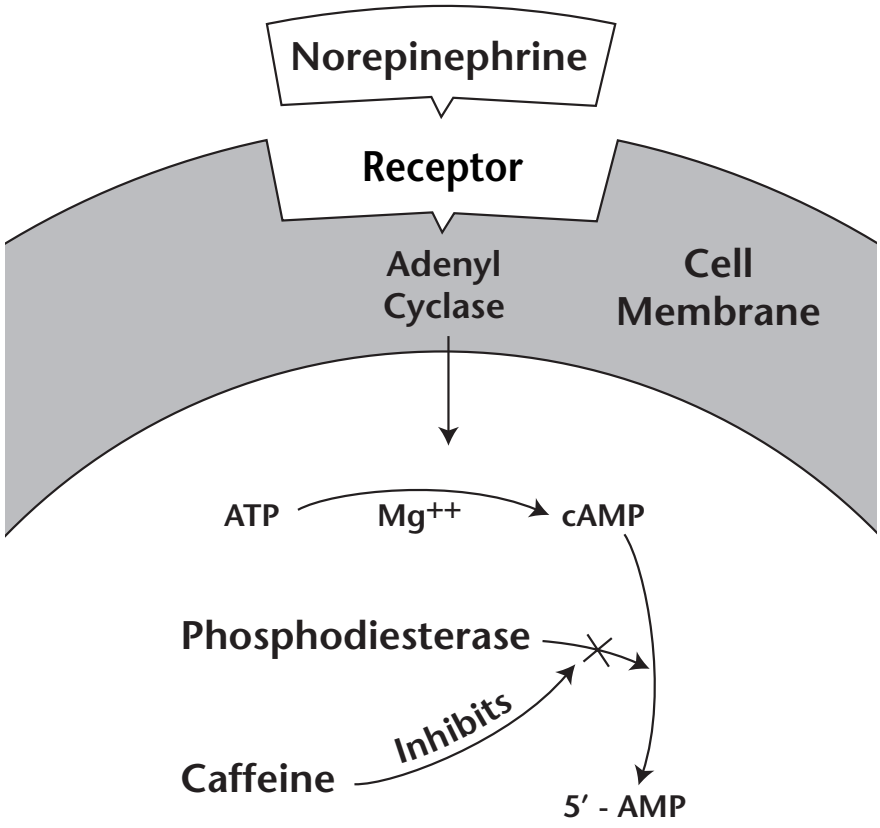
Ion Channel-Coupled Receptors

These receptors bind the neurotransmitter (ligand) and immediately undergo a conformational change which opens an ion channel (to Na^+, Ca^{++}, K^+, Mg^{++}) and generates either an IPSP or an EPSP. They have a short duration of response (1 to 2 msec).



Second Messenger-Coupled Receptor
Activation and Inactivation of cAMP



These receptors bind the neurotransmitter (ligand) and turn on a second messenger system (in the above case, cAMP.) This is usually done through a large molecule transducer called a G-protein which resides in the cell membrane (not shown above.) The responses generated by these receptors can last from milliseconds to minutes or longer.

Cranial-Spinal Centering Procedures

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Abstract

This paper will describe a new procedure for centering the nervous system for treatment. It evolved from the Tomatis techniques shared in previous papers to an investigation of the pyramidal pattern of weakness. This led to a discovery of focusing the body through various tongue positions, which relate to cranial and spinal bones. A simple, yet highly effective procedure resulted which encompasses all previous techniques.

Introduction

My desire to find the best way to center the nervous system so that the most comprehensive correction can be given to a patient has led me through a variety of techniques and discoveries. The basic theory is that how you address the nervous system determines to a great extent how it responds back to you. It is the same as if you were to address someone named Dr. Bob Smith, as 'Dr. Smith', 'Bob', 'Bobby' or 'hey you'. Each way of addressing Dr. Smith will determine how he responds. The nervous system is no different and if we look at the body as only a mechanical system, for example, then we will primarily find only mechanical information. The more we can center the body, the less adaptation and peripheral distortions we will have to deal with. The procedures at the end of this paper show how to center the body in a very special way. In order to appreciate these simple procedures however, I am including the various techniques that were developed along the way. They are valid and usable procedures and the cranial-spinal gaits demonstrate some interesting principles. If all you are interested in is the final technique then skip to the last section on 'Tongue Positions'.

The Pyramidal Distribution of Weakness

The pyramidal distribution of weakness (PD) is one of the pivotal neurological lesions in the body. It is caused by hyper polarization of the thalamus on one side of the brain from deafferentation of the opposite cerebellum. It is characterized by functional inhibition (weakness) of the distal extensors of the upper limb and the ipsilateral distal flexors on the lower limb. The muscles appear functionally inhibited because the pyramidal pathways that are over stimulated are inhibitory pathways. Therefore, in muscle testing you would find functional inhibition of the ipsilateral wrist extensors or finger abductors and foot dorsi flexors upon testing. There is hypertonicity of the wrist flexors and foot plantar flexors, which would only be seen if you tap Gv20 first[i], which unlocks hypertonic muscles so that they momentarily show their functional inhibition. There are other factors found with the PD such as a difference in the measurement of the blind spot of each eye, but for muscle testing purposes the above pattern is a good indication there may be stress in this pathway. This lesion can be found in many people upon testing. More often, it is hidden and needs to be brought to the surface. For instance, placing a tongue depressor between the left upper and lower teeth will often bring out the PD. What I have found extremely helpful is to use the finger abductors or

wrist extensors, on the PD side, as the indicator muscles to use when therapy localizing (TL) or testing for allergies, affirmations, etc. Many distortions are found that do not show with any other indicator muscle. This is how the following new techniques were developed.

Eric Peet wrote a paper in the 1998 Proceedings of the ICAK, The Walking Triad[ii], which describes the mechanics of the skull in relation to both gait and the primary respiratory mechanism of the cranial dura. His concept is that the cranial bones have a different movement pattern in relation to bipedal gait than in relation to the dural reciprocal tension mechanism. He also correlates the pyramidal distribution of weakness with these cranial patterns.

Walter Schmitt, Jr. discusses in his "Centering the Spine" workshops, the three ways the spine moves-flexion/extension, side bending and cross gait movement. He has correlated many endocrine, nutritional and other applied kinesiology procedures with these three movement patterns. Based on Peet's observations, I found that one easy way to determine which side the pyramidal distribution of weakness is active in a person is to perform the following procedure. Have the person do one cross crawl followed or preceded by one homolateral crawl or visa versa. The same result occurs if you have the person do one or more cross crawls followed or preceded by flexing and extending both feet. In other words, use any two of the three gaits or all three. Then test the wrist extensors, finger abductors or extensor hallucus muscles on each side. There should be functional inhibition of these muscles on only one side of the body, the side of the PD. This should correlate with measurement of the blind spot and other indicators.

The previous two years I presented papers based on the work of Alfred Tomatis[iii], the French ear surgeon who developed a system of auditory training and therapeutics. His concept that the right ear is dominant in the normal human being is based on an anatomical asymmetry of the recurrent laryngeal nerves. I found this aspect to have great application in balancing patients and this was the subject of several papers. When I began to test the body with the muscles of the PD in relation to the laryngeal centering techniques, many exciting things developed. I found that any neurological stimulus (depending upon the patient) could activate the PD if the stimulus accompanied sound in the right ear (or the first half of the laryngeal centering technique)[iv]. This produced some excellent results, but required a lot of work to stimulate the different types of nerve pathways in conjunction with sound in the right ear. I eventually discovered that once all possible nerve stimuli had been activated through the right ear, that through various means it was possible to do certain cranial techniques that would cause the PD muscles to functionally inhibit on the other side of the body as well. In essence, the PD would exhibit bilaterally. This led to an entirely new focus and eventually returned me to the gait patterns implicated by Peet in his excellent paper. Since the discovery of the procedures below, it has been unnecessary to use any of the laryngeal centering techniques. They are still valid ways of working, but these new protocols work at a much deeper level.

Discussion

Cranial-spinal Gait Patterns

At the beginning of this paper, I discussed a method of determining which side the PD was active in a patient. This involved the use of two or three of the three different gaits (cross gait, homolateral gait and hopping gait). We return to these gait patterns to activate the PD only with a bit more sophistication. The goal is to activate the spinal gait in conjunction with the appropriate cranial movement and breath phase. I found that different combinations would activate the PD and that when all three gaits were implemented the PD would then manifest bilaterally. Ultimately however, I found that if each gait were done correctly, then each cranial-spinal gait (CSG) would by itself elicit the PD.

Peet's question, "Why would the pelvis walk but the cranium only breathe?"[v] is the basis for the following cranial-spinal gait pattern. We are all familiar with the respiratory movement of the cranial dura as it relates to cranial techniques. Peet's suggestion is that there is another type of cranial movement that occurs in relationship to bipedal gait. He explains how the skull needs to shift its center of gravity to one occipital condyle in order to maintain its balance when we are standing on one foot in bipedal gait. One temporal moves internal while the opposite temporal bone moves external in a counter torque fashion. This helps stabilize the cranium during the shift of the weight when we walk. This cranial gait pattern has received little acknowledgement until now. Breath is not involved here since it is beyond the dynamics of the primary dural respiratory mechanism. It is an important mechanical adaptation of the occipital condyles and the temporal bones during gait (see Peet's paper for a more elaborate discussion of the mechanics). To activate the PD using this knowledge is fairly easy.

- The patient holds his breath.
- While the patient plantar flexes the left foot and dorsi flexes the right foot, the doctor gently pulls inferior on the left mastoid and lifts superior on the right lateral occiput.
- While the patient plantar flexes the right foot and dorsi flexes the left foot, the doctor gently pulls inferior on the right mastoid and lifts superior on the left lateral occiput.

The cranial contacts are extremely light contacts. This is not a cranial adjustment or inspiratory assist treatment. It is only necessary to barely move the skin on the mastoid and occiput. The mastoid contact is easiest if you contact the superior portion of the mastoid for the inferior vectoring movement. If you are sitting at the head of the supine patient, it is easiest to place your hands beneath the patient's head so that the index fingers are on the mastoids and the middle fingers are on the lateral occiput. Then as the patient moves one foot up and the other down, you can very gently vector the mastoid inferior and opposite occiput superior. Following this brief procedure, the PD muscles will test functionally inhibited on one side.

The cranial-spinal gait pattern we are more familiar with is the primary sacral respiratory mechanism as taught by Sutherland, DeJarnette, Upledger and others. The midline bones (sphenoid and occiput) flex and extend while the paired bones internally and externally rotate. The pelvic bones respond in reciprocal motion to their cranial Lovetts. This is quite different from the previous CSG where the innominates and temporal bones counter torque for stability in a normal cross gait. Breath is important in this CSG of course as the movement of the cranial bones with breath is well established. Dorsi and plantar flexion of both feet at the same time to activate cranial dural movement is a part of SOT, Vector Point Technique, cranial osteopathy and other procedures. It is useful here as well. This CSG is a bit more complex than the previous pattern due to the many possible cranial dural imbalances that can occur. We need to make this CSG specific to each patient and each time it is activated.

- The patient inhales and dorsi flexes both feet, and then exhales and plantar flexes both feet.
- Using a functionally facilitated muscle, the doctor tests for a positive therapy localization (TL) somewhere on the occipital bone.
- While lightly holding the occiput contact, the patient inhales and dorsi flexes both feet and then exhales and plantar flexes both feet.

Again, the contacts are subtle and only one breath cycle is necessary after which the TL disappears. If done properly, the PD muscles will demonstrate functional inhibition. If this is the second CSG performed, then the opposite PD will be activated so that the PD is now bilateral.

The third CSG relates to the homolateral gait. The homolateral gait is often considered a vestigial pattern found in early development, but meant to be grown out of. It sometimes considered a form of switching or developmental imbalance when it is found in applied kinesiology testing. I believe this CSG relates to the ipsilateral patterns of the nervous system such as the pyramidal system and has great importance. Bipedal gait is considered abnormal if done in a homolateral fashion, but it would also be abnormal if we hopped everywhere as in the previous CSG. That does not diminish the importance of having this system balanced for what it represents. All three gaits have importance as can be seen in Schmitt's centering the spine patterns. The lateral spinal movements, which we refer to here, relate to the adrenal and thyroid axis[vi]. To activate the homolateral CSG we need to breathe through one nostril at a time and use the ipsilateral foot movement to activate the cranium.

- The patient closes the right nostril in order to breathe through the left nostril.
- The patient inhales and dorsi flexes the left foot and then exhales and plantar flexes the left foot.
- The doctor now can test for a positive TL somewhere on the occiput while the patient continues to breathe through the left nostril.
- The doctor holds the occipital contact while the patient again inhales through the left nostril with left foot dorsi flexion and then exhales through the left nostril with left foot plantar flexion.
- The doctor maintains the occipital contact while the patient switches nostrils so that he inhales through the right nostril while he dorsi flexes the right foot and then exhales through the right nostril while he plantar flexes the right foot.

This all moves quite quickly and once the four breaths have been done with the occipital contact, the TL disappears. This procedure will also functionally inhibit the PD. If it is the second CSG, then it activates the other side of the body. If it is the third CSG to be activated, then a facilitated muscle will test functionally inhibited if the patient places his tongue to the roof of his mouth or holds his breath with a TL to anywhere on the occiput. (See next section for full explanation of this phenomenon).

The three CSG patterns may be done in any order. Following activation of the CSG patterns, I like to scan the body to see what distortions or inhibitions have been exposed by these procedures (in addition to the functionally inhibited PD muscles and the occiput TL with breath holding). Most frequently I find that there is a spinal imbalance that needs correcting. Correction can be done directly to the spine, but most of the time I challenge the five extremities (counting the neck and skull) to see which one needs a gentle Injury Recall Technique (IRT)[vii] traction. Note that IRT as taught by Dr. Schmitt is generally performed only on the feet. It is my experience that when the nervous system is challenged in the systemic manner described in this paper, then there is a possibility of finding the IRT challenge on any one of the five extremities. If you are fixing a local TL associated with trauma and have not involved systemic neurological pathways such as the CSGs, then IRT only to the feet is appropriate. In a previous paper I correlated the five extremities with the five elements and this still holds true[viii].

Tongue Positions

What began as an investigation into the pyramidal distribution of weakness led me to find that it could be activated, from a testing standpoint, on both sides. But with three CSG patterns each capable of activating the PD, I found that the third gait would activate a pattern in the skull previously not seen-the inhibition of a facilitated indicator muscle with the tongue placed on the roof of the mouth. It seemed to me that this pattern opened a deeper neurological area than the PD, or activated the PD more completely. Upon study-

ing this tongue position, I found that it related to the occipital bone if the breath was held. I eventually found that the tongue placed in different positions would have a similar effect upon other areas of the skull and pelvis. This led to the discovery of the following procedures.

There are ten positions to place the tongue in the mouth and each position has a different impact on the nervous system. Each relates to a different vault bone of the skull, the sacrum or the coccyx. The positions are: the tongue on the floor of the mouth, the roof of the mouth, above and behind the soft palate both touching and not touching the bony surface, the tongue not touching anything and the tongue touching somewhere on each of the four quadrants of teeth. There is also a position where the tongue is touching the front teeth (upper and lower) or is just between the slightly closed front teeth.

The first tongue position found was with the tongue on the roof of the mouth. This position relates to activation of the third of the three cranial-spinal gait patterns. In Chinese medicine, placing the tongue on the roof of the mouth is said to connect the governing and conception vessels. It is very important in certain meditation and chi gong techniques. From a muscle testing standpoint we can demonstrate that it equates to a TL to the occiput and holding the breath. If a patient places his tongue on the roof of his mouth, holds a TL to the occiput and holds his breath, it will inhibit a functionally facilitated muscle. In other words, there is no difference between the tongue position and the occipital TL with breath holding.

Placing the tongue on the floor of the mouth equates to a TL to the sacrum with the breath held. This tongue position also activates the lambda area of the skull (where the parietals and the occiput join) if the breath is simultaneously held along with the lambda TL. This tongue position is the beginning point of this technique. (More on this later.) This tongue position centers the nervous system to the spine.

The next tongue position is used along with the previous position in beginning this centering technique. Place the tongue up against the upper and lower front teeth at the same time or lightly between the anterior upper and lower teeth (this second method is easier). This is the only other tongue position that relates to two areas on the body with breath holding. It relates to both the glabella and the area one inch below the navel. These are both areas of great significance as the glabella is associated with the third eye and the area below the navel is associated with the Tan Tien, a major energy center in oriental philosophy. This tongue position relates to the movement of energy through the skull. Placing the tongue lightly between the front teeth will usually eliminate switching as long as the tongue position is maintained. Touching the glabella or the area below the navel, with breath holding, will do the same since they are equal maneuvers. This is a very quick method of temporarily eliminating switching if you want to test something without making a permanent change in the patient's current condition.

The sphenoid bone also relates to a specific tongue position with breath holding. If the rare person is able to swallow his tongue and place it behind and above the soft palate on the undersurface of the sphenobasilar (obviously not everyone can do this), it equates to holding a TL to the greater wings of the sphenoid along with holding the breath. This tongue position is an ancient yogic technique used in deep meditation.

If the tongue is placed above and behind the soft palate, but is not touching the undersurface of the sphenobasilar area it equates to a TL to the coccyx with breath holding.

When the tongue does not touch anything, it equates to a TL to the frontal bone with breath holding. Have the patient open his mouth so that the tongue is not touching above or below.

Placing the tongue on any of the left upper teeth equates to a TL to the left parietal with breath holding. The same holds true for the right upper teeth and the right parietal bone. It does not seem to matter if the patient has dentures or no teeth at all, as long at the tongue is in that position and touching something.

Touching the tongue to the left lower teeth equates to a TL to the left temporal bone and holding the breath. Likewise, touching the tongue to the right lower teeth with breath holding relates to the right temporal bone.

The tongue position on the floor of the mouth (sacrum) focuses energy to the spine. The tongue position between the front teeth (glabella) focuses energy to the skull. The tongue position on the roof of the mouth (occiput) relates to the yin/yang energies of the body and the breath. The other seven tongue positions relate to the seven chakras. All the details of how to use these observations haven't been worked out yet and will be the subject of another paper.

The tongue positions can be used to focus the body/nervous system on specific areas of the skull/pelvis so that an individualized attunement with each patient may be made. Actually, the tongue positions are unnecessary, however they are useful at times, especially the two starting positions. Since each tongue position equates to a TL to a specific cranial bone, sacrum or coccyx, you have the choice of using either way of finding the positive indicator. Have the patient hold his breath while finding the appropriate TL on the cranial bone or use the tongue position. Either method can be utilized since they are equal to each other.

The Technique

As with any technique, there are many ways to capitalize on the observations concerning the tongue positions and their correlations. What follows is a fast two-step method of using these observations to profoundly center a patient. This will maximize the corrective force when the treatment is applied. This protocol is designed for taking the patient 'in the clear' and finding an area to work on by centering him. If you are working with a positive TL, there is no difference in the procedure except that the first step will cancel the positive TL. The following protocol will give both options, the tongue positions as well as the breath holding plus cranial/pelvic TL. The obvious advantage to using the tongue positions is that the patient can breathe during that phase of the testing.

The two tongue positions that have two body correlations (floor of mouth and between the front teeth) work together to center the spine and the skull.

1. First, have the patient place his tongue on the floor of his mouth (breathing) while the doctor searches for a positive TL somewhere along the spine. Hold this TL for a few seconds while the patient stops breathing.

   Or, touch the sacrum or lambda with breath holding and then find the area along the spine that gives a positive TL and hold this TL for a few seconds.

2. Next, have the patient place his tongue between his front teeth while the doctor searches for a positive TL on one of the cranial vault bones, the sacrum or the coccyx. Hold the positive TL for a few seconds while the patient holds his breath.

   Or, touch the glabella or the area one inch below the navel while the patient holds his breath. Find a positive TL on one of the cranial vault bones, the sacrum or the coccyx and hold this TL for a few seconds.

3. Once the previous two steps have centered the patient, there should be functional inhibition of the PD muscles bilaterally. There will be functional inhibition of any facilitated test muscle if the patient places his tongue on the roof of his mouth or if he holds his breath. There will be a positive TL over the thymus area and all the chakras give a positive TL using hand modes or direct contact with the chakra area. It is obvious that this simple procedure has had a great impact on the nervous system. Evaluate the body to see what specific area needs treatment, by scanning the spine, using the set points on the face or any other method of your choice.
4. Treat using a slight IRT traction to the one of the five extremities that tests the need for it. Or do the indicated treatment (adjustment, neurolymphatic, etc,) directly.
5. Repeat the entire procedure until no more patterns can be opened.
6. Test any nutrition, allergen, affirmation, structural area etc. while the patient uses one of the two starting tongue positions, TLs to the thymus or uses the PD muscles as indicators. These indicators will enhance the information you can gather from the patient. The patient is now very clear for testing concerning a chronic or complex problem if you use one of these methods of investigation.

Tongue Positions and Cranial-Pelvic/Breath Holding Correlation

Tongue Position
Cranial/Pelvic Correlation
(With breath held)
Floor of mouth
Sacrum/Lambda
Between and touching front teeth
Glabella/1” below the Navel
Roof of mouth
Occiput
Not touching anything
Frontal
Above soft palate touching sphenobasilar
Sphenoid
Above soft palate, not touching anything
Coccyx
Left upper teeth
Left Parietal
Right upper teeth
Right Parietal
Left lower teeth
Left Temporal
Right lower teeth
Right Temporal

Conclusion

I have been using the tongue positions with excellent results for approximately 10 months now. Patients enjoy the clarity they experience from the treatments and the use of the IRT is a great advantage for many patients. Many patients receive standard manipulations, neurolymphatic work, cranial adjustments etc. as well. The choice is yours in how you want to treat a specific patient. Once the centering treatments are completed, then the use of one of the two starting tongue positions or the other indicators as mentioned above, give greater insight into the needs of the body than we would have from a general indicator such as a pectoralis sternal muscle. It's all right to use the pectoralis muscle as the indicator muscle as long as the patient is also touching his thymus, or maintaining one of the two tongue positions. The benefit of these procedures is that it focuses the patient by centering him through his cranial-spinal system so that the effect of the treatment is leveraged to have more impact on the system.

The tongue enjoys a rich nerve and blood supply and along with the TMJ, lips and teeth has a very large amount of representation in the homuncular map. Our use of the tongue to evaluate the nutritional status of a patient demonstrates how much sensitivity and importance the body places on this sensory organ. The correlation of the teeth with the organ/muscle/meridian system in applied kinesiology and the emphasis of oriental energy traditions on tongue positions further show how much there is to be gained by a full understanding of this system.

Summary of the CSG patterns (for interest only, use Tongue patterns instead):

1. Activate the three cranial-spinal gait patterns (in any order):
   - The patient closes the right nostril so as to breathe through the left nostril. (Start with either side.)
   - The patient inhales and dorsi flexes the left foot and then exhales and plantar flexes the left foot.
   - The doctor now can test for a positive TL somewhere on the occiput while the patient continues to breathe through the left nostril.
   - The doctor holds the occipital contact while the patient inhales through the left nostril with left foot dorsi flexion and then exhales through the left nostril with left foot plantar flexion.
   - The doctor maintains the occipital contact while the patient switches nostrils so that he inhales through the right nostril while he dorsi flexes the right foot and then exhales through the right nostril while he plantar flexes the right foot. The PD muscles should now test functionally inhibited on one side.

   - The patient inhales and dorsi flexes both feet, and then exhales and plantar flexes both feet.
   - Using a functionally facilitated muscle, the doctor tests for a positive therapy localization (TL) somewhere on the occipital bone.
   - While lightly holding the occiput contact, the patient inhales and dorsi flexes both feet and then exhales and plantar flexes both feet. The PD muscles should now test functionally inhibited on both sides.

- The patient holds his breath.
- While the patient plantar flexes the left foot and dorsi flexes the right foot, the doctor gently pulls inferior on the left mastoid and lifts superior on the right lateral occiput. (Start with either side.)
- While the patient plantar flexes the right foot and dorsi flexes the left foot, the doctor gently pulls inferior on the right mastoid and lifts superior on the left lateral occiput. There should now be a positive TL to the occiput if the patient holds his breath.

2. The extensors of each wrist, the finger abductors bilaterally and both extensor hallucus muscles should now test functionally inhibited. (The bilateral pattern of the pyramidal distribution of weakness.) There will also be functional inhibition of a facilitated muscle if the patient stops breathing and holds a TL to the occiput. There will also be an area of functional inhibition such as a localized muscle, TL to the spine, set point, etc. associated with the distortion that has just been exposed by this procedure.
3. Treat by performing IRT to the one of the 5 extremities that tests the need for it or fix what you find directly.

Cranial-Spinal Centering Using Tongue Positions

1. First, have the patient place his tongue on the floor of his mouth (breathing) while the doctor searches for a positive TL somewhere along the spine. Hold this TL for a few seconds while the patient stops breathing.

   Or, touch the sacrum or lambda with breath holding and then find the area along the spine that gives a positive TL and hold this TL for a few seconds.

2. Have the patient place his tongue between his front teeth while the doctor searches for a positive TL on one of the cranial vault bones, the sacrum or the coccyx. Hold the positive TL for a few seconds while the patient holds his breath.

   Or, touch the glabella or the area one inch below the navel while the patient holds his breath. Find a positive TL on one of the cranial vault bones, the sacrum or the coccyx and hold this TL for a few seconds.

3. Evaluate the body to see what specific area needs treatment, by scanning the spine, using the set points on the face or any other method of your choice.
4. Treat using a slight IRT traction to the one of the five extremities that tests the need for it. Or do the indicated treatment directly.
5. Repeat the entire procedure until no more patterns can be opened.
6. Test any nutrition, allergen, affirmation, structural area etc. while the patient uses one of the two starting tongue positions, a TL to the thymus or the PD muscles as indicators.

Tongue Position

Cranial/Pelvic Correlation

(With breath held)

Floor of mouth

Sacrum/Lambda

Between and touching front teeth

Glabella

Roof of mouth

Occiput

Not touching anything

Frontal

Above soft palate touching sphenobasilar

Sphenoid

Above soft palate, not touching anything

Coccyx

Left upper teeth

Left Parietal

Right upper teeth

Right Parietal

Left lower teeth

Left Temporal

Right lower teeth

Right Temporal

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CO_2 Excess Challenge

Implications in Metabolic Acidosis

Barton Stark, D.C.

Abstract

The carbon dioxide excess challenge is proposed for critical review as a kinesiological indicator of metabolic acidosis. The challenge is accomplished by testing a previously strong muscle for weakening following the patient rebreathing their own CO_2. When the challenge is positive, the resultant muscle weakness can be utilized to expose causal relationships. Clinical findings have shown to be multifactorial and should be evaluated as part of the individual patient's history. Common findings relate to toxicity, protein imbalance, adrenal, kidney, liver, and intestinal disturbance. The author concludes the CO_2 excess challenge to be a kinesiological window into the acid-alkaline balance of the body and should be evaluated further.

Introduction

Acid control is a primary issue in the maintenance of homeostasis. Traditionally, applied kinesiologists have used various indicators such as oral and urinary pH, teres major function, and the many signs and symptoms of acid-alkaline imbalance discussed thoroughly by Goodheart and others.[i ii] These indicators usually make any pH imbalance clear. What I have sought is a challenge specifically related to acid imbalance which can be exploited to reveal causal relationships.

Since I began incorporating Schmitt's neuro-metabolic assessment challenges two years ago, I have observed a group of patients who exhibit a weakening response to the CO_2 challenge. The normal CO_2 challenge is accomplished by testing a previously weak muscle for strengthening upon having the patient rebreath their own air 8-10 times using a small bag held over the nose and mouth.[iii] In a certain subset of patients this same challenge creates a weakness of a previously strong indicator muscle. Goodheart has discussed the respiratory structural relationships of this phenomenon in his discussion of the latent vital capacity problems.

Discussion

This phenomenon also suggests an excess of acid in the body, or perhaps a diminished ability to process even a momentary increase in acidity. These patients usually have an oral pH of 6.0 or less. Possible explanations for this include pathological and functional scenarios.

The primary pathological causes of acidosis are discussed in Guyton and include: diarrhea, vomiting, uremia, diabetes mellitus, and prescription drugs which inhibit carbonic anhydrase (Diamox).[iv] Interestingly, the common denominator in all of these is loss of water. Functionally, it is important to note that food allergy, toxicity from various sources, negative emotional states, and structural balance are of primary importance when attempting to balance an over-acid patient.[v] When these are reasonably addressed and the patient continues to show signs of acidosis, I use the CO_2 excess challenge to screen for additional causes.

At this point I tend to find disturbance of the adrenal, kidney, liver, and intestinal systems. Before discussing these an understanding of acidosis is beneficial.

To understand acidosis we need to consider the physiology involved. CO_2 is produced in every cell in the body by the Kreb's cycle. Excess CO_2 is then picked up by the circulating blood. In the red blood cells it is instantly combined with water in the presence of carbonic anhydrase to form carbonic acid, or H_2CO_3. Almost as instantly as it is formed, H_2CO_3 dissociates into hydrogen (H+) and bicarbonate (HCO_3-) ions. The majority of CO_2 is transported to the lungs as part of red blood cell bicarbonate ion.[vi]

Normally, the bicarbonate, phosphate, hemoglobin, and protein buffer systems of the blood quickly prevent the resultant hydrogen ion concentration from rising too high. If this is not sufficient the respiratory center will increase respiration to expel excess CO_2 thereby slowing production of carbonic acid. If the hydrogen ion concentration is still too high the kidneys will begin secreting excess hydrogen ions while resorbing sodium, bicarbonate, and water. This requires an intact renin-angiotensin-aldosterone system and a healthy tubular epithelial cell lining. It also requires adequate sodium and bicarbonate ions.[vii]

The task for the kidneys is to move excess hydrogen ions across the tubular epithelial cell lining into the lumen of the kidney tubules. This is accomplished by a mechanism called sodium -hydrogen counter-transport. In response to aldosterone from the adrenals a sodium ion inside the lumen will attach to one end of a carrier protein. The other end of the same protein is positioned inside the tubular epithelial cell, and accepts a hydrogen ion from carbonic acid. At this point, due to the lower concentration of sodium inside the epithelial cell, the protein flips, thus providing the energy needed to move the hydrogen ion across the tubular membrane into the lumen where it can be buffered and flushed out in the urine. The sodium ion is then actively transported into the extracellular fluid, pulling water with it, and combines with bicarbonate ion (from carbonic acid) to form sodium bicarbonate. This replenishes the blood buffer system.[viii] Thus, the importance of the ability of the adrenals to supply the needed aldosterone. In true hypoadrenia or kidney dysfunction this pathway responds inadequately, at best, leading to dehydration, loss of sodium and bicarbonate, and hydrogen ion imbalance.[ix]

The biochemistry involved in this process is complex, however, using this challenge is relatively simple. If I suspect a person is too acidic I will have them rebreath in a bag and check for weakening of a previously strong indicator muscle. When positive, the resultant muscle weakness may last a few seconds or many minutes in some patients. Challenges which negate this weakness should then be investigated and are outlined below.

Structural, chemical, and mental stressors should be addressed in this investigation. Neurolymphatic reflexes for the adrenals, kidney, liver, and intestines are the most common positive challenges. When an organ reflex therapy localizes I prefer to test the related muscle, alarm, and pulse points to reveal nutrient and structural relationships. These relationships will also negate the CO_2 excess challenge directly. The related muscle often reveals protein imbalance, adrenal and kidney nutrient deficiencies, and, less often, liver and intestinal nutrient deficiencies. Questioning the patient may also reveal overconsumption of carbohydrate, protein, or ascorbic acid as possible causes for acidosis.[x]

Protein imbalance is suspected when amino acids, RNA, or protein itself strengthens the weak indicator muscle. Often these patients are vegetarian or semi-vegetarian and simply need to be more diligent in obtaining sufficient dietary protein. This pattern resembles slow starvation and places great stress on the adrenals. If protein intake seems adequate then the patient should be checked for absorbtion and utilization factors such as HCl and RNA. If these do not show, the liver function should be evaluated as it is a protein factory in the body. Cellular and plasma protein helps counter acidity in the blood and is, in fact, the most plentiful buffer system.[xi]

When the adrenal neurolymphatic is involved check vitamin C, A, adrenal glandulars and protomorphogen, sodium, potassium (Organic minerals-SPL), and protein. In addition to being a major adrenal nutrient vitamin C is influential in blood oxygenation, protein conservation, and hemoglobin formation.xii Phytodren (PRL) is also useful, especially for vegetarians, as it conserves vitamin C[xiii] in the adrenals.[xiv xv] Vitamin A protects the integrity of all epithelial tissues, including endocrine glands, kidney tubules, intestinal mucosa, liver, etc.. When needed, I prefer Drenamin (SPL) for low dose adrenal glandular because it has shown not to overstimulate the gland. This is likely due to the presence of vitamin "G," organic minerals, and adrenal protomorphogen, all of which provide a soothing, balancing effect on the gland. Sodium is most important for extracellular bicarbonate buffer, and may be necessary especially in the exhaustion stage of the general adaptation syndrome. Likewise, potassium may be needed for intracellular bicarbonate buffers usually in the resistance stage of the GAS.[xvi]

With positive therapy localization to kidney neurolymphatic check for Cataplex A-C-P, Kidney PMG, Renafood, Arginex (SPL), manganese, kidney cooling herbs, Vapornil (PRL), and water.[xvii] These patients often have chronic bladder irritation, history of NSAID usage, exposure to chemical vapors, and/or soy allergy. Urinalysis may show albuminuria, many epithelial cells, and specific gravity less than or equal to 1.010.[xviii]

Positive therapy localization to intestinal neurolymphatics generally shows need for leaky gut and malabsorbtion nutrients, and possible excess dietary carbohydrate. These include vitamin A, UltraClear Sustain (MET), PMG for epithelial tissue (Dermatrophin PMG-SPL), Glutamine Plus (NW), parotid, bentonite clay, CoQ 10, and probiotics. Antronex (SPL) and pancreas NL should also be screened as indicators of food allergy.[xix]

Positive liver therapy localization may reveal any of a plethora of problems. It is good to review Schmitt's Liver Detoxification Pathways protocol.[xx] A good general liver support is A-F Betafood or Livaplex (SPL), and also Arginex (SPL) which supports urea production and thereby helps flush the kidneys.[xxi] Also check vitamin "B," homeopathics for various toxins, and food allergy indicators.

All patients should be questioned for excess carbohydrate in the diet, especially refined carbohydrate, as this is a very common source of acidity. Liver and intestinal neurolymphatics and "B" vitamins are strong indicators of this. Conversion of excess carbohydrate to lactic acid by gut fermentation eventually overburdens the buffer systems. "B" vitamins are helpful here as they oxidize lactic and pyruvic acids.[xxii]

Conclusion

Most of these nutrient relationships are predictable and only represent those with which I have experience. I have found it intriguing, however, the number of patients who show signs of protein deficiency together with adrenal stress. Traditional wisdom correctly states that too much protein increases acidity.[xxiii] It also appears that too little may do the same especially in the vegetarian and semi-vegetarian. This may be due to the stress placed upon the adrenals. So much discussion has occurred in recent years regarding protein balance, and I have been pleased to find this challenge useful in advising individuals more specifically about protein.

The CO_2 excess challenge has also shown to be enlightening in cases showing signs of both acidosis and alkalosis. One patient that comes to mind presented with typical symptoms of alkalosis. She was stiff and sore in the morning but got better as she became active. Her Kroenisberg test was 55, indicating her adrenals were failing to retain sodium. Her oral pH was 5.0. This patient's case seemed paradoxical but the CO_2 excess challenge created a very obvious weakening and made her treatment much less confusing.

It is also likely that this phenomenon has implications in dysoxia. Lee states that when the bicarbonate system is overburdened CO_2 builds up at the tissue level, and oxygen utilization is hindered.[xxiv] The end result is an increased cancer risk as well as a host of other low oxygen problems.[xxv]

In conclusion, one can readily discern the importance of acid control in our patients and the sometimes ambiguous constellation of effects occurring when balance is jeopardized. These effects have been discussed in relation to common findings using the CO_2 Excess Challenge, particularly in regard to function of the adrenal, kidney, liver and intestinal systems. I have found that a disciplined applied kinesiology exam is pivotal in unraveling the very individual clinical picture in these patients and facilitates graphic improvement in their ability to maintain homeostasis.

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Subscapularis CO_2 Excess Challenge

Implications in Coronary Artery Disease

Barton Stark, D.C.

Abstract

The subscapularis CO_2 excess challenge is proposed for critical review as a provocative muscle testing procedure which may have implications in coronary artery disease. The challenge is accomplished by testing a previously strong subscapularis muscle for weakening upon having the patient rebreath their own CO_2. When weakening occurs and other causes have been ruled out, the author feels the patient should be evaluated for underlying causes of coronary artery disease.

Introduction

Coronary artery disease is one of the leading silent killers in our society. This author has been asked by many a concerned patient if he or she should consider chelation therapy, aspirin, or more radical medical intervention. As a special application of the CO_2 excess challenge, described in a previous paper, the subscapularis CO_2 excess challenge is felt to reflect decreased coronary circulation under certain circumstances. This implication evolved from an attempt to understand why the subscapularis continued to show a weakening response to the CO_2 challenge even following treatment which negated weakening of other muscles to CO_2. In fact, many times the subscapularis weakness to CO_2 is a stand alone phenomenon likely because it is testing a local, not systemic, build up of metabolic acid. The challenge is performed by testing the subscapularis following the patient rebreathing their own CO_2 eight or ten times with a bag held over the nose and mouth. In this author's experience when neurogenic, myofascial, and emotional causes for this phenomenon are reasonably ruled out the likely explanation is circulatory.

Discussion

As discussed in the author's article, "CO_2 Excess Challenge-Implications in Metabolic Acidosis," metabolic acids are generally buffered and/or carried away by the circulating blood.[i] In coronary artery disease however, the circulation may be inadequate due to arterial obstruction. One would think the resultant decrease in circulating oxygen would also cause the metabolic rate of the heart muscle to slow, and thereby slow the production of CO_2 and carbonic acid. This does not appear to happen, however, until significant build up of metabolic waste, including acid, has occurred. According to Guyton, the average cell can sustain normal metabolic processes with only 1-3 mm. Hg oxygen pressure. The average oxygen pressure supplied to the cell is 24 mm. Hg.[ii] This offers a considerable safety margin to most tissues. When plaquing occurs in the coronary circulation, the blood and oxygen supply to the heart muscle is certainly decreased. The metabolic rate, however, is believed to continue at a relatively normal pace. CO_2 is therefore being produced but not removed due to the diminished blood supply. Fortunately, this process only

occurs in the tissues effected by the occluded vessels as opposed to the entire heart muscle.

Kinesiologically, this situation may or may not cause the subscapularis to become weak. If it does not, a weakness may be provoked using the CO_2 challenge. The subsequent weakness is best analyzed after spinal, cranial, emotional, and myofascial subluxations have been treated with standard methods. If a subscapularis weakness is still evident after all these avenues have been addressed then nutritional chelation and support is strongly suggested.

This author feels the most important organs to consider in coronary artery disease are the liver, gallbladder, intestines, kidneys and thyroid. These are the cholesterol and free radical controllers (or creators) in the body, and their related kinesiological indicators should be evaluated. For a better understanding of cholesterol metabolism, it is recommended to review Schmitt's article, "Liver, Gallbladder, Cholesterol, and Such", which can be found in the Compiled Notes.[iii] Also, it is important to remember that estrogen replacement therapy places females at higher risk for elevated cholesterol. In addition to the organs involved, the reserves of vascular tissue supporting nutrients should be evaluated (A, Betaine, B6, B12, C, P, K, E, E2, "G", and omega 3 and 6 oils).[iv, v] Attempting IV chelation therapy without addressing these key organs and nutrients is a recipe for recidivism.

This author has found it unnecessary to engage in IV chelation therapy a majority of the time if the above items are addressed. Bypassing Bypass by Cranton is recommended reading on the subject of I.V. chelation therapy and coronary artery disease.vi Should chelation be required, herbal methods are recommended prior to IV chelation. The author prefers Vein-Lite (Chi Enterprises), and Rumex Venosus(PRL). Both of these are especially effective when heavy metal toxicity is involved. In addition, phosphorous can be useful for thinning the blood and resisting the calcium component of atheromatous plaque. It occurs naturally in lecithin or can be supplemented as ortho-phosphoric acid. This is especially warranted when blood clotting time is less than two minutes. The author preferred method for mobilizied cholesterol from arterior deposits is Cyruta (SPL). It is strongly recommended to prime the cholesterol excretion mechanisms with a combination of lipotrophic nutrients (A-F Betafood-SPL vit. "G", lecithin, choline, inositol, etc.) prior to chelation so that the cholesterol released from plaque does not simply change addresses in the body.[vii]

There are many possible mechanisms for insult to the coronary arteries which ultimately lead to plaque formation. Dietarily, these include food allergies, over consumption of refined carbohydrates, saturated and synthetic fats, inadequate fiber intake, high homocysteine levels, and xanthine oxidase from mechanically homogenized cow milk, just to name a few. Lifestyle issues include smoking, alcohol, stress, inadequate aerobic exercise, and over use of pharmaceutical and recreational drugs. Toxic metals are also a direct insult. These factors are paramount as they are the root sources of toxicity in the intestines, liver, gallbladder, and kidneys.

Conclusion

Most coronary artery disease patients are given a very limited choice of angioplasty, bypass surgery, and aspirin from their allopathic physicians. The few who refuse this approach for alternative therapies are told they may die at any time if they leave the hospital. The fact is, however, many live with even 70% blockage indefinitely and experience little, if any, symptoms. When confronted with the expense, danger, and misery of surgery, the effectiveness and safety of alternatives such as chelation, nutrient support, and dietary and lifestyle modifications are greatly appealing. The role of the doctor is to educate the patient objectively and conservatively as to their holistic options. The decision, however, remains in the hands of

the patient. In conclusion, there is no more impressive investigative and therapeutic approach than that utilized by an applied kinesiologist with a fundamental knowledge of physiology, nutrition, and structure. These afford the applied kinesiologist the unique ability to uncover even the earliest subtle changes such as those revealed by the subscapularis CO_2 challenge.

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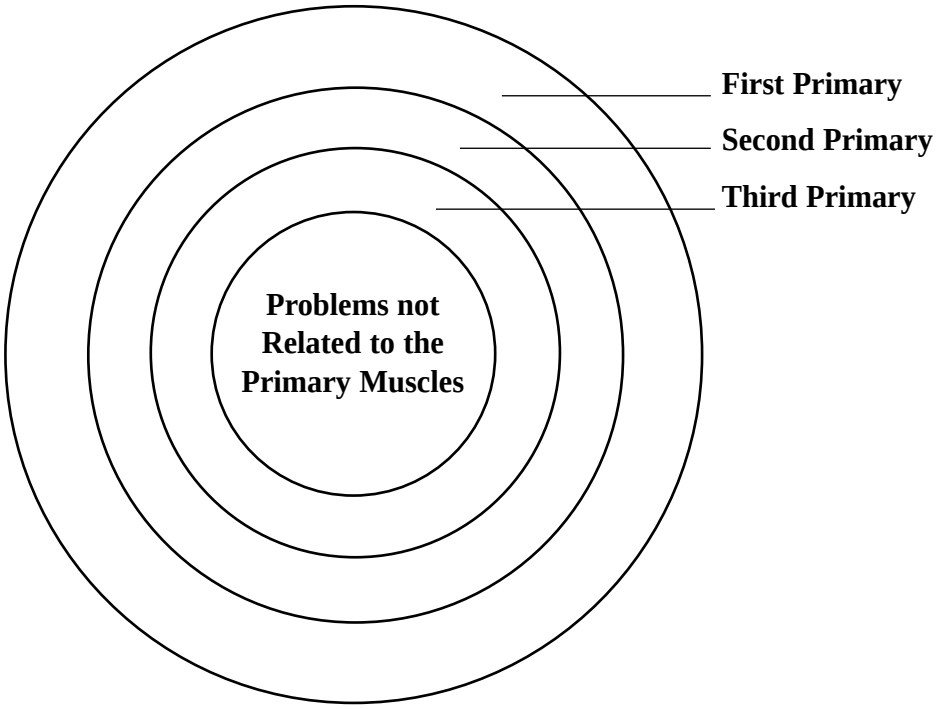
The Relationship Between Weak Muscles, Hypertonic Muscles, Leg Length Inequality, Switching and the Primary Muscle Technique

M. Jay Wiles, D.C.

Abstract

Clinical evidence over the past several years has lead me to believe that several different phenomena in AK are directly related to the primary muscle. Weak muscles, hypertonic muscles, leg length inequality, switching, and cloachial synchronization seem to be related to the primary muscle phenomena. Not all problems are related to the primary muscle. There can be several primary muscles that occur on a patient at any one time, but only one of these will be demonstrated at a time. The others will be hidden until either the first primary is corrected or one of the hidden primaries is brought to the surface by stimulation of certain reflexes.



Introduction

In the fall of 1996 and the spring of 1997 I attended a series of seminars taught by Dr. Robert Rakowski. In this series, he taught the primary muscle technique1 which I find to be very valuable. I have been using this clinically for nearly three years and have discovered relationships between the primary muscle and certain standard AK findings.

Discussion

I predict that if we correct the primary muscle, we will eliminate the leg length inequality, switching, weak muscles, hypertonic muscles, and cloachial synchronization. I would like to discuss each of these conditions one at a time.

Leg Length Inequality

Dr. John Bandy[2] teaches a clearing procedure for leg length. He checks the leg length of a supine patient and checks several positions of the patient to see if these positions affect the leg length. There are six positions: head left, head right; left shoulder off the table, right shoulder off the table; left hip blocked off the table right hip blocked off the table. Dr. Bandy is looking for a position that will cause unequal legs to become equal. I have modified this slightly by looking for any of these positions that will change the leg length. Surprisingly, you will always find one body position out of the six that will cause a change, never two, and never three.

The body position that causes a change in the leg length also causes weakness of a strong indicator muscle. For example, if a block under the right hip causes a change in the leg length, this will also cause a strong indicator muscle to weaken. Dr. Bandy uses the weak indicator muscle while he therapy localizes around the body to determine how to correct the problem. He then, for example, adjusts the spine, the leg length becomes equal, and the indicator muscle no longer is weak when the body is in that particular position. All is well.

I combined Dr. Bandy's method with Dr. Bob Rakowski's primary muscle technique and made an interesting discovery. In the event that a block under the right hip causes a change in the leg length and results in a weak indicator muscle, Dr. Bandy would likely adjust a vertebra. However, you can also therapy localize the neurolymphatic reflexes to find one that will negate the indicator weakness, which is the primary muscle technique developed by Dr.Rakowski. Thus, the body position and leg length change seem to be related to a primary muscle. It wasn't until months after this discovery that I found another interesting thing. If a block under the right hip causes change in the leg length, it inevitably will also cause weakness of a previously strong muscle. In fact, all the muscles of the body will now be weak, except one! This one muscle of the body that is strong will be the primary muscle. In our example in which the block under the right hip causes weakness and subsequent therapy localization to the liver neurolymphatic reflex negates the weakness, then all the muscles of the body become weak except the pectoralis major sternal. Take the block away and the pecoralis major sternal weakens again, since, as the primary, it will be weak in the clear.

Cloachial Synchronization

The next question that came to mind was, "What do the body motions have to do with anything?" It seemed reasonable to assume that the motions that we described earlier must have something to do with gait. So I tried to see if there was any correlation between gait and the motion of the body that caused the weakness. I found no such correlation. However, I did find a direct relationship between the test for cloacal synchronization[3] and body motion. For example, if a block under the right hip causes a change, an unusual type of weakness is demonstrated after the block is removed. Pushing the right leg and the left arm away from each other exhibited a weakness. Yet, if you pushed the leg by itself it was strong, and if you pushed the arm by itself it would be strong, but if you push them at the same time they test weak. Another interesting observation: If you put the block under the hip, all the muscles of the body become weak except the

primary. If you test the two muscles of the cloacal synchronization separately, they also will be weak. But if you test them simultaneously, they test strong.

Weak Muscles

A cornerstone of our practice is finding weak muscles and trying to fix them. If a patient comes in with an injured low back, I look for the location of the injury (disc, pelvis, or SI Lesion, etc). I then look to see if the muscles that support this area are strong. Very often, as you know, there will be some that are weak. I look at the abdominal muscles (usually rectus abdominus), quadratus lumborus, psoas, gluteus maximus, and erecti spinae. Therapy localization of a weak muscle does not cause a strong indicator muscle to weaken, but if you test a weak muscle and therapy localize over the belly of that muscle immediately after the test, you will get a weak indicator muscle (Rakowski). You can then test the neurolymphatic reflexes and the indicator muscle will strengthen when you touch the reflex related to the primary muscle. You will find that the indicator is weak and the primary muscle will be the only strong muscle.

You will note that I said that you touch the neurolymphatic to find the primary muscle. If you challenge the neurolymphatic by rubbing, you are apt to find more than one that will change the strength of the indicator muscle. I believe that this is because there may be more than one primary (diagram above). If you touch a neurolymphatic, only the first primary (in the outer circle) will show up, but if you stimulate by rubbing, this will bring a primary in one of the inner circles to the surface. This idea came to me during a lecture given by Dr. Schmitt[4]. Dr. Schmitt was demonstrating a technique whereby the patient is placed in a reverse "C" position and their adrenal reflex was stimulated. If this causes weakness of an indicator muscle, a hyperadrenal condition is indicated. Because of Dr. Rakowski's lectures on this subject, I was aware that if hyperadrenal was the primary (on the surface), putting the patient into the reverse "C" curve would immediately cause weakness without stimulating the adrenal reflex.

I don't want to drift too far from our discussion of weak muscles, but there is a diversion I think is best injected at this point. If the primary muscle indicates hyperadrenal, the patient must be put into the "C" curve to work with an indicator muscle. I thought that it would be easier to work with this if we could find a muscle directly related to the hyperadrenal condition. This is, after all, one of the most common problems that we see. The next time I observed hyperadrenal as the primary in a patient, I put him into the curve and got a weak indicator. I then went to the T.S. line on the patient to see if any points would cause a change in the indicator muscle. As it happened, there was a change in the strength at a point on the line that corresponded with the upper trapeseus clavicular on the T.S. line in "The Pocket T.S. Line Manual"[5]. As a result, if I find an adrenal problem and sartorius and gracilus are not the primary, I will look at upper traps clavicular as the primary. For example, assume we have a patient with weak abdominal muscles. Test the abdominals and have the patient touch over the abdominals immediately after the test. Have the patient lay back still touching the abdominals. A strong indicator muscle will then test weak. Touch the neurolymphatic for the adrenal (usually on the right side) and the indicator muscle tests strong. Test sartorius and test gracilus; if they are weak, test upper traps clavicular. Upper traps clavicular will be strong. This then is the primary. I realize that this conflicts with what has been found in the past regarding this upper traps muscle, but it is consistent with with my observations in the clinic and ask you to try it for yourself. Upper traps clavicular is simply having the arm abducted 90 degrees from the body with the hand in a neutral position. Test by adducting the arm toward the body.

Hypertonic Muscles

A hypertonic muscle is too strong; it is always shortened or tight. By definition, this muscle will not weaken when the sedation point on its meridian is stimulated. If the muscle were normal, stimulation of the sedation point corresponding to it would cause temporary weakening. Touching the belly of this muscle will cause weakening of a strong indicator muscle. As a result, we had a weak indicator muscle. Since I had been finding "primary muscles" from weak indicator muscles regularly, I asked myself, "What would happen if I looked for a primary muscle from the hypertonic muscle?" Upon checking, I did find a primary, which I corrected. I then rechecked the hypertonic muscle and it had normalized. What did this mean? I had previously believed that you had to "turn down" the spindle fibers to fix a hypertonic muscle. I fixed a primary and the hypertonic muscle corrected. I have concluded that the hypertonic muscle is as much a part of the primary muscle phenomenon as the weak muscle.

When I am challenging for a disc problem or subluxation on patient in the prone position, I like to use the hamstring as an indicator muscle. I know that the prevailing research has shown this to be inaccurate. However,I think that it was inaccurate because it is often hypertonic. If you use a hypertonic muscle as an indicator muscle, nothing will cause it to weaken. The only way to weaken a hypertonic muscle is to touch the belly of that muscle. I always test this way before I will use it as an indicator. I was surprised at how often the hamstring would test hypertonic.

This brings me to another point. Regardless of what the primary muscle is, there seems to be a similar pattern of weak muscles and hypertonic muscles. I had noticed that the following muscles would be weak a great deal of the time: sternocleidomastoid, subclavius, rectus abdomminis, quadraceps, peroneus longis and brevis, and unilateral gluteus maximus. Muscles that are often hypertonic are hamstrings, piriformis, and deltoid. I found these because I use the hamstring as an indicator muscle and had to determine that they were working properly. If you use Dr. Bandy's method of finding lumbar disc problems, which I highly recommend, you will on occasion find piriformis syndrome. If all three muscles that indicate disc involvement (rectus femoris, anterior tibialis, and gastrocnemius) are weak, you may have piriformis syndrome: a hypertonic piriformis. It is really interesting to note that these three muscles are weak, and yet they can be fixed by finding the primary and correcting it without touching the piriformis muscle.

It was a mystery to me why certain muscles would be consistantly weak and others would be hypertonic. Joshua Dubin, DC, CCSP stated6 that the abdoninals, gluteus maximus, gluteus medius, and quadriceps are fast twitch muscles that he called "phasic." Dr. Dubin said the phasic muscles react to "disturbance" by weakening. He also stated that the slow twitch muscles (including hamstrings and piriformis) would react to "disturbance" by shortening and tightening. He did not realize that the disturbance was coming from a "primary muscle." In other words, the disturbance is caused by a system in the body (meridian or organ) that is under stress. The weak muscle and the hypertonic muscles can be corrected by finding the stressed system and fixing it. Here is typical example: The patient complains of low back pain. You test the three muscles related to the lumbar discs and find a disc problem at L3 causing weakness of the right rectus femoris that strengthens when you bend the patient's legs to the left. You test the abdominals (rectus) and find them weak. Touch the abdominals immediately after testing them and a strong indicator muscle weakens. Touch the neurolymphatic reflexes, and the reflex for the sinus causes the indicator to strengthen. When sinus is the primary I always look at a food allergy as the cause. Very often the patient will be allergic to milk or wheat. If you take the patient off the allergen or use N.A.E.T. to eliminate the allergy, this pattern of weakness will disappear. This can usually be accomplished in a fifteen-minute office visit.

Switching

Switching or neurological disorganization is such a significant problem that it should never be overlooked. When I fail to check for it I find that it makes a fool out of me. I started looking for this when I was working with the SI Lesion. If the patient was switched, I would be working on the opposite side of the problem. More than once a patient would say, "Why are you working on the right hip when the left hip hurts?" Then I found that, if the patient were switched, and I challenged on the medial aspect of the ankle (talus), I would get weakness of a strong indicator muscle. You can temporarily remove the switching by adjusting lateral to medial on the talus (usually used an activator). I found that the switching can be removed permanently if the primary muscle is fixed. Basically, this is another way to find the primary muscle.

This leads us into another tool that I find useful. It is an offshoot of the tool I learned from Dr. Rakowski, in which he tests a weak muscle and has the patient touch the belly of that muscle. This technique holds that weakness in suspension until the patient takes their hand away from the of the muscle. I took this one step further. A challenge to the talus results in a weak indicator muscle, but this weakness is temporary and doesn't allow enough time to find the primary muscle. However, if you have the patient touch the belly of the indicator muscle (hand over the shoulder, etc.) when you challenge the talus, the indicator muscle will become weak, so long as the patient holds the belly of the indicator muscle. This technique can be used to hold or "lock in" any temporary weakness resulting from a challenge to a vertebra or even a mental image. As long as the patient holds the indicator muscle that weakened following the challenge,the weakness is held in suspension. I have found this to be quite valuable on many occasions. Let me give an example. With the patient supine, you test the right arm in the upright position (90 degrees of flexion). The arm tests strong. Have the patient put their left hand on the right shoulder; this will not change the strength of the indicator muscle. Challenge the left and right talus by tapping on the medial aspect of the ankle. Check the strength of the indicator muscle each time. If one side causes weakening, the indicator will remain weak until the patient takes their hand away from their shoulder. You can then proceed to find the primary muscle. After you fix the muscle, you can recheck the switching and it will have disappeared.

Fixing the Muscle

The traditional way to fix the primary is to consider all the five factors of the IVF. This is described in Dr. Walther's book[7]. One of the pamphlets sold by Systems DC8 compares the system under stress to a circuit overload in an electrical system. This meshes with my own observations. Where are the circuit breakers in the body? I believe the spine contains all the body's circuit breakers, and if we reset the circuit breakers that are blown, we will turn that circuit on. For example, the sinus becomes overloaded because of the allergy to milk, and the system causes subluxation of C2 and T6. In addition, the neurolymphatic for the sinus becomes active, the neurovascular becomes active, SCM becomes weak along with many of the fast twitch muscles, occasionally the slow twitch muscles become hypertonic, switching may occur, and the meridian system is inevitably affected. Yet, if you adjust C2 and T6, the entire pattern will change because this resets the entire circuit. To make that adjustment to hold, fix the milk allergy. In my experience, you can correct the subluxations at C2 and T6 by working on the neurolymphatic and the neurovascular, or cranial. I'm sure that an acupuncturist could fix the subluxations at C2 and T6 with needles, but no one can fix them as fast as we can by simply adjusting C2 and T6.

I researched numerous sources to determine which subluxations might be caused by what organ systems in the body. I consulted Walther's book, Renae Espie's books, basic information from chiropractic, etc. The information I found is not complete, and I don't present it as the final word. Usually, the subluxations caused by a system (heart, sinus, liver, etc.) will be consistent in a given patient over time and from one

patient to another. This is not a hard and fast rule, however, and you will find variations. Thus, I test each time to determine which subluxations are present, and I use the attached chart as a guide. Whichever way you found the primary (off a weak muscle, off a hypertonic muscle, off the switching, off leg length, etc.), a weak indicator muscle will be present. Touch the spine at the areas listed to determine if there is a change in the strength. The indicator muscle will change if there is a subluxation at that vertebra. I use the palmar surface of the middle finger on the spinus process, and if you turn the finger over you will find anterior subluxations. You can challenge for direction of correction, but I don't believe this is essential. If you get some motion in it, you have reset the circuit. If it doesn't adjust one way, try another. Activator usually works, and I've even used a sparker to do this, but my daughter who works in the office didn't like that one bit.

One system that is consistently inconsistent is the hyperadrenal. You will find that occiput or C1 will be involved, but on occasion C2 must be adjusted. In addition, D8 and L3 are sometimes present. I have also found that any chronic problem may show up with this one. Let us say that a patient has had a sinus problem with pain at T6 for years. You have corrected the sinus problem, and it doesn't show up as a primary. However, T6 may need adjusting when hyperadrenal shows as a primary. Hyperadrenal shows up at some level on almost every patient. I see this more often than any other primary. It is caused by the adrenal being overactive,and whose isn't? I believe that this is why upper cervical work has done so much good for so many people.

Another primary that is very common is the heart. This is probably the second most common primary, yet I didn't understand why for a long time.I now believe that it is common not because of the mechanics of the heart, but because the heart is profoundly affected by the emotions. Many people have "heartaches," "broken hearts," grief, etc. All of these are the underlying causes of a "heart primary," and they are not easy to remedy. Of course, nutrition is often helpful (selenium, magnesium, CO Q10, etc.) , but many of the other primaries are much easier to fix. Techniques affecting the psychological side of the triangle might be very useful here.

Fixing Allergies

I have been finding food allergies for many years and have always, realized their importance, now more than ever since I began using Dr. Rakowski's techniques. Sinus, heart, liver, stomach, large intestine, and small intestine can all be related to allergies. I have found food allergies in many different ways. The accepted method is to find a strong indicator muscle and have the patient taste or smell the test substance. I prefer this method because I don't have to get into any "strange stuff" that I have to try to explain to the patient. You can tell the patient that the taste buds have picked up the substance and the nerves transmitted those signals to the brain. Now, the reality is somewhat different. If you have the patient hold a glass or plastic vile with a substance in it, you will get the same weak indicator muscle. I can't explain this to patients, which can be awkward. Nevertheless, this tool is too valuable not to use, even though it is not recommended by many and it just seems strange.

I had a patient that we found to be allergic to wheat. She responded greatly to her abstinence from wheat. A couple of years later, she changed medical doctors and happened to mention to her new doctor that she had this allergy. He gave her a reprint from an article he had read about N.E.A.T. She brought it to me, and I said "ya, ya, sure" and threw it on my desk. After looking at it lie on my desk a couple of weeks, I picked it up and started to read it. After reading it, I think it was at noon on a Friday, I found that she (Nambudripad) was using a weak indicator muscle to find the allergy, and then doing something that eliminated the weakness. It seemed to me to be very similar to what I was doing with the primary muscle tech-

nique. I couldn't wait to get home, where my wife had a convenient wheat allergy to work with. I reasoned that if the body's reaction to the allergy could be changed, I could find it using the tools I learned from AK.

The wheat caused a weak indicator muscle in my wife, and I touched all of the neurolymphatic reflexes without any positive reactions. I then went to the T.S. line and carefully looked for any change without luck. The third place I checked was the B and E points on the face. When I touched (with the pad of my middle finger) the B and E point for the heart/small intestine, I found the indicator muscle strengthen. Now what? From the primary muscle work, I knew that one muscle should be strong and that it would indicate the meridian (or the primary muscle) that is involved. I found that subscapularis (related to the heart) was weak and that the rectus femoris (related to the small intestine) was strong. With her still in contact with the wheat, I touched along the spine until I found points that also strengthened the indicator muscle. These points were related to the small intestine. At each point (C6, D4, and L3), I adjusted. My wife no longer reacted to the wheat with a weak indicator muscle. I have included a table which I developed as a guide to correct both the primary muscles and the allergies.

I attended the N.A.E.T. seminar in December, 1998, and I continued to use my method of correction, but instead of adjusting manually, I used the activator. I knew that this procedure would stop the allergen from causing a weak indicator muscle, but did this really change the body's reaction to the substance? Some proof came when I corrected a milk allergy on a patient who had SCM as a primary (sinus) and didn't instruct the patient to avoid milk. The primary didn't return, and the patient no longer tested allergic to milk. This was unheard of in my experience, because the primary will always return if the allergic patient is exposed to milk.

The N.A.E.T seminar was very worthwhile, and I recommend that anyone interested in allergy work attend. Dr. Devi S. Nambudripad, D.C., L.Ac., R.N., O.M.D., Ph.D. has had many years of experience using her method. She has written several books on the subject, including Say Goodbye To Illness10, which is a "must read" for anyone interested in this work. Her technique differs significantly from what I describe above, and I have modified my method slightly as a result. I don't consider the treatment complete until I can touch all of the six B and E points and go through the three hand modes without the indicator muscle weakening. The three hand modes are (1) thumb and first finger, (2) thumb and second finger, and (3) thumb and third finger. They indicate physical, chemical, and emotional causes. Dr. Nambudripad's experience shows that if the patient is not cleared for many of the common nutrients before being treated for the more complex allergies, bad reactions may occur, especially in some of the weaker patients.

chart

chart

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Division III

Comments

Unconditional AK, A Commentary

What's Yoga Got To Do With It?

Bill Conder, D.C.

Fall 1998 Issue No. 2 of The International Journal of Applied Kinesiology and Kinesiologic Medicine features an excerpt from ICAK U.S.A.'s Health Capsules Newsletter titled "Q&A."[1] The first question deals with yoga: the questioner wants to know if yoga is a good type of stretching. The answer suggests that the questioner is hurrying through his yoga routine and may obtain more benefit riding a stationary bike or walking. Essentially, the questioner is led away from his interest in yoga and directed to aerobic exercise for "general fitness, relaxation, and stress reduction."

Although I do have some exposure to the practice, I am not a master or expert of yoga, and I suspect that the answerer of the question is not, too. But I think it is important that questions about yoga be answered by those who have some expertise in it, as is the case with any subject. Chances are that asking an orthopedist if chiropractic is good for low back pain will yield biased, one-sided answers. Furthermore, it seems inappropriate for one to attempt to lead another away from a personal interest and into what one presumes to be something better. Does this not establish a karmic responsibility?

"Yoga" is from a Sanskrit word that means union, or to yoke or join. B.K.S. Iyengar explains that it means "...to concentrate one's attention on."[2] Acknowledged yoga master Patanjali, who wrote the Yoga-Sutras in the 3rd century A.D., explained yoga as "the cessation of the modifications or fluctuations of the mind or consciousness."[3] These descriptions appear to have no relationship to the yoga stretches or poses, but a closer examination may reveal one.

Asana, or pose, aside from its benefit to the musculoskeletal system, seems also to have an effect on the mind. To understand this, one might think of doing asana not as posing, which simply involves stretching, but as disposing of chronic habits of posture. The effect of disposing of chronic habits of physical posture is to make it easier to dispose of habitual postures of mind, which probably is where the more troublesome inflexibility resides anyway.

Disagreement regarding the importance of asana in the practice of yoga exists among the different schools of yoga and throughout its tradition. But there is agreement on the notion that the practice of yoga liberates the mind from the effects of illusion and leads to a realization of higher, if not ultimate, truth - an honorable pursuit. Meditation, moral disciplines, breath-control exercises, and devotional practices are among the other aspects of yoga practice.

It was breath-taking!

As I understand it, yogasana, the physical-positioning-stretching, is performed with controlled breathing exercises called pranayama.

Pranayama, or breath control, is one member of Patanjali's Eightfold Path, a systemized method of achieving the goal of yoga. In any case we are aware of the relationship between breath, bio-chemical function, emotional and mental states, and relaxation. Proper breathing during exercise is necessary to meet the

demands of muscular activity and it is thought to have a therapeutic effect on the whole body-mind when the musculature is relaxed, as is the case with pranayama.

Conscious, controlled breathing is an important tool in some psychotherapies. AK doctors use the controlled breathing phase-of-respiration adjunctive to cranial and other adjustments, and in other ways. Since we breathe automatically, as respiration is under autonomic nervous system control, but can also control intentionally the rhythm of our breath cycle within certain limitations, some researchers have proposed that controlled breathing is a window to the control of other autonomic functions, especially what we think of as the stress reaction. Since many of us habitually and chronically are controlled by the stress reaction, and as chronic elicitation of the stress reaction without adequate recovery from its effects causes chronic disease, acquiring the ability to modulate "stress" with conscious breathing should have a healthful effect. The phrase "control the breath and calm the nerves" has truth in it.

Furthermore, the yoga poses are performed with a mental focus which is called meditation. But meditation, a very prominent branch in the science of yoga, also is to be practised by itself. Lots of research has demonstrated the beneficial effects of meditation (and prayer, which some say is similar to meditation). Drs. Herbert Benson and Deepak Chopra have written about it extensively. The Transcendental Meditation organization, centered in Fairfield, Iowa, has made meditation big business and helps individuals worldwide. World-class athletes use meditation techniques to improve their performance. I, too, believe that meditation is important and I recommend it to my patients.

Still, there may be a mechanical and mundane explanation for the beneficial effect of yogasana on the mind. Researcher Valerie Hunt describes in her book Infinite Mind discovering that electrical activity was higher or stronger when measured on the bodies of individuals who had just received Rolfing deep-tissue massage in spite of the fact that her instrument readings of muscle tension indicated greater muscular relaxation.[4] She believes, as others do, that connective tissue conducts electricity, and that the field-emanation of electromagnetic energy some call the aura is the mind.

Therefore, if the asanas of yoga practice, by disposing habitual posture in the body, produce a stretching in the connective tissue, and if this stretching enhances the electrical activity of the connective tissue, and if this electrical activity is associated with what we call the mind, then it is conceivable that this kind of physical activity promotes mental clarity.

How much is that six-pack in the window?

Of course other forms of exercise are valuable, especially the aerobic ones like walking, jogging, cycling, swimming, and so on. Of these, walking is probably the best if only because it is the most accessible for most of us. Swimming, in my opinion, is the best of all forms of exercise for a number of reasons, but there are limitations associated with it. Cycling, again in my opinion, is the worst for many good reasons, not the least of which is the chronic mal-position of the spine, the almost exclusive use/overuse of the lower extremity, absence of cross-crawl reinforcement, and, for men, irritation of the prostate gland. The high cost of equipment makes it prohibitive for some people - and what about that funky-looking headgear, which is necessary because riding a bike can be dangerous.

Of course, riding a stationary bike in a fitness center is not so dangerous. In fact it's boring - so boring that riders read books or listen to music on portable headsets while exercising. This form of exercise, it would appear, is drudgery performed only because "it's good for you." It is mindless activity for the body.

Yoga, on the other hand, purports to unite mind and body, enhance awareness, and elevate consciousness. In recommending mindless drudgery over mindful rebalancing, Health Capsules Newsletter seems to have answered before making a thorough examination of the subject matter. Our modern scientific, technological, and commercial environments may blind us with facts that encourage us to be hasty in our reasoning. Yoga is an holistic health science that's been around for thousands of years. It is not perfect, but it is a beneficial and worthwhile pursuit.

Are those buns of steel, or are you just too big for your britches?

One does not have to wear a turban, change one's name to "Swami," and take up residence in a cave to practice yoga. Understanding the fundamentals and purpose of yoga, and separating these from cultural entrapments, one can practice at home from a book with periodic visits to a yoga class for guidance and to exchange ideas. Time spent in yoga practice is physical and mindful, restorative and therapeutic.

I'm guessing that the answer to the question in question was an attempt to elevate a point-of-view about exercise and sports medicine that has flourished in AK and about which we have techniques to test and verify. A lot of people don't want the holism and self-responsibility of yoga, but a lot of thoughtful people do. Putting-off yoga so casually under the AK banner in order to promote a specialized activity will put-off many of these people and potential AK practitioners. This is unfortunate since AK and yoga seem to have many similarities. Both pursue structural balance and integrity with the understanding that bio-chemistry and the mind/emotions are involved cat's cradle-like.

In his article Joint Complex Dysfunction and the Decondition Syndrome, Philip Maffetone, D.C. discusses David Seaman, D.C.'s modernized conceptualization of the chiropractic subluxation and introduces "deconditioning syndrome" as terminology to further explain this "contemporary" idea.[5] In this article, Dr. Maffetone says that a component of the so-called "joint complex dysfunction" manifests as degeneration and atrophy of spinal tissues, reduction in cardiovascular fitness, and pain, as described by Mayer and Gatchel, authors of a book with a sports medicine orientation who actually coined the phrase "deconditioning syndrome."

According to Dr. Maffetone's article, deconditioning of muscles and cardio-vascular capacity is due to sedentary living and may exist long before the event of traumatic injury to tissue. He suggests that the deconditioned patient needs more than "spinal manipulation and the additional treatment procedures related to the five factors of the IVF." He proposes that lifestyle factors, including rehabilitation and dietary adjustments, must be addressed for "true corrective care." Presumably, what Dr. Maffetone is referring to when he says "rehabilitation" includes the prescription of a form of aerobic exercise as recommended in the answer to the question in the above-mentioned "Q&A."

"Deconditioned" apparently is being used by Dr. Maffetone and the others in contrast to "conditioned" which is a term that has been used in athletics and fitness circles to refer to the state of the body necessary to undertake a specific physical or sports activity. "Conditioning" may also refer to the physical training and exercise process undertaken for improved health.

"Was it the Boogie-man? What was he wearing?"[6]

"Conditioning" is also a concept used by psychologists in reference to a specific stimulus causing a specific response. "Deconditioning," that is the extinction of behavior, is thought to occur in psychotherapy to correct what happens when a specific stimulus causes a response that is inappropriate or unhealthy. Some

experts would argue that conditioning response in specific, acceptable ways is a primary function of education and enculturation.

That our behavior is conditioned by environmental cues, that we respond automatically in a specific way to a specific stimulus, ultimately is not desirable. We like to see ourselves as independent entities in control of our lives, which we may be more or less, until something triggers the stress reaction in us. Some researchers, including Hans Selye, M.D., propose that even subliminal chronic elicitation of the stress reaction, warranted or not, is the cause of many of the chronic diseases in our culture that, mysteriously, are most puzzling to us.

The stress reaction is automatic and mediated by the autonomic nervous system, specifically the sympathetic branch. But chronic sympathetic activity unbalances autonomic function, diminishes potential energy, and leaves one in a state of low-grade, anesthetic shock; the effects are experienced in all systems of the body. The sympathetic reaction is designed to help us survive threatening situations and adapt to less than optimal conditions, but temporarily.

Conditional Love

The worst part is that we love the excitation provided by the elicitation of the stress reaction. Our entertainment and pasttimes are filled with triggers to which we are conditioned to respond with increased sympathetic nervous system activity. We seem to be addicted to this activity, which we may examine for clues to the problem of addiction in general. In any case, most of us react as conditioned by the cultural boogieman with whom we have a junkie-pusher, co-dependent, love-hate relationship.

Education, especially in the professions, uses stress as a conditioner to make us act like “professionals” - whatever that means. Eventually, the stress reaction becomes environmental and, therefore, invisible. Ironically, in the true sense of the word, doctors treat patients with diseases that can’t be understood or explained but that seem to be associated with chronic stress. Soon we accept arthritis and cardiovascular disease simply as a part of the aging process. Eventually, as younger people develop these diseases, they will be just a fact of life. Meanwhile, the exciting reports of genetic research will give us a rush of adrenalin followed by a trickle of cortisol as a fix for keeping empty promises and inverted hope alive.

This sympathetic/fight-or-flight/stress reaction is anaerobic, over-rides the preferred oxidative respiratory metabolism, and unbalances sympathetic/parasympathetic function. It makes sense that the fight-or-flight reaction should over-ride the calm, aerobic, intentional state if one’s survival is in question. But it doesn’t make sense if we’re running around like rats in a maze - or on a treadmill.

“Joint complex dysfunction/decondition syndrome” as described by Drs. Maffetone and Seaman sounds like it could be arthritis with the beginnings of heart disease - degenerative diseases caused by chronic stress conditioning. What’s new here is that the point-of-view of these degenerative diseases is “sports medicine” whose primary treatment modality is rehabilitative exercise. Is not this “sports medicine” the latest craze that seems to be taking health care and sports fanatics by storm?

Of course, regular mild to moderate physical activity is a condition of good health. And, in our culture, so is adequate rest, stress reduction, and awareness. A side-effect of interaction with the artifacts of our cultural environment is the subliminal elicitation of the stress reaction. Periodically disengaging one’s self from this entanglement is one of the things one gets from meditation; rest, stress reduction, and awareness are other benefits.

Above all, one should pursue one's interests, making informed, conscious decisions as one goes. As for "general fitness, relaxation, and stress reduction," yoga is hard to beat. As a recommendation to the questioner in "Q&A": Spend less time on the rat-race treadmill giving it up for the boogey-man, and more time in repose taking care of self.

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