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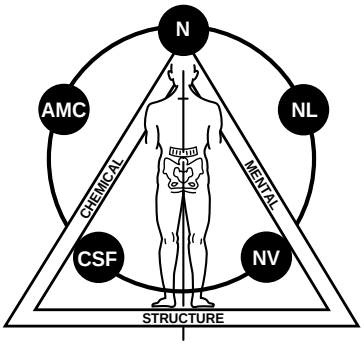

Message From the Chairman

Dr. David Leaf

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

Past history shows that the observations of one doctor stimulate the minds of others and the end result can be "another piece of the puzzle," as Dr. Goodheart often says.

Thank you and congratulations to all of our contributors. And a special thanks to Drs. 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 22-25, 2000 in Palm Springs, CA.

Introduction

This forty-second collection of papers from members of the International College of Applied Kinesiology-U.S.A. contains 21 papers by 17 authors. The papers will be presented by the authors to the general membership at the Annual Meeting of ICAK-U.S.A. in Palm Springs, CA, June 22-25, 2000. 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

Evaluation of TMJ in One Case Study with Congenitally Absent Right Forearm

Janet Calhoon, D.C., DIBAK

Abstract

A case history of temporomandibular joint (TMJ) treatment in a 13 year old female with a congenitally absent right forearm, wrist, hand and fingers.

Introduction

Temporomandibular joint (TMJ) problems are certainly a common presenting symptom to the applied kinesiologist. The standard approach includes a two-handed therapy localization, the appropriate hand to the ipsilateral temporomandibular joint. When a patient presented with TMJ pain and a congenitally absent right forearm, wrist, hand and fingers, the approach took some rethinking.

Discussion

A 13 year old female presented with an inability to open her mouth wide enough to accommodate two knuckles of her hand. The problem had started one week previously when she fell during physical education class and struck the left side of her head against the floor.

Examination revealed an upper cervical fixation, cervicothoracic fixation and thoracolumbar fixation.

Her right upper extremity is normal in size and function from the shoulder to the elbow. Her forearm ends approximately two inches below the elbow with no carpals or metacarpals and rudimentary digits, each approximately one-eighth inch in length. When I asked her to therapy localize the right TMJ with the right upper extremity the limb came closer to her nose then to the TMJ. She wanted to help by changing the position of her head and neck which of course was not acceptable because of the many variables that would throw into the equation. With a little patience and persistence, she did therapy localize the right TMJ with the right upper extremity. Examination and evaluation demonstrated a need for neuromuscular spindle cell technique to “turn down” the right internal pterygoid. The right sternocleidomastoideus was evaluated and demonstrated a need for neurolymphatic reflex treatment as well as golgi tendon organ and neuromuscular spindle cell techniques.

As we proceeded through the evaluation, at times I assisted her in placing the distal end of the right arm on the body part to be therapy localized. When I observed her putting her head and neck into a distorted posture trying to make contact with the arm I just spoke candidly, explaining the need to accomplish the task and asked if I could assist. I was met by a simple matter of fact, go-ahead-and-do-it, attitude from the patient. Many times, in my experience, teenagers are extremely self conscious and very aware of anything about their persons that make them feel different from their peers. This young woman was very used to being self sufficient and yet when I spoke honestly, explaining how stretching her head and neck and mov-

ing the jaw to meet the arm would produce misinformation, she readily allowed me to place the rudimentary limb where it needed to be.

At that same office visit, the upper cervical, cervicothoracic and thoracolumbar fixations were also corrected, as was a universal interosseous cranial fault. The right gracilis was also treated via neurolymphatics.

At the end of the treatment, she opened her mouth almost wide enough to accommodate three knuckles.

I have treated her on subsequent visits and she improved with each visit. She ran into some stumbling blocks by jamming her jaw against her normal hand while studying. Some instructions regarding good posture and study habits were offered and her progress continued.

Conclusion

Bilateral therapy localization is a very valuable tool in evaluation of the TMJ as well as many other situations. A rudimentary limb can be used to therapy localize and therapy localization using such a limb is as effective as using a normally formed limb. Applied Kinesiology techniques over the decades have given us answers for which we sometimes do not understand the questions. However as Dr. Goodheart has said countless times, “look with eyes that see.” By investing a little extra time and effort this case was brought to a conclusion that was more than satisfactory to the patient, her parents and myself. As an interesting aside, the last time I saw this young woman, her complaint was pain in three fingers of her left hand caused by a hyperextension injury playing basketball! There’s just no setting of limitations for her.

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1. Walther, David S., Applied Kinesiology, Synopsis, 2nd edition, pp. 413-416.

2. Walther, David S., Applied Kinesiology, volume II, pp. 424-437.

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Applied Kinesiology Management of Asthma: A Case History

Cecilia A. Duffy, D.C., DIBAK

Abstract

A case history of applied kinesiological management of asthma in a six-year-old female is presented.

Introduction

Incidence of asthma is estimated to occur in three to eight percent of children and is diffuse airway inflammation provoked by allergy, virus, irritants, and exercise, characterized by episodic dyspnea, cough, and wheezing due to bronchospasm. Medical intervention consists of medications for bronchodilation and to decrease submucosal edema.[1]

Discussion

A six year, six month old female was evaluated for a primary complaint of asthmatic symptoms. History revealed a previous diagnosis of asthma from a medical pediatrician at the age of ten months with continual use of prescription medications (Proventil and Ventolin) for symptom control. Asthmatic episodes were generally intermittent prior to presentation at my office. When she experienced her sixth asthmatic reaction within five weeks, her mother sought alternative care. The remaining medical history was unremarkable as reported by the mother.

Physical examination revealed: weight 54 pounds; height 3'11"; axillary temperature 98.4 degrees; salivary pH 6.8; blood pressure seated 80/60, standing 80/58, and supine 90/50; pulse seated 100, standing 100; Lingual Ascorbic Acid Time was elevated at 15 seconds on the right and left sides of the tongue; hematocrit 38; breath holding time diminished at 10 seconds; vital capacity diminished at 650, (normal for age is 980), right hand and foot dominance with left eye and ear dominance; and first morning urinalysis negative via dip stick, with Sulkawich testing for calcium levels elevated at grade 4, and Koensberg testing for chloride (indirect sodium) elevated at 30 plus. Auscultation of the lungs revealed scattered rales and wheezing throughout both lung fields.

Based on the history and physical findings of a positive Ragland's sign, mild vitamin C deficiency, and elevated urinary chloride level, adrenal dysfunction was presumed and examined for via applied kinesiology testing. Examination revealed the following and was corrected on the first evaluation and treatment: conditionally inhibited left upper trapezius and left sartorius, and positive challenges at the following levels for subluxation-Category II pelvic lesion, T9, T2, and a fixation at the right C7-1st rib junction.[2] Utilizing the left sartorius, nutritional supplements relating to adrenal function were tested via oral administration and Drenamin (Standard Process Laboratories)[3] tested positive by negating the conditional inhibition of the left sartorius. Drenamin contains bovine adrenal nucleoprotein extracts and vitamins C, riboflavin, and niacin, among others. She was placed on Drenamin at a dose of one, three times a day.

Dietary analysis revealed heavy consumption of refined grains, refined sugars, and dairy in the form of whole milk and cheese; there was no vegetable intake, 1-3 fruits per week, and only refined/processed proteins like bologna. She was instructed to totally restrict milk, and refined grains and sugars, and to consume only vegetable, fruit, non-processed proteins, high quality fats, and non-processed whole grains.

The patient complied fairly well with the diet restrictions and supplementation. The mother discontinued both medications shortly after the first visit. The patient was treated a total of five times in three months. Structural examinations and treatments were based on evaluations of the patient's posture, temporosphenoidal line, and manual muscle testing.[4] During the three months of therapy, the patient experienced one episode of an asthmatic reaction and two brief episodes of a congested cough. The asthmatic reaction was preceded by heavy consumption of refined grain and sugar earlier in the day. On the last visit, she was instructed to finish the Drenamin and continue with diet changes, and placed on a self-schedule basis for treatment.

At seven months and ten months following initial presentation the patient again returned after experiencing one asthmatic reaction and several asthmatic reactions respectively. Each time, the patient was seen twice in two weeks for applied kinesiology evaluation and treatment and prescribed Drenamin as well as Congaplex (Standard Process Laboratories).[3] Congaplex contains bovine thymus extracts, vitamins A and C, and other vitamins and minerals. On each occasion, after the first visit, the asthmatic reactions stopped.

At seventeen months following initial presentation, she returned with an upper respiratory infection, and mild wheezing was noted in the lung fields upon auscultation, but no asthmatic reactions occurred. She was seen at twenty-two months following initial presentation for acute neck pain, and at thirty-two months following presentation for an upper respiratory infection. There were no episodes of asthmatic reactions noted to have occurred.

Conclusion

Successful applied kinesiological management of asthma over a seventeen month period in a six year old female is presented. Further investigation of the effectiveness of applied kinesiology management of asthmatics is warranted. The primary approach of medical management of asthma is medication, whereas the approach of the applied kinesiologist is to diagnose underlying dysfunctions that when corrected safely produce optimum health rather than drug-induced "control."

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Nitric Oxide - Placental Tissue Correlation

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

Abstract

Nitric Oxide has been associated with blood pressure and immune function. Various nutrients have been demonstrated to influence its production. There has been discovered another product which appears to influence the oral challenge of arginine, and that is placenta (Ecological Formulas).

Introduction

Nitric Oxide is an end product of the arginine cycle and is responsible for maintaining the endothelial relaxing factor as well as part of the immune response via the phagocytes. Dr. Goodheart proposed a procedure to test for the adequacy of nitric oxide by an oral challenge of arginine. If this produced a positive challenge various nutrients were utilized to test for the negation of the weakening response. These included arginex (Standard Process), calcium/magnesium, folate B/12, and superoxide dismutase. In addition to these, another substance has been found, placenta (Ecological Formulas).

Discussion

The May 1992 Scientific American classified nitric oxide as a new class of neurotransmitter. Nitric oxide is produced from the internal lining of blood vessels, neurons, and phagocytes. It has been correlated with blood pressure, immune system, cholesterol, platelets, and sexual function.

Dr. Goodheart has described a screening procedure for nitric oxide. Arginine is placed on the tongue and a previous strong muscle is tested for response. If the muscle tests non-intact, then nutrients are introduced on the tongue, one at a time while the arginine is still present there. These are arginex. (Standard Process), calcium/magnesium, folate B/12 and finally superoxide dismutase. The doctor is to continue testing until the weakening is negated, leaving the previous substance on the tongue. The patient is then prescribed the nutrients which negated the weakening effect from arginine.

Another factor has been discovered by this author to negate the weakening effects of arginine. That factor(s) is placental tissue (available from Ecological Formulas). With the arginine on the tongue, if a weakening response occurs, simply place some of the placental tissue orally, recheck your indicator for strengthening. If positive, supplement the patient with placenta. This may or may not negate the need for factors mentioned above, but is an additional factor which appears to be clinically beneficial to the patient if so indicated. The dosage is usually three to nine capsules daily depending on patient need and response.

Conclusion

There is another factor(s) involved in negating the weakening response via oral insalivation of arginine, and that is placental tissue. This appears to have a positive clinical effect on the patient response if so indicated via manual muscle testing. Exactly what is present in the placental tissue responsible for this effect in unknown at this time to the author. However, no adverse side effects have been reported.

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Why Can't Sports Medicine be More Like Football

Dr. Evan Mladenoff

"It must be remembered that there is nothing more difficult to plan, more doubtful of success, nor more dangerous to manage than the creation of a new system. For the initiator has the enmity of all who profit by the preservation of the old institutions and merely lukewarm defenders in those who would gain by the new ones."

from "The Prince," by Machiavelli, 1513

The attention of the majority of doctors has been directed toward pathology and trauma which are demonstrated by significant abnormalities observed in the laboratory, on x-ray or MRI, and by standard physical diagnosis. The great strides that have been made in these fields are commendable, yet there remains the athlete who complains of low back pain, neck pains and stiffness, muscle spasms or joint disturbance - among numerous other symptoms - but is pronounced "game ready" after a thorough diagnostic work-up. These subjective symptoms are often diagnosed as fatigue, a muscle pull, muscle strain, joint sprain, the athlete is a head case, functional disturbance[1] or frankly ignored because no objective findings are present. Limited diagnostic procedures cause the physician to only occassionally be able to evaluate the cause of these symptoms. There is an absence of laboratory findings because these conditions are usually functional[2] rather than pathological.

When an athlete has been diagnosed with a lumbar disc injury that is not severe enough for surgery to be an option, is an example of aberrant function of the body. This injury may be permanent and prevent a return to any sense of a 'normal' career performance style. It is clear that the athlete will have incentive in seeking innovative solutions for injuries, improving pain reduction and preventing degenerative changes. What you will discover is just how difficult it is to obtain an unbiased opinion.

Just as one would not go to IBM to ask if Macintosh makes a good product, the athlete and team management needs to be at least wary of asking a group of medical doctors if chiropractic or applied kinesiology is any good (the AMA was found guilty of conspiracy to eliminate the chiropractic profession[3]), or asking orthopaedic surgeons if a new enzyme formulation (that will produce anti-inflammatory effects, pain reduction, repair to disc fibres and will eliminate the need for surgery) is any good. Unfortunately, the tendency in the world of sports medicine is to want to defer assessment of medical innovation to a group of medical experts.

Thomas Chalmers of Harvard School of Public Health said "A few years ago I looked at review articles of subjects like radiotherapy for patients with radical mastectomies, coronary artery surgery and emergency surgery for bleeding peptic ulcer. The opinions of the 'experts' who wrote reviews were always dependent on how they were trained, not on the body of evidence. That convinced me that on the average the opinion of experts is no good."

To most people not involved in scientific research it seems odd that there are innovative therapies existing today that offer solutions to debilitating injuries and some of our most pressing health problems. Surely, one would think, discoveries and breakthroughs offering great promise in the treatment of injuries and dis-

ease would be immediately communicated and embraced by the scientific/medical community. Historians of scientific progress conclude otherwise.

Historical citations of science resisting new ideas are too numerous to review in any depth, from Copernicus to Galileo to Darwin, Pasteur, Einstein, Watson , Fleming ... the list goes on and on.

This presents a dilemma. How can the athlete or team management assess sports medicine? The answer, I believe, is to simply focus on results. It should not matter to the athlete that solutions to various injuries come from different modalities. If the injury does not require surgery, then the quickest, least toxic, performance enhancing, career and health enhancing treatment should be the athlete's and management's concern, not the kind of treatment. Either a physician has therapeutic modalities that have good results or he doesn't.

Convinced of the need to focus on results, the athlete and team management must be careful not to fall into the trap of throwing out the first tomato. Historically it is referred to as the "Tomato Effect" in medicine. This means that instead of accepting an effective treatment or examination procedure you throw a tomato, that is, an immediate rejection of a highly effective therapy because it does not "make sense" in light of accepted theory, OR, more importantly the majority of doctors and team trainers don't do that or would never do that. Remember, everyone thought it was crazy for Columbus to sail across the ocean for the world was believed to be flat and Columbus will sail off the end; what do you mean man will land on the moon - he can't even flap his arms and fly like a bird; it's not baseball if you have a designated hitter; it's impossible for a human to run a mile in less than 4 mintues; a goalie to wear a mask in hockey, it will never happen; it doesn't matter where you shoot from it's 2 points for a basket; a hockey player to score 50 goals in 40 games or less is ridiculous; a hockey player to score more than 200 points in a season is absurd, it will never happen; a 260 pound linebacker running the 40 in 4.5 or less not possible, a running back rushing for 2000 yards in a single season unthinkable, winning the Super Bowl 3 years in a row insanity etc. etc.

Introduced in 1984 in the Journal of American Medical Association, the tomato effect is derived from the history of the tomato in North America. By 1560 the tomato was becoming a staple of the continental European diet. However, it was shunned in America until the 1800's. Why? Because it was poisonous. Everyone knew it. It was obvious. Tomatoes belong to the nightshade family. The leaves and fruits from several plants in this family can cause death if ingested. The fact that Europeans were eating tomatoes without harm was not relevant. It simply did not make sense to eat poisonous food.

In the Journal of the American Medical Association May 11, 1984 discussing the "Tomato Effect" shows us how easy it is to get lost in the details of the language of medicine when it states: "we are asked to use a new arthritis drug because it stops monocytes from crawling through a filter, a new antidepressant because it blocks re-uptake of serotonin but not epinephrine into rat synaptosomes,..."

What gets lost in these discussions are the only three issues that matter in picking a therapeutic approach or a physician:
1. Does it help?
2. How toxic (poisonous) is it?
3. Does it restore or lengthen the athlete's normal competitve performance?

In this atmosphere, the athlete and more importantly team management is at risk of rejecting a safe, effective therapy in favor of an alternative treatment perhaps less effective, more poisonous, health deterring and life threatening. Such an attitude also increases the risk that we use a medication to "normalize" a lab test regardless of whether it improves the athlete's state of health and even if it increases risks for morbidity and mortality."

Simply, the athlete, his team and his agent should be careful of Ceremonial Technique.[4]

We should be more demanding and stick to the basic premise that results are what matter. When looking at a new anti-inflammatory drug e.g., we should remember that its impact on erythrocyte sedimentation rates is secondary to more basic questions such as: Did it resolve clinical symptomatology? Did it create a toxic liver reaction? Did it deplete the metabolic enzyme pool? Did it cause destruction of normal tissues? Did it get rid of the pain? Does its repeated use cause shortening of an athlete's career? What post-career affects does it leave?

Cortisone injections have been a treatment modality in medicine because it "allows" the athlete instant pain relief. When looking at a cortisone injection in the form of a spinal block for a lumbar disc injury, the New England Journal of Medicine[5] indicates that these injections remained "an expensive treatment of unproven efficacy" until the late 1980s. Developers claimed good success rates but no controlled trials were done. Now it has been shown by trials in Europe (1989) and North America (1991) that these injections " were to be of no significant value" and that "too much research on back problems consists of case series that serve the entrepreneurial purpose of legitimizing expensive new forms of technology, rehabilitative centers or surgical programs of uncertain effectiveness."

An example of the Tomato Effect is the opinion that the medical treatment of painful spinal disorders via rest, massage, heat, diathermy[6] and orthopedic supports is the most "effective" recommendations, and therefore, alternative treatment such as spinal adjustments and soft tissue manipulation must be experimental, fraudulent or harmful. The overwhelming majority of professional sports franchises chose to have the athlete with a spinal or a disc injury sleep in the training room while the rest of the team practices, rather than explore every treatment strategy possible.

Indeed to the opposite, there is NO LACK of evidence supporting the alternative treatment of painful, spinal syndromes. There are a number of controlled clinical trials directly comparing the alternative medical treatment of spinal adjustments with such approaches as bed rest, heat massage, diathermy, and orthopedic supports. These studies conclude the superior treatment of choice in reducing pain and restoring the patient to regular activities is spinal adjustments.

Parker and Tupling[7] reported that 82% of the patients seen by chiropractors had been treated unsuccessfully by conventional methods, then were treated with spinal adjustments. Three out of four were sufficiently relieved, and satisfied with their improvement to seek chiropractic care again.

Edwards[8] compared massage, exercises, and heat with spinal adjustments for the treatment of various low back pain disorders. The patients with low back pain and no radiation responded 83% of the time in both the physical group and the spinal adjustment group. Those patients receiving spinal adjustment experienced the same level of improvement, in 50% less treatment time. For patients with low back pain with radiation into either buttocks, thigh, or calf, spinal adjustment was able to improve a greater percentage of patients while still requiring less treatment time.

Farrell[9] conducted a controlled study of treatment via spinal adjustments. He concluded that spinal adjustment offers greater pain relief, shorter duration of symptoms, and increased range of motion compared with patients receiving microwave diathermy and an exercise program.

In a patient population of over 4500 cases experiencing low back pain treated with spinal adjustments, 90% were completely relieved, 5% received at least some benefit, and only 5% received no relief from spinal adjustments.[10]

A 6 year study of 283 patients with low back pain and leg pain treated by spinal adjustment by chiropractors under the supervision of an orthopaedic surgeon[11] were graded in the following manner:

Grade I: Symptom free with no restrictions for work or other activities

Grade II: Mild constant pain or intermittent pain with no restrictions for work or other activities

Grade III: Improved but restricted in their activities by pain

Grade IV: Symptoms not significantly affected by adjustment

The patients in this study did not respond to simple conservative measures, they had all suffered from low back pain for may years, and they were in Grade IV (disabled by pain) at the start of treatment. Kirkaldy-Willis concluded that the best results were obtained in patients with dysfunction due to a posterior joint or sacroiliac syndrome or a combination of these:

- Some patients with Instability were improved
- 50% of patients with lateral entrapment were markedly improved and avoided surgery.
- 36% of patients with central stenosis (not fit for surgery) were significantly improved.
- No patient in this series was made worse by adjustment.

The message from the outcomes of a recently released research study by the United States Department of Health and Human Services, Agency for Health Care Policy & Research, reveals that chiropractic manipulation plays a key role in the treatment of low back pain by offering safe and effective relief for low back problems without drugs or surgery. This study reinforces the referral of patients from medical physicians to doctors of chiropractic.

The Ontario Ministry of Health[12] funded a study to determine how to contain and reduce health care costs. Some of their findings include:

- spinal manipulation applied by chiropractors is shown to be more effective than alternative treatment for low back pain. Many medical therapies are of questionable validity or are clearly inadequate.
- chiropractic manipulation is safer than medical management of low back pain.
- literature review revealed much greater need for clinical evidence of the validity of medical management of low back pain, several medical therapies are contra-indicated on the basis of existing clinical trials.
- chiropractic management of low back pain is more cost-effective than medical management.
- there would be a highly significant cost savings if more management of low back pain was transferred from physicians to chiropractors - the potential savings would be many hundreds of millions of dollars annually.

The medical literature is bursting with volumes of these types of studies for many, many different conditions. This paper could be filled with research on the superior results of the integrated approach to low back pain only! Inspite of this overwhelming evidence doctors, teams, agents and athletes continue to throw tomatoes.

Realistically, one can conclude that it is not in the athlete's best interest to accept a team doctor's treatment recommendations without investigating the potential detrimental effects of the therapy. A few examples of short term gain at the expense of lifelong permanent residual problems are Bobby Orr of the Boston Bruins, Mike Palmateer of the Toronto Maple Leafs, Jim Otto of the Oakland Raiders, Dick Butkus of the Chicago Bears.

What Does This Have to do with Football?

A vivid example occured in the 1995/96 NFL playoffs in which the Kansas City Chiefs' starting quarterback Steve Bono had a dismal first half. By the 10 minute mark in the fourth quarter the Chiefs were losing 10-7 to the heavy underdog Indianapolis Colts, the starting quarterback has 3 interceptions, the untested rarely used backup Rich Gannon was put into the game to make something happen. Gannon had no time outs with 3:58 remaining on the clock. He needed a field goal to tie or a touchdown to win. He drove the team to the Colts 32 yard line. With 11 seconds to go he put the ball on a wide receivers finger tips. Was it catchable ? This is questionable, but he did move the team under adverse conditions and came close. The field goal kicker missed his 3rd kick of the game. Will the backup get a shot at the starting job in mini camp and at training camp? The evidence points to giving him the opportunity to prove himself. He certainly deserves it. If the backup or rookie quarterback's performance and statistics continue to improve at every opportunity and, they hold up over a longer period of time, he eventually replaces the veteran.

As the reader can clearly see, the misinformation of the AMA, and the innuendoes of the insurance industry of experimental/fraudulent or unneccessary treatment is viewed differently from the scientific research community. Yet another rotten tomato. The confirmed value of spinal adjustments is indisputable in its effectiveness, compared to conventional methods. This form of alternative integrated medicine has demonstrated the accelerated ability to relieve pain, restore function, and succeed where conventional methods alone have failed.

The examples discussed above, represent a small fraction of the integrated medicine with therapeutically successful treatment that is available today through a qualified Sports Performance Applied Kinesiologist. However, the medical establishment, professional sports teams, athletic trainers and therapists continue to enforce by inference or by intent "The Tomato Effect".

There's Proof in the Pudding

Athletic performance is state management. Professional athletes will control their own destiny or will be forced to control their own destiny. Those players seeking the performance edge are outright defying their team's medical staff, or, are quietly going behind their backs to seek the integrated medicine approach that will return them to the playing field, that will improve their performance and that will extend their career.

Even fewer, some courageous team trainers and doctors will seek out expert alternative care on behalf of their players because they intuitively and scientifically know there are other choices. It is a rare, dedicated and even keen business minded team executive that pursues innovative results producing practitioners who utilize applied kinesiology.

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Using AK Muscle Balancing Techniques to Improve Specific Exercise Movement Patterns (Pilates)

By: John K. Moore, D.C., CCN, CCSP

Abstract

The Pilates Method is a unique style of exercise that was pioneered by Joseph Pilates in the 1940's. He was a man who suffered many chronic ills and used his method of "physical therapy" to regain his health. This method was later adopted by skaters and dancers because of its focus on posture, alignment and breathing. Strengthening the abdomen, lower back and hips (core strength) are the primary focus of this technique. Exercises can be done on a mat or specialized equipment. The equipment consists of a reformer, a bed-like unit with a sliding carriage that has springs for resistance, the trapeze table and a Pilates chair.

Discussion

Having done Pilates for four years, I have noticed a significant difference in my abilities following a treatment with AK. Since this work is so movement specific, I wondered if this might be a good way to evaluate the effects of "AK muscle balancing" on performance. A group of elite athletes who had been doing Pilates for over one year were my test subjects.

Method

The questionnaire on the following page was used with five people during a Pilates class before and after assessment and correction of muscular imbalances/weakness. Both the individual performing the exercise and the instructor were polled as to any perceived improvements. The results will be discussed during the 2000 ICAK Meeting as well as Pilates movements demonstrated.

Conclusion

The exercise routing (Pilates) was improved in most cases by utilizing basic AK procedures to correct any muscular and spinal imbalances that I found. This demonstrated that the more specific the exercise, the better the perceived results will often be by the patient. At the meeting in Palm Springs, I will also discuss how "show and tell" is extremely valuable in these easy to do studies and how it can be a practice builder.

Pupil*	1-Very Difficult	2-Difficult	3-Somewhat Difficult	4-Easy	5-Very Easy
1. Jenny			# (#)	X	(X)
2. Sarah	(#)	(X) # X			
3. Lisa			#	(#)	X (X) (excellent)
4. Steve				# (#)	X (X)
5. Richard	# (#)		X (X)		

Key:

 # Before Tx
X After Tx
(Instructor Evaluation)
Patient Evaluation

*Recorded best scores of three (3) tries

Warm up 1st then perform: Ribcage/Arms Rollup

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Lessons from Our Ancestors, A Historical Review with Pertinent Clinical Applications

Heath Motley, D.C.

To help each patient gain the maximum benefit from each treatment, we as doctors must gain the confidence of our patients, give sound advice and compel our patients to adjust their diet and lifestyle accordingly. Depending on the patient, this can be joyous interaction or similar to pulling teeth. Few patients are as difficult to sway (at least diet wise) as the long-term vegetarians. The information presented here is directed toward treating and informing the stout vegetarian who is somewhat dogmatically positioned in their dietary beliefs. These patients tend to show symptoms of wasting, adrenal stress, ligament laxity, protein deficiency, zinc and vitamin B-12 deficiency, thyroid dysfunction and host of others.

My recommendation not only stems from my professional education and clinical experience but also from dozens of peer reviewed journals. It is my professional duty to inform my patients of the risks associated with a vegetarian diet. The remainder of this paper will list the specific reasons why a vegetarian diet can be detrimental to one's long term health. I will focus on the benefits of cholesterol, the historical overview of man's prehistoric diet and the problems associated with vegetarian diet choices.

The public is currently encouraged and instructed to minimize fat intake, especially from saturated fats (i.e. meat, butter, dairy products, palm and coconut oil). "Low-fat" and "non-fat" food products typically dominate the supermarket shelves; virtually making consumers very fearful of fat. A lack of fat (particularly essential fatty acids) can be instrumental in promoting degenerative diseases. Studies suggest approximately 80% of the U. S. population is deficient in essential fatty acids. The U.S. Department of Agriculture publishes nutritional recommendations represented by the "food pyramid." Fats and oils are placed at the top pyramid — to be consumed in the smallest amount. Grains and pasta are at the bottom of the food pyramid - indicating these should be eaten in the greatest amount. "This may encourage an essential fatty acid (EFA) deficient diet which can lead to an increased incidence(s) of artherosclerotic disease, among other health problems," explains Dr. Edward N. Signed and Dr. Robert H. Lermas, of Boston University Medical Center Hospital. Moreover, many doctors and researchers believe many patients with coronary artery diseases and heart attacks have EFA insufficiencies so prevalent that the deficiency is considered "prediction" of the disease. By following the "pyramid' diet, the common consumer is more likely prone to EFA deficiencies.

Looking back at history, we can see a gradual shift toward declining health in our population. Heart attacks were practically unheard of in the United States a hundred years ago. The first artheroslerosis case was recorded in 1910; the first reported heart attack in 1912. There was no record of Alzheimer's disease. Alzheimer's disease did not even exist. One out of 100,000 people had diabetes and cancer caused 3.4% of all deaths. Today, approximately two-thirds of Americans develop atherosclerosis; half of these cases die from cardiovascular disease. Alzheimer's is one of the top 10 causes of death. One in 20 has some form of diabetes. One in four (28%) develops cancer - 500,000 of these die. Other degenerative conditions, which have exploded in numbers since the turn of the century, include multiple sclerosis, kidney degeneration and others.

Back in the early 1900's, people ate fresh, whole foods including plenty of meat, butter and lard. But they did not ordinarily eat any refined, processed/chemicalized foods or refined/altered oils and fats. All these are commonly used now.[1] Almost all of the oils sold in health food stores are also refined and almost identical to regular supermarket oils.2 Hence, care must be taken to obtain unrefined oils. The refined fatty acids inhibit the function of the thyroid gland, impair intercellular (between the cells) communication and may be toxic to the mitochondria (cellular energy factories).[3]

Cholesterol is an important tissue substance and not a substance to be avoided. Cholesterol is a hormone precursor that rises and falls in the blood stream in proportion to hormone levels more than with dietary factors. Cholesterol contains Prostaglandins 1, 2 and 3, all of which are vital to nearly every body process including blood pressure, childbirth, blood clotting, platelet aggregation, immune responses and stomach secretions as well as hundreds of other interactions. Cholesterol is essential in the production of progesterone as well as adrenal and reproductive hormones. Every cell in the body produces cholesterol and is covered by a double lipid cholesterol membrane. Cholesterol acts as a very powerful antioxidant in that it quenches free radicles. Decreased levels of cholesterol may increase the likelihood of schizophrenia or worsen persistent schizophrenia conditions.[4] Low-fat/high carbohydrate diets have been shown to increase plasma-glucose, insulin, triglycerides and VLDL-TG in diabetic patients.[5]

A 1938 report in the Journal of Biological Chemistry show test animals fed cholesterol produce less cholesterol in their livers. Hunter-gatherer societies of today have exceptionally low serum cholesterol levels despite their high dietary cholesterol intake.[17, 41] The Masai in Kenya consume 600 to 2000 mg of cholesterol daily, yet their serum cholesterol levels remain low, generally ranging from 115 to 145 mg/dl.[17] It has been shown that linoleic acids present in unsaturated fats will reduce cholesterol, but arachidonic acid in beef will reduce it almost twice as fast.[42] Natural fats have cholesterol mobilizers; as well as other nutrients.

In today's culture, people are constantly told to avoid fatty substances, particularly butter. Let's examine the beneficial properties of butter. For instance, butter is very rich in vitamins A, D, E, and F. Butter is considered one of the best sources for vitamin A.[6] Butter contains both Vitamins A and pro-vitamin A (carotene).[7] (Keep in mind that beta-carotene is not a carotenoid complex, and vitamin A acetate or palmitate is not Vitamin A).[8] Butter has enough vitamin D to be considered a preferred source of this vitamin. The natural vitamin D found in one pound of butter is equal to that of 10 quarts of milk.[9] The most potent form of vitamin F (unsaturated fatty acids) is known to be associated with arachidonic acid.[10] Arachidonic acid is also found in butter.[11] Normal patients fed 100 gm of butter (slightly more than a normal day's supply) showed an immediate increase in the unsaturated fatty acid content of the blood.[12] Hart and Cooper[13] found it to be a successful curative agent in the treatment of prostate disorders.

Dr. A. B. Grubb has used butter as a dietary agent in the treatment of psoriasis, xerophthalmia, tuberculosis, dental caries (cavities) and rickets.[14] Substituting vegetable oils for butter has been proven to cause a decalcification of bones; resulting in bone fragility.[15] In addition, swine fed saturated fats (butter, eggs and meat) in increasing proportions, but not unsaturated fatty acids, had normal cholesterol balance. The animals with the highest level of saturates in their diet had the lowest amount of plaguing in their aortas.

In 1992, the magazine Circulation reviewed all the cholesterol work that had been done up until that point. The conclusion they drew revealed that there is no level of cholesterol that puts women at greater risk for heart disease. So even if one has high cholesterol, the risk for heart disease is no greater. For men, there was a slightly greater risk of heart disease with cholesterol levels over 350; this level is only small portion of the population.

Another reason why a vegetarian diet is a risky approach can be supported by evidence from our ancestors' dietary choices. Upon close examination of the human dietary habits since the Paleolithic Era, it is

well documented that animal consumption was the mainstay of the diet for at least 2 million years (100,000 generations). Grains have only been incorporated in the diet in the last 10,000 years (500 generations). Throughout history, the longest-lived societies on this planet were not raw-food eaters or even vegans.[16] In addition, a dentist, Dr. Weston Price, spent many years researching the dietary lifestyles of the indigenous tribes of the world. He physically visited and documented a large variety of indigenous tribes. When he visited Africa, he discovered some tribes were almost entirely meat-eaters while other tribes were largely vegetarian. The vegetarian tribes had more tooth decay and disease, were less robust, less athletic, and were dominated by the meat eating tribes, even though they were still eating native foods, and were certainly healthier than Europeans, and Americans. He said it was his greatest disappointment that he did not find a healthy tribe that was largely vegetarian. He had hoped he would but he did not.[16]

Many Paleolithic anthropologists reported that there was a universal drop in height, muscularity and even cranial capacity once grains were introduced and became the mainstay in human diets. The size of the human brain has decreased by 11% in the last 35,000 years, 8% of that in the last 10,000 years (Interestingly enough, the absolute brain size is paralleled by the similar decrease in overall body size during the same period). Overall, health dropped significantly.[16, 17, 35, 37] Moreover, because human teeth resist deterioration - they are abundantly found at archaeological sites. The condition of these teeth fossils reflects the health status and lifestyle similar to bony remains. It is clear the Paleolithic people had limited exposure to sugar. Only 2% of fossil teeth from the late Paleolithic show evidence of cavities; and even these are shallow and small. In contrast, about 70% of the teeth in some recent industrialized population (England 1900 for example) have had cavities and these cavities were frequently very large.[17] Fully 99% of our genetic heritage dates from the period before our ancestors became human and over 99% of this remaining 1% dates from before the development of agriculture.[17]

Additional evidence for the need of animal food comes from the International Atherosclerosis project, which looked at 31,000 autopsies in 15 countries. They found vegetarians had just as much atherosclerosis as non-vegetarians did.[18] Many vegetarians often make the claim that apes are vegetarians. Looking back at the first primates (65 to 70 million years ago), we find their diet consist primarily of insects and meat. They also ate foods that were tougher than what we would generally call fruits today.[19] In the wild, primates will eat flesh whenever they can acquire it. The most common prey of apes in the wild is young bushbucks (Tragelaphus scriptus), bushpigs (Potamochoerus porcus), baboons (Papio anubis) and young/adult red colobus monkeys (Colobus badius). Occasionally, chimpanzees may catch a redtail monkey (Cercopithecus ascanius) or a blue monkey (Ceropithecus mitis). The Chimps of Gombe consume a wide array of insects, bird eggs and chicks as well.[20] Many primates actually prefer flesh over any other food source. They are basically “opportunistic” in nature.

Modern hunter-gatherer societies showed animal food consumption ranged from 20-90% of diet, with the average being 50%. The evidence does not support the view that flesh-eating was an exception.[16, 17] The fossil record clearly shows our prehistoric ancestors were omnivorous; they ate both plant and animal foods. They are not vegans, fruitarians, or even vegetarians. All prehistoric diets have been a mixture of plant and animal foods; depending on season and habitat. There are no “strictly” vegetarian hunter-gatherers or purely “carnivorous” hunter-gatherers. (The traditional Inuits diet, for example, being 90-95% flesh). Mixed diets predominate. Arctic Eskimos, Kenyan Kikuzu and Masai, Solomon Islanders, Navajo Indians, Australian Aborigines, Kalahari San (Bushmen), New Guinea Highlanders, and Zairian Pygmies are among the peoples shown to be protected from the signs and symptoms of atherosclerosis even though they are predominately meat eaters.[16, 17] Among people such as the San (Bushmen), the Eskimo, the Australian Aborigines, the Tanzanian Hadza, and many other pre-industrialized groups, blood pressure remains low throughout life.[17]

Vegetarian animals (herbivores) have more than twice the length of digestive tract as humans. Herbivores have two to four stomachs which act like fermentation vats; humans only one. Humans have very few grinding teeth (molars); most are for cutting and tearing; and some herbivores have well developed incisors. Although cooked meat moves slowly through a human, raw meat passes through a human in 10 to 16 hours. Raw food passes through a herbivore in 48 hours.[21] In addition, there are many carnivores that eat a great deal of plant material (i.e. wolves).

It is well known many vegetarians are deficient in vitamin B-12. Let me elaborate further as to the reasons why.

Daily dietary intake of 1 mcg (vitamin B-12) can be expected to sustain average normal adults. To allow for variations, the RDA is set at 2.0 mcg. It is only made by bacteria. Plants and animals do not synthesize vitamin B-12. The very limited amount (usually only traces) of vitamin B-12 in plants comes from the uptake of soil or surface contaminants (bacteria which produce vitamin B-12). Vitamin B-12 is essential for humans and non-human primates.[22] Many captive primate species enter into hypovitaminosis B-12 (deficiency) when maintained on vegetarian diets. Vitamin B-12 is the least readily available vitamin to omnivorous primates. Deficiency diseases have not been identified for any wild primate populations. Studies do show that primates are omnivorous and get adequate vitamin B-12 from insects, grubs, animal flesh, feces, etc....

To a great extent, vitamin B-12 is recycled from liver bile in the digestive system. This is one reason why vitamin B-12 deficiency is rare among vegans, even those who do not use supplements or supplemental foods. Herbert summarizes the recycling below:[23]

> The enterohepatic circulation of vitamin B-12 is very important in vitamin B-12 economy and homeostasis. Non-vegetarians normally eat ~2-6 mcg of vitamins B-12/d and excrete from their liver into the intestine via their bile 5-10 mcg. If they have no gastric, pancreatic, or small bowel dysfunction interfering with reabsorption, their bodies reabsorb ~3-5 mcg of bile vitamin B-12/d. Because of this, an efficient enterohepatic circulation keeps the adult vegan, who eats very little vitamin B-12, from developing B-12 deficiency disease for 20-30 years.
>
> Unlike the vegetarian whose absorption machinery is normal, the person whose absorption machinery is damaged by a defect in gastric secretion, by a defect in pancreatic secretion, or by a defect in the gut that produces intestinal malabsorption will develop vitamin B-12 deficiencies in 1-3 years because these absorption defects block not only absorption of food vitamin B-12, but reabsorption of vitamin B- 12 excreted into the intestinal tract in the bile.

The reduction in stomach acid (promoting vitamin B-12 deficiency) is very common in vegetarians (to various degrees). This reduction of acid is associated with the development of bacterial colonies in the stomach producing analogues of vitamin B-12; which can accelerate or promote vitamin B-12 deficiency.[24]

The loss of gastric acid may also occur in iron deficiency. The iron in plant food is of much lower bioavailability than in animal foods. The common grain-based vegan diet contains antinutrient factors that may inhibit iron absorption (discussed later). Vegetarians, especially vegans, are at higher risk of iron deficiency.[23] Iron deficiency is twice as common in vegetarians as in omnivores.[23] Herbert also mentions that prolonged iron deficiency damages the gastric mucosa and promotes atrophic gastritis and gastric atrophy, including loss of gastric acid and intrinsic factor secretion, and therefore diminished vitamin B-12 absorption. This would cause vitamin B-12 deficiency in twice as many vegetarians as omnivores.[23]

At the American Natural Hygiene Society annual convention in 1979, a total of 83 vegetarian volunteers provided blood samples for vitamin B-12 testing. Each volunteer gave a detailed dietary information sur-

vey asking the individual's consumption of animal foods (including eggs and dairy), and their typical daily diet. The ANHS emphasized raw food at the time and at least some of the subjects in the vegan category presumably were raw/predominantly raw vegans. The results: serum B-12 levels of vegan natural hygienists were below the lower limit of normal range.[25] Dong and Scott stated:

> Among subjects who did not supplement their diets with B-12 or multiple vitamin tablets, 92% of the vegans, 64% of the lacto-vegetarians, 47% of the lacto-ovo-vegetarians and 29% of the semi-vegetarians had serum B-12 levels less than 200 pg/ml [the lower limit of the normal range]. Mean serum B-12 levels of the dietary groups increased with increasing dietary sources of B-12. The data indicates that increasing diversity of animal products consumed increase the serum B-12 level.

Additional studies revealed that diversity of animal products consumed increases serum B-12 level. Note that the normal serum B-12 level is 200-900 pg/ml.[25]

> The standard American diet was found in one study to be significantly higher serum B-12 levels (average 311 pg/ml) than the living-food vegans (average 193 pg/ml). Rauma in a 2 year longitudinal study reported that 57% of the living food vegans had B-12 levels below 200 pg/ml.[26]

Long-term vegans had an average serum B-12 level of 164 pg/ml; the control group (standard western diet) had an average serum B-12 level of 400 pg/ml.[27] Crane indicated that over 80% of those people who have been vegans for 2 or more years are deficient in CBL (cobalamin).[28] This study is noteworthy for two reasons. First, it was very thorough and included tests for levels of homocysteine, methylmalonate, and several other factors involved in vitamin B-12 metabolism. Secondly, it discusses the use of oral vitamin B-12 supplements and recommends vitamin B-12 supplements be chewed, rather than swallowed whole, for best absorption. Some early studies also reported poor results from oral doses of 100-250mcg of cobalamin per day. Oral doses of 300-1000mcg per day have proven effective for treatment of pernicious anemia.[28] This is also mentioned in five relevant studies.[29]

Some vegetarians often claim there is adequate vitamin B-12 in spirulina and other plant foods, but the U. S. Pharmacopoeia's microbial assay method for vitamin B-12 is unreliable.[24, 30] The assay measures total corrinoids - that is, true vitamin B-12 plus analogues (forms of vitamin B-12 not metabolically active in the body) and the analogues have the potential to block the absorption of true B-12 by occupying vitamin B-12 receptor sites.[30] Now the most accurate bacteriological assay for vitamin B-12 is Ochromonas malhamensis, but even it may report values for some analogues.[31] Claims that vitamin B-12 is produced in the intestines may be at best based on insufficient evidence. Currently it is unclear whether the bacteria would produce similar amounts of vitamin B-12 needed under the conditions present in the intestines.[32] Dietary advocates tend to ignore this with ideological interest in minimizing the requirement for vitamin B-12 in the diet. Exactly what contribution vitamin B-12 production and absorption from the intestines makes toward the daily intake of vegans remains to be clarified.[35]

To get the daily requirements of vitamin B-12 from soybeans, one must eat 2.64-4.78 kg or 5.81-10.52 pounds of soybean sprouts per day. For barley kernels, it would take 0.49-1.70 kg or 1.07-3.74 pounds while spinach requires 1.33-3.44 kg or 2.94-7.57 pounds. These amounts are in the range of possibility for a person to eat; though it would be difficult. In addition, the B-12 levels in soil are highly variable, not to mention losses in shipping and processing. There is also some talk about direct coprophagy, "night soil", uses of sludge and raw manure in plants. All of which are not recommended.[34]

Many fruitarian circles believe the cobalt in vitamin B-12 undergoing oxidization causes the vitamin B-12 deficiency from the heavy-metal action (specifically inorganic mercury) of dental fillings (amalgam). This has been disproved by the fact that wild primates (whom have no fillings) become B-12 deficient in captiv-

ity when fed vegetarian diets. Inorganic mercury is chelated by tryptophan. This suggests that high protein diets might provide limited protection from inorganic mercury uptake.[38]

The poor assimilation of B-12 at the sites synthesized by bacteria in the human colon and the fact that plant foods are not reliable sources of B-12 supports our adaptation to animal foods due to long-term consumption.

The American Journal of Clinical Nutrition (Feb.1999) published a study on China which compared the rates of heart disease by countries showing rural Chinese men and women have lower rates of heart disease than American men and women. However, the study does not differentiate between rural and urban Americans. When you look at the urban Chinese, their rates of heart disease is about the same as the average American. In fact, the women are even higher.[37] There are areas in China having some of the highest cancer rates in the world. Liver and stomach cancer are 30 times more common in China than the U.S.[43]

In places like China, Japan or areas where diets are lower in animal fat, there are high rates of gastric cancer. The Oriental diet is "perceived" as a healthy diet. This is a myth. The cancer rate is just the same in Japan as in the U.S. Japanese have as much prostate cancer as Americans. The life span in Japan has increased and infant mortality rates declined since the war, but the Japanese have added more animal fat in their diet. They fry their food in lard, eat eggs, beef, and oily fish. It's not as much fat as we eat, however, they don't use the vegetable oils and hydrogenated fats. They eat primarily natural fat. Westerners should not necessarily adopt the grain- eating habits of the Orientals. The Orientals have a larger pancreas, larger saliva gland, and longer intestines than the Westerners. These traits make the Orientals better suited to a grain-based diet.

We often hear high-protein diet causes bone loss but when we examine the actual studies, these studies used protein powder (low in cholesterol). If meat was used as a protein (whole food), then there is no the bone loss.[18]

Vegetarian men have a slightly greater all-cause death rate than non-vegetarians but vegetarian women have a much higher all-cause death rate than non-vegetarian women. Women just do not do well on vegetarian diets. There is some evidence that children raised exclusively on a vegan diet have slow growth and development.[17] There are many vegetarian groups that are very healthy, like the Hunzas, the people of Soviet Georgia, and many of these groups eat largely dairy products.

The longest living man in history was Old Par in England. He lived to be 153 years old and he worked in the fields until he died. His diet was milk products and grains.

Vitamin A in plant foods is in the form of carotene, and it's really not true vitamin A.[8] Beta carotene is pro vitamin A. The vitamin A you need for health is found in fats, and it comes from splitting the carotene in half. The human body can do the splitting, except for babies and children. They don't have the enzymes. Certain adults cannot get vitamin A from carotene, such as diabetics, those with thyroid problems, and other conditions. These people need animal products. But even those who can convert, to get the minimum daily requirements of Vitamin A from carotene, you would have to eat 2 cups of carrots a day, 1 cup of broccoli or 17 cups of chard.

Zinc deficiency is another culprit known to be detrimental to the health of many vegetarians. Zinc deficiency has a tendency to make people on the vegan-diet "spacey". The mental cloudiness they confuse with religious/spiritual euphoria is a lack of zinc (can be easily tested by zinc tally test). There is zinc in a lot of plant foods, but it is often blocked by phytic acid. {Note: Medical doctors are not getting enough nutritional training in medical school to be able to recognize the fallacies in these arguments}.

The soy products of the Orient are fermented and this is very important. Soy has a lot of phytic acid which blocks minerals and contains potent enzyme inhibitors (trypsin inhibitors) which can cause cancer or interfere with protein digestion. There are other known toxins in soybeans very resistant to denaturing by cooking. Only fermentation gets rid of them. Soy isolates can cause deficiencies in vitamin B, increasing adrenal stress. Some sources from the soy industry say it causes B-12 deficiencies.[18] Another reason to limit soy is zinc. Soy products block zinc. This is well known. The producers of soy baby formula know this. Every time it's been tested, soy has been shown to cause a negative zinc balance. Meat is a wonderful source of zinc. A study was done which compared soy to cows milk as far as mucus formation and they were equal.18 Allergies to soy are just as frequent as they are to milk. (Although we find they may not be true allergies as opposed to too much soy intake). Oats have the most phytate of all the grains. Rice may be much lower down. In legumes, soy is at the top. Rice cakes are horrible and I don't recommend them. The rice is puffed at a very high temperature. When they fed puffed wheat to rats they died in a week, much quicker than those did that got nothing at all. Raw cabbage could cause a thyroid problem. Spinach and similar vegetables have oxalic acid, which can cause a lot of irritation (i.e. arthritis, gout). Potatoes, like soy, have a growth inhibitor called hemogluten.

In all, I am critical of fruitarian diets. A fruitarian diet would be very beneficial for healing some ailments in the short run; but it may be dangerous for other maladies. The question is long-term problems of this or any other diet. I am not saying (nor do I tell my patients) that a person cannot be healthy on a vegetarian (animal-free diet), it's just that it is risky. One should strive for a complete balanced diet, not eliminating a food group due food "prejudice." The fact that meat is a natural part of the evolutionary diet does not imply that one must, or even should, eat meat. Circumstances alter cases and we are all our own experiment in diet and lifestyle. A balance is what is required.

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Division II

Critical Review

A Functional Approach to the Treatment of Urinary Incontinence

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Abstract

Many people have trouble holding their urine for one reason or another. This paper mainly deals with those cases of urinary incontinence found in otherwise functional patients who are free from frank pathology. The idea here is to present a way to examine the pelvic diaphragm muscle's involvement in this condition. The usual medical point of view on this subject deals mostly with the integrity of the detrusor muscle, but this paper takes a different course. We will consider the effect of the signals that arise from the detrusor muscle and the muscles of the pelvic diaphragm that reach the spinal cord and ultimately the brain, and the resultant influence of the increased cerebral outflow on detrusor and pelvic muscle function, resulting in greater autonomic control of bladder function as expressed by detrusor integrity. The resulting greater expression of newfound autonomic modulation will benefit the patient through enhanced global neurologic display.

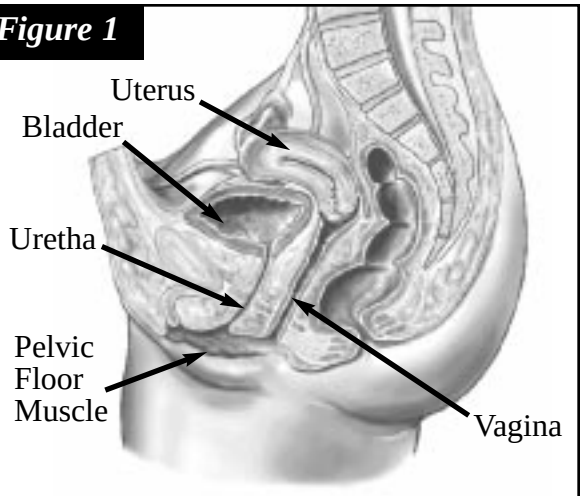
Introduction

Millions of people have a common problem - urinary incontinence (UI). This problem refers to the lack of voluntary control of urination. More than 13 million people in the United States — male and female, young and old - experience UI. It affects up to 30 percent of the community-based elderly, roughly one third of hospitalized elderly patients, and half of institutionalized elderly patients. It is often temporary, and it always results from an underlying medical condition.

The Agency for Health Care Policy and Research (AHCPR) cites studies indicating that one in four women ages 30 to 59 has experienced UI. If this percentage stays constant, the number of cases will rise dramatically as the baby boomers age. Incontinence, which usually can be cured or improved, causes unnecessary social isolation, expense, and secondary medical conditions. AHCPR estimates that the annual costs for caring for people with UI are $11.2 billion in the community and $5.2 billion in nursing homes. Most of this money is spent on management measures, such as pads and diapers, rather than on treatment.

Both men and women of all ages can have bladder control problems. They can become incontinent from strokes, multiple sclerosis, and physical problems associated with old age. Women, however, experience UI two times more often than men. Although the problem is so common, it should by no means be considered a normal consequence of aging. Normal aging does not cause incontinence, but age-related changes in the lower urinary tract can predispose to incontinence, especially in the presence of additional pathological, physiologic, or pharmacological insults usually outside the urinary tract. Age-related changes include reductions in bladder capacity, inability to postpone voiding, and urinary flow rate; maximal urethral closure pressure and urethral length probably decline in women.

Figure 1



Cross section of female pelvic anatomy

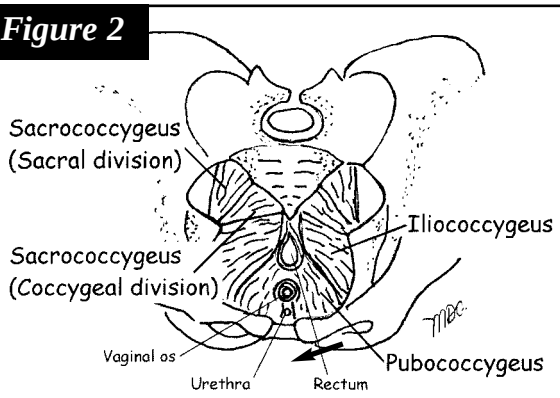
(*Figure 1*) Postvoiding residual volume may increase, but it does not exceed 50 to 100 ml. The prevalence of uninhibited detrusor contractions probably increases, although little comparative data in younger persons are available. Age-related increases in prostatic size develop in men, and both men and women older than 70 years may experience one to two episodes of nocturia that may be age-related.

Brief Anatomy

The pelvic diaphragm (*Figure 2*) is composed of the Levator ani (Ileococcygeus and Pubococcygeus) and Coccygeus muscles together with the fasciae covering their internal and external surfaces. The expanse of the pelvic cavity has been compared to that of a hammock. It is the most caudal portion of the body wall, closing the abdominopelvic cavity, restraining the abdominal contents, and giving support to the pelvic viscera. It is pierced by the anal canal, the urethra, and vagina, and is reinforced in the perineum by the special muscles and fasciae associated with these structures. The pelvic diaphragm and the structures of the perineum are intimately associated both structurally and functionally and an accurate knowledge of one cannot be obtained without study of the other.

Most of the bladder control system lies inside the pelvis. (*Figure 3*) The floor of the pelvis is composed of muscles that should be strong and tight, and hold the bladder in its proper place. The urethral sphincter muscles keep the urethra closed by squeezing like tight rubber bands. Urine stays inside the body when the pelvic floor and sphincter muscles are tight and the bladder is relaxed.

Figure 2



The Pelvic Diaphragm

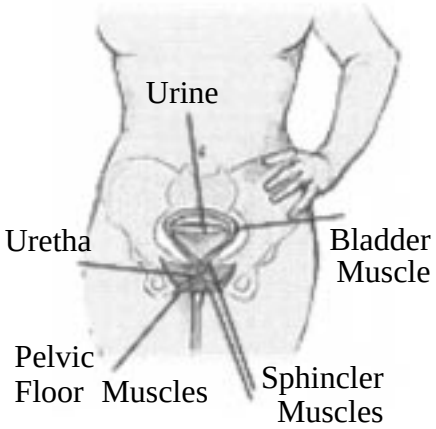
There are four autonomic systems working inside the pelvis. They are the:

- Bladder control system (bladder, urethra and sphincter)
- Female system (uterus and vagina)
- Digestive system (rectum and anus)
- Nervous system (nerves and muscles)

The Bladder Control System

Three sets of pelvic floor muscles support the bladder and control urine. (*Figure 4*) One set is the bladder muscle itself. If these muscles weaken, the bladder can move downward, pushing slightly out of the bottom of the pelvis toward the vagina. The second set of muscles is the sphincter muscles that open and close the urethra. If they become weak, urine can leak into the urethra during moments of physical stress causing "stress incontinence."

Figure 3



Female bladder control system

The third set is the muscles at the bottom of the pelvic bowl. They are called the pelvic floor muscles themselves, and they support the uterus, rectum, and bladder.

Sometimes pelvic muscles get stretched and weak causing organs in the pelvic bowl to sag. Especially in women, this leads to trouble holding their urine. Fortunately, strengthening the pelvic muscles can often make them strong again.

Bladder control means you urinate only when you want to. For good bladder control, all parts of the system must work together:

- Pelvic muscles must hold up the bladder and urethra
- Sphincter muscles must open and shut the urethra
- Nerves must control the muscles of the bladder and pelvic floor.

Stress incontinence can worsen during the week before a woman's menses. At that time, lowered estrogen levels might lead to lower muscular pressure around the urethra, increasing chances of leakage.

Stress incontinence may also occur when women are pregnant or after childbirth as a result of stretching and weakening of the pelvic floor muscles. These problems are common during pregnancy and may even show up months to years after childbirth.

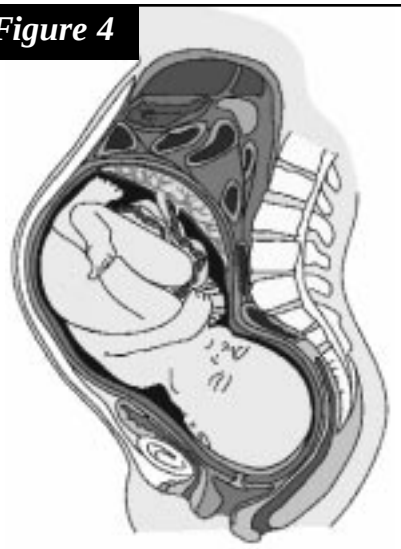
The incidence of stress incontinence increases following menopause because of the same muscle weakness. They weaken because they no longer get female hormones like estrogen, which keeps the lining of the bladder and the urethra plump and healthy.

The new onset or exacerbation of incontinence may occur when either an acute illness or iatrogenic intervention aggravates the underlying age-related changes. This "transient incontinence" is usually temporary. It can be triggered by medications, urinary tract infections, mental impairment, restricted mobility, and stool impaction (severe constipation), which can push against the urinary tract and obstruct outflow. Medical professionals describe such a bladder as "unstable," "spastic," or "overactive."

Reversal or treatment of the factors precipitating incontinence may be sufficient to restore continence without correcting any underlying urologic abnormality, if one is present. The majority of patients who first become incontinent during hospitalization will be cured with this approach. UI that cannot be explained by one of these transient causes may be the result of abnormalities in the bladder, the bladder outlet, or both and is termed "established incontinence."

Especially in the elderly, more than one type of incontinence may be present. Four basic types of malfunction occur: (1) the bladder contracts when it should not (detrusor over activity; the most common cause of geriatric incontinence), (2) the bladder fails to contract appropriately (detrusor under activity; the least common cause of incontinence in older patients), (3) bladder outlet resistance is high when it should be low (obstruction; the second most common cause of incontinence in older men), or (4) bladder outlet resistance is low when it should be high (outlet incompetence; which is secondary to pelvic muscle laxity, and is the second most common cause of incontinence in older women.

Figure 4



Cross-section of a pregnant woman

The Female System

There are several reasons for the increased prevalence of UI in women, like the effects of pregnancy and childbirth, menopause, and the structure of the female urinary tract. Some younger women find they cannot hold their urine after having a baby. Others have problems when they stop having periods.

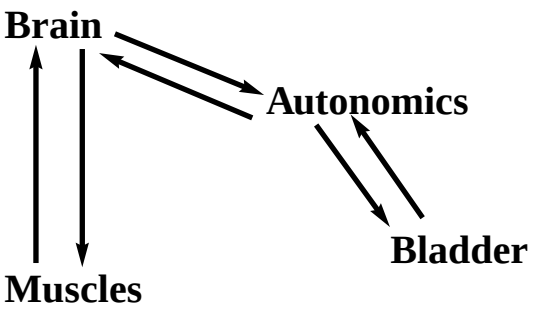
The added weight and pressure of pregnancy can weaken the muscles of the pelvic floor. Other aspects of pregnancy and childbirth can also cause problems:

- Changed position of the bladder and urethra
- Vaginal delivery
- Episiotomy (the cut in the muscle that makes it easier for the baby to come out)
- Damage to bladder control nerves.

The Digestive System

The autonomic difficulties involved with UI can also include the digestive system in general through their connections with the intermediolateral cell column. However, because of their further connection to the muscles of the pelvic diaphragm, any weakness there should be considered in cases of rectal incontinence.

Figure 5



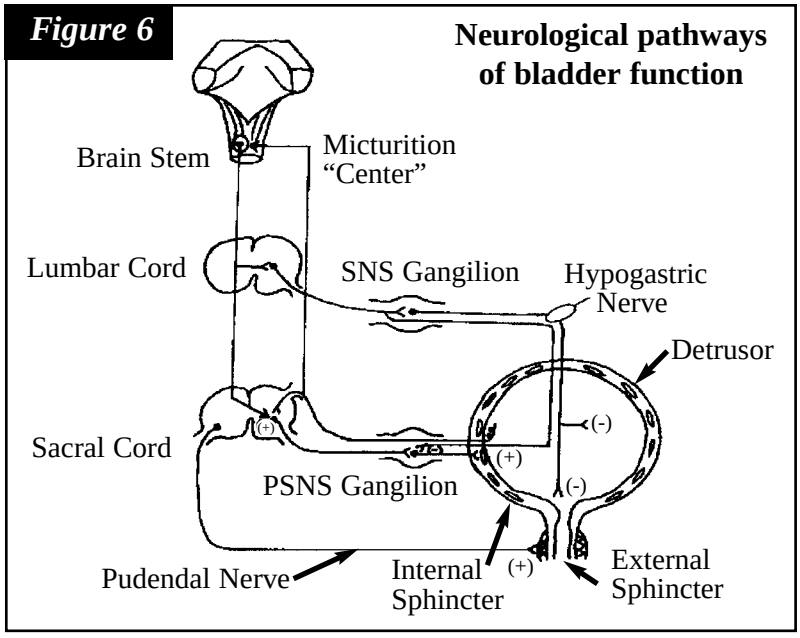
Schematic of neurological pathways for bladder function

The Nervous System

All the above systems send afferent input to and receive efferent signals from the brain. This is important when it comes to treatment of UI.

During urination, muscles in the wall of the bladder contract, forcing urine out of the bladder and into the urethra. At the same time, sphincter muscles surrounding the urethra relax, letting urine pass out of the body. Incontinence will occur if the bladder muscles suddenly contract or muscles surrounding the urethra suddenly relax.

Control of the musculature of the urinary bladder is predominantly a parasympathetic function, which although purely reflexive in infants, is brought under voluntary regulation in normal adults. The preganglionic fibers of the parasympathetic nerves to the bladder (*Figure 6*) have their cell bodies in the intermediate region of the gray matter of sacral cord (S2-4). They enter the pelvic splanchnic nerves and terminate on ganglia that are located in the wall of the bladder, and then on to the detrusor muscle which guards the internal orifice of the urethra. Stimulation of the parasympathetic nerves of the bladder contracts the detrusor, relaxes the internal sphincter, and empties the bladder.



The sympathetic nerves originate in the lumbar region (T12-L2) and extend to the inferior mesenteric ganglia where they synapse and become known as the hypogastric nerve. This post-synaptic nerve innervates the bladder smooth muscle in the body and the base and also the proximal urethra (intrinsic sphincter). The precise function of the sympathetic stimuli is not known. However, it is thought that the sympathetic nervous system acts primarily to facilitate storage of urine by 3 mechanisms: 1) stimulation of certain receptors in the bladder body resulting in increased accommodation which causes filling/storage of urine, 2) increased outlet resistance that occurs through stimulation of certain other receptors of bladder and base and proximal urethra causing retention of urine, and 3) inhibition of bladder contractility via a blocking effect on parasympathetic nervous system transmission which is thought to be mediated by a different type of receptors. This latter effect is activated by afferent impulses in the pelvic nerves (signaling bladder filling), which impinges upon and influences parasympathetic outflow directly (spinal reflex) and indirectly (supraspinal reflex).

As the bladder fills, many micturition contractions begin to appear. These are the result of a stretch reflex initiated by stretch receptors in the bladder wall, especially by the receptors in the bladder neck when this neck begins to fill with urine at the higher bladder pressures. Sensory signals are conducted to the sacral segments of the cord through the pelvic nerves and then back again to the bladder through the parasympathetic fibers in these same nerves.

Once a micturition reflex begins, it is "self-regenerative". That is, initial contraction of the bladder further activates the receptors to cause still further increase in afferent impulses from the bladder, which causes further increase in reflex contraction of the bladder, the cycle thus repeating itself again and again until the bladder has reached a strong degree of contraction. Then, after a few seconds to more than a minute, the reflex begins to fatigue, and the regenerative cycle of the micturition reflex ceases, allowing rapid reduction in bladder contraction. In other words, the micturition reflex is a single complete cycle of (1) progressive and rapid increase in pressure, (2) a period of sustained pressure, and (3) return of the pressure to the basal tonic pressure of the bladder. Once a micturition reflex has occurred and has not succeeded in emptying the bladder, the nervous elements of this reflex usually remain in an inhibited state of at least a few minutes to sometimes as long as an hour or more before another micturition reflex occurs. However, as the bladder becomes more and more filled, micturition reflexes occur more and more often and more and more powerfully.

When the bladder is full, nerves in the bladder signal the brain. That is when you get the urge to urinate. Once the person reaches the toilet, their brain sends a message down to the sphincter and pelvic floor muscles. It tells them to relax. The brain signal also tells the bladder muscles to tighten up. That squeezes urine out of the bladder.

The micturition reflex is a completely automatic cord reflex, but it can be inhibited or facilitated by centers in the brain. These include strong facilitatory and inhibitory centers in the brain stem, probably located in the pons, and several centers located in the cerebral cortex that are mainly inhibitory but can at times become excitatory.

Normally, the micturition reflex is the basic cause of micturition, but higher neuraxial centers normally exert final control of micturition by:

1) The higher centers keep the micturition reflex partially inhibited all the time except when it is desired to micturate.

2) The higher centers prevent micturition, even if a micturition reflex occurs, by continual tonic contraction of the external bladder sphincter until a convenient time presents itself.

3) When the time to urinate arrives, the cortical centers can (a) facilitate the sacral micturition centers to help initiate the micturition reflex, and (b) inhibit the external urethral sphincter so that urination can occur. However, even more important, voluntary urination is usually initiated in the following way: First, the person contracts their abdominal muscles, which increases the pressure of the urine in the bladder. At the same time, the pelvic floor muscles are relaxed, which lengthens the bladder neck and allows extra urine to enter the neck under pressure, thus stretching its walls. This then excites the stretch receptors, which excites the micturition reflex and simultaneously inhibits the external urethral sphincter. Ordinarily all the urine will then be emptied, with rarely more than 5 to 10 milliliters left in the bladder.

Voluntary suppression of urination is dependent on fibers that descend in the pyramidal tracts from the cortex of the paracentral lobules of the cerebrum. It is generally believed that these fibers exert an inhibitory effect on the detrusor reflex. The sensation of increased bladder tension and the desire to void are conveyed by sensory impulses in the afferent fibers of the pelvic nerves and ascending tracts of the spinal cord.

Lesions of the dorsal roots of sacral nerves, or of the posterior funiculi, which interrupt afferent reflex fibers produce an atonic bladder. The bladder wall is flaccid; its capacity greatly increased. Sensations of fullness of the bladder are entirely lost. As the bladder becomes distended there is incontinence and dribbling. Voluntary emptying is still possible, but it is incomplete and some urine is left in the bladder.

As long as the lower lumbar and sacral cord remains intact, any lesions involving the corticospinal pathways or even the motor portion of the cortex of the paracentral lobule produce a spastic paralysis of the bladder and rectum. The bladder will be automatic, that is, will empty automatically by reflex action when it becomes filled to a certain level. Also, stimulating the perineum or its vicinity often produces a reflex contraction of the bladder. Voluntary control is no longer possible.

Diagnosis

A clinical evaluation that includes a history of the incontinence, a targeted physical examination, and a stepwise laboratory investigation can identify factors that precipitate incontinence.

There are lots of medical diagnostic procedures for UI. Some are listed here:

- Stress test-Have the patient relax, then cough vigorously as the doctor watches for loss of urine.
- Urinalysis—Urine is tested for evidence of infection, urinary stones, or other contributing causes.
- Blood tests—Blood is taken, sent to a laboratory, and examined for substances related to causes of incontinence.
- Ultrasound—Sound waves are used to "see" the kidneys, ureters, bladder, and urethra.
- Cystoscopy—A thin tube with a tiny camera is inserted in the urethra and used to see the urethra and bladder.
- Urodynamics—Various techniques measure pressure in the bladder and the flow of urine.

The patient could also be asked to keep a daily diary of when they void for a day or more, up to a week. This diary should note the times of urination and the amounts of urine produce.

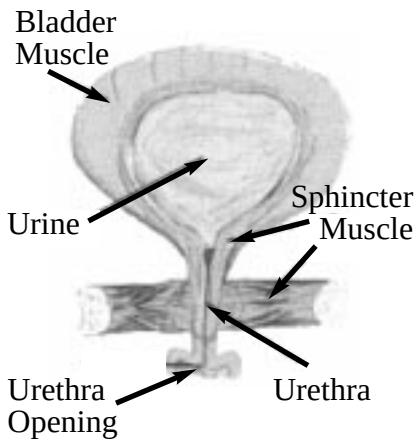
Treatment

Medical Treatment

Good bladder control takes teamwork from many organs, muscles, and nerves in the body. (*Figure 7*) Most bladder control problems happen when muscles are weak or too active. Problems may also happen when nerve signals don't work properly.

There are several types of "Do-it-yourself" treatments, like pelvic muscle exercises (or Kegel exercises) bladder training, weight loss, and watching the consumption of specific food and drink (like coffee, tea, cola, or chocolate and alcohol).

Figure 7



Cross-section pelvic diaphragm and urethra

In many patients, incontinence can be cured, and in most patients, an appropriate treatment program can substantially improve the condition.

The first step toward relief is to see a doctor who is well acquainted with the functional considerations of UI. Gynecologists and obstetricians specialize in the woman's reproductive tract and childbirth. An urogynecologist focuses on urological problems in women. Family practitioners and internists see patients for all kinds of complaints. Any of these doctors may be able to help you. But primarily, doctors of chiropractic also fit right in nicely as an important part of this health care team.

Certain medical treatments have also been shown to be helpful, from electrical stimulation and biofeedback, medical devices like the pessary, urethral inserts, and urine seals to help control

bladder leakage, to medicines and surgery to change the position of the bladder and urethra. There are even several dryness aids like pads or diapers, bedside urinal, health care assistance, and residential renovations. None of these, however, correct the problem.

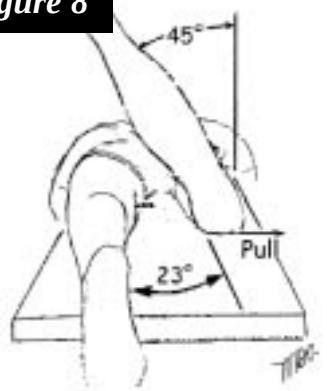
Applied Kinesiology Treatment

Testing the muscles of the pelvic diaphragm is only one aspect of proper care of UI. There are several other considerations. They might range from subluxations and fixations to nutritional, emotional or even reflexive considerations.

Probably the most common direction of evaluation and treatment of UI from an applied kinesiological perspective is to check the muscles of the shin and ankle. The Peroneal (the longus, brevis and tertius) muscles, and the Anterior tibialis muscles are all related to the bladder.

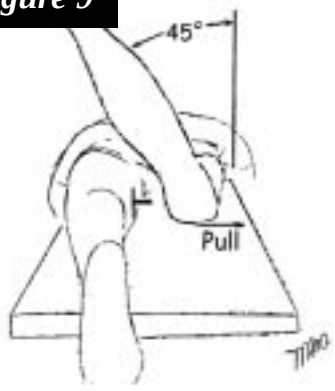
When this author finds any combination of bilateral inhibition of these muscles, my experience leads me to consider a fixation at the cervicodorsal spine. This makes neurological sense to me because of its relationship to tonic neck receptor function and normal gait mechanisms, but it is not the purpose of this paper to go into these processes right now. However, their involvement in the treatment of UI is closely related to the subject at hand. Producing facilitation of these muscles at optimal times is primary to recreating appropriate stability of not only that area they support, but also to the organs these muscles represent.

Figure 8



Coccygeus
(Sacral division)

Figure 9



Coccygeus
(Coccygeal Division)

Local muscles of the pelvic diaphragm — the Levator ani (Iliococcygeus and Pubococcygeus) and Coccygeal muscles — can be tested and treated by applied kinesiological means. The following is a brief description of the muscle tests and their associated nutrition, reflex, structural and acupuncture points according to Alan Beardall, D.C.

Coccygeus (Sacral division; 740) (*Figure 8*)

O: Ileum & Pancreas Duct System N: Cataplex C NV: Zygomatic bone at level of inferior portion of orbit NL: 6th intercostals space, parascapular area VL: T1R MM: S3 Cr: Palatine MAP: Liv 5 (posterior medial aspect of tibia) F: 1st Metatarsal (Beardall, Vol 2, pg 16)

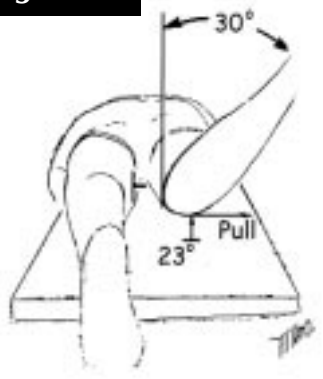
Coccygeus (Coccyx division; 742) (*Figure 9*)

O: Stomach (Pyloric Canal) & Thymus N: Ferrofood; Saw Palmetto NV: Frontal bone (orbit of eye at 10 o'clock on left at frontozygomatic suture) NL: R 9th intercostals space, just lateral to transverse processes MAP: LI10 (lateral posterior elbow just below head of radius) VL: C4R MM: S2 Cr: Maxillary
A-P F: 3d cuneiform, 3d metatarsal (Beardall, Vol 2, pg 16)

Pubococcygeus (744) (*Figure 10*)

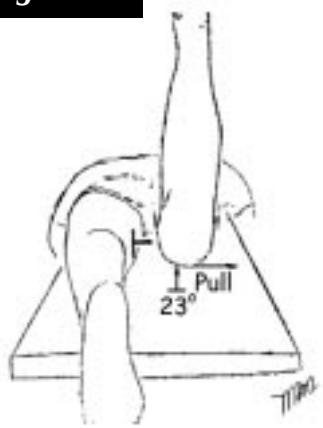
O: Lungs & Heart N: Okra-Pepsin NV: Sagittal suture (halfway between anterior and posterior fontanel NL: GV 11.5 (between T4-T5 spinous process) MAP: GV26 (midway between lips and nose) VL: L5R MM: S2 Cr: Occipital torque F: Calcaneus (Beardall, Vol 2, pg 16)

Figure 10



Pubococcygeus

Figure 11



Ileococcygeus

Ileococcygeus (746) (*Figure 11*)

O: Thyroid & Prostate or Uterus N: Hy-C (Solgar) NV: Occiput (halfway between posterior fontanel and EOP) NL: R 3d intercostals space just lateral to spine) MAP: GB41 (foot between 4th and 5th metatarsal VL: L2R MM: S2 Cr: Lacrimal F: 2d proximal phalanx (Beardall, Vol 2, pg 16)

[NOTE: O=Organ; N=Nutrition; NV=Neurovascular; NL=Neurolymphatic; MAP= Muscle Acupuncture Point; VL=Vertebral Level; MM=Myomere Level; F=Foot.]

Conclusion

From an applied kinesiological perspective, the treatment of UI should be directed toward re-establishing the optimal proprioceptive signal that comes from the muscles of the pelvic diaphragm. A properly functioning muscle maintains its optimal distance between its origin and its insertion. This orientation is essential for the production of optimal signal. Otherwise, the muscles become deafferentated leading to structural and functional compromise and eventual pathology.

While neurological expression much of the time is “by the book”, not necessarily is the cause of that expression from person-to-person by the same book. The display of a condition’s signs and symptoms does not point to the same cause every time, and treating patients with the same signs and symptoms in same ways can be harmful to the patient. The point is that a person may have the display of UI without needing the traditional medical intervention for that condition, some of which have been described above. More probably they need a reintegration of their functional neurological display in order to reduce their manifest pathology to the greatest degree.

Urinary incontinence may be the result of practical inhibition of neuromuscular proprioception resulting in the display of functional pathology absent frank pathological processes. Treatment, in these cases, should be directed toward facilitation of the pelvic muscles through increased stability of their reflex control systems, and the provision and availability of the essential components necessary for their repair.

Once re-integrated, the neurological reflex display will become more normal enhancing the functional capacity of the entire nervous system.

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The Temporomandibular Joint Revisited

Hans W. Boehnke, D.C., DIBAK

Abstract

Most of us are all familiar with the standard diagnostic work up that is taught in the textbooks of Walther and Leaf.[1, 2, 3] In this paper, I would like to quickly review those, and add some other diagnostic factors that I have found very useful when faced with cases that are more difficult to analyze, not showing the usual stomatognathic findings.

Introduction

The standard approach to the temporomandibular joint that I use is a combination of David Walther's and David Leaf's methods and is as follows:

Visual examination

- Look for postural and facial asymmetries.
- Use standard Applied Kinesiology techniques to level the head and shoulders.
- Correct any cranial faults found.

Palpation

Palpate for muscle hyper and hypo tonicity as well as referred pain from trigger points, dural tension points etc.

Therapy localization

I usually use the high gain therapy localization[8] approximating the little finger and thumb and using the index and second finger for the therapy localization.

This is done in the rest position and then with the following additions:

Open the jaw slightly

- *This activates the inferior division of the External Pterygoid.*

Open the jaw fully

- *This activates the inferior division of the External Pterygoid and the anterior belly of the Digastric and possibly the Geniohyoid.*

Close the jaw with gentle occlusion

- *This, if a positive therapy localization ensues, will indicate a possible prematurity with malocclusion or a neurologic tooth or both.*

Bite on the right side

- *This activates the closing muscles on the right side, the Temporalis, the Masseter the Internal Pterygoid and the superior division of the External Pterygoid.*

Bite on the left side

- *This activates the closing muscles on the left, the Temporalis, the Masseter the Internal Pterygoid and the superior division of the External Pterygoid.*

Bite with the incisors

- *This activates the anterior fibers of the Temporalis and the superficial fibers of the Masseter.*

Bite with the molars

- *This activates the posterior fibers of the Temporalis and the deep fibers of the Masseter.*

Protrude the jaw

- *This activates the superficial fibers of the Masseter, Internal Pterygoid and inferior division of the External Pterygoid.*

Retrude the jaw

- *This activates posterior fibers of the Temporalis with assistance from both the anterior and posterior Digastric as well as the Geniohyoid and Stylohyoid.*

Laterally deviate the jaw to the left

- *This activates the posterior fibers of the left Temporalis as well as the right Internal Pterygoid.*

Laterally deviate the jaw to the right

- *This activates the posterior fibers of the right Temporalis as well as the left Internal Pterygoid.*

Swallow (Deglutition)

- *This activates the hyoid muscle group: the Omohyoid, Sternohyoid, Thyrohyoid, Stylohyoid, Geniohyoid, Mylohyoid, and both bellies of the Digastric, as well as the muscles of the tongue: the Genio-glossus, Hyo-glossus, Chondro-glossus, Stylo-glossus, and Palato-glossus.*
- *It also activates the muscles of the pharynx: the Inferior constrictor, Middle constrictor, Superior constrictor, Stylo-pharyngeus, Palato-pharyngeus, and Salping-pharyngeus.*

UNFORTUNATELY, THERE ARE NO TESTS AT THIS TIME THAT ARE IN THE LITERATURE OF APPLIED KINESIOLOGY FOR THESE MUSCLES OF THE PHARYNX.

Speak

- *This activates the hyoid muscle group: the Omohyoid, Sternohyoid, Thyrohyoid, Stylohyoid, Geniohyoid, Mylohyoid and both bellies of the Digastric. It activates the muscles of the tongue and pharynx listed above.*

- *It also activates the muscles of the larynx. The muscles of the larynx are eight in number with five being muscles of the vocal cords and three connected to the epiglottis. The five vocal cord muscles are: Crico-thyroid, Crico-arytenoideus posticus, Crico-aryteniodeus lateralis, Arytenoideus, and Thyro-arytenoideus*

Reactive muscles

For reactive patterns I have them do as Walther recommends in his text such as wag your jaw side to side and stop to the left, then repeat stopping to the right etc. Then sequential movements as I feel might be involved such as the following:

- Abduction to open
- Abduction to closed
- Open to abduction
- Open to closed
- Protrusion to retrusion
- Retrusion to protrusion

These are examined to see if the indicator muscle is strong after the first movement but not after the second movement.

These previous tests can also be done in the following positions:

- Head and neck in flexion and or extension
- Swallowing or phonation
- Weight bearing in various combinations
- Chewing something such as almonds which challenges the stomatognathic system.

I have over some time observed some other factors that have helped me in treating disorders related to the stomatognathic system. These are given below.

Observations and Methods

Additional observations that I would like to share

Hidden cranial faults

I also like to use tongue depressors as recommended by Dr. Leaf in his video on the temporomandibular joint. However, here I like to add a concept that was originally put forth by Dr. Bob Blaich,[4] to stress the body to find which components are going to break down next. This, Bob Blaich did with speed reading, taking a person to a point of neurological disorganization and then correcting the findings present and testing for improved performance. I use tongue depressors to bring out hidden cranial faults. I do the normal challenges for cranial faults: for instance, external and or internal faults and then, if the challenge is negative but their symptom pattern indicates that there may be an involvement of that cranial fault, I have the patient put a tongue depressor between the left molars and/or right molars and redo the challenge.

Many times I find a hidden fault that needs correcting.

Hidden muscle imbalances related to common lesions

I have also found that at times, you will have patients who show a problem such as a category II posterior ischium which would usually have a related Hamstring inhibition pattern (weakness) on the ipsilateral

side[5, 1] and I would not be able to find the Hamstring weakness. I found that if I had the patient hold a static challenge on the hyoid in various vectors, I would often find the Hamstring would show a weakness in one vector of challenge yet not with another indicator muscle. In this case, I would find which hyoid muscle would be involved and correct it by spindle cell or other activity, and follow it by a second challenge to see that the correction held.

Postural imbalances that are only partially corrected after the usual AK procedures are done

Dr. Leaf in his flow chart manual,[2] states, "When imbalances in the muscles of the hyoid are found, they tend to cause the patient to be unable to maintain perfect structural balance." I therefore check my patients for postural distortions that come back or partially come back after correction. For instance, when I have someone who showed the classical signs of a high pelvis, shoulder and head on the same side, it is an indication of a Gluteus Medius weakness (inhibition) on the involved side. After that is corrected, I find that frequently, although the high pelvis etc., was corrected, that a lateral shift of the pelvis remains.

This I then try to address with the hyoid challenge which in one direction of challenge will only show a weakness (inhibition) of the Gluteus Medius on the same side.

The hyoid muscle responsible for the imbalance is treated and the Gluteus Medius does not weaken again with the hyoid challenge and a better postural balance is established.

Then the patient is asked to exercise the involved muscle as well and to reeducate themselves in front of a mirror in maintaining better postural balance.

I have found this helpful in patients who show foot pronation. You work on the Tibialis Posterior and the dropped navicular, possibly the posterior calcaneous, the shortened Triceps Surae if applicable etc., but when they stand the pronation is still obvious. I could then test the above factors again and not find any associated inhibition or weakness pattern. In these cases I find that the Tibialis Posterior and or Tibialis Anterior will show a weakening (inhibition) if the hyoid is challenged in a certain direction. Correcting the hyoid imbalance will often improve the postural findings of pronation with less tension of the plantar fascia on erect palpation.

This would also fall in line with the principles of the Alexander technique[6] in which Matthias Alexander stated "When I was experimenting with various ways of using myself in an attempt to improve the functioning of my vocal organs, I discovered that a certain use of the head in relation to the neck and of the neck in relation to the torso.....constituted a '*Primary Control*' of the mechanisms as a whole."

Alexander was also able to demonstrate improvement of pronation with using his improved body usage technique which included this area of primary control which he talked about.[5] This technique of balancing the hyoid can help in this regard.

To determine a weak muscle that you cannot test in the usual way

Just as our status statement says that Applied Kinesiology procedures are to be used as an enhancement of standard diagnosis, I like to have more than one finding to make a diagnosis. I therefore like to use my palpatory skills as well as muscle testing etc., to determine a problem. Most muscles that are weak (inhibited) will exhibit decreased tone to palpation especially when compared to the same muscle on the opposite side and or the synergists of the involved muscle. Some muscle weaknesses are not obvious and do not therapy localize in the normal manner as we are taught to do as for the Sacrospinalis. I found that they will therapy localize easier when the muscle is contracted.

- *I thought that this was an original observation, but was reminded that at the ICAK-USA meeting in New Orleans, John Corneal had demonstrated this and presented a paper including this form of therapy localization.*[13]

Therefore, my finding of this therapy localization done with the involved muscle being contracted is a confirmation of Dr.Corneal's original observation. On review, if I find that on palpation and observation that the hyoid is inferior on the right, it would indicate either one or more hypertonic infrahyoid muscles on the right or weakness of one or more of the suprahyoid muscles on the right. I therefore palpate for lack of tone in one of the suprahyoid muscles, which would be one indicator of possible weakness (inhibition). Then I have the patient therapy localize the involved muscle. If positive, I know to treat the muscle, but if the therapy localization was not positive, I find that simultaneous therapy localization and contraction of the involved muscle will often show a weakening (inhibition) of the indicator muscle. When this finding is present, you know to apply treatment that will help facilitate (strengthen) the muscle involved.

Much of the information in our literature dealing with the Stomatognathic system deals with toning down the hypertonic muscles. This goes against the primary tenet of Applied Kinesiology, which is to address the weak (inhibited) muscle first, and that in most cases the hypertonic muscle which is often the symptomatic muscle will be relieved and normalized.[7, 8, 11]

In our previous treatments we were falling into the same trap that most professions prior to George Goodheart's observations did with muscles, that is only addressing the hypertonic muscle. A case in point is a book by Goldman and McCullough,[12] which reports that "TMJ is basically a muscle-spasm problem" and report using splints, dry needling and equilibration with the main purpose of relieving spastic External Pterygoid muscles and thereby a "Tooth Gearing Discrepancy."[12] The above procedures, palpation for hypotonicity, and combined therapy localization with muscle contraction, helps us to determine which muscles are not functioning adequately and need tonification by the various methods available to the Applied Kinesiology practitioner.

Please also keep in mind that occasionally, the apparently hypertonic muscle needs tonification, as Goodheart teaches in relation to "Repeated Muscle Activation Patient Induced, RMAPI."[8] In this case a muscle does a contraction response to its weakness (inhibition). I have noticed that in a number of cases that, using the technique given in the next section, the spindle cells of the muscle that was palpated as short (appearing hypertonic) responded by spreading the spindle cells apart (which tonifies) rather than together (which reduces tone) as would normally be the case. I suspect that these cases would fall into that category but I have not yet tested that idea.

The main reason for not testing for RMAPI above is that it is difficult to devise a motion to have the patient do 10 times that singles out a hyoid or TMJ related muscle and puts it through its full range of motion. I could possibly devise such a motion, but to teach the patient to do it would likely be difficult and time consuming.

The use of a static challenge on spindle cells

I have found that a static challenge of holding the spindle cells apart or together can be done when a muscle is found that is causing a remote dysfunction by its contraction.

For instance, I have a patient who came in for a severe plantar fascitis and treated him in the usual ways, foot pronation, tarsal tunnel syndrome treatment, flexor hallucis limitus treatment, Triceps Surae lengthening, myogelosis treatment etc., with good results. He was beginning to run some distance again with minor discomfort. I still found that he demonstrated foot pronation in the erect position, but that his Tibialis

Posterior tested strong (facilitated) when supine. I had him stand and palpated his arch and had him clench his teeth together and felt a slight contraction under his plantar fascia and elevation of the arch.

This finding was more consistent when he clenched his teeth together on the ipsilateral side to the side that I was palpating. I had him lie prone and felt the same finding with the clenching action. I then had him assume the supine position and open his mouth wide open, contracting the anterior belly of the Digastric and possibly the Geniohyoid and the Tibialis Posterior muscle I was testing weakened dramatically (*we will discuss this muscle activation more in the next section*). I had him then keep his mouth wide open and therapy localize over the anterior belly of the Digastric on the ipsilateral side and the Tibialis Posterior muscle strengthened dramatically. I then had him use two fingers to pull the spindle cells apart with his mouth wide open while I tested the Tibialis Posterior again and the muscle was strong. I then had him do the same but hold the spindle cells together and the Tibialis Posterior muscle weakened again.

The treatment then was obvious - to pull the spindle cells apart. These findings were present on both sides and treated accordingly. On standing, his plantar fascia on the left was less tense and the arch more well formed - therefore less pronation. The left foot was less problematical. The right planar fascia was less tense but the pronation on the right was only slightly improved. I then had him lie supine again and hold his hyoid in various challenge directions while testing the right Tibialis Posterior. When he held the hyoid superior and to the left and superior, the right Tibialis Posterior weakened again dramatically. I had him maintain the challenge and therapy localize the various right sided infrahyoid muscles until one caused the right Tibialis Posterior to strengthen.

When the muscle was found I had him again hold the spindle cells apart for one test and together for the second test while he did the challenge and found that the Tibialis Posterior required the spindle cells to be pulled apart as in the above treatment for his Digastric. After the treatment, his plantar fascia on the right exhibited less tension, his arch on the right more well formed, and his pronation visibly improved.

The activation of a Stomatognathic muscle to bring out a related hidden weakness (inhibition pattern).

I have found that if a muscle was previously treated and tested intact but still showed some signs of the original weakness, that not only can a hyoid challenge bring out a hidden weakness, but contracting the stomatognathic muscle involved can bring out the hidden weakness as well.

Case # 1 For example, I have a patient who is an older woman with a left hip joint problem, who I treated successfully in the usual ways but after a day of shopping with numerous uses of her car and much walking, her symptoms returned. Erect, I palpated her hip joint and found a tender area at the posterior aspect. The tenderness was reduced when she shifted her weight to the outside of the left foot (supinating the foot). I therefore tested the Tibialis Posterior which appeared intact, but if I had her open her mouth up wide contracting the anterior belly of the Digastric, it weakened dramatically. This finding was confirmed with the previously mentioned hyoid challenge earlier in this paper.

Case # 2 Another case was of a patient with a category II right posterior ischium but the usual Hamstring weakness (inhibition) was not found until I had him hold a static challenge on the hyoid to the left and superior and then it weakened dramatically. Using therapy localization along with the challenge, I determined the problem to be in the Omohyoid muscle. Rather than just treat it, I decided to have the patient contract the Omohyoid which is done by holding the shoulders superior and anterior, a Beardall test.[9] When he contracted the Omohyoid, the right Hamstring weakened dramatically. It was then treated successfully.

Case # 3 A male patient with a chronic right rotator cuff injury that had been according to him successfully treated medically, but still occasionally bothered him a little, showed a weakening of the right Teres Minor muscle on a hyoid challenge superior and to the right. The same weakening response could be shown with contraction of the Omohyoid muscle which needed treatment.

Case # 4 This patient was mentioned in the above section where there was a dysfunctional anterior belly of the Digastric and on activation, caused the remote weakness in the Tibialis Posterior to occur.

Useful information and exercises for the temporomandibular joint.

A. When discussing the temporomandibular joint, Hertling and Kessler[10] focus a lot of attention on the postural distortion referred to as "forward head position FHP" as being a contributing factor to a TMJ problem. Theirs is a very valid point. You can palpate your own Masseter, Temporalis, External and Internal Pterygoids when the head is in a neutral position and again when it is in a forward head position FHP and will find that they contract. This corresponds well with the teachings of Mathias Alexander that were mentioned earlier in this paper. We, in Applied Kinesiology, can influence the forward head posture by testing for and correcting neck extensor weakness by correcting sacral fixations and by using strain and counterstrain and other methods on the muscles of the anterior neck lexors and the hyoid muscles.

B. I have also found that I can reduce hypertonic neck flexors by adjusting the upper ribs superiorly. When this is necessary the palpation findings of muscle hypertonicity will reduce if a static superior challenge is held on the first or second rib. After the rib is adjusted you can check for the need of strain and counterstrain to the intercostal muscles related to the adjusted ribs.

C. If translation begins before 11 mm of opening as measured by the intercisal distance, it is called premature translation as the initial motion should be rotation. According to Hertling & Kessler,[10] normal mandibular opening has been reported to be between 35-50 mm. To complete 40 mm of functional range 25 mm is rotational and 15 mm translation. They feel that if there is a large anterior translation at the beginning of opening when rotation should be occurring it is an indication of excessive joint mobility, which can lead to joint subluxation. They propose the following exercise:

The patient is asked to place his or her tongue on the hard palate as far back as possible. This effects retrusion. While keeping the tongue on the hard palate, the patient opens and closes his or her mouth slowly and rhythmically within pain limits several times in succession practicing mandibular opening with only condylar rotation taking place.

The next step is to have the patient contact the chin and exert resistance as the mouth is opened.

If the suprahyoid muscles demonstrate poor tone, an exercise described by Hertling & Kessler[10] which they indicate came from Shore, is the following:

They teach the patient to use isometric contraction of the suprahyoid muscles in front of a mirror. The patient closes his or her mouth with the teeth in light contact. The patient then tries to retrude the jaw and depress the floor of the mouth without actually moving it. Each day a little more mouth opening is introduced to the exercise working to achieve coordinated muscle action of the mandible.

The patient can eventually guide the movements of opening and closing in front of a mirror correcting for deviation or protrusion thereby increasing the rotation aspect of the mandibular opening and reducing the translation aspect to create a more balanced motion and develop a new engram.

Discussion

The practitioner of Applied Kinesiology is faced with a great number of therapeutic intervention possibilities and subtle variations of the classical findings that can make the use of the techniques perplexing and difficult. Just as earlier discoveries of EID and BID, PiLUS and other procedures have helped take some of the difficulties out of finding hidden problems, it is my hope that the above observations will help to more easily unravel some of the body's more subtle problems.

I would be remiss if I did not add to all of the above, that when you are therapy localizing anywhere and are not finding any weakening of your indicator muscle, where you are pretty sure there should be, then be sure to check that it is not in a hypertonic state. This could be the reason for your not finding a lesion which is there. *If a muscle is in a hypertonic state, then it will not weaken when its spindle cells are pushed together and or its meridians sedation point is stimulated.*

For this observation I have the work of Dr. Gerz to thank[11] as I have found a number of cases that I would not have succeeded in helping unless I brought the indicator muscle back to a normal responsive state.

Conclusions

All practitioners who work diligently to advance the treatment of patients will occasionally observe findings that are novel and helpful in practice. In this paper I share a few of my observations as well as some information from other sources. If any of my above observations have been written about previous to this paper, I have missed them in the literature and apologize to the original author. I did find (with the help of my good friend Kathy Conable), that one of my observations was a rediscovery of a finding of Dr. Corneal and acknowledged it earlier in this paper.

It is my hope that these observations will help practitioners of Applied Kinesiology to better help their patients. Some of these observations should also be useful in demonstrating to patients the complex interactions in the body that can be related to the stomatognathic system. My good friend and mentor Dr. Lance West who taught so much to many of us and who was a master at patient communication, always told me to spend more time demonstrating AK to the patient. He called it "SHOW AND TELL", just like we had to do in primary school. He believed that it helped the mental side of the triad of health and was just as necessary for the patient to get better as the physical treatment.

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P.L.U.S. and Balance

Bill Conder, D.C.

"That individual who, because of personal inclination and initiative, turns to the study of the healing arts with the intention and desire to investigate the issue beyond the rather obsolete teachings of the traditional hypothesis, is indeed in for a very exciting and exhilarating life." Illi.

Abstract

Goodheart's P.LU.S. observation is discussed, explored, and interpreted. Right piriformis muscle inhibition is proposed as the mechanism of right leg lengthening as found in other chiropractic techniques using patient feedback. This piriformis inhibition as observed in P.L.U.S. is proposed as a passive muscle test and an important applied kinesiology and chiropractic evaluation tool.

Introduction

The initials P.L.U.S. stand for the muscles **p**iriformis, **l**atissimus dorsi, **u**pper trapezuis, and **s**ternocleido-mastoideus. The observed phenomenon indicated by this acronym is a pattern of muscular inhibition caused by flexion or extension of the lumbar spine as follows: right piriformis and sternocleidomastoideus, and left latissimus dorsi and upper trapezius. This same pattern of muscle inhibition is found in normal human gait when taking a right step forward.[1]

Goodheart discovered P.L.U.S. after research by Illi[2] that concerned spinal torsion as a biomechanical adaptation to prevent "undue tension upon the spinal nerve radicles and their enveloping dura matrix sheaths" during flexion or extension of the spine.[3] Goodheart, using healthy patients and athletes, found that spinal torsion was possible due to inhibition of the right piriformis muscle which permits the sacrum to rotate around its perpendicular axis, initiating the subsequent torsion-effect of each vertebral segment up the spine and what we know as the P.L.U.S. phenomenon.

Illi found that this torsion-effect was not reproduced by each segment in identical fashion but that vertebral groups participated in this in distincly different ways: the lumbar vertebrae each turn around the axis of the segment below, the thoracic vertebrae undergo a lateral torsional shifting that increases the kyphosis, and the cervical vertebrae glide anteriorly.[4] Illi states specifically that "Vertebral Rotation Concomittant To Spinal Flexion [is] Essential If Undue Traction Upon [the] Spinal Cord Is To Be Avoided."[5] Illi notes also that the integrity of the intervertebral disc is maintained by this torsion and its effect on the fibers of the annulus fibrosus.[6]

a. The piriformis muscle.

The piriformis muscle originates on the anterior surface of the sacrum at the capsule of the sacro-iliac joint, the margin of the greater sciatic foramen, and the sacrotuberous ligament; it inserts into the superior

border of the greater trochanter of the femur. Its action is to externally rotate the thigh and abduct it when the hip is flexed. Nerve supply is provided by L-5, S-1, and S-2 nerve roots via the sacral plexus.

The piriformis can be muscle-tested with the subject standing, seated, and lying supine or prone. In the supine and seated patient, the hip must be flexed for proper testing as compared to the case with the prone patient where the hip is neither extended nor flexed. Please refer to Walther's Synopsis[7] for AK details.

The anatomical relationship of the piriformis muscle in its origin at the sacrum, sacro-iliac joint, and sacrotuberous ligament, and in its insertion at the superior border of the greater trochanter of the femur is noteworthy. From the anterior surface of the sacrum the piriformis muscle passes obliquely anterior and slightly caudad, transitting the greater sciatic foramen, and attaching anteriomedially[8] on the femur's greater trochanter. The sciatic nerve passes out of the pelvis below the piriformis and above and posterior to the obturator and gemelli muscles. Cailliet says that the sciatic nerve passes between the two bellies of the piriformis, and that the muscle inserts posteriorly on the greater trochanter.[9]

The femur appears to hang from the piriformis. The sacral apex seems to act as it were the pointer of a balance scale under the influence of the tension of the bilaterally paired piriformis muscles. The sacrum itself may be thought of as hanging from its dural attachment like a plumb-bob or pendulum. Chiropractors often use a plumb-bob as an evaluation tool in analyzing the standing patient's spine. Both Illi and Goodheart have described the motion of the pelvis in gait as gyroscopic. These metaphors deepen the suggestion that the sacrum and its muscular and meningeal accessories can be considered an organ of balance in motion and stillness.

b. Loss of balance

Properly in the P.L.U.S. phenomenon the right piriformis muscle is inhibited upon flexion or extension of the lumbar spine, and not the left piriformis. In normal gait, the right piriformis is inhibited to permit stepping forward with the right leg and the left piriformis, in its turn, is inhibited to permit stepping forward with the left leg, and so on. In gait, this leg lengthening contributes to stride length as the body sends the leg/foot/heel forward and downward to meet the ground. The alternating or oscillating effect of the gait pattern may be seen as an elaboration of the adaptive spinal torsion reaction to the loss of verticality experienced in flexion or extension of the spine that is loss of balance. Gait, therefore, may be considered an organized, restorative, adaptive response to the loss of vertical balance. In a childhood development view, we may say that gait is the intermediate adaptive response of learning to come into upright balance (that is, gait serves coming into balance in the upright posture and is not an end in itself). As an initiator of this adaptive response in neuromuscular physiology, it is proposed here that piriformis inhibition may also be an indicator of loss of balance in other analog systems of the body-mind.

In general terms, loss of balance, or a stressful challenge or stimulus to the body-mind that may cause a loss of balance literally or analogously or that mimics the body-mind's reaction to loss of balance, will elicit right piriformis muscle inhibition and the right leg lengthening response.

c. The right leg-lengthening response: the Illi - Belli connection.

Properly, the prone subject's right leg will appear to lengthen, upon observation of his/her heels or medial maleoli, in response to the introduction of a stressor to his/her body-mind. (More accurately, the leg itself does not increase in length but, due to a momentary inhibition of the piriformis muscle which results in the muscle's lengthening, the leg transits inferiorly from the hip, sometimes remarkably so.) This stressor may be in the form of an AK rebound challenge to the spine, temporary stimulation of an "active" reflex point, subsequent to extension of the spine,[10] and so on. This response, it is proposed, results from inhibition of the right piriformis muscle and is the equivalent of the inhibition/weakening of an "intact indicator mus-

cle."[11] This right leg-lengthening and the weakening of an indicator muscle are designated here for convention as a "positive" response demonstrating the identification of a positive current that exists properly in the body or of Becker's "current of injury" which is a positive current in a normally negative field caused by injury.[12]

Specific stimuli should cause the positive response, while others should not.[13] In the indicator muscle test, only one of two events can occur: positive ("weak" muscle) or negative ("strong" muscle) response. A false negative, as in the case of the so-called hypertonic or hyperfacilitated muscle, that is one that does not demonstrate autogenic inhibition,[14] is another possibility. In the leg lengthening response, although the same digital neuromuscular functioning abides as in the muscle test, conditions exist in which the left leg may lengthen instead of the right, in which neither leg may respond by lengthening, and in which one or the other is significantly longer and neither responds to specific stimuli. The digital positive-or-negative response is common to both but, in the case of the leg lengthening response, the ability to compare sides of the body that are oppositely polarized gives an added dimension for evaluation. Moreover, the subject-passive nature of the leg lengthening procedure limits variables.

The leg lengthening response is valuable as a tool to validate a muscle test, among other things. In muscle testing we are assured by the "strong" muscle and work to strengthen a "weak" one. In the leg lengthing response we are working with the positive reaction to know that nothing inhibits its occurrence, and to determine what triggers it. A muscle that will not demonstrate autogenic inhibition upon testing will cause right leg lengthening as if the stressed, adaptive state of the tested muscle[15] is unmasked and read correctly through the leg legthening response, suggesting imbalance. Goodheart's pincer palpation[16] of an injured muscle will cause the right leg lengthening response. In this sense, the right leg lengthening response can be used as a a tool to interpret the results of muscle testing.

To fine tune the leg lengthening response, one must correct injuries that will compromise the response, in the same way and for the same reasons that one would prepare a muscle for use as an indicator muscle. Correcting structural imbalances, especially subluxations, improves the right leg lengthening response in the interest of detecting other injuries and injurious substances relative to the whole body-mind. This structural fine tuning in preparation to use the leg lengthening response is basic chiropractic and should be completed unless the prone patient's heels appear fairly even and the right leg lengthens subsequent to his/her extension of the lumbar spine, that is unless the patient responds properly.

The right leg lengthening response can be elicited properly in ways other than by extension or flexion of the lumbar spine. Elicitation of the stretch response in bilaterally paired muscles should always cause the right leg lengthening response (and inhibition of an indicator muscle). Belli has shown the usefulness of the muscle stretch response (a.k.a. deep tendon reflex) in the evaluation of a muscle's functional neurological integration, and in posture and gait, in general. Dr. Belli's procedure is to test a muscle subsequent to applying 2 to 3 pounds of pressure to its musculotendonous junction, whereupon the proper response is inhibition or weakening of the muscle for one contraction only, noting that any other muscle response indicates malfunction.[17] In the right leg lengthening response, the same positive reaction (right leg lengthening in this case) is elicited by applying 2 to 3 pounds of pressure simultaneously to the musculotendonous junction of bilaterally paired muscles. For example, squeezing both Achilles tendons will elicit properly the right leg lengthening response. Apparently, simultaneous stretching of bilaterally paired muscles is a stressor to which the body-mind responds by making it possible to step forward with the right foot in order to recover balance.

d. Tuning the sacral pendulum.

The prone patient's right leg should appear to lengthen upon the doctor's simultaneous squeezing of the patient's Achilles tendons. If this is the case the doctor can proceed to use the right leg lengthening response as he/she would use an indicator muscle test.

If neither leg responds by lengthening upon elicitation of the muscle stretch response as above, one can assume that the right piriformis does not demonstrate autogenic inhibition. Muscle testing of the right piriformis with the patient in flexion, extension, or in right-leg-forward gait will demonstrate its failure of inhibition. Correcting this imbalance by searching the Five Factors may prove fruitful, however this researcher has found that rebound challenge of the 3rd lumbar vertebra will evoke the right leg lengthening response, indicating a need to adjust that segment. In this case, adjustment of 3rd lumbar restores proper function to the right leg lengthening response.[18] One may find, further, the need to strengthen the left latissimus dorsi and upper trapezius by adjusting thoracic 7 or 8 and cervical 2. The posterior latissimus dorsi neurolymphatic reflex also may require stimulation.

If the left leg lengthens upon elicitation of the muscle stretch response as above instead of the right leg the pelvis is considered switched.[19] Rebound challenge of the 4th lumbar usually elicits right leg lengthening and adjustment of that segment often restores the proper response. In addition, adjustment of either first rib head and/or balancing Bladder meridian may be necessary to restore fully the right leg lengthening response.

If either the right or the left leg is significantly longer and neither responds by lengthening upon elicitation of the muscle stretch response as above, a sacroiliac or sacral subluxation, or an SOT Category II may be present. These imbalances must be corrected to restore the proper right leg lengthening response.

Consideration to the quadratus lumborum muscles must be given in all of the above cases.

(Procedures presented here for sacral balancing have not exhausted the possibilities.)

When "local" injury interferes with proper right leg lengthening the specific challenge indicating the specific correction will cause the right leg lengthening response. Once the correction is made and other local stressors to the proper response are relieved, the right leg lengthening is free to respond to stressors that effect "globally" in the body-mind. This is true also of the indicator muscle test.

e. The condition of the right leg.

That the right leg lengthens properly in response to a stressor, and not the left leg, is remarkable and mysterious. Generally, the right side of the body, the anterior or ventral surface of the body, and the extremities are more electro-negative than their left, dorsal, and axial counterparts. This may precondition by neuromuscular activity the right leg/foot/heel forward motion in gait, and concomitant right piriformis inhibition.

We know from research conducted by neuroscientists that an increase in negative current anticipates neuromuscular function - anticipates even the decision in the brain - as if it is necessarily preparatory, in other words, a condition of neuromuscular function.[20] This researcher has proposed that the increase in negative current induces neuron depolarization that causes muscular contraction. It is further proposed that this increase in negative current is a condition of neuron depolarization without which the neuron will not function. The more negatively polarized right leg preconditions flexion of the right hip and inhibition of the right piriformis muscle to restore lost balance. Although right leg lengthening acts through reflex action and not conscious decision, the reflex itself is a built-in condition of gait function.

f. The human dynamo.

Gait can be understood as the means by which human mechanical motion is converted into electromagnetic energy in the manner of a generator or dynamo. The inhibition/facilitation oscillation of the piriformis muscles at the sacrum in gait indicates the periodicity of the semiconductiong perineural system of the lower extremities in the ambient magnetic field of the earth, thus producing electrical energy in-around the body that restores the energetic systems of the body-mind. In this way mild to moderate aerobic exercise that utilizes the cross crawl mechanism, as in walking, makes one feel good even though it uses energy, and ta'i chi chuan, which is a cross-crawl tour de force, energizes.

In gait, the sacrum indicates the "O" of balance toward the ground even as its ala trace horizontal figure eights; the gyroscopic pelvis maintains stability in motion adapting to the forward lean necessary for efficient gait; and the oscillating polarity of the legs in motion generates electromagnetic energy in the body via the yin energy channels. Central to all of this, as both cause and effect, is the inhibition of the right piriformis muscle.

Method

In order to be comfortable and confident with the right leg lengthening indicator the doctor will want to prove to him/herself that the phenomenon is predictable and reproducible. The following procedures are presented in that interest. (Note: It is recommended that a healthy, balanced subject be used for the following procedures as the right leg lengthening response properly appears in the healthy, balanced subject given these stimuli.):

1. Muscle test the prone subject's right piriformis. It should test strong in the clear and weaken when the subject extends the spine into the lumbar region. (Note: Bilateral contraction of the gluteus maximus muscles will inhibit right piriformis inhibition during flexion or extension of the lumbar spine and, therefore, the right leg lengthening response will not occur.)
2. Pinch the insertions of both Achilles tendons at the same time and observe a momentary lengthening of the right leg at the heels or medial maleoli, that is the right leg lengthening response. As per Belli, this is a way to stimulate the muscle stretch response (which unilaterally should cause a weakening of the specific muscle).
3. Stimulate the muscle stretch response by sweeping caudad to cephalad bilaterally over the posterior crus (the gastrocnemius or soleus muscles) with the doctor's hands, and observe the right leg lengthening response.
4. Stimulate the muscle stretch response of all bilaterally paired muscles by sweeping insertion to origin over the bellies of the muscles with the doctor's hands, and observe the right leg lengthening response.
5. Introduce the positive/south pole of a 3 inch by 5 inch diagnostic magnet to within 2 inches of the plantar surface of the subject's right foot and observe the right leg lengthening response.
6. Tap for 4 or 5 percussions the yin jing-well points of the feet and hands and observe the right leg lengthening response. Tap the yang jing-well points and observe no change.
7. If the subject falls asleep, tap for 4 or 5 percussions the yang jing-well points of the feet and hands and observe the right leg lengthening response. Tap the yin jing-well points and observe no change.

These represent proper responses such that any other response indicates imbalance.

Discussion

Goodheart's observation of the piriformis muscle in gait and the P.L.U.S. phenomenon, on the heels of Illi's research, are rich with potential for a comprehensive understanding of body-mind function and for an acceptance of the right leg lengthening response as a valid and useful evaluation tool. Goodheart's observation is presented here in the context of the discovery of Becker and others regarding the electrical nature of the perineural cell system, and Becker's discovery of the "current of injury." Now we have a grammar and syntax with which to read and interpret body-mind language holistically, not merely objectively, quantitatively, or analytically.

Becker's research is useful in understanding neuromuscular function in the right leg lengthening response and in muscle testing. The polarized current in the perineurium establishes the environment and condition necessary for neuron depolarization. Local injury causing diminution of this negative current, which results in a relatively positive current, inhibits local proper nerve function. Under normal conditions the introduction of a positive current anywhere in the body properly causes the right leg lengthening response by inhibition of the right piriformis muscle, or the weakening of an indicator muscle.

After a few seconds in the relatively strong body, if the injurious positive current is not overwhelming, adaptation to the positive current of injury results in a failure of the right leg lengthening response, and in a muscle test that does not demonstrate autogenic inhibition. Chiropractic adjustment of 3rd lumbar, in the case of the right leg lengthening response, usually restores normal function unless the current of injury is local or specific to the right piriformis. The accumulation of small injuries has the same effect on neuromuscular function as one or two larger injuries.

When local positive currents of injury specific to the right piriformis or the indicator muscle are normalized, the right leg lengthening response and indicator muscle test then may be used to detect global positive currents of injury. In AK we normalize positive currents of injury when we strengthen a weak muscle by searching the Five Factors of the IVF. Global currents of injury may be accessed or retrieved by TL or other AK challenge.

Conclusion

Leg length change, and the muscle test, are traditional chiropractic evaluation tools. Variability of the response and lack of understanding, however, have reduced the credibility of both. Furthermore, the usefulness of the leg length reaction and the indicator muscle test are limited by demands for research whose parameters do not include recent discoveries concerning perineural/meningeal function - discoveries which would open the door on a comprehensive understanding of not only the muscle test but also of the whole human body-mind.

Acknowledging the electrical condition of neuromuscular function bestows confidence in leg lengthening and muscle testing methodology and will help us design better research projects. The right leg lengthening response, as a passive muscle test of the right piriformis, provides a back-up or alternate to muscle testing, and potentiates our technique. No metaphor in the AK vernacular expresses so much so succinctly about kinesiologic function and chiropractic as Goodheart's P.L.U.S.

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not from this level but from 5th lumbar and 1st and 2nd sacral nerve roots. The iliacus muscle, which is inhibited in the same way as the piriformis and which is an aspect of the P.(i).L.U.S. phenomenon, is served by the 3rd lumbar nerve root and, therefore, must be included in this discussion.

19. The left leg will lengthen properly if, instead of a conventional AK challenge, a question requiring a "yes" answer is asked of the body-mind. Brent Davis, D.C. has demonstrated the left leg lengthening response in his excellent "Holographic Scanning" procedure. Other doctors use the muscle test and ask questions of the bodymind as it were to obtain a yes or no answer via the muscle test. In this case, the yes or strong response is analogous to "negative" and the no or weak response is analogous to "positive," while in the leg lengthening response a yes is indicated by a lengthening of the left leg and a "no" by no change. Great potential for controversy exists here because we are unable to legitimize observable phenomona with our present rationalism and we tend to resist new ideas that would modify this rationalism.
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Spinal Subluxation/Bilateral Muscle Syndrome Correlations

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

Abstract

Dysfunction in the body may be caused by spinal subluxations. These subluxations may create specific muscle dysfunction which may only display when the same muscles are tested bilaterally at the same time, but may not show dysfunction when tested individually on the right or left side of the body.

Introduction

It is well known and recognized in applied kinesiology that subluxations (spinal and extraspinal), as well as fixations, are one cause of dysfunction in the body which can create muscle dyspoiesis. This dypoiesis may be discovered by manual muscle testing; however, there are times when a muscle may manually test intact and yet there is subluxation present. This subluxation muscle pattern may only display when both the right and the left side of the body are tested simultaneously. For example the teres major tests strong individually, but when tested together at the same time they test weak which indicates a thoracic fixation. Besides fixations there exists spinal subluxations which also demonstrate this phenomena.

Discussion

The following bilateral muscle dysfunctions have been correlated to these spinal subluxations.

Simultaneous Muscle Dyspoiesis	Spinal Subluxaton
1. TFL	L5 - Anterior
2. Gluteus Medius	L4
3. Adductors	Pubes (Goodheart)
4. Piriformis	Sacrum-Base Posterior (Frank) L1 - Anterior
5. Rectus Abdominous	Ilium - Post Inferior
6. Hamstrings	Sacrum - Base Posterior L5 Cat III Cat I (Leaf)
7. Iliacus	Base Posterior L5 - Anterior
8. Latissimus Dorsi	T7 (Frank)

Simultaneous Muscle Dyspoiesis	Spinal Subluxaton
9. Deltoids (Mid)	C5 - Anterior
10. Teres Major	Thoracic Fixation (Goodheart) C1 T5 - Anterior T8 - Anterior
11. Middle Trapezius	Occiput T5 - Anterior
12. PMC	T5 - Anterior
13. SCM	Occiput C7 T5 - Anterior Frontal Fault (Goodheart)
14. Cervical Extensions	Lumbar Fixation (Goodheart) T3 - Anterior L5 - Anterior
15. Supraspinatus	Occiput T2

All of these muscles must be tested bilaterally simultaneously to display the dysfunction. Look for the subluxation as indicated by utilizing therapy localization and challenge to vector for correction. Recheck the muscles bilaterally simultaneously for verification of correction procedure.

Conclusion

These bilateral simultaneous muscle tests are an addition to the previous correlations by this author and others. They are helpful clinically when searching for the structural causes of a patient's symptom.

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The Universal Stress Receptor

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

Abstract

Cranial stress receptors were among the first reflexes to be used in applied kinesiology. Each of these reflexes corresponds to a particular muscle/organ and are turn on/off switches. There exists a universal stress receptor correlates to all muscle/organs.

Introduction

In applied kinesiology it is well known that there are various cranial stress receptors which have influence over their corresponding muscle/organs. In addition to the already known applied kinesiology cranial stress receptors there also exists a universal stress receptor which appears to influence all muscle/organs and in addition the neurovascular reflexes, acupuncture and even many of the cranial - sacral faults are apparently corrected after the patient is allowed to walk and move around for a period of time after correction of this stress receptor.

Discussion

The location of this universal stress receptor is on the forehead and involves one horizontal line, a bisecting vertical line, and a circle which encompasses the previous two lines (*see diagram*). The procedure for utilizing this universal stress receptor follows the same rules as for the other cranial stress receptors in regard to therapy localization, and challenge respiration. However, first therapy localize to the horizontal and vertical stress receptors and after correction challenge the circular stress receptor first counter clockwise, (fix what you find) and then clockwise (fix what you find). You may have to correct in both directions (counter clockwise and clockwise). Recheck the horizontal and vertical stress receptors, and if positive for therapy localization, then challenge and correct. Continue this procedure until all therapy localization is negative. Recheck the original indicators (muscles, neurovascular, alarm points, cranials) after patient walks for a short time and most if not all should be negated.

Conclusion

This newly discovered universal cranial stress receptor has proved extremely valuable for patient care. It is simple, efficient, and very effective. It makes a wonderful addition to your existing armamentaria for helping humanity.

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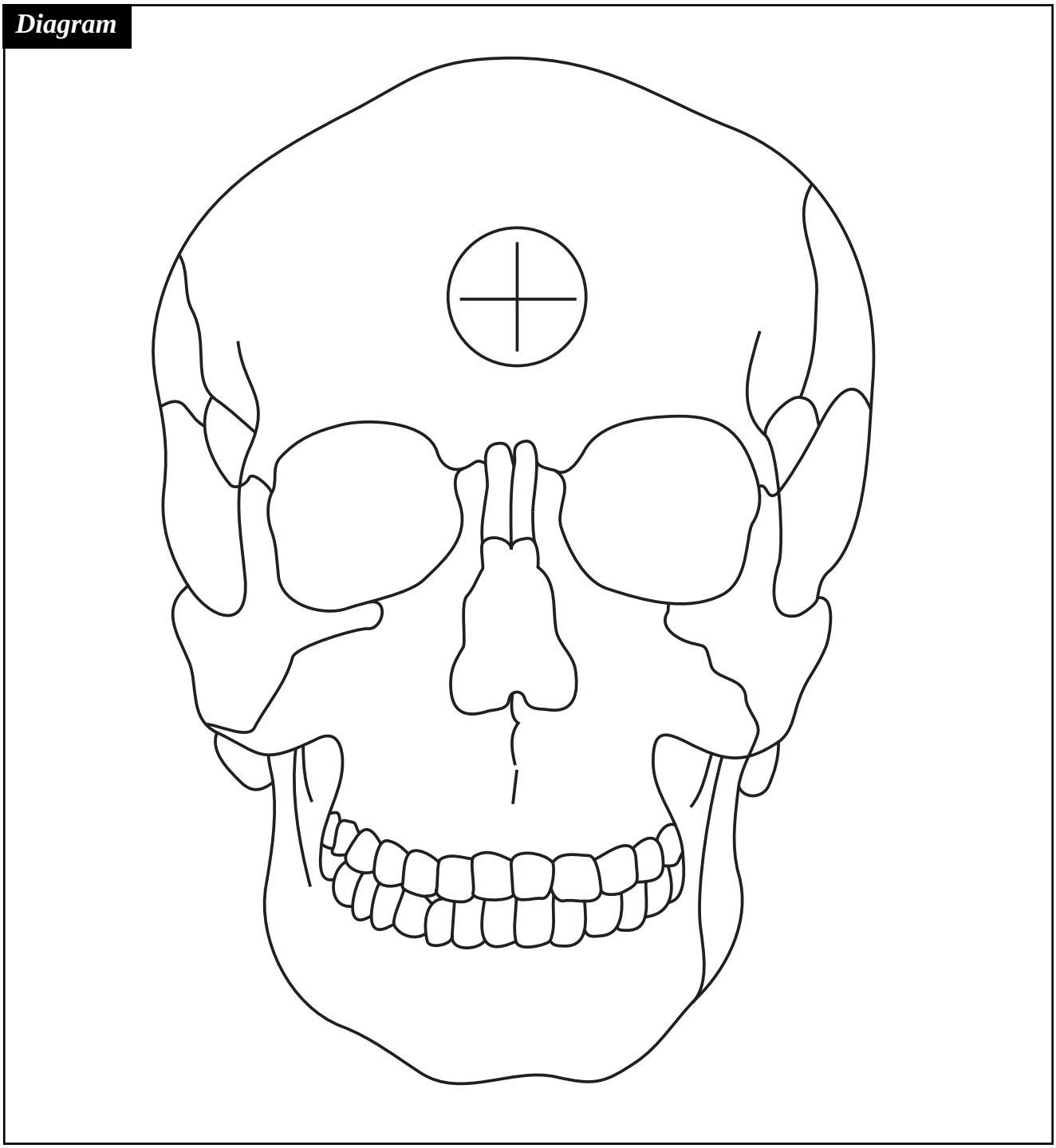
Diagram



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The Relationship between Ho points, Pincher Palpation and Gluteus Maximus Dysfunction

John N. Kane, D.C.

Abstract

Dr. Goodheart has observed that when using pincher palpation to identify areas of gelosis, the weakness that occurs on muscle testing is ipsilateral.[1] Fixing the gluteus maximus muscle coccygeal division for fascial adhesions allow one to use contralateral muscles in pincher palpation and often removes the need for repeated treatment of Ho points.

Materials and Methods

Subjects were first screened for the presence of an area of gelosis by pincher palpation using ipsilateral or contralateral tensor fascia lata muscles for testing. Patients were asked to therapy localize the anterior or posterior elbow or knee to see if a Ho point was involved and then to see what point negated the weakness as described by Dr. Goodheart.[2] The gluteus maximus coccygeal division[3] was then tested and stretched and retested for the presence of fascial adhesions. If present the muscle was fascial flushed.

Results

After treatment of the myofascial adhesion of the gluteus maximus coccygeal division, pincher palpation over an area of gelosis would inhibit contralateral muscles as well as ipsilateral muscles. After walking, Ho points were retested and found to be corrected.

Conclusion

Correcting for myofascial adhesions of the gluteus maximus muscle restores bilateral pincher palpation and often eliminates the need for recurrent Ho point technique.

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The Reticular Formation and Its Role With The Beginning and End Points of The Governing Vessel

Datis Kharrazian, D.C., C.C.N., C.C.S.P., C.S.C.S.

Abstract

This paper expands on techniques that were presented last year on a treatment protocol that was proposed to normalize aspects of the reticular formation. The reticular formation is the bridge for many neurological pathways between segmental and suprasegmental areas. A technique was discussed in which patients that had minimal facilitation to therapy localization, gustatory motor responses, and afferent pathway stimulations would be enhanced by performing a simple procedure. This paper discusses therapy localizing the beginning and end points of the governing vessel meridian to assess if this technique is required.

Introduction

Last year I made the observation that I had numerous discrepancies between my findings with laboratory assessment and applied kinesilogy indicators.[1] For example, if laboratory findings indicated high levels of heavy metals or low thyroid function, I would not find muscles to facilitate or inhibit with challenges or stimulants in addressing these imbalances.

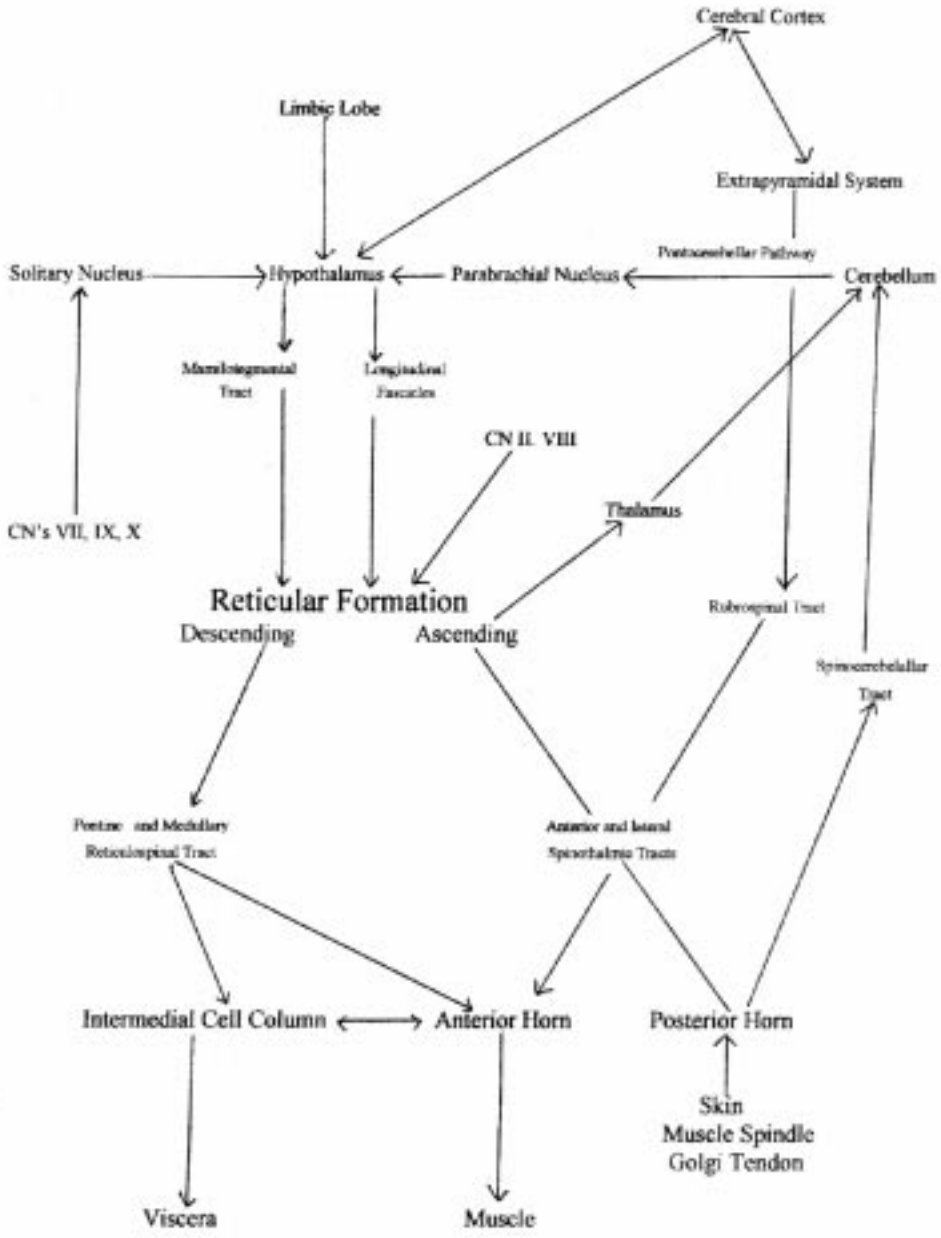
I theorized that the laboratory numbers were accurate and therefore there may be aberrant information being sent to the alpha motor neuron by the neurological pathways that we use in our receptor challenges. For example, when we have the patient therapy localize and use light touch afferents, or challenge a vertebra and induce mechanoreceptor afferents to cause a change in the central integrative state of the alpha motor neuron, we are dependent on a properly integrated thalamohypothalamoreticulospinal loop. And if we use gustatory challenges in order to induce a response of the alpha motor neuron central integrative state we are dependent on properly integrated pathway from cranial nerves VII, IX, and X to the salitory nucleus and down the hypothalmoreticulospinal tract to the anterior horn.

The common denominator between these pathways is the reticular formation and its medial and lateral reticulospinal tracts influence on the anterior horn through the final common pathway.

Since the reticular formation is the bridge between segmental and suprasegmental pathways, its function is crucial in applied kinesiology receptor challenges such as therapy localization and mechanoreceptor challenges.

It also plays in important role in allowing us to accomplish changes in the anterior horn and the intermediolateral cell column from manual manipulation. When vertebral motion is compromised there will be a state in which there is decreased afferent input from type Ia mechanoreceptors and muscle spindles into the posterior horn. This deafferentated state will cause cerebellar and thalamic hyperpolarization that will cause decreased thalmohypothalmoreticulospinal outflow and decreased afferent impulses to the intermediolateral cell column and the anterior horn. If there are decreased impulses to these areas of the spinal cord then there will be decreased autonomic outflow and decreased muscle efferent outflow and will result in decreased visceral and motor functions.[2] Having these neurological loops and pathways optimalized will enhance the effects of manipulation on visceral and motor function.

Diagram



Discussion

The reticular formation is composed of many scattered groups of diffuse nuclei (formations) in the large portions of the tegmentum. 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 extrapyramidal system) goes to the nuclei or formations of the descending reticular system. From these nuclei, axons form the ponitine reticulospinal tracts and the medullary reticulospinal tract, which are the medial and the lateral reticulospinal tracts respectfully. 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 intermedio-

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

The reticular formation also know as the reticular activating system has a general function to regulate the level of consciousness and arousal that are general from the somatosensory, auditory, visual and visceral systems.

Initially, it was found that a loud noise such as clapping by the patient's ears would stress out the reticular activating system and creates a suprasegmental muscle inhibition pattern that includes all three types of muscle conditional weakness.

The correction is made by clapping and finding which beginning and end acupuncture point negates the inhibition pattern. Once the B and E point is identified. The treatment consists performing injury recall technique after clapping and stimulating the B and E point. Nutritional factors related to the corresponding neurotransmitters can also be tested to find which cofactors are indicated in normalizing the imbalance.

It is now known that therapy localizing the beginning and end points of the governing vessel can indicate if the technique is required. Generally, any facilitated muscle will inhibit when the B and E points of the governing vessel are therapy localized.

Performing this technique can result in dramatic improvements in therapy localization and other afferent receptor challenges that are commonly used in applied kinesiology. It is also theorized that it will complement the function of the thalmohypothalamoreticulspinal loop and optimize the impulses sent to the intermediolateral cell column and the anterior horn.

Conclusion

Applied Kinesiology is a technique which different receptors are challenged to determine changes in the central integrative state of the anterior horn. This allows the clinician to apply specific afferent stimulus to the central nervous system and normalize the neuraxis.

However, we always assume the neurological pathways from the receptor challenges to the anterior horn are modulated appropriately. This may not always be the case. It is important to assess and correct these pathways in the initial states of treatment. This will allow optimal afferent input to facilitate the anterior horn and interomediolateral horn which will result in improved receptor challenge responses and improved treatment outcomes.

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The Role of the Transverse Ligament in Suprascapular Nerve Entrapments

Datis Kharrazian, D.C., C.C.N., C.C.S.P., C.S.C.S.

Abstract

This paper expands on the work of Dr. David Leaf. It discusses the role the transverse ligament plays in this syndrome. In addition this paper also elaborates upon the mechanism of neuroischemia as the model for peripheral nerve entrapments.

Introduction

Dr. David Leaf has written extensively about the suprascapular entrapment syndrome. He has found that activities that require scapular stability exacerbate the patient's symptoms. These symptoms run the gamut from diffuse shoulder pain, scapula-thoracic instability, to atrophy of the infraspinatus muscle. A stretching of the suprascapular nerve when the scapula is unstable causes the mechanism of entrapment. This entrapment becomes evident when the infraspinatus muscle is inhibited as it is tested with the arm flexed to 90 degrees and the shoulder rotated anteriorly. The entrapment is corrected by normalizing function to the scapular stabilizers. This correction can be accomplished by implementing applied kinesiology techniques such as: origin and insertion, strain-counter strain, fascial stretch reactions, muscle spindle techniques, reactive muscle patterns, etc.[1, 2]

I have experienced numerous successes by implementing the procedures discussed above. Surprisingly, one "typical" shoulder patient demonstrated only minimal results with the above protocol. I was compelled to further investigate other possible mechanisms involved. I was treating a professional tennis player who originally hurt his shoulder two years ago while attempting to hit a ball out of his reach. His arm was in external rotation, extension, and 180 degrees of abduction when the ball made contact with his racket. He noticed an immediate stabbing pain in his shoulder that has diminished to a constant boring pain over the two-year period. Since that time he has developed severe atrophy of the infraspinatus muscle that was obvious with inspection of the scapula. Two years of rubber band exercises to strengthen the infrapspinatus and other rotator cuff muscles were of no value. Somewhat amazingly, he demonstrated proper facilitation and strength of the standard infraspinatus muscle test. However, when his arm was flexed to 90 degrees and his shoulder rotated anteriorly - inhibition of the infraspinatus muscle was dramatic. Therefore, indications of a suprascapular nerve entrapment were present.

After, normalizing scapular stabilizer muscle function to normal with the symptoms of the entrapment still present I searched for other causes. Reviewing Netter's "Atlas of Human Anatomy" I noticed that the suprascapular nerve passed through the foreman created by the scapular notch of the superior border of the scapula and the transverse ligament. I theorized that the transverse ligament, if constricted, might be the cause of the entrapment. Challenging the ligament to a stretch appeared to cause inhibition of all muscles. This challenge appeared to be due to the rebound phenomenon, which is commonly demonstrated, in applied kinesiolgy. This rebound phenomenon is noticed with vertebral and ligament challenges. After, applying forceful pressure to the transverse ligament for approximately 60 seconds there was a dramatic

facilitation of the infraspinatus muscle when the muscle was tested in 90 flexion and anterior rotation of the shoulder. The clinical results suggested that the patient's suprascapular nerve entrapment sign and symptoms were not only caused by an unstable scapula, but also by an entrapment at the transverse ligament as well.

Discussion

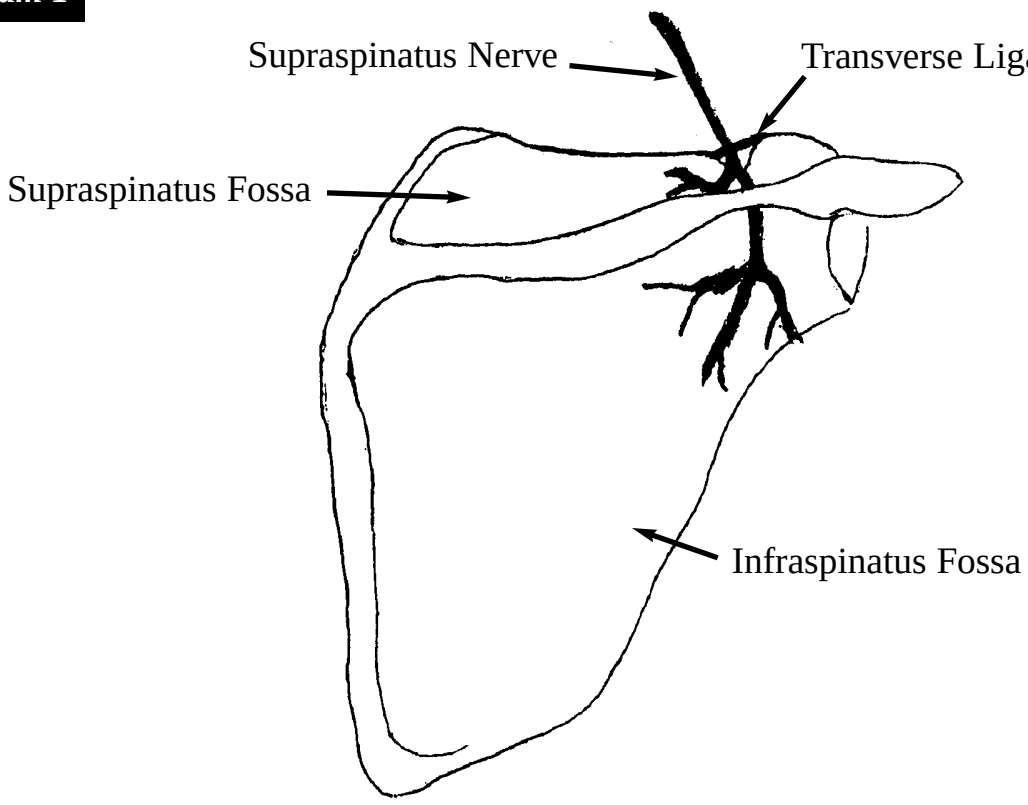
The suprascapular nerve innervates the supraspinatus and the infraspinatus muscles. It originates from the fifth and sixth cervical nerve roots. It then travels underneath the omohyoid and upper trapezius muscles. On its way to the supraspinatus fossa the suprascapular nerve travels in a foramen created by the scapula and the transverse ligament (see diagram). In this area, the suprascapular nerve may become entrapped.[4]

In a true sense, what occurs in this poramen is not actually direct pressure on the nerve, but it is more likely due to neuroischemia caused by pressure on the blood vessels supplying the suprascapular nerve. Because the blood vessels supplying the nerve tissue are softer and more susceptible to compression than nerves, it is the most probable explanation.[3] In addition, if there was actual pressure on the nerve there would be signs and symptoms of Wallerian Degeneration and axonal degeneration. This cannot be the mechanism because there are immediate changes in muscle facilitation when the transverse ligament is restored to normal integrity. The more plausible explanation is neuroischemia that we experience when "our arm or leg falls asleep" as it is put under constant pressure. When this occurs we notice immediate loss of motor strength that is normalized quickly when the pressure is removed.

It appears when the arms are outstretched on front of the body the diameter of the foramen created by the scapular notch and the transverse ligament is reduced. This appears to be caused when the scapula is unstable. Therefore, restoring proper function to the scapula stabilizers (rhomboids, levator scapula, pectoralis minor, etc.) by means of applied kinesiology techniques (origin and insertion, fascial flush, strain/counter strain, muscle spindle and/or golgi tendon procedures, etc.) will correct the syndrome (leaf notes). However, at times the transverse ligament itself may be causing the entrapment due to a constricted transverse ligament.

After, recognizing the important role of the transverse ligament in its role in the suprascapular entrapment syndrome I began checking for its involvement in all cases in addition to scapular instability. I found that the infraspinatus muscle entrapment would be corrected by treating just the transverse ligament itself. However, in patients that had scapular instability the correction would not maintain itself if they were involved in activities that required scapular motion or scapular stability. On the other hand, with some patients that had the entrapment and had no signs of scapular instability the correction would be maintained by only correcting the transverse ligament. This group was definitely the minority. These patients probably have some incident in which there was direct trauma to the transverse ligament itself.

Diagram 1



Conclusion

In conclusion, it is important to check for the involvement of the transverse ligament in cases of suprascapular nerve entrapments. It is usually secondary to scapular stability, but at times there will be minimal results from stabilizing the scapula unless the ligament integrity is normalized. It is the author's recommendation that the involvement of the transverse ligament be addressed in all cases of scapular instability

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Common Threads Between Muscle Therapies

David W. Leaf, D.C., DIBAK

Abstract

When injury occurs, physiological changes occur which can alter normal functions of muscles, tendons and ligaments leading to at least a prolonged healing time or establish a chronic health problem. This paper reviews the underlying tissue response and lays the groundwork for an oral presentation on a proposed protocol for addressing these problems.

Introduction

A general concept in pathology is that the inflammatory response was developed through evolution to protect against bacterial insults. If a bacterial insult starts, there is a massive inflammatory response to wall off the infection. This same response to a noxious stimulus is the same whether the insult is physical, chemical or infectious. When this response occurs to a non-infectious cause, the result is an excessive and potentially harmful process.

When an injury occurs, cellular and humoral responses are initiated. The physical signs of these responses are pain, redness and localized warmth. These are the signs of an acute inflammatory response. In chronic overuse syndromes, the stress is insufficient to create these generalized symptoms. However, inflammatory changes are taking place at the microscopic level.

Trauma to the skin, muscles, tendons and ligaments results in an increase in friction between the structures and a release of chemical factors that precipitate the inflammatory response. When the inflammatory response is due to infection, resting or immobilization of the area afflicted is indicated to reduce damage to the surrounding tissues. In the case of trauma, treatment involving rest may result in chronic disability due to fibrous changes that result due to the inflammatory response. Consequently, many therapeutic tissue mobilization techniques have been developed. The basis for most of these is the creation of motion between parallel structures.

Figure 1 **Normal collagen - a triple helix with parallel fibers for strength and flexibility.**

Discussion

In the first hours after an injury occurs, an extensive network of fibrils begins formation around the fibroblasts. This formation continues at a rapid pace for 48 hours at which time the fibroblasts are completely covered. By the 12th day, these fibrils are dense and heavy. In the first stage, the fibrils are layed down in random directions. But as forces of motion

Figure 2 **Fibers shown slightly separated with a cross link between two parallel fibers. This leads to decreased strength and inflexibility.**

and stress are applied to the injured structure, the fibrils become parallel to the directions of the stress. The sooner that normal motions are applied to the joints, the less the fibrils are deposited in a random manner. At the same time, the initial inflammatory respnse should be minimized as greatly as possible. If there is insufficient motion and controlled stress through the injured structures, the normal parallel fibers become disorganized, reducing the normal strength and flexibility of the structures.

After an injury, the following are basic guidelines:

1. Ice - utilize until swelling is gone
2. Elevation - utilize until swelling is gone
3. Compression - until swelling is gone
4. Passive range of motion - until swelling is gone
5. Active range of motion - when swelling stops until a full range of motion is restored
6. In the first two weeks following a severe injury, motion should be restricted as the strength of the surrounding tissues may be compromised

Most injuries are the result of a sudden trauma that exceeds the loading capacity of the muscle, tendons or ligaments resulting in tissue damage. Even in the minor injuries, microtrauma causes disruption of some of the fibers and localized inflammation. With this inflammation comes swelling and enlargement of the structures within their fascia sheaths. This expansion causes friction between the injured tissue and the fascia resulting in more damage and the formation of adhesions or fibrosis.

During the healing process, tension in the granulation tissue lines up the cells along the lines of stress. Immobilization may limit in the formation of a strong scar with potential adhesions to surrounding structures including bone.

After 72 hours, the repair or regenationa phase begins. This can last up to 6 weeks or longer in very severe injuries. In this state, collagen is deposited at the injury site to regenerate the damaged tissue. Following the removal of damaged tissue by granulocytes and macrophages, a capillary network is expanded to increase blood flow into the injured site. The oxygen and nutrients brought by the increased blood flow stimulate the fibroblasts to secrete collagen. Collagen, a triple helix, is formed from 3 polypeptide chains, protied, and requires vitamin C and oxygen to be formed properly. As the collagen is formed, cross-links are formed between the triple helix individual strands and between adjacent triple helixes. From these cross-links, the collagen gains additional strength.

The final stage of healing is termed remodeling. This stage may take up to one year or a little as 3 weeks. This is a continuation of the regeneration stage. Here the normal motions that occur through the previously injured structures cause restoration of hopefully normal function and strength. Here, ground substance is added and the final alignment of the collagen fibers is firmed in relation to the direction of the forces that the structures are subject to.

Conclusion

When a patient is seen in the office, a choice must be made between which of the many procedures that have been developed are correct for the patient. Another way of looking at this is if there is a common thread between all of the different procedures in the muscle therapy domain.

Between strain counterstrain, or as it is known today, positional release, the myofascial techniques of Barnes, cross frictional massage, Active Release Technique, percussion, the spray and stretch procedures of Travell, and proprioceptive neurological facilitation, one common thread exists. This thread is the breaking and preventing of cross-linking between structures.

To maximize treatment outcomes, a combination of many of these procedures needs to be done on an office visit with appropriate follow-up care.

A model of this treatment protocol will be presented.

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Magnetic Model of Acupuncture Points

In E. Moon, D.N., L.Ac., O.M.D., Ph.D.

Abstract

An acupuncture meridian is the channel through which Qi flows.[1]

An acupuncture Point is the three dimensional space where Qi gathers. Qi is both energy and matter.[2] Qi flows like as liquid and /or group of particles. The turbulence of this Qi at acupuncture point creates multiple dipoles of Qi-magnets. The disturbance and unbalance of this Qi-magnet is the cause of excess and deficiency of the acupuncture point and meridians. This distorted point can cause another distortion of opposite character at nearby acupuncture points as well as other therapy points such as neurolymphatic or neurovascular. The distorted magnetic induction causes misdiagnosis of other points and meridian jamming which we understood partially as neurological disorganization and switching.

Introduction

The electricity flows through a metal wire like a fluid in a tube, and the electric flow is understood as flow of electrons which are tiny particles.[3] The conductor of electricity is usually metal in which there are plenty of free electrons. And these electrons can move freely between the structure of the metal.

The magnetic field is created by circular circuit of electricity, the magnetic field is perpendicular to this circuit. The electric magnet is made of circular coils connected to the electricity. The permanent magnet consist of well aligned atoms and molecules where the electrons moving in circular motion the same as electricity in circular coil. Not only spinning electrons but also spinning protons generate magnetic field.[3]

It is not difficult for us to imagine that the circular movement of Qi in acupuncture meridian create a magnetic field, because Qi includes electricity too. Here we can expand our imagination that the circular movement of Qi create Qi-magnet. The Qi-magnet includes electric magnet and other field created by other energy beyond electricity. The swirling or eddying or spinning Qi creates Qi-magnet.

Hypothesis

1. Qi flow within acupuncture meridian is linear and unidirectional.
2. The acupuncture meridians connect acupuncture points, the diameter of the space of the acupuncture point is bigger than diameter of meridians which connect that point.
3. A healthy normal acupuncture point is magnetically neutral and well balanced.
4. A deficient acupuncture point shows characteristics of magnet South. (on northern hemisphere of the earth)

5. A excess acupuncture point shows characteristics of magnet North. (on northern hemisphere of the earth)

6. The magnet north is the same magnetic polarity of north pole of the earth, and the magnet south is the same magnetic polarity of south pole of the earth.

Diagrams of Magnetic Model of Acupuncture Points

Diagram 1

Normal healthy acupuncture point on northern hemisphere

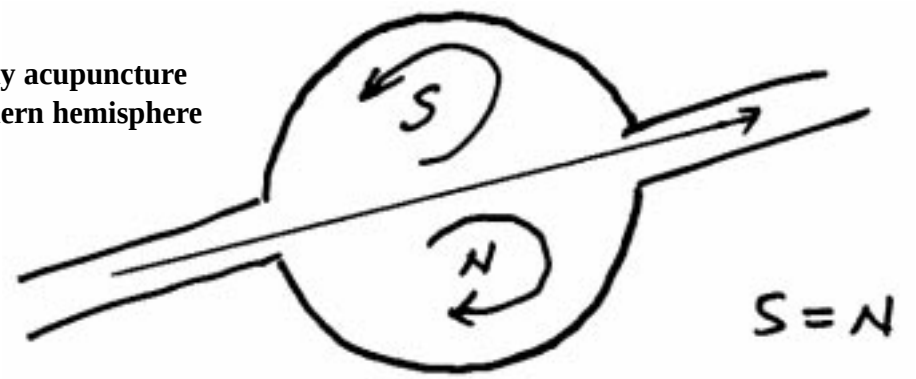


Diagram 2

Deficient acupuncture point on northern hemisphere

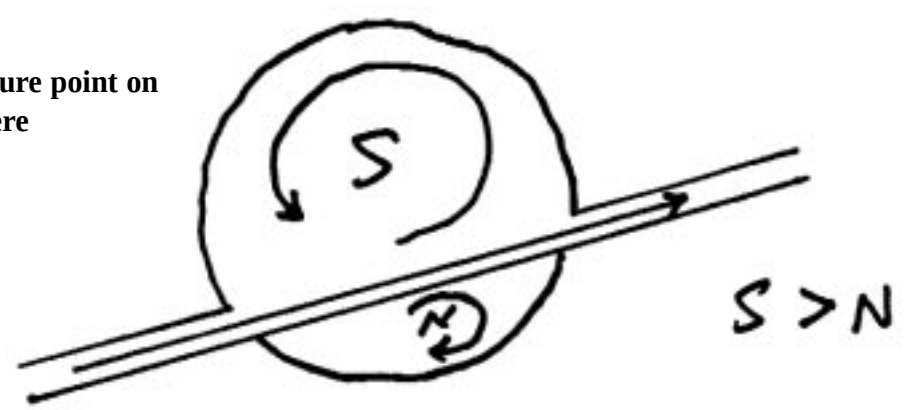
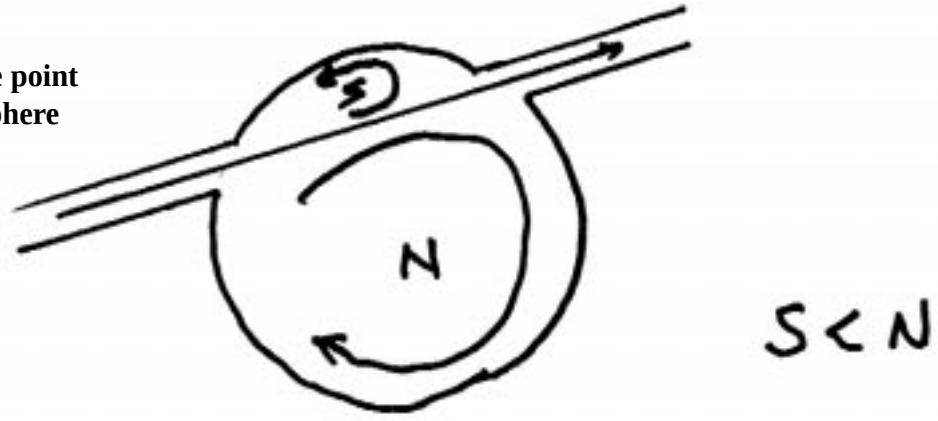


Diagram 3

Excess acupuncture point on northern hemisphere



Applied Kinesiological Observations

Example 1:

When right Large Intestine meridian is deficient, we find that right front Mu-point of LI-meridian(ST25) is deficient. When patient touches right ST25 with one hand, then patient's SIM(=strong indicator muscle) become weak.[4] But previously weak right brachioradialis become strong. This is common AK response of autolysis and odd TI(=therapy identification).[5, 6]

Then we place a south pole of magnet at right ST25, the previously weak brachioradialis become strong, but SIM does not become weak. This is positive extralysis.

If we place a north pole of magnet at right ST25, SIM becomes weak, but previously weak brachioradialis stays weak. This is negative extralysis.[7]

ST25 was magnetized as south already, when we place north pole of magnet, it exaggerate the magnetizing further south, i.e. cause more distortion. But we place south pole of magnet, we reduce the magnetic strength of south at ST25, because magnetic south repels other magnetic south.

Now we found deficient ST25 was magnetized (magnetically distorted) as south pole of a magnet.[8]

Example 2:

When right Large Intestine meridian is excess, we find that right front Mu-point of LI-meridian(ST25) is excess. When patient touches right ST25 with one hand, then patient's SIM(=strong indicator muscle) does not become weak. But previously strong right brachioradialis become weak. SIM becomes weak if patient touches at ST25 with two hands. This is common AK response of autolysis and even TI(=therapy identification).

Then we place a south pole of magnet at right ST25, the previously strong brachioradialis become weak, but also SIM become weak. This is negative extralysis.

If we place a north pole of magnet at right ST25, SIM does not become weak, and previously strong brachioradialis stays strong. This is positive extralysis.

ST25 was magnetized as north already, when we place south pole of magnet, it exaggerate the magnetizing further north, i.e. cause more distortion. But we place north pole of magnet, we reduce the magnetic strength of north at ST25, because magnetic north repels other magnetic north.

Now we found excess ST25 was magnetized (magnetically distorted) as north pole of a magnet.

Therefore, a deficient acupuncture point respond exactly opposite to a excess acupuncture point as we saw in Example 1 and 2.

Experiments

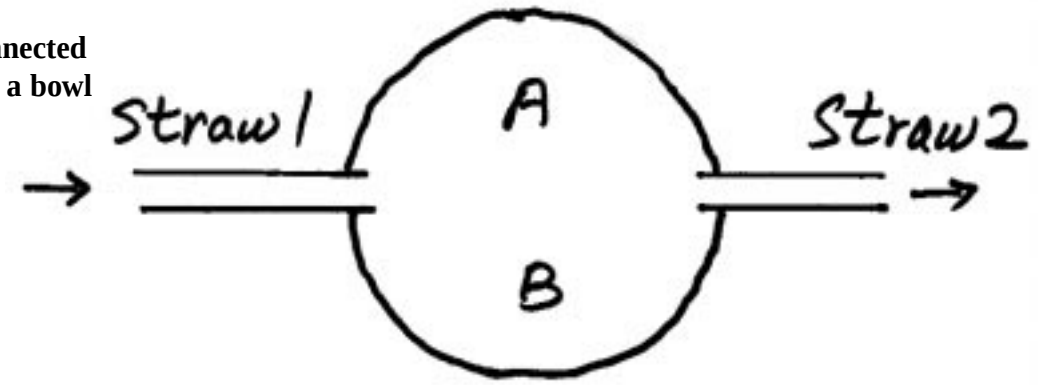
Experiment 1:

Here is a bowl with full of water and two straws as in Diagram 4.

Let's supply water into this bowl through straw 1, and simultaneously drain water from the bowl through straw 2.

Diagram 4

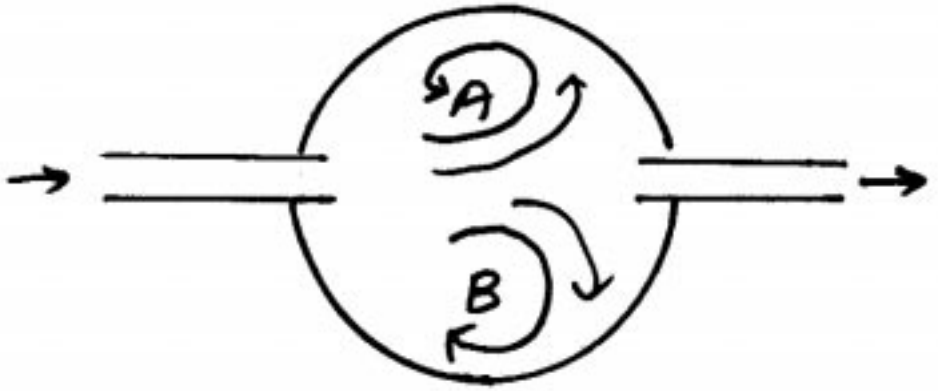
Two straws connected opposite end of a bowl



If we do it steadily long enough, we will develop counter clockwise swirling current at A part of bowl, and clockwise swirling current at B part of bowl as in diagram 5.

Diagram 5

Equal size and opposite direction Vortex at A and B

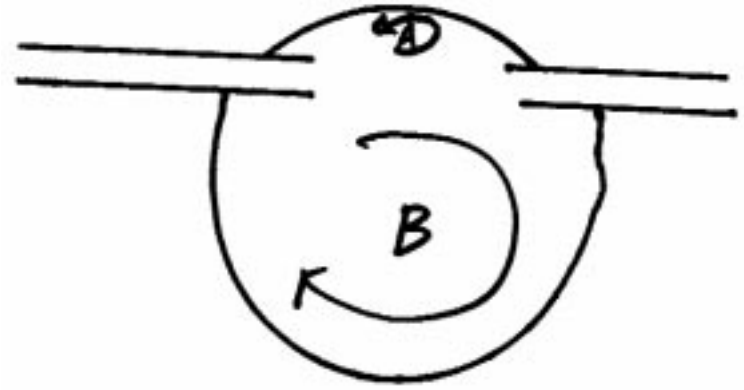


Experiment 2:

Let's place the straws lopsided as we see in Diagram 6, we will get smaller vortex at A than vortex at B.

Diagram 6

Vortex A < Vortex B

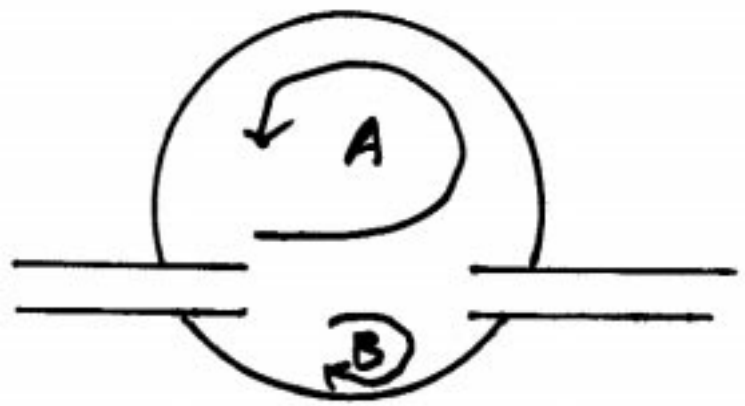


Experiment 3:

If we place the straws as in Diagram 7, we will get bigger vortex at A than vortex at B.

Diagram 7

Vortex A > Vortex B

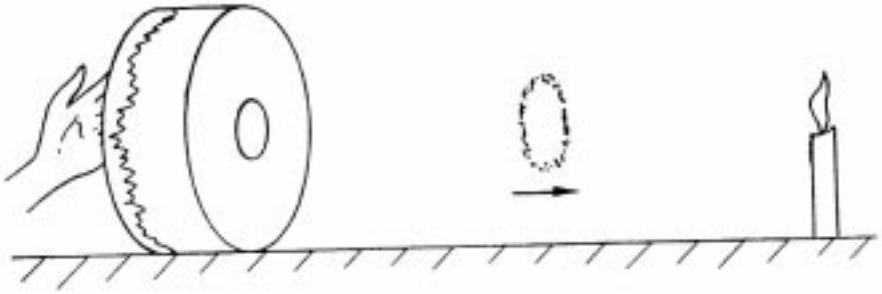


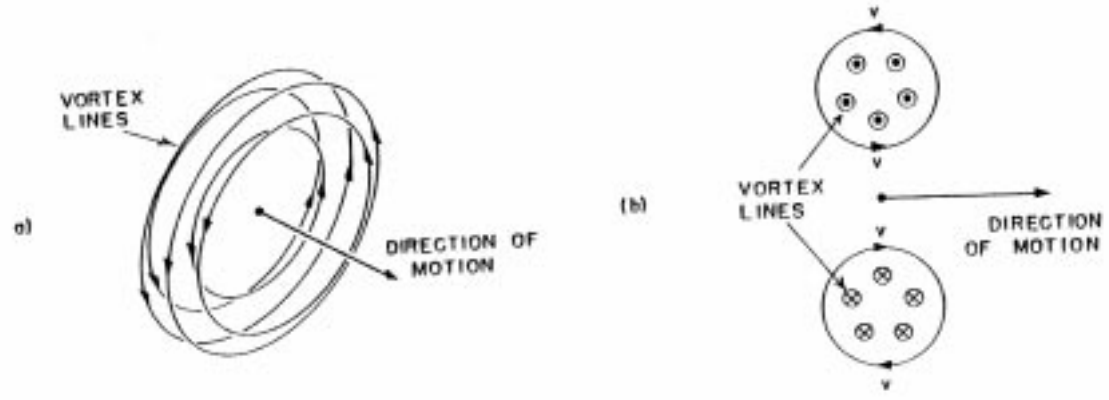
Experiment 4:

There is a nice demonstration of a vortex ring which is made with the simple apparatus of Diagram 8. It is a drum two feet in diameter and two feet long made by stretching a thick rubber sheet over the open end of a cylindrical box. The other end is solid except for a 3-inch diameter round hole. Then let's blow some smoke into this drum through the hole. If you give a sharp blow on the rubber diaphragm with your hand, a smoke vortex ring is projected out of the hole. The energy of this vortex ring can blow out a candle 10 to 20 feet away.[3]

Diagram 8

Vortex Ring





Discussions

Let's assume the liquid in the bowl is full of electric charge as in our body electrolyte. Obviously, Vortex A will create the magnetic field of south pole, and Vortex B will generate the north magnetic field.

In diagram 6, the south magnetic field is smaller than the north magnetic field, therefore whole bowl will act like north magnet. This is what happen exactly on excess acupuncture points.

In Diagram 7, the south magnetic field is bigger than the north magnetic field, therefore whole bowl will represent as a south magnet. This is what happen exactly on deficient acupuncture points.

Because the acupuncture points are situated in the three dimensional space, we can picture a vortex ring at the balanced normal acupuncture point. This vortex ring looks like a smoke ring. (see Diagram 8)[3]

Since we live on north magnet of the earth, in order to keep our acupuncture point healthy and balanced our homeostatic life energy will compensate against magnetizing influence from north magnet of the earth.

When this compensating energy is not strong enough that point become magnetized as south because north magnet of the earth attract and create south magnet.

The other case, when the compensating energy is too strong and over compensate against north magnet of the earth, the point become north, which is excess point as we found applied kinesiological examination in example 2.

As modern science found that our body is electric,[9] therefore also our body is magnetic, it is evident that our body create radio waves because there is radio waves so called electromagnetic wave where electricity and magnetism interact.[10, 12]

It is very easy for us to imagine that the acupuncture points are broadcasting and receiving radio waves which is vital communication not only between organs of our body also interaction to our environment which may be other person, animal, plant, lifeless objects, the sun and the moon, and stars, etc.

Clinical Applications

There are numerous clinical applications of magnets in the past,[10, 11, 12] now we can use it more precisely with scientific knowledge and applied kinesiological examinations.

We can test a SIM(Strong Indicator Muscle) place a magnet north or south on any desired acupuncture points or other therapeutic points, then we can identify if that point is deficient or excess. If we found excess, treat with north magnet on that point. If we found deficient, treat with south magnet on that point. Place a magnet with adhesive tape and change it to other side of the body midnight and midday since excess and deficient point change its side near midnight and midday.

Qi-magnet can be made easily by circling concentrated Qi at the doctor's finger tips, and this Qi-magnet is more powerful than any other instrument tested by the author. The test method was priority muscle testing according to the relativity.

Conclusion

The magnetic model of acupuncture point is useful to explain diagnostic and therapeutic application of magnets scientifically.

Also it is very convenient to understand meridian jamming which causing neurological disorganisation and switching and dejamming procedure.

Further research is needed for the reason why left right change pattern of magnetic response AM and PM.

Also further research is needed for changing characteristics of magnetic model of acupuncture points in different respiratory phases.

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The Role of the Scaleneus Anticus Muscle in Dysinsulinism and Chronic Non-traumatic Neck Pain

Thomas A. Rogowskey, D.C., DIBAK

Abstract

Investigation into why dysinsulinism often relates to symptoms of cervical spine imbalances led to the discovery that the scaleneus anticus muscle was conditionally inhibited when tested as part of an applied kinesiological exam. This conditionally inhibited muscle is implicated in many of the symptoms associated with chronic neck pain, brachial plexus syndromes, and an unstable cervical spine. Treating dysinsulinism facilitates the scaleneus anticus muscle and ameliorates the cervical spine related symptoms. Using applied kinesiology, one can tailor a program that is patient-specific for better insulin tolerance.

Introduction

The focus of this paper is to demonstrate that dysinsulinism is the source of many presenting problems in our patients. Discussion is focused on the stages and the symptoms of dysinsulinism. Among the symptoms to be discussed are cervical spine related syndromes that have not been addressed previously; a rational for the presence of these symptoms will be given. General discussion of remedies will be made in the context of using applied kinesiology to determine the specific needs of the patient. Discussion of the mechanisms leading to the cause of dysinsulinism will be left to other authors.

Commentary and Clinical Observations

Applied kinesiology combines the scaleneus anticus, scaleneus medius and the scaleneus posticus muscles into a test for the medial neck flexors and associates them with sinus conditions.[1] Beardall, in his text on Clinical Kinesiology, associates the scaleneus anticus muscle with the bladder and ductus deferens.[2] Investigating chronic neck stiffness in my patients has led me to a new association of the scaleneus anticus muscle with the sugar metabolism mechanisms of the body. I have observed that when the patient presents with chronic neck pain, there consistently will be a scaleneus anticus conditional inhibition (CI) along with other signs and symptoms consistent with dysinsulinism. Reflex points for this muscle are under investigation and appear to be along the costal cartilage bilaterally, approximately two inches from the xiphoid process. If the patient is successfully treated for dysinsulinism, it often eliminates the need to treat this muscle; therefore, it becomes necessary to discuss a protocol for measuring and treating dysinsulinism.

Patient cases

The following presenting symptoms and accompanying information of typical patients is given to highlight the degree to which cervical instability is associated with dysinsulinism and should be an area for further investigation.

Mary Jane, age 44, sought care from my office for the chronic neck pain that she suffered. Her case history revealed that she often craved refined carbohydrates, was sleepy after lunch, had difficulty returning to sleep after waking during the night, and had a tendency to easily sprain her ankles when hiking. Her fraternal grandmother had type 2 diabetes, and her father was on hypertension medication.

Louis, age 48, sought care for his low back pain that would come and go chronically, often shifting from one side to the other. He also had joint pain in arms and legs during the winter months. A stiff neck bothered him in the morning, and sometimes it woke him at night. He admitted to eating fast foods and had put on about ten pounds in the last two years that he could not lose. There was a familial history of heart disease and high cholesterol, with his cholesterol level at 230. His medical doctor wanted to put him on a cholesterol-lowering drug.

Pat, age 65, came in for dietary advice after an eye operation. He was on a cholesterol-lowering drug, he was about 25 pounds overweight, and he was on a hypertensive drug. He had difficulty sleeping, waking about 3:00 am and remaining awake for one and one-half hours before returning to sleep. He was an admitted "sugar addict". He was physically very active, playing tennis in the morning and walking for forty-five minutes in the evenings. He was bothered by a decreased range of motion in his cervicals and upon waking would have to stretch and take a hot shower to loosen his neck before it was comfortable.

Tiffany, age 15, was obese and suffered from acne. She had painful menses and pain over the area of her ovary premensturally. She could not control her eating of sugar and was often "obsessed" with eating it. She had no musculo-skeletal complaints. There was a familial history of hypertension and heart disease. No one in her immediate family had any weight problem.

Dysinsulinism

The common thread among the patients above and many of the patients I treat is that they display some form of dysinsulinism. Phil Maffetone and Jeff Bland have lectured and written about the spectrum that exists in the severity of dysinsulinism. Both authors have divided the spectrum into three areas or stages of carbohydrate intolerance or insulin resistance.[3, 4]

The dysinsulinism spectrum ranges from patients that may display only subtle sugar handling problems or an inflammatory condition, to patients with insulin dependent diabetes. The early part of the spectrum is characterized by the typical low blood sugar symptoms such as headaches, dizziness, weakness if a meal is missed or craving sweets. The early part could also include sleep disturbances, acne, inflammation,[4, 5, 6] hormonal imbalances,[4, 6, 7, 8, 9, 19] and functional adrenal problems. I have observed that neck stiffness, brachial plexus disturbances and cervical instability also fall into this area of the dysinsulinism spectrum. Adrenal dysfunction with accompanying ligament problems often results in musculo-skeletal problems. The ligament stretch test is often positive in these cases.[1] Schmitt has hypothesized that increased aldosterone is associated with the ligament stretch phenomena.[10] "The best predictors of plasma aldosterone [increases] were abdominal obesity measured as [increased] waist/hip ratio or by CT scan, and insulin resistance."[11] Increased waist/hip ratio is also an indication of insulin resistance. [6, 4]

As dysinsulinism becomes more severe, additional symptoms develop beyond those of the early stage. These include polycystic ovary syndrome (PCOS),[7, 9] hirsutism,[9] peripheral nerve symptoms, Syndrome X (which includes hypertension, hypercholesteremia, increased LDL, decreased HDL, increased uric acid, increased triglycerides and heart disease),[4, 12] frequent urination, dry mouth, scalloped tongue, psoas CI demonstrating dehydration, and functional hypothyroidism as in some cases classified as Wilson's syndrome. Cognitive function can be affected,[4, 6] and cases of breast cancer[8] have been associated with this syndrome.

Syndrome Identification

The degree to which dysinsulinism is part of the functional illnesses that applied kinesiologists treat daily is evident by the list of symptoms above. It is important to identify this syndrome and take the appropriate steps to alleviate it. Schmitt teaches two methods of identifying dysinsulinism using applied kinesiology. Each requires a challenge to a previously facilitated long head of the biceps muscle with the arm in full extension and flexed at the shoulder 45 degrees. (Author's note: if this muscle tests as conditionally inhibited, it may also indicate hyperinsulinism.) Pinching the pancreas visceral referred pain point (VRP) located below the left anterior rib border, or hard rubbing the pancreas NL are interpreted as stimulating insulin activity.[13] A conditionally facilitated muscle becoming inhibited when the body is exposed to 6 or 8X homeopathic insulin can be interpreted as a dysinsulin type condition, probably insulin resistance. Schmitt has found these two techniques to be equivalent in indicting hyperinsulinism.[13] I have found the insulin 6X challenge to be equivalent to a biomagnetic challenge of samples of insulin.

After exposing the patient to insulin and finding a weakness or using the Schmitt procedure with the biceps muscle, it is necessary to correlate these finding with the patient history as it relates to the dysinsulinism symptoms previously mentioned. Various muscle tests when found as conditionally inhibited can also corroborate these findings. These muscles include triceps, latissimus dorsi and the above mentioned scalenius anticus. According to Beardall, the scaleneus anticus muscle is tested by flexing the head and neck on the trunk 45 degrees, and rotating the head 20 degrees away from the side tested. The doctor places the lateral aspect of his/her hand on the maxillary and frontal bone and attempts to extend the neck on the trunk in a sagittal direction.[2]

When indicated, laboratory tests should be used to substantiate the findings of dysinsulinism. The initial stages of dysinsulinism may be too subtle to show positive on a lab test. The most accurate laboratory test for measuring insulin levels is with an euglycemic hyperinsulinemic clamp;[14] however, this method requires hospitalization. The next best measurement is with a short glucose tolerance test that measures the glucose levels and the insulin levels simultaneously.[14, 15]

If a correlation to dysinsulinism can be established, I proceed to identify the main causes of dysinsulinism. The causes that are currently associated with dysinsulinism are:

1. Disturbances to the beta cells of the pancreas caused by an apparent antibody response to particular foods.[4]
2. Missing nutrients that participate in the insulin receptor site and in the cellular glucose transport system.
3. A general state of inflammation.
4. A down regulation of the receptor sites due to consistent and excessive glucose levels.

Treatment

Using the muscle that is conditionally inhibited, or any muscle that is demonstrating the general weakness found with insulin exposure, the above causes of dysinsulinism can be investigated. First, attempt to find if the dysinsulinism is a primary or secondary problem with a patient. Finding a receptor stimuli that would override the insulin response would indicate that the dysinsulinism is secondary. Conversely, not finding a receptor stimuli to override the insulin response would indicate that the dysinsulinism was the primary

problem to treat. The following list indicates the common overriding stimuli and their associated primary problems.

Stimulus	Possible Primary Problem
Emotional Neurovasculars	Emotional Problems
NSAIDS	Inflammatory condition
Arachidonic Acid (AA)	Inflammatory condition
Hydrogenated oil	Inflammatory condition
Hydrogen Peroxide[21]	Overoxidation or free radical pathology
Chlorine bleach	Overoxidation or free radical pathology
Oil or Fat[16]	Too large a percentage of fats in diet or not enough protein
Glucosamine[17]	Wrong supplementation
Sugar	Down regulation of the receptor sites
Foods (Dairy)[4]	Antibody response to particular foods
Homeopathic food	Antibody response to particular foods

Next, test the nutrients associated with dysinsulinism to determine which would abolish the conditionally inhibited muscle. Nutrients that are useful in treating dysinsulinism are: vitamin E, sources of essential fatty acids particularly EPA, chromium, vanadium,[4, 19, 6] alpha lipoic acid, biotin, inositol, zinc, choline,[4, 6] l-arginine,[4] DHEA,[24] magnesium[20] and antioxidants.[4, 19, 6]

Once these have been determined, treatment can be directed to reverse the dysinsulinism. The receptors that override the insulin weakness should be treated in the manner appropriate for that particular receptor. The emotional NVs can be treated in the methods that are traditional to applied kinesiology, or in related emotional techniques that abolish the positive response to the receptors. For instance, in NET the pancreas is related to self-esteem, and this issue could be treated.[23] If inflammation appears to be an issue due to the response to a source of AA or NSAIDS, then IRT can be performed to the reflexes that are indicated by two-pointing when the patient is exposed to AA as follows.[22] The conditionally facilitated muscle will become inhibited when exposed to the AA. All reflexes that reverse the muscle response should be noted. These reflexes are then digitally stimulated and an IRT is performed. The same technique can be performed when hydrogenated fats, bleach, peroxide, sugar and food reactions are the culprit. If insulin requires the primary treatment, the IRT technique described for AA should be used to reset all the insulin-affected reflexes.

In particularly difficult cases, the insulin will not inhibit a conditionally facilitated muscle. When this occurs the above receptor stimuli could be introduced to the body while continued exposure to the insulin is maintained. These receptor stimuli by themselves will usually not inhibit a muscle when exposed to the body. If one or more of the receptor stimuli in concert with insulin does inhibit the muscle, find which nutrients will reverse this inhibition, and then perform the IRT technique used for AA described above.

It is necessary to counsel the patient on lifestyle adjustment regarding dysinsulinism if it proves to be the primary condition of treatment. This information is contained in excellent texts on the subject and is outside the scope of this paper.

Conclusion

Dysinsulinism, particularly insulin resistance is being recognized as a major condition in the field of functional medicine. It is particularly pertinent to the practitioner of applied kinesiology because of the prevalence of insulin resistance in the general populace in addition to the essential place it holds in the physiological matrix of the body. Specific muscle weaknesses, inflammation, cognitive behavior, sleep disturbances, acne and other skin disorders, cervical spine disorders, dysfunctional adrenal disorders, hormone imbalances, syndrome X and functional hypothyroidism are some of conditions that relate to dysinsulinism. Applied kinesiologists have the means and the methods to help reverse this condition. Associating the scaleneus anticus muscle weakness with dysinsulinism can help the practitioner of applied kinesiology reach the root causes of a problem and facilitate a more timely clinical intervention.

Summary of Procedures:

- If symptoms seem to indicate a form of dysinsulinism:
  Test the
  - scaleneus anticus muscle
  - triceps muscle
  - latissimus muscle
- A positive test is when the muscle is conditionally inhibited (CI)
- Test the long head of the biceps with extended elbow
- Pinch the VRP for the pancreas
- Retest the long head of the biceps
- A positive test is either when the biceps is CI when first tested or after the VRP is pinched
- Test a conditionally facilitated muscle after exposing the body to insulin
- A positive test is when the muscle becomes CI
- Using a positive test check for overriding stimuli
- Emotional NVs, exposure to a non -steroidal anti-inflammatory drug or a source of arachidonic acid (AA), a source of hydrogenated oil, a source of oil like olive oil, hydrogen peroxide, chlorine bleach, sugar and/or foods such as dairy or homeopathic food sources.
- Check for nutrition needs: vitamin E, sources of essential fatty acids particularly EPA, chromium, vanadium, alpha lipoic acid, biotin, inositol, choline, zinc, DHEA, l- arginine, and antioxidants.
- Correct the priority receptor stimuli by finding all the reflexes that two-point to the CI muscle. Stimulate digitally and perform and IRT ipsilaterally on the talus. If it is a centerline reflex, perform the IRT bilaterally.
- Recheck all CI muscles for refacilitation.
- Retest the vitamins found to determine if still needed.
- Discuss diet and lifestyle changes relative to a sugar-handling problem.

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Fixations, A Muscular Correction

Scott J. Vrzal, D.C.

Abstract

One of the fundamental principles in Applied Kinesiology is that muscles move bones. This paper applies the principle to the commonly treated clinical entity of the vertebral fixation and discusses the muscular corrections that have been successfully applied.

Introduction

The continued quest to reach the deepest cause of patient ailments includes searching for the potential causes of the many vertebral fixations seen in clinical practice. In this paper the many muscular imbalances typically found opposite to the associated vertebral fixation levels are discussed. The vertebral fixation was first published by Goodheart[1] in 1973. Later taught and discussed in detailed text format by Walther[2] with the neurologic cause promoted by Schmitt[3] in 1975. There has been much written about the vertebral fixation since. The 1991 index of Collected Papers of the ICAK lists 26 papers by 21 different authors, none of which approach the muscular correction of the vertebral fixation.

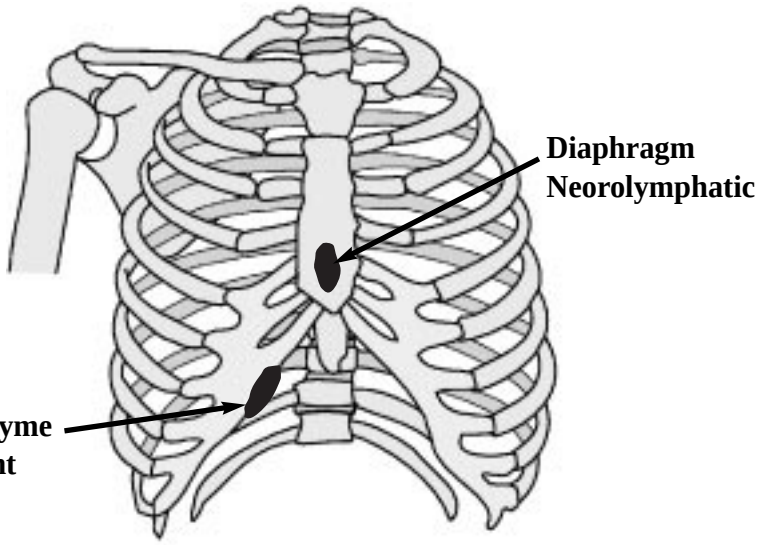
Materials and Methods

The following insight was gathered via literally thousands of patient treatments in private practice over the last several years. It has been tried and refined by the author and his associates with increasing continuity and success. Corrections will be discussed from caudal to cranial fixation locations.

Unilateral sacral-iliac fixations create an ipsilateral splenius capitus neurophysiologic inhibition. We have found that correction of the associated structural, chemical, acupressure, or emotional faults related to the ipsilateral iliacus muscle will negate the previously inhibited splenius capitus muscle. The primary association we find here is an open iliocecal or valve of houston ipsilateral to the spenius capitus and iliacus inhibitions.

Lumbar fixations lead to a bilaterally inhibited splenius capitus pattern. We find that correcting neurophysiologically inhibited abdominal muscles will negate the lumbar fixation. There are many associations for abdominal weakness. Most are in some way associated with small intestine function,[4] hydrochloric acid secretion and/or protein metabolism and jamming of the sagital suture.[4] We find our best success to be to stimulate the function of (upregulate) pancreatic enzyme function. Proper upregulation will immediately eliminate tenderness of the pancreatic enzyme point located at the right costal margin one-inch inferiorolateral from the xiphoid process (diagram a). This point will also therapy localize until clinically negated by proper treatment to T6, pancreas tonification or stomach 1 acupressure points, and/or digestive enzyme supplementation.

Diagram a



Thoraco-lumbar fixations create a bilateral neurophysiologic inhibition of the lower trapezius muscles. Many[2, 3, 5] have discussed associated diaphragm dysfunction in relation to the thoraco-lumbar fixation. Functional correction of the diaphragm has also been well detailed by Maffetone[6, 7] and Walther.[8] In addition to the structural, chemical, emotional, and acupressure therapies outlined by prior authors we find trans fatty acid intake to be a common diaphragm inhibitor. In this case treating the weakness created by ensalivation of the offending food (or similar trans fat) will negate the bilateral lower trapezius weakness and thoraco-lumbar fixation. Concomitant elimination of trans fatty acids is then typically recommended along with testing for the potential need for omega 3 fatty acids.

Mid-thoracic fixations respond to correction of bilaterally inhibited pectoralis major clavicular muscles. This may be accomplished by correction of the jammed sagital suture or again addressing the concept of pancreatic enzyme upregulation and enhanced protein digestion as previously discussed.

Cervico-thoracic and lower cervical fixations and their associated neurophysiologic inhibition of the bilateral deltoid and popliteus muscles respectively have been found to respond to correction of the lower thoracic fixation. The accompanying lower trapezius hypotonicity creates hypertonicity of the upper trapezius muscles. Correction is accomplished by improvement of diaphragmatic function. Facilitation of the splenius capitus muscles as described under lumbar fixations may also need to be addressed.

Upper cervical fixations and their concomitant neurophysiological inhibition of the gluteus maximus muscles bilaterally have been found to respond to facilitation of the cervical flexors. Most commonly inhibited as a group indicating a frontal fault. This then typically leads to correction the offending pterygoid muscles.[9] Occasionally there will be a singular inhibition of an individual scalene or sternocleidomastoid muscle. When the offending anterior cervical muscle is corrected, the bilateral inhibition of the gluteus maximus should no longer be present.

Occipital fixations and their indicative bilaterally neurophysiologically inhibited psoas muscles are often corrected with the previously described therapies. We find that most commonly there is an associated unilateral splenius capitus inhibition present with its attachment to the occiput, which would then be corrected via the unilateral iliacus as previously described. Secondarily, (approximately 40%) when an inhibition of the splenius capitus is not determined, there may be a hypertonic pterygoid muscle. In some cases a left deltoid dysfunction leading to a universal cranial fault as described by Goodheart[10] may need correcting. In either case when the appropriate correction is made the previously inhibited psoas muscles will function properly with lasting success.

When there are multiple fixations present octacosanol supplementation is the tried and true recommendation. In these cases the patient will often have a negative (universal, neurophysiologic inhibition) reaction when ensalivating refined sugar. By applying the indicated therapy to the organ that negates the previous inhibition, typically the liver or pancreas, all 3 reactions should no longer occur. Meaning most of the fixations should no longer present, the need (indication for) octacosanol should no longer present, and sugar should no longer generate a universal muscular inhibition.

Results

The previously outlined corrections have been safe and effectively utilized on hundreds of patients with encouraging, lasting results when the underlying cause has been corrected. Two associates with equally encouraging results have also duplicated the application.

Discussion

Muscles move bones. When inappropriate muscular facilitation/inhibition occurs, muscles aberrantly move bones. Schmitt[3] when outlining the possible neurologic cause of fixations states that "inhibition of a muscle group cannot occur without reciprocal facilitation of antagonist muscle groups and vise versa." It may be that the larger extrinsic muscles discussed here are the antagonists to the spinal intrinsic muscles that become facilitated causing the fixation. Dr.'s Goodheart[10] and Leaf[9] have extended the philosophy to the cranium for many of us to apply and further enhance our clinical excellence.

Conclusions

The goal of this study was to discuss the clinical findings that have produced lasting corrections. Several doctors have applied the aforementioned material safely and effectively. Fixations have been treated and sometimes retreated for more than two decades. The discussed material needs to be applied and confirmed by others in order that it's clinical reproducibility may be validated and incorporated.

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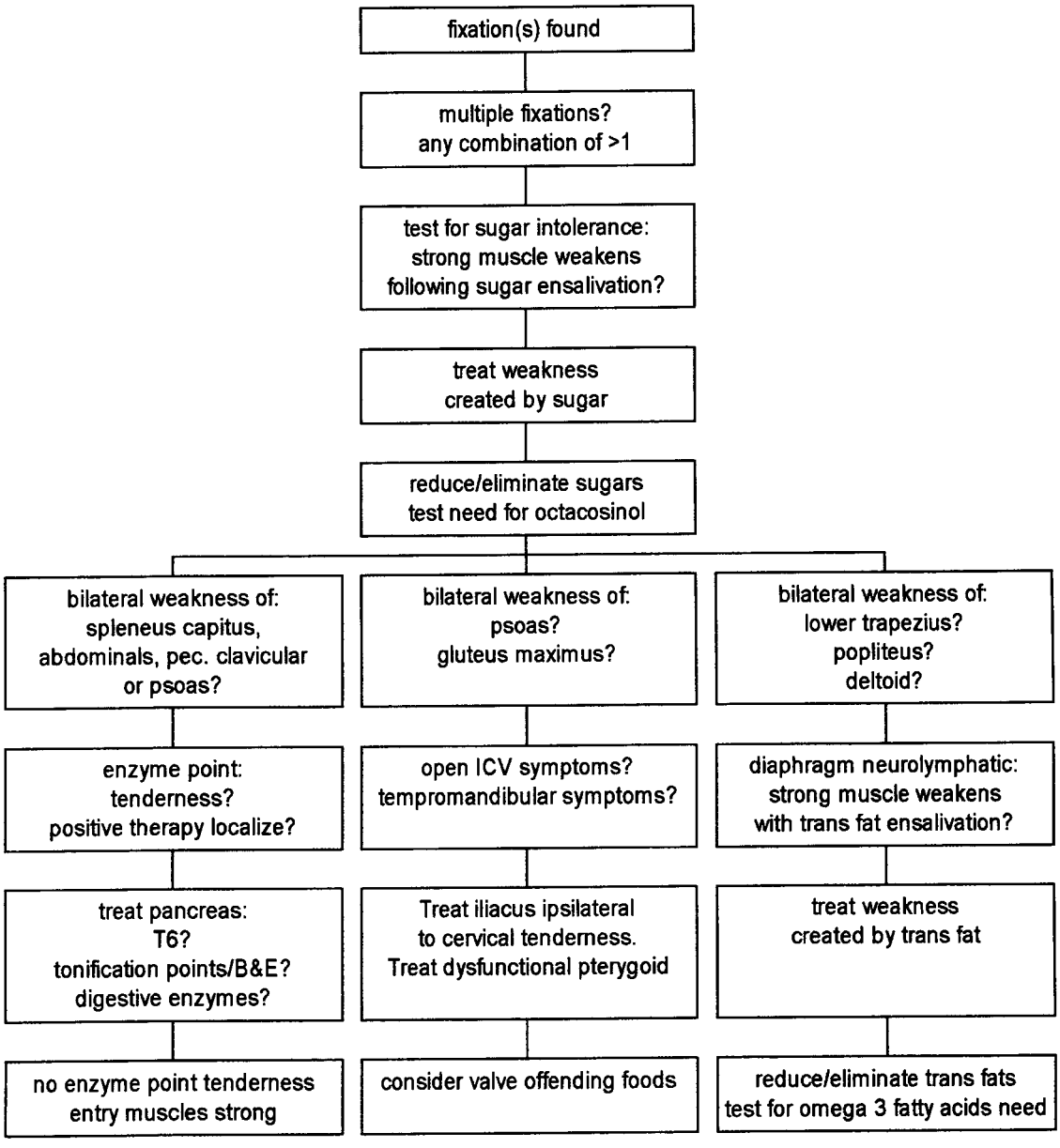
Diagram b **Fixation Flow Chart**



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Division III

Comments

Commentary: Chiropractic is Energy Medicine - AK is Advanced Khiropractic

Bill Conder, D.C.

Body Language/Mind Language

In his *Synopsis* David Walther, D.C. writes: "The body has a language providing information which can lead to the discovery of the cause of health problems; the key is an ability to understand the language." In the next paragraph he continues: "A major aptitude that Goodheart possesses is an ability to decipher the language the body presents."[1]

The body language referred to here is the expression of the body that, when properly read, points to functional disturbances that cause or contribute to the patient's manifest symptoms. These expressions include posture and other physical signs, urine and blood tests, orthopedic and neurologic indicators, and results of applied kinesiology muscle testing.

Yet in addition to these expressions is what we might think of as an information storage and retrieval system upon which these more superficial signs depend, by which the apparently different systems of the body-mind inter-communciate, and to which the muscle test can provide access. Dr. Walther's use of the word "language," though he may not have intended it so comprehensively, is neither merely incidental nor coincidental - it is exactly appropriate.

But applied kinesiologists are not the first or the only to use a body language information storage and retrieval system:

Major Bertrand De Jarnette, D.C.'s Sacro Occipital Technique features an evaluation tool he called "mind language." This mind language, in A.K. language, is an indicator muscle test using the deltoid muscle and therapy localization. Mind language is a prominent aspect of SOT: On page 1 of the 1984 *Sacro Occipital Technic Manual* the second paragraph is about mind language.[2] Though in this paragraph Dejarnette says " Mind language is not a muscle test...", I think what he is referring to is the kind of gross muscle test that would be performed by an orthopedist, neurologist, or physical therapist to determine muscle strength only. On page 5 of the *Manual*, Step One in the Category One Procedural Outline is "Mind Language." Dejarnette says, "...this is as accurate as most clinical examinations... offers clues as to the procedures to follow." Pages 24 through 30 give a more detailed expositioin of the value of mind language including a brief method for testing cranial faults. The "arm-fossa test," used in SOT pelvic categorization, is an anterior deltoid indicator muscle test with a specific doctor TL to the inguinal ligament.

Yoshiaki Omura, M.D., Director of Medical Research, Heart Disease Research Foundation, and President of the International College of Acupuncture & Electro-Therapeutics, claims to be the originator of the Bi-Digital O-Ring Test (BDORT) which is a muscle test of groups of hand muscles. In the BDORT, the thumb and one other digit are opposed forming an o-ring; the operator attempts to open this o-ring. The Board for Dr. Omura's Japanese Association for the BDORT includes the founder of Sony and former Deans of Japanese medical schools. In his Greeting at the opening ceremony of the 3rd International Symposium on the Bi-Digital O-Ring Test in Tokyo, he said that with proper study and application of the BDORT any physician can make an accurate diagnosis and provide effective treatment, and that the exact

location of a pre-cancer or cancer can be located and mapped on the body. In this Greeting, he quotes a doctor-friend of his who said that the o-ring test "will remain forever for future generations to use as a means to make progress and revolutions in future medicine." Dr. Omura is not shy in proclaiming his confidence in the BDORT. Moreover, Omura has obtained a U.S. patent on this form of the muscle test.[3]

Roy Martina, M.D. blended Omura's BDORT, Versendahl's reflex points, German doctor Schimmel's VEGAtest, French doctor and acu-puncturist Nogier's ear acu-puncture, homeopathy, some AK odds-and-ends, his own knowledge of bio-energetics, and came up with "Bio-kinesiology" and a powerful technique for prescribing homeopathic medicines he calls "Integra."[4] This technique is as effective using the arm-pull-down test as it is using the BDORT. I attended a seminar of Dr. Martina's a few years ago and observed some participants using a modified Toftness device as an indicator in lieu of the muscle test or the BDORT. Martina has said that his Bio-kinesiology is more accurate than Applied or Behavioral kinesiology because it uses homeopathic filters in its evaluation and supplement prescription.

Behavioral Kinesiology is the work developed by John Diamond, M.D. which uses the indicator muscle test to monitor what he calls an individual's "Life Energy" and the effect on that Life Energy of a variety of influences including food, emotions, environments, even printed symbols.[5] In discussing emotional imbalances and their relationship to the acupuncture meridian system, Dr. Diamond often employs the etymology and exegesis of words that describe emotional conditions.

David R. Hawkins, M.D., Ph.D in his book *Power vs Force* calibrates the level of consciousness of historical figures, cultural artifacts - of everybody and everything.[6] Dr. Hawkins is a psychiatrist and lecturer, and co-authored *Orthomolecular Psychiatry* with Linus Pauling. In his foreward to *Power vs Force* he mentions being influenced by the work of Drs. Goodheart and Diamond. For his calibrations, Dr. Hawkins uses the indicator muscle test.

The January 1998 issue of the *International Journal of Integrated Medicine* published by Menaco Publishing of Albuquerque, New Mexico, features an article titled "Kinesiology for Patients of a General Practice: Empirical Findings," by Hans-Jurgen Schramm, M.D., of Germany. In the article Dr. Schramm says, "Kinesiology is a diagnostic method..." and "...varying electromagnetic potentials can be pin-pointed..." and "Because of its broad range of application, kinesiology promises to become a significant diagnostic tool in the hands of physicians."[7] This is just a sample of the articles that one can find in various homeopathic and acupuncture journals that promotes the use of the indicator muscle test as a diagnostic tool.

Recently, I received some materials in the mail from the American Academy of Neural Therapy/Neural Kinesiology promoting its seminars and products. Neural Kinesiology is a healing technique developed by Drs. Louisa Williams and Dietrick Klinghardt that combines neural therapy and kinesiology methodology; they identify it as a treatment of autonomic nervous system dysfunction. A "Biological Dental Seminar" that featured Hans Lechner, D.D.S., from Germany was promoted in these materials. Dr. Lechner's evaluation protocol includes the use of the "reflex arm technique." The packet of materials from AANT/NK included an order form for hand mode cards and the availability in the near future of a hand mode *dictionary*.[8]

Brent Davis, D.C., in his article in the 1998-1999 ICAK *Proceedings* titled "Holographic Scanning," reports using "a type of leg length check observed in other chiropractic techniques" as his indicator in conjunction with the conceptual framework of his holographic scanning.[9] I know Activator Technique doctors who run the activator protocol in their minds while looking at the patient and are able to evoke the same leg length changes that occur as when the patient actually performs the isolation tests. Some doctors read a word or sentence or verbally state a question as the form of a challenge in conjunction with the indicator

muscle test. Richard Van Rumpt, D.C.'s DNFT also uses the leg length check. I use the leg length check as a general reference to muscle testing - to identify hyperfacilitated muscles, adaptation-causing supplementation, and so on. I think that the leg length check is similar to Goodheart's toe-in evaluation and that both work on the same principles as the muscle test.

There are perhaps dozens of excellent techniques in addition to these that use the muscle test or some type of neuro-physio-muscular feedback as its indicator. Beardall, Versendahl, Walker, Frank, Callahan and many others have developed protocols all of which depend on the outcome of the indicator muscle test. *Common principles are at work in these techniques*: they all function on the yes/no, all-or-none, binary response of neuro-physio-muscular function, and a method of asking questions of the body-mind to which it responds either positively or negatively.

Sound Mind, Sound Body

One may question the use of "language" and "asking a question" as proper metaphors to use in understanding muscle testing techniques. But even in the grossest form of muscle testing, that is simply to determine muscle strength, we are examining as if asking "Is there injury here that will compromise function?" to which test/question a weak muscle is yes or positive and a strong muscle is no or negative. In AK muscle testing we know further that a muscle test can retrieve injury or imbalance from an associated organ, meridian, nutrient deficiency, or reflex as if the cause of the weak muscle is somewhere else in the body-mind. The muscle-organ-meridian-nutrient-reflex complex, in addition to other factors, may be thought of as an analog system injury in which testing its specific muscle retrieves. That testing a muscle can provide a window on otherwise invisible and unmanifest processes in this way is one of the beauties of AK.

Using the indicator muscle test of kinesiology we raise the intensity of the AK muscle test such that it may include any muscle, organ, or meridian globally - any component, known or unknown, of any analog complex - but indicated with a specific TL/challenge/question. *Any* muscle that is strong in the clear, demonstrates autogenic inhibition, and is free of influence from its 5 factors (that is its analog complex) can be used as an indicator muscle. Using the indicator muscle test this way we probe for and retrieve information digitally: 0 or 1, no or yes, negative or positive.

Restating, the analog complex of the muscle that is to be used as the digital, kinesiologic indicator must be free of disturbance or injury before it may be used to probe the analog complex of other muscles, or of the whole body-mind. This suggests further ideas: the analog system has priority over the muscle's digital function; preparing muscles to be indicators can become a treatment protocol in itself; and global or higher order injury or imbalance can be found using the digital indicator muscle test than what is found staying within the muscle's analog complex.

Professor of English Walter J. Ong, S.J. in his book *The Presence of the Word*, says "There is only one point in the knowledge process where the question of truth or falsity directly applies: this is the point at which... we join a subject and a predicate. ...only in predication can truth and falsity be formally tested."[10] In language we test for *truth or falsehood* by interfacing or joining a subject with a predicate and putting it in motion in the form of an articulated question as a sentence or statement. "Yes, this is true" or "No, this is not true" (uncertainty is also a possibility) is implicit in every sentence we read or write or speak. And this is the form of body-mind language as when we question a joint's articulation by testing its muscle.

This body-mind language seems to transcend artificial boundaries (language barriers, if you will) between D.C's, M.D's, Ph.D.'s, and so on. Though its use has come out of the chiropractic tradition its genius is not lost on other professions. Unfortunately, however, this powerful tool is not universally embraced by chiro-

practors. In any case, the neuro-physio-muscular feedback, along with the subluxation and innate, belong to chiropractic and coincide with an holistic understanding of the body-mind and its language. Moreover, recent conventional research steeped in scientific method presents information that can expand our understanding of these traditional chiropractic concepts and expose us as being on the forefront of all of health care, conventional and traditional.

The Water Flows *Outside* of the Hose

Some of us hope to define neurological pathways to prove or confirm for scientific convention what others of us have observed experientially with the muscle test. Unfortunately, those to whom we are trying to prove the validity of muscle testing don't care, aren't listening, and their minds are closed on the issue. In any case, the neurological hypothesis will not be confirmed in its present form.

Conventional neurology suggests that information is transferred in or by the neuron with the conduction of action potentials. Although there is some speculation about bio-chemical neurotransmitters and neuropeptides, the identity of the stuff that comprises the information remains unknown. Conventional modern communications theory, sometimes referred to as the Shannon-Weaver model, proposes the same mechanism: An information source emits a message that enters a pipeline-like container; the message may be modified as it is sent to a destination which receives the message.[11] That both modern neurological theory and modern communications theory believe in similar paradigms may suggest a kind of validation of one or both - or it may mean that the dominant perception in both fields is modified by the same conditioning.

Even chiropractors have used the pipeline concept of nerve information-transmission. Attempting to describe the subluxation as a pinching of the nerve, they suggest the image of the crimping of a hose that has water flowing in it. In other words, a spinal subluxation crimps the hose, or neuron, and the water, or nerve impulse, stops flowing. The information is interrupted on its way to the tissues which are adversely affected by not getting enough of this information. This water-in-the-hose/pinched-nerve concept to explain subluxation has been abandoned by some chiropractors for more general ideas of "joint dysfunction." Oddly, chiropractic neurologists who promote the joint dysfunction concept maintain the notion that somehow information flows in the neuron, even as they trash the crimped-hose image, the subluxation, and other traditional chiropractic ideas.

German neurologists in the 1960's measured and examined changes in the voltage of brain signals that anticipated simple physical movement.[12] This voltage change came to be called the **readiness potential**. The work of Robert O. Becker, M.D.[13] demonstrates the presence of an electric current in perineural cells. This current is characterized as steady-state, polarized, and DC, and emanates an electromagnetic field pattern. This DC appears to reside in the perineural cell system, not in the neurons, and seems to be the source of the readiness potential.

Neuroscientist Benjamin Libet experimented with the readiness potential and found a measurable increase in negative DC that *anticipates even the decision in the brain* to raise the arm, for example.[14] Scientists think this readiness potential prepares the neuron to fire. It is suggested here that the readiness potential *induces neuron depolarization* and that this view presents a priority of function without which neurological activity does not occur. Becker says his research suggests that "the perineural cells were more primitive than the nerve cells and likely represented the more primitive analog data-transmission and control system" and that "...perineural cells were the precursors for the nerve cells."[15] Neurological function depends on the polarized DC and field pattern a priori, sine qua non.

The electromagnetic field emanation is steady-state but comprised of many micro-pulsations. Valerie Hunt

measured continuous frequencies in the 500 to 20,000 cycles per second range on the human body after filtering out brain, heart and muscle frequencies which she says exist at 0 to 250 cycles per second. She attributes these extremely high frequency-low amplitude signals to the presence of an energy field.[16] The field apparently surrounds and permeates the more dense physical body, reacts to changes in and around the body, and is considered by some to be the mind. This analog information repository also is extra-neuronal, that is non-neurological.

The neuron can only depolarize and repolarize, or not, according to the influence of the DC perineural current and field pattern.

In addition, Becker identified and measured what he called a **current of injury** which he defined as *a positive current in a normally negative field* whose presence can be attributed to a break in the current caused by an injury such as a broken bone. Becker discovered that in higher vertebrates this positive current decreased as healing occurred until it became normally negative or, in some cases, slightly more negative than normal. This current of injury provides chiropractors a metaphor to describe subluxation.

"Here's your joint complex dysfunction, right here"

The verb "articulate" means to utter distinctly. "Articulation" means joint or juncture of bones. A joint is where two things or parts come together or are united. This etymology makes the idea of body language more real though it seems coincidental.

The two parts of a joint or articulation are not really connected in a fixed, that is fixated, way but interface to permit appropriate motion, movement, kinesis, expression, utterance. This relationship is observed also in the chemical bond. Scientists, including Linus Pauling, have identified chemical bonding not as a fixed entity but as a resonant, electromagnetic relationship between atoms and molecules.

It is proposed here that a subluxation is analogous to a broken bone and a luxation in the sense that its presence constitutes an interruption of the normal negative polarity pattern of the spine, an extremity, or any polarized system in the body, but that it differs from a broken bone by *an order of magnitude*. Hence, it can be identified as an analog. In fact, the human body-mind is a system of analogous, polarized systems, from the electromagnetic field pattern on a macro- scale to the electron transport and oxidative phosphorylation potentials inside the mitochondria of the cell on a micro- scale. Interruption, weakness, or breaks in any of these potential systems can be identified as a 'current of injury' or subluxation, and may be located using predication in body-mind language and the indicator muscle test.

Becker's current of injury and steady state, polarized, direct current electromagnetic field pattern are literally analogous to chiropractic's subluxation and innate intelligence. "Above - down, inside - out," pithy as it is, describes the direction of the polarized, DC system. That it may have been borrowed from classical homeopathy as good fit and fashion for chiropractic aligns the two modalities in the field of energy medicine. Where chiropractors sensed or intuited the disturbance of the subluxation and the presence of a polarized energy pattern in the human body, Becker, exploring bone healing and limb regeneration, measured these phenomena. This represents great synchronicity and resonance - even poetry, which is a highly propitious expression of language of any kind.

Therefore, we may say that *a subluxation is a positive current that affects the energetics of articulation by reducing induction and, in general, limiting the expression of potential*. This may be said of the spine, any muscle-joint complex, electron transport potential, the thought process, and every other polarized system

of the human body-mind. It represents a truly holistic perception of body-mind function as found nowhere else. The muscle test is a tool for applying this perception.

In human phylogenetic and ontogenetic evolution, gesture precedes verbal utterance as communication; “articulation” communicates the idea of communication in both gesture and words. The walking body communicates the whole body-mind from place to place, and, talking, we articulate ideas and ex-press our minds by shaping and stopping the breath. The chiropractor, by adjusting spinal articulations in the appropriate manner, facilitates the DC information system in the patient, strengthens the negative field, promotes healing of the current of injury/subluxation, and improves the ex-pression of potential of the whole person walking and talking. This is not merely a philosophical statement or a show of belief in some alternative and complementary health-care modality - it is a statement of logical induction based on a natural principle which the human body-mind abides and in which it inheres.

“She moves like poetry.”

Your Body Lies like a Son-of-a-Gun

It has been observed that the human body doesn’t lie. However it seems more accurate to say either that lies and deceptions are as common to the body as they are to the personality, or that the human person, mind, and body often attempt to adapt over time to the truth in bits and pieces which are not wholly true. The latter, being more forgiving and less judgemental, is preferred.

Specific to the muscle test, the muscle that does not exhibit autogenic inhibition is lying. Though the muscle is not functioning properly it appears upon testing it that it is functioning properly, that is the muscle tests strong. We might explain this phenomenon as the muscle’s adaptive response to less than optimal conditions.

This process can be observed in the following way: On a test subject who has removed all electronic devices from his body, find a muscle that is strong in the clear and weakens demonstrating autogenic inhibition. Also determine that there is no influence on the muscle from any of its 5 factors. Therapy localize to all of the subject’s acu-puncture alarm points and note which are negative and which are positive; preferably none cause the indicator muscle to weaken. Place a battery-powered wrist watch on the solar plexus of the test subject and retest the indicator muscle: it will weaken. Allow the watch to remain on the subject’s body for a minute and retest the muscle: it will be strong. However, one will find that upon attempting to elicit autogenic inhibition, the muscle will not weaken approproately (that is, it will lie about its function). And upon retesting the alarm points one will note that with the watch on the subject’s body there will be a change in alarm point indications, most notably that the pericardium alarm point, having been negative, now is positive; testing muscles associated with the pericardium meridian that previously were strong may now be weak. This shifting complex of positive alarm points and muscle weakness/hyperfacilitation is a sign of the adaptive response of the body-mind.

The immediate cause of this adaptive response is the positive current introduced by the watch battery which the body-mind perceives as injury, albeit, in this case, a small injury. In any whole system there can be only one positive or source pole; all other sources of positive polarity and current will be experienced as injury and will require adaptation. The process of identifying the positive current as injury and adapting to it consumes energy, thus devitalizing the whole person and contributing to a fragmented sense of awareness.

This may be extrapolated to the personality to explain our tendency to prevaricate. In other words, a lie is an adaptive response based on one's fragmented awareness. This observation has further implications for the fragmented and fragmenting tendencies of conventional and conventionally applied so-called alternative and complementary medicine.

Kinesiology/Applied Kinesiology

A distinguishing feature of applied kinesiology is its focus on strengthening the weak muscle. It is believed that the weak muscle is the immediate cause of skeletal imbalance, irregular posture, subluxations, inefficient bio-mechanics, and so on, and that it may indicate metabolic disturbances or energetic imbalances within the body. Therefore, the cause of a weak muscle can be manifold: physiological, neurological, psychological, energetic, or kinesiological. The approach to strengthening the weak muscle includes chiropractic techniques, nutritional prescriptions, meridian therapy, reflex-point stimulation, soft-tissue manipulations, and other methods.

Walther in his *Synopsis* describes the indicator muscle test as one "...in which a muscle not known to be associated with a reflex or other factor is used to evaluate therapy localization...."[17] As noted above, Dejarnette may have been the first in recent history to use an indicator muscle test, as in his mind-language and arm-fossa test, to obtain information about the precise nature of a patient's complaints. However, with the development and reputation of applied kinesiology, the indicator muscle test has been adopted by a variety of practitioners and used in a variety of methods as "kinesiology." Hence, the distinction between applied kinesiology and kinesiology is blurred and confused for almost everyone, except applied kinesiologists.

Upon closer inspection a muddy distinction between applied kinesiology and kinesiology is understandable. The indicator muscle test is used in AK and in kinesiology; both use reflex points, nutritional supplements, emotional challenges, and so on. A big difference between them is that in kinesiology the same muscle or muscle group is tested against all challenges, while in AK several or more muscles might be tested for weakness and subsequently strengthened by one or more of a number of factors. In this sense, AK stays close to the correction of musculoskeletal or structural imbalances while kinesiology may be applied to the evaluation of any imbalance anywhere in the body-mind.

Wild geese and Cheap Thrills

Some methods that strengthen the weak muscle may cause the muscle to lie and bring about the side effect of an adaptive response elsewhere in the body-mind.[18] In other words, we may only be moving the current of injury from one place to another, chasing the wild goose, providing cheap thrills and a great show for the patient, but we're not facilitating health. Worse are conventional, complementary, and alternative health care modalities practitioners who do not abide the natural principle of the polarized DC of the human body-mind, and especially those who do not use any form of neuro-physio-muscular feedback; these practitioners are lost or guessing, and doing harm. An holistic doctor is one who perceives the importance of this polarity and the principle it implies, and applies methods to enhance that polarity; this is one whose word is consistent and synchronous with his role. Health means whole.

AKprovides immediate access to the polarized systems of the human body-mind.

Adjusting the Chiropractor's Perception and the Perception of Chiropractic

Kinesiology is present in applied kinesiology as the indicator muscle test. The phenomenon of the neuro-physio-muscular response, as in the indicator muscle test and leg length reaction, is a chiropractic tool that other professions have borrowed and used. The energetics of articulation, which we have just begun to explore, can adjust our perception of chiropractic, health, adaptation, and evolution.

Chiropractic adjustments address musculoskeletal imbalances. But adjustments have an energetic impact on the whole body-mind, which impact can be demonstrated within the context of applied kinesiology and kinesiology protocols, and which eventually will be "validated" by high-tech hardware. The energetics of articulation abide the principles of the polarized current and field pattern - the mind - of each whole person. Ultimately and comprehensively, chiropractic is energy medicine, and AK stands for advanced khiropractic.

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ICAK-Australasia

Abstracts

Cranial Nerve Examination Using Manual Muscle Testing as Functional Neurology

Richard P. Cheyne, D.C.

A method for examining the functional status of cranial nerves is discussed. Procedures using manual muscle testing based on sensory and motor based diagnostic challenges are employed. Challenge procedures for individual cranial nerves are demonstrated. These include mechanoreceptor and sensory receptor stimulation. The cranial nerve or nerves involved help guide the practitioner to the appropriate, cranial, pelvic, spinal, shoulder, foot or dental correction.

Dural Shear

Kieth Keen, D.O., D.C.

A cranial fault which is not in AK textbooks and which may appear only while the patient is in cervical Kemp's position is described. Correction of this fault in cervical Kemp's position reveals subluxations which did not previously therapy localise or challenge. The configuration of the subluxations revealed indicates that this cranial fault causes a unilateral shearing of the dura matar. It's correction assists patients with recurrent problems which do not respond adequately to standard AK treatment.

The Eyes

Frank Marcellino, D.C., D.O., M. Chiro. Sc.

In this presentation, we are basically concerned with a direct mechanical approach to the eyeball, an area of organ correction that has been neglected by most chiropractors using Applied Kinesiology.

Hyoid Treatment With Taping

Hirofumi Nakatsuka, D.C.

This paper presents a new method of Applied Kinesiology treatments for Hyoid muscle imbalance. It will give the maximum therapeutic benefit for the least amount of intervention.

ICAK-Russia

Abstracts

About Peculiarities Of The Effect Of Muscle Functional Weakness

L.F. Vasilyeva, T.N. Chernysheva, V.I. Korenbaum, T.P. Apukhtina

An adequacy of supposition that there are different physiological mechanisms of support of prolonged muscle contraction effort is confirmed by pilot experiment using electromyographic examination of voluntary contraction during manual muscle testing. A possibility of reliable objective evaluation of muscle reaction to problem provocations is shown.

ICAK-UK

Abstracts

A Protocol To Assess the Need For Foot Orthotics and How To Accurately Fit Them

John Pichler, D.O., MBChA.

To evaluate the accurate fitting of foot Orthotics using both pain and Applied Kinesiology as diagnostic tools.

Colour Therapy & Its Uses In AK

Tracy S Gates, D.O., DIBAK

Colour has been used for much longer than previously imagined to determine and alter mood.

Applied Kinesiology and Neurology: Procedures for Functional Evaluation of the Cranial and Peripheral Sensory Nerves

Joseph Shafer, D.C., CCSP, DIBAK

At first glance, understanding the reasoning behind the following test procedures might seem a bit confusing. In reality, once the idea is understood, evaluation of the cranial nerves and peripheral nervous system becomes quite simple.