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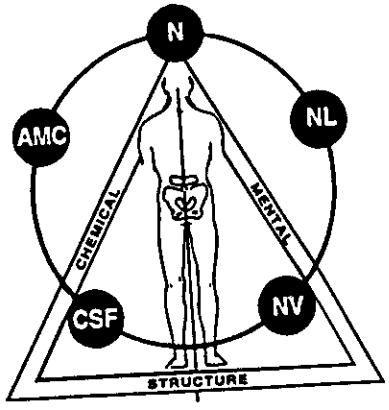
INTERNATIONAL COLLEGE OF APPLIED KINESIOLOGY - U.S.A.

Experimental Observations of Members of the ICAK

Presented August 3 through August 6, 1995
Boston, Massachusetts



A MESSAGE FROM THE CHAIRMAN

Dr. Robert Porzio

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 a Mecca in which members have the opportunity to present their observations and research results. These published works document the first steps toward the furtherance and development of the authors' hypotheses, concepts and procedural techniques. We invite all members to participate in this endeavor in the future.

Congratulations to all of the contributors and a special thanks to Dr. John Heidrich, Publications Committee Chair, and Dr. Daivd Leaf, *Proceedings* Committee Chair, for all of their help. We look forward to seeing you in Boston.

INTRODUCTION

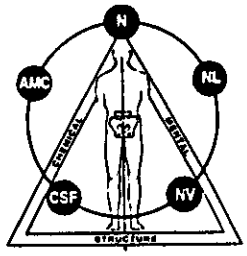
This thirty-seventh collection of papers from members of the International College of Applied Kinesiology-U.S.A. contains 23 papers by 14 authors. The papers will be presented by the authors to the general membership at the Annual Meeting of ICAK-U.S.A. in Boston, Massachusetts, August 3-6, 1995. The 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 Summer 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.



INSTRUCTION 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 conform 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 text-

book references is reviewed at the conclusion of this article. Because of reproduction processes, however, we have provided examples eliminating italics, underlining, etc. All papers must follow these requirements. No font formatting is acceptable. The only exceptions are papers which are Commentaries or Critical Reviews. (See explanation listed below.)

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 art work 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. Photographs must be original black-and-white glossy prints.

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) Each page of the paper should be identified by an abbreviated title, the author's last name and a page number, flush left with a 1 inch margin.

9) The publication standards for the health care 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 2 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).

10) The body of the article should be double-spaced on plain paper. Articles must be flush left. No papers typed on office letterhead will be accepted. Use a plain, nondecorative font (i.e., Times New Roman, New Century Schoolbook, etc.). The manuscript must be an original with dark print, on one side of the paper only, to ensure adequate reproduction in *The Proceedings of the ICAK-U.S.A.* The margins on both sides of the paper must be a minimum of 1 inch, and the top and bottom margins must be a minimum of 1 inch when relating to 8 1/2 inch x 11 inch letter-size paper. European authors should make note of the copy height of the American standard 11 inch paper size, which relates to approximately 28 cm.

11) Authors are encouraged to send articles to the Central Office on computer disk. This will be allowed as long as all formatting procedures mentioned above are followed. Disks should be sent to the Central Office in a padded envelope with the marking "Magnetic Computer Disk Enclosed" to ensure safe delivery. Authors must note on the disk the name and author of the document, and on what type of software it is written (i.e. Microsoft Word 6.0, MacWrite 4.0, etc.).

Please reread, in its entirety, the Instructions to Authors to insure that your paper will be suitable for publication.

Manuscripts that do not meet the above qualifications will be returned to the author, with recommendations for bringing the paper under ICAK-U.S.A. guidelines for possible future publication.

The articles to be published should be sent to the Publications Committee in triplicate (the original and two copies), c/o ICAK-U.S.A., PO Box 905, Lawrence, KS 66044-0905, (913) 542-1801, FAX(913) 542-1746.

APPLIED KINESIOLOGY STATUS STATEMENT

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

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

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

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

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

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

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

On the basis of response to therapy, it appears that in some of these conditions the primary neuromuscular dysfunction is due to deafferentation, the loss of normal sensory stimulation of neurons due to functional interruption of afferent receptors. It may occur under many circumstances, but is best understood by the concept that with abnormal joint function (subluxation or fixation) the aberrant movement causes improper stimula-

tion of the local joint and muscle receptors. This changes the transmission from these receptors through the peripheral nerves to the spinal cord, brainstem, cerebellum, cortex, and then to the effectors from their normally-expected stimulation. Symptoms of deafferentation arise from numerous levels such as motor, sensory, autonomic, and consciousness, or from anywhere throughout the neuraxis.

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

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

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

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

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

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

An applied kinesiology-based examination and therapy are of great value in the management

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

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

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

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

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

Approved by the Executive Board of the International College of Applied Kinesiology-U.S.A., June 16, 1992. Status Statement will be submitted to the International Council for review.

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DIVISION I - INFORMATIVE PAPERS

MOVEMENT PATTERN ANALYSIS OF HIP EXTENSION

William Lerner, D.C.

ABSTRACT

Active hip extension is a useful tool in the functional assessment of muscle coordination patterns and can be used as a screening tool for subluxations, fixations and reactive muscles affecting the gluteus maximus.

INTRODUCTION

In standard Applied Kinesiology diagnostic testing muscle imbalance is most commonly identified by three forms of analysis, they are: static postural analysis; gait analysis; and temporal sphenoidal analysis[1]. Vladmir Janda, M. D., a neurologist from Czechoslovakia has developed a series of functional tests to evaluate muscle coordination or activation sequence during performance of key movement patterns. Janda used these tests to evaluate which muscles are overactive or "tight" and which muscles are inhibited. Janda treated these imbalances with joint mobilization, muscle lengthening and proprioceptive retraining using balance and wobble boards, trampoline exercises and the wearing of sandals with hemispheres attached to the soles to help restore proper coordination and proprioception[2 3]. This author has found that these tests are a good screening tool that allow the examiner to evaluate joint dysfunction and reactive muscles.

DISCUSSION

Muscles are the medium through which central motor commands or reflex spinal activity will compensate for any disturbance. Certain muscles will typically react when specific joints are injured or dysfunctional. Janda has identified those muscles that tend to become hypertonic as primarily postural muscles and those that have a common tendency to become hypotonic as primarily phasic. Those that tend toward hyperactivity are the gastrocnemius, soleus, hamstrings, adductors, rectus femoris, tensor fascia latae, psoas, sacrospinalis, quadratus lumborum, pectoralis, upper trapezius, sternocleidomastoid, suboccipital and masseter muscles. The phasic muscles which tend toward hypoactivity are the following: tibialis anterior, gluteus maximus, gluteus medius, rectus abdominus, lower and middle trapezius, scalenii/longus colli, deltoids and digastric muscles[4]. These muscles are often grouped as paired antagonists and appear to be effected by Sherrington's Law of Reciprocal Inhibition. For example, a hyportonic psoas muscle can neurologically inhibit the gluteus maximus.

When muscular or joint imbalance are present the patient's performance of certain stereotypical movement patterns will be altered. When a movement pattern is altered the activation sequence or firing order of different muscles involved in a specific movement is disturbed. The prime mover may be slow to activate while synergistic or stabilizers substitute and become overactive. This abnormal performance may be self perpetuating and can become ingrained into the central nervous system via cerebellar pathways, these abnormal patterns hence become "memorized.[5]"

PROCEDURE

One of the most important movement patterns to evaluate is active hip extension. With the patient prone have the patient extend their hip with the knee held in extension. The doctor should observe the activation of the hamstrings and the gluteus

maximus first, the contralateral lumbar erector spinae second and the ipsilateral erector spinae third. The test is positive if the erector spinae contracts before the gluteus maximus. In extreme cases, the contralateral neck extensors may fire before hip extension takes place.

Once faulty hip extension is identified Applied Kinesiological evaluation is very useful in finding the underlying cause of the abnormal movement pattern. Subluxation and fixation patterns in the lumbosacral, thoracolumbar and upper cervical should be ruled out first. Walter Schmidt's Movement versus Imagery Technique[6] is very useful in finding the primary subluxation in these cases. Reactive muscle patterns between the gluteus maximus and psoas, sacrospinalis, neck extensors, quadriceps and hamstrings should be diagnosed and treated[7]. Finally fascial involvement of the gluteus maximus should be evaluated and treated either by the Travell[8] or Jones Technique[9].

CONCLUSION

Examination of movement patterns is another way to evaluate muscle interplay and imbalance in patients. It is especially useful in diagnosing difficult patients where all muscles appear intact upon manual muscle testing. Examination of hip extension is a useful screening tool that can help evaluate the entire spine and it's commonly associated muscles involved in flexion and extension quickly and effectively.

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HIP ABDUCTION AS A SCREENING TOOL FOR REACITVE MUSCLES

William Lerner, D.C.

ABSTRACT

Active hip abduction is a useful tool in evaluating muscles affecting the gluteus medius.

INTRODUCTION

Movement pattern analysis as described by Vladmir Janda[1] is another method for diagnosing muscle imbalance. This author has found that active hip abduction is a good screening tool to find reactive muscles[2] affecting the gluteus medius.

DISCUSSION

The prime movers involved in hip abduction from the side lying position are the gluteus medius and the tensor fascia lata. The quadratus lumborum serves to stabilize the pelvis[3].

PROCEDURE

1. With the patient side lying with the lower knee flexes and upper leg extended place the pelvis in a slightly untucked position.

2. Have the patient raise the upper leg into abduction and hold for two seconds.

3. Note if there is limited range, any shaking or twisting, and any hip flexion or hip external rotation, excessive hip hiking or posterior rotation of the upper ilium. If any of these occur it is a positive test of gluteus Medius inhibition[4].

4. If there is excessive hip flexion, rule out a psoas muscle or rectus femoris that is over reactive or requiring fascial work[5 6].

5. If there is decreased range of motion rule out an adductor that is over reactive or requiring fascial work.

6. If there is excessive flexion with external rotation rule out a tensor fascia latae that is over reactive or requiring fascial work.

7. If there is excessive rotation without flexion rule out a piriformis muscle requiring fascial work or is over reactive.

8. Examine the gluteus medius for the five factors of the IVF, fascial involvement, origin and insertion work or muscle spindle work. Strengthening exercises for the gluteus medius are usually given. It is very important for the patient to perform these exercises without over recruitment from the other muscles. To accomplish this have the patient perform hip abduction with the leg slightly internally rotated. This will isolate the fibers of the gluteus medius and prevent over contraction of the tensor fascia latae.

CONCLUSION

Movement pattern analysis is another tool that can be easily utilized and incorporated in applied kinesiology practices to help identify and treat structural muscle imbalances.

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SOMATIC THERAPY AN APPLIED KINESIOLOGY APPROACH

Douglas P. Hetrick, D.C.

ABSTRACT

The following paper is a description and an explanation of muscle testing procedures to determine the need for the application of somatic therapy.

INTRODUCTION

Somatic therapy is a process of neuro muscular re-education that was developed by Thomas Hanna. Hanna was inspired by his association with Moshe Feldenkreis as well as others.

I was exposed to this work in a seminar taught by Jim Dreaver, D.C. I highly recommend those interested to take this seminar. It is a simple and effective technique in neuromuscular therapy. Typically the assessment for the need for this type of therapy is based on observation, posture analysis, gait and palpation. Due to my experience with applied kinesiology, I always prefer a testing procedure that at least to some degree will confirm the appropriateness of any therapeutic efforts I apply. As a result of this perspective, I experimented and found a way to test for somatic therapy to a particular muscle group.

DISCUSSION

The basic process of somatic therapy is directed to re-educate or integrate neuro-muscular communication from the cortex, cerebellum and undoubtedly other levels of the brain, to muscle function. This is another potential method to address the problem of deafferentation.

For a brief description of somatic therapy, I will use the gluteus maximus as an example. Patient is prone, bring the thigh into maximum extension, instructing the patient to become fully aware of contracting that particular muscle against the doctors pressure. Maintain this for five seconds, then instruct the patient to allow the muscle to smoothly lengthen with control in response to the doctors pressure, for approximately 20% of its range of motion. Then have the patient re-engage the muscle fully, allowing it to re-extend the thigh about 5%, holding full contraction three to five seconds and again smoothly, with control, allowing the muscle to lengthen for another 20%. Then re-engage full contraction at that stage of ROM for three to five seconds, and again the patient smoothly allows the muscle to lengthen. This process of the patient fully engaging and controlled release of the muscle at various stages of the muscles ROM is repeated until the thigh is fully relaxed on the table. Then have the patient fully extend the thigh against the doctors pressure for five seconds, after which the patient is instructed to fully release the muscle, so that it rests on the table, all in one motion. Then the doctor proceeds to stimulate the muscle by tapping or rubbing, after which the patient is instructed to contract the antagonist group of muscles, that is to pull the anterior part of the thigh down towards the table for a few seconds.

This technique is very gratifying to its response to increased range of motion, muscle relaxation and bio-mechanical balance of associated structures. It is also too time consuming to apply to muscles that may possibly require other procedures.

In an effort to establish some type of muscle testing protocol, I followed an intuition to have the patient visualize a muscle test immediately after performing the muscle test, testing another muscle. As the patient visualized doing the test just performed, I tested a different muscle, i.e., test the gluteus maximus, have patient visualize gluteus maximus function, as I tested the hamstring. At times this would fail and illicit a hamstring weakness as the patient visualized the muscle test. This response would generally not be present without first activating the muscle group by testing prior to visualization. I then apply the somatic therapy procedures, after which the above described testing and post testing visualization would effectively be processed by the patient and no hamstring weakness would result.

There would also be an associated decrease in pain if pain was associated with the function of the muscle test, ROM would increase, along with a good clinical response of the patient in general. Further evaluation of this technique should possibly be pursued by those with a deeper grasp of the complexities of modern neurological knowledge. It appears obvious that it does increase conscious control of the muscles involved as well as integration of cerebellar function.

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APPLICATION OF SOMATIC THERAPY TO GAIT IMBALANCE

Douglas P. Hetrick, D.C.

ABSTRACT

Certain types of gait imbalance may be treated effectively with somatic therapy. This can be determined by muscle testing with associated post test visualization.

INTRODUCTION

After some experience using somatic therapy with the diagnostic muscle test procedure of post testing visualization, I was confronted with a patient that exhibited lumbo sacral pain aggravated by the activity of walking. After the utilization of the process there was a marked improvement in gait function.

DISCUSSION

To apply the testing procedure to gait imbalance, I test all of the gait muscle relationships in the supine position. Test opposite leg and arm flexors, extensors, abductors, adductors and the pectoral sterna vs. Psoas. If positive, treat appropriate acupuncture points. After establishing that the gait muscle relationship test strong, retest each group and then have the patient immediately visualize the muscle test as you test a specific muscle, i.e. , pectoralis clavicular. If the pectoral is clavicular tests weak with the visualization, that muscle group would receive the application of somatic therapy. If for instance, the visualization of gait flexors produced weakness, have the patient bring opposite arm and leg flexors into contraction directing the patient to become aware of what they are doing as they contract arm and leg muscles against Dr. 's pressure for five seconds. The patient then allows some decrease of their resistance as they allow the muscles to lengthen smoothly to the Dr.'s pressure to approximately 30% of range of motion. Then the patient is instructed to fully re-engage the muscles for three to five seconds and again to smoothly allow the muscles to lengthen with control another 30% and re-engage the muscle for three to five seconds and continue the smooth controlled lengthening of the gait muscles until the limbs are relaxed on the table.

The arm and leg are fully flexed again against Dr's tension for five seconds, after which the muscles are released smoothly under Dr's pressure down to a relaxed state on the table. The Dr. then stimulates the muscle groups by tapping or rubbing and then activates the antagonist gait muscles, in this case the extensors. After this process the post test visualization will be negative.

If the extensors are found to be positive apply the therapy in a prone position, all other gait relationships are easily done supine.

CONCLUSION

Somatic therapy used with the gait muscles provides one more method of neuro muscular re-education to further enhance the balance of certain types of gait imbalance. The post test visualization offers some degree of assessment for the appropriate application of the technic.

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A COMPARISON OF NUTRIENT BLOOD TESTS WITH ORAL NUTRIENT MUSCLE TESTING

John K. Moore, D.C., CCN

ABSTRACT

A new method of assessing nutrient status via blood testing was introduced just over a year ago by Spectracell Laboratories Inc. in Houston, Texas. This method claims to be a look at how well specific nutrients function in the body by intracellular analysis of lymphocytes. This author thought a functional nutritional blood test may have a high degree of correlation with the oral nutrient muscle testing used by many applied kinesiologists.

INTRODUCTION

Two blood tests were run on a sampling of 6 patients. These were the EMA and SPECTROX tests, and will be described later. The patients ranged from 30 to 65 years of age. 3 men and 3 women were tested.

How the EMA (essential metabolic analysis) test works. Lymphocytes are placed into a chemically defined tissue culture media and stimulated to grow. The growth response is measured by the incorporation of radioactive thymidine into the DNA. Growth is measured by counting the radioactivity within the lymphocytes. This is the patients maximum, or optimal, growth response. This test is then repeated while removing an essential nutrient from the growth media. The cells are again stimulated to grow and the growth is now dependent upon the cell's reserve of the removed nutrient. These results are expressed as a percentage of the optimal growth response. Spectracell claims this indicates how well the cell is using a nutrient and therefore determines the availability and utilization of the nutrient within the lymphocyte. Lymphocytes are an excellent system to monitor nutrient status, as the metabolic systems encompass carbohydrate, lipid and protein metabolism. A reference range has been established by Spectracell Laboratories and a graph is furnished with the results that express the patients values as fitting somewhere in between 1 and 100%. Being below the 75th percentile is noted as a deficient state. Nutrients tested for are as followed:

BIOTIN
CALCIUM
ZINC
MAGNESIUM
THIAMINE
RIBOFLAVIN
B6
PANTOTHENIC ACID
NIACIN
FOLIC ACID
CHOLINE
INOSITOL
B12

How the SPECTROX (antioxidant function) test works. This test is also done on lymphocytes by addition of a peroxide to induce oxidative stress into the growth media. Lymphocytes growth response with the added peroxide is reported as a percentile from a reference range. Values below the 25th percentile are reported as deficient. Please note this test is designed to asses overall antioxidant function and not individual antioxidant vitamins such as C, E, or selenium.

PROCEDURES

For the EMA testing patients were screened via the T.S. line for muscle inhibition. Manual muscle testing was performed on a selected muscle that was found to have both a gamma 1 and gamma 2 inhibition. Nutrients were placed on the tongue of the patients being tested for approximately 5 seconds and the muscle was re-tested by both a gamma 1 and a gamma 2 muscle test. If the muscle strengthened either to gamma 1 or gamma 2 testing a

positive response was noted. As it turned out in all 6 patients tested, whenever a gamma 1 response was positive so was a gamma 2 and visa versa. This worked out well for being able to delineate a positive muscle testing response. The procedure to cross check the SPECTROX test using A.K. was very simple. A strong indicator muscle was found and was screen for inhibition immediately after the patient sniffed clorox. This screening test was chosen by the examiner based the work of Walter Schmitt D.C. Dr. Schmitt found many patients he suspected of being under oxidative stress would have a normal, facilitated muscle become inhibited when the patient sniffed clorox. I have used this as a general screening tool in my practice for many years and decided to see if this test correlated with a deficient SPECTROX test.

In both of these tests the blood draws were done within 24 hours of the nutrient muscle testing. Two of the blood draws were done before the muscle testing and 4 were done after. No treatment of any kind was performed before the nutrient muscle testing. Each patient would be described by me as being in "reasonably good health". The nutrients chosen for testing were provided by either Nutri-West or Thorne. In several cases a nutrient from both companies was tested such as folic acid-s (Nutri-west) and folacal (Thorne). In all cases when 1 companies nutrient strengthened the patient so did the other companies. All nutrients were independently taken from a box and tested without the examiner or patient being aware of the nutrient tested.

RESULTS

A total of 11 nutritional deficiencies were reported by spectracell Laboratories on the 6 patients that underwent EMA testing. Manual muscle testing revealed 24 nutrients to have strengthened an inhibited muscle. The correlation of the two tests was very poor. Of the potential 35 deficiencies reported (11 by Spectracell and 24 by Applied Kinesiology) only 3 of them were found by both tests to correlate. Of the 6 patients tested with the spectrox test 2 were reported by the laboratory to be deficient in antioxidant function. Three of these patients showed weakening on the clorox sniff test, however these two tests matched only once.

CONCLUSION

The great discrepancy in positive findings between these two tests shows that this form of nutritional blood work will likely not be the proving ground for applied kinesiology oral nutrient testing that many of us would like to find. I believe both types of testing have their merits and would encourage practitioners to utilize the test that their patients, and themselves, feel most comfortable with. If both tests are used on the same patient it will most likely be confusing as to what course of action to take. Possibly a larger sampling of patients being tested or clearing switching factors or other A.K. findings may have shown a better correlation. Further investigation by other A.K. practitioners is encouraged.

DIVISION II - CRITICAL REVIEW PAPERS

AUTOGENIC INHIBITION

A Look at the Importance of the Golgi Tendon Organ

Richard Belli, D.C.

ABSTRACT

The symphony of autogenic inhibition working autogenic facilitation provides the central nervous system with the negative feedback necessary for smooth movement. Autogenic inhibition is one of the neurological components affected by osseous manipulation. Manual muscle testing is a simple and extremely valuable tool in evaluating autogenic inhibition. Dysfunction of autogenic inhibition dysfunction is correctable by components of osseous manipulation.

Golgi tendon organs are the receptor organs for a systematic reflex commonly referred to as autogenic inhibition (AI). AI is the result of firing of the Golgi tendon organ (GTO) and resultant disynaptic post synaptic inhibition of the involved muscle. AI involves the afferent Ib fiber from the tendon organ, internuncial neurons in the spinal cord, descending modulation, and efferent fibers back to the involved muscle. AI in conjunction with autogenic facilitation, the muscle spindle bundle (MSB) provides a negative feedback system that allows the central nervous system to provide the synchronous contraction of muscles necessary for smooth movement. Dyskinesia of joints as a result of AI dysfunction are commonly a factor in musculoskeletal pain, as dyskinetic joints result in increased firing of the nociceptive system.

Failure of AI can have an adverse effect on the outcome of osseous manipulation. One function of high velocity osseous manipulation is fast stretch of the Golgi tendon organ of the involved hypertonic muscle which depolarizes the Golgi tendon organ and induces AI of the previously hypertonic muscles. Fast stretch of the muscle and subsequent depolarization of the GTO results in disynaptic post synaptic inhibition of the hypertonic muscle, resulting in normalization of motion of the involved segment and resetting of the tone of the involved muscles and normalization of afferent mechanoreceptor activity . If normal AI does not take place, the opposite effect occurs, potentially resulting in iatrogenic effects of the manipulation. Manual muscle testing can be a valuable tool in evaluating AI. AI can be induced by digital manipulation of a tendon associated to the muscle to be tested. If AI does not function correctly, the inappropriate response will be demonstrated with manual muscle testing. Often failure of AI is the result of the loss of normal supraspinal modulation as well as segmental cord dysfunction. Bearing in mind that osseous manipulation (Wyke 1985) has a supraspinal effect one can see that manipulation is a valuable tool in normalizing AI. Manipulation will also normalize segmental cord dysfunction by normalizing mechanoreceptor afferent activity from the involved segments.

DISCUSSION

The GTO is an encapsulated stretch receptor, generally classified as a mechanoreceptor (Fitzgerald 1985). The GTO are graded pressure receptors that are depolarized by being squeezed by adjacent tendon fibers when tension develops. GTO receptors are not stretch receptors, but are pressure receptors that are the functional part of a mechanism to detect stretch. This prevents development of too much tension in the muscle (Snell 1987).

The GTO does not differentiate between tension provided by contraction or stretch of the muscle (Burt 1993), however, it's primary task is to measure the effort of the individual muscles

on the basis of returning signals and to maintain muscle tension within physiological limits by inhibiting impulses (Duus 1989).

The major difference between the function of the GTO and the MSB is that the spindle detects changes in muscle length while the tendon organ detects muscle tension (Guyton 1991). The GTO very specifically monitors the tension generated by muscle contraction and it is currently considered to play an active role in the process by which the nervous system controls motor activity (Nolte 1993) .A muscle, by virtue of these two types of receptors, can simultaneously monitor it's own length and tension (Nolte 1993).

The tendon organs are exquisitely sensitive and respond to contraction of even one muscle fiber (Snell 1987). And provide the nervous system with instantaneous information on the degree of tension in each small segment of muscle (Guyton 1991). The tendon organ has both a dynamic and static response. The tendon organ responds very intensely when the muscle tension suddenly increases (dynamic response), but within a small fraction of a second settling down to a lower level of steady-state firing that is almost directly proportional to the muscle tendon (static response) (Guyton 1991). The GTO are necessary for fine motor control, leading to decreased contraction on contact (Burt 1993). In other words, in the process of touching something, the tension on the tendon organ is immediately increased making delicate tactile movements possible.

The afferent fibers to the encapsulated receptor organ are large, fast conducting type Ib. (Burt 1993). The Ib afferents exert negative feedback in homonomous motor nerves, in contrast to the positive feedback exerted by IA fibers from MSB. This effect is called AI, and the reflex is disynaptic. There is an accompanying reciprocal excitation of motor neurons supplying antagonist muscles (Fitzgerald 1985). The influence of the Ib inhibitory interneuron on motor neuron excitability depends on the combined input from many sources, both central and peripheral. Thus every muscle is under the control of two feedback systems: 1). its length is controlled by a system in which the MSB act as the measuring sensors, and 2.) it's tension is controlled by another system, in which the GTO are measuring sensors (Duus 1989). In addition to tendon organs the Ib interneuron receives IA from muscle spindles, low threshold cutaneous afferents, and joint afferents, as well as both excitatory and inhibitory input from various descending pathways.

When tension on the tendon becomes extreme, the inhibitory effect from the tendon organ can be so great that it leads to a sudden reaction in the spinal cord and instantaneous relaxation of the entire muscle. This may be a protective mechanism (Guyton 1991). This function is also vitally important in high velocity osseous manipulation. When adequate velocity is achieved during manipulation, the AI mechanism shuts the hypertonic muscles off and apparently resets the baseline tone of the hypertonic muscles resulting in a return of normal motion to the involved segment. When baseline motion is returned than appropriate baseline mechanoreceptor activity is emitted from the motion segment to the central neuraxis. It is second order joint mechanoreceptor afferents from dorsal columns that maintains the central integrative state of the thalamocortical system. When the AI system is not functioning properly this whole aspect of mechanoreceptor activity is compromised resulting in a compromised central neuraxis.

Although AI is important as a protective mechanism, it's main function is to provide the central nervous system with information that can influence voluntary muscle activity (Snell 1987). Another function of the GTO reflex is to equalize the contractile fibers of the separate muscle fibers. These fibers that exhibit excess tension become inhibited by the reflex and that exhibit too little tension get no inhibition (Guyton 1991). The synchrony of the two result in homonomous smooth muscle contraction.

The local cord signal excites a single inhibitory Ib interneuron that in turn inhibits the anterior horn cell (Guyton 1991). Both GTO and MSB generate afferent signals that are important for spinal cord reflexes, supraspinal reflexes, and cerebellar function.

(Burt 1993). The GTO transmit signals both in local areas of the cord and through long fiber pathways such as the spinocerebellar tracts into the cerebellum and through still other tracts to the cerebral cortex. Some of the information generated by these receptors reaches the cerebral cortex via the dorsal column-medial lemniscal system and contributes to a conscious awareness of posture and movement of limbs and the body in space (Burt 1993).

Our upright posture is maintained by a servomechanism based on feedback circuits that continuously carry action potentials for retaining the muscle tone necessary for standing and walking (Duus 1989). Tendon organ input provides a negative feedback for regulating muscle tension parallel to the negative feedback from muscle spindles that measures weight and length or stretch. The GTO determines stiffness and is important for posture and function (Burt 1993). The dorsal spinocerebellar tracts carry instantaneous information from both the MSB and the GTO directly to the cerebellum (Guyton 1991). Additional pathways transmit similar information into the brain stem reticular regions and also all the way to the motor cortex (Guyton 1991). The combination gives us maintenance of upright position, a sense of the position and the appropriate autonomic responses.

Due to the inherent design of the tendon receptor mechanism they can be conveniently depolarized. The receptors are pressure receptors that are depolarized by the squeeze of the tendon fibers on them. Therefore, to depolarize them you simply squeeze in the area of the musculotendinous junction of the muscle that is to be tested. The normal sequence of response should be a weakening or neurological inhibition of the muscle then an immediate refacilitation on the next subsequent contraction.

As the term AI implies, there is an function of muscular inhibition that leaves us with the ability to perform a manual muscle test as an analysis tool. Once AI is induced by depolarizing the receptor organs in the tendon of a given muscle, there should be a normal neurological inhibition of the motor neurons associated to that muscle. This is easily and conveniently tested with a manual muscle test. Once the manual muscle test has been executed, the Renshaw cell mechanism that inhibits the inhibitory function of the interneurons that induced the initial motor neuron inhibition, inhibits the inhibitory neuron leading to refacilitation of the pathway (Fitzgerald 1985, Guyton 1991). In other words, Renshaw inhibition of the inhibiting interneuron allows the motor neuron to refacilitate and the AI cycle is complete.

CONCLUSION

It is easy to conclude that AI is important for smooth movement, posture, fine motor control, and protection of the musculoskeletal system. AI is also a fundamental component of the high velocity osseous manipulation, and if it is not functioning correctly treatment may be rendered ineffective, or even worse, involve iatrogenic problems. With this in mind it is easy to conclude that examination and treatment of the AI system should be a basic tool in the Applied Kinesiology treatment protocol.

PROCEDURE

1) Apply 2-3 pounds of pressure to the musculotendinous junction of a strong indicator muscle, then immediately test it. The muscle should demonstrate neurological inhibition for one contraction only.

2) If the muscle demonstrates a pattern other than neurological inhibition for only one contraction, correct fixations of any motion segments that will therapy localize using an intact indicator muscle, then re-test.

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STRESS WITHOUT DISTRESS THROUGH NUTRITION

John W. Brimhall, D.C.

ABSTRACT

Ongoing research is necessary to upgrade the structural and nutritional approaches we use in practice. This paper couples old information with new research and clinical trails to formulate new improved results.

INTRODUCTION

Stress is a constant fact of our lives. To have stress without distress takes nutrition as well as structural balancing. This paper couples the body-mind connection by combining nutrient synergists that prevent and hopefully reverse distress.

DISCUSSION

In 1977 I wrote a paper on the use of glandulars for the reversing of the general adaptive syndrome. Hans Selye in his book "Stress With Out Distress", showed his mastery of research in the effects of stress on the glands in the body. He found excessive work, play or emotional upset would cause exactly the same reaction. He found the adrenal cortex to hypertrophy, the shrinking of the thymus gland and the lymph nodes took place simultaneously as well as the appearance of gastro intestinal irritation and ulceration in a very short time.

There are three stages of the general adaptive syndrome: 1. The alarm reaction, 2. The stage of resistance, and 3. The stage of exhaustion. Dr. Selye made an interesting analogy to our childhood, our adulthood and then on to senility. We have found the specific use of glandulars and cofactors to minimize long term damage and help rebuild past insults. The glandulars to be used are adrenal, to help offset the adrenal hypertrophy and later atrophy in the final stages. The stomach substance to help the damage to the stomach lining, thymus and spleen to help replenish the diminution of those tissues and parotid because of the synergistic connection to the entire process.

It has also been found that the parotid glands are important to tag other nutrients as we in-salivate them. The parotid is also quite helpful in the detoxification of heavy metals from the body. Dr. Versendaal's research, has found toxins in the system of any kind and particularly heavy metals respond to parotid.

The co-factors that are necessary to allow for repair and healing are pantothinic acid in the form of (calcium and d–panatothinate), vitamin C, Vitamin B6, Vitamin B2 (riboflavin), niacinamide, proanthocyanidins, along with zinc, iodine and chromium.

It is important that the trace minerals accompany the glandulars to allow enzymatic activity to be adequate. Enzymes are the building blocks of our existence. The major building blocks of sea water are sodium, potassium, calcium, magnesium combined with chloride, phosphate and carbonate to provide the general matrix for our cells.

In Dr. John A. Myers opinion, metabolic disease and health loss have happened most abundently from our loss of mineral elements. He states depletion of our soils have yielded plants with almost no trace minerals. He further states, that washing, soaking and boiling of vegetables further reduces the mineral elements. We then discard our cooking water which further depletes our availability and the spring water we use to get deep in the wells is now no longer how we acquire our water. The foods that we use to get our vitamins and minerals from have also been cooked, microwaved, pre-packaged and devitalized further.

So not only should we add the glandulars to rebuild glandular deficit from stress but also the trace minerals necessary to

allow the body to utilize them in repair and in normal function.

Trace minerals act as catalysts and regulators in both the plant and the animal kingdom.

We are told that stress is the cause of many conditions or the aggrevator. Yet stress comes to us physically, emotionally and nutritionally in a constant barage. The illustration in this article shows any stressor causes the autonomic nervous system to yield stomach irritation almost instantly with the hypothalamus-pituitary action overstimulating the adrenal cortex and causing hypertrophy and the thymus as well as other parts of the immune system diminishing in size and function.

The adrenal stretch reflex was demonstrated by Dr. Gereld Deutsch many years ago and still holds constant with any stress patterns effecting the adrenals. You can check any strong intact muscle and stretch it. If it becomes weak, the adrenals need nutritional support. The DSF or the De-Stress Formula will neutralize that action. Postural hypoadrenia will also manifest itself many times by the blood pressure dropping when we stand and or Rogoff's Sign with the pupil dilatation to light, DSF is also indicated. Any other adrenal depletion symptoms is a tipoff to DSF necessity, such as a person getting dizzy when they stand.

Stabilizing the blood sugar sometimes can be difficult with multiple hyper or hypo glycemic symptoms. DSF is indicated on both of the glycemic indexes due to the adrenals action and considering stress can be a major factor in either condition.

Having all the glandulars supported by their cofactors will support both the adrenal cortex and medulla. They will also support the digestive and immune systems.

I have seen in practice, as I'm sure you have, patients that are chronically reversed. Reversal or switching may be indicated as we would normally check in muscle testing by the two point bilateral therapy localization of K 27 or the coccyx area to GV 21. It may also be manifested in the patient that takes vitamin C and gets a cold or someone that takes different nutrients that seem to irritate the gastro intestional track or causes the opposite of the desired effect.

A challenge I developed for this is to check a persons general muscle strength and then giving them sugar. If sugar does not make them weak, give them a natural nutrient, e.g. and apple or banana and see if that weakens them. We have found some people get weak on good food and not weak on devitalized food. We refer to them as chemically switched. We then have them chew the DSF and they will then weaken to sugar and not to the good natural nutrients. This is an over simplification of the test as not all people will show the non weakness to sugar that still show need for the DSF. They may show an allergic reaction to certain foods or sensitivities as it might more correctly be called, which disappears after the ingestion of the DSF.

I have found this combination to be the best adrenal product I've ever used. I also find it the most helpful in any stress related insult. We use it instead of any other adrenal, parotid or general stress product.

As an example, let me give you one case history: Four years ago a man came in with a positive disc was recommend for surgery. He opted for us to do conservative treatment instead.

Treatment went well and he has been stable with only periodic tune-ups as he calls them. That is, until he tipped the scale of stress.

He received a new supervisory job with accompaning emotional stress. To offset the emotional stress, he played racketball. This was within body tolerence until challanged by a younger player in a prolonged match.

Remember stress is interpreted by the body and added together:

+ Emotional Stress
+ Physical Stress = GAS -:- Nutritional Support
+ Nutritional Stress

The persons weak link will usually be the first to show. In this persons case, it was his back. With AK and DSF he made a quick recovery.

I hope with continual nutritional support and a better understanding we can prevent further insult from ever developing.

SUMMARY

Applied kinesiology and current literature, coupled together with clinical trials, gives us ammunitation for reversing the effects of stress. Applied kinesiology also allows us to develope and improve products on an ongoing basis.

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BLOOD SUGAR REGULATION AND VANA-CHROME

John W. Brimhall, D.C.

ABSTRACT

Glucose regulation is one of the major factors that promotes and maintains energy and health. Any wide range from hyper to hypo or dysinsulinsin lays the foundation for dis-ease. The combination of chromium, vanadyl sulfate and the co-factors that allow them to work are presented as blood sugar regulators with great promise.

INTRODUCTION

We examined the patient kinesologically and by darkfield examination. We compared improved muscle strengthening by oral nutrient challenge to improvements of the blood picture on darkfield examination.

DISCUSSION

We have been monitoring the effects of nutrients on muscle strength by oral ingestion for many years. Dr. Goodheart has told us we need to measure our tests and results by as many other comparisons as possible. This past year we have been checking applied kinesiology findings to regular glucose levels on standard blood exam and darkfield examination.

On microscope examination we have identified liver stress, glucose mismetabolism, undigested fats, poor protein digestion etc. We then challenged the results of nutrition by oral ingestion of nutrients. Then we compared this persons follow-up darkfield in 15 minutes after ingestion of the nutrients.

We altered formulations of products until we accomplished both positive muscle test challenge and positive darkfield improvements. A new product was developed, Vana-Chrom, after this manner of testing. A literature search was used for helpful nutrients and clinical trial proved their usefulness.

Vanadyl Sulfate plus chromium picolinate, with the co-factors of glucose metabolism may prove to be a revolutionary breakthrough.

Oral use of vanadyl has been shown to produce a long lasting optimum balance of blood sugar and glycogen. Vanadyl has an effect on glucose production, storage, and breakdown. It increases the glycogen synthesis in fat cells, making more glucose available in the blood for transport to the working tissues such as muscles and potentially reducing body fat.

Vanadyl increases the rate of glucose transport relieving the insulin mechanism. This allows for greater energy production.

Research shows by blood sugar normalizing with proper diet and exercise, one could loose fat and protect lean muscle mass. Vanadyl sulfate aids in the synthesis and retention of muscle as the protein from muscle becomes less available for fuel.

Research indicates there is more energy providing glucose by vanadyl increasing the uptake of nutrients in the active working muscle cells. Less insulin is needed as the vanadyl as well as the chromium picolinate cuts down insulin requirements.

Insulin uptake is enhanced by these cooperative actions, plus the co-factors of glucose metabolism are added for the synergistic effect. Also, cholesterol lowering effects can be expected due to the synergistic effects of these bioavailable nutrients working as a team.

Blood sugar normalization has been prolonged and facilitated in studies using vanadyl.

Chromium has been hailed as a blood sugar regulator for many years. It has proven to be effective in blood sugar normalization and cholesterol reduction. It's been found to be in large amounts in the heart cells on autopsy of those that do not die of blood vascular accidents as compared to those that do.

Chromium effects on glucose metabolism has been shown to potentiate the action of insulin at the cellular level. To convert chromium to usable glucose tolerance factor it needs niacin and other co-factors which have been added to this product.

Biotin is added since it is necessary in the initial process of glucose utilization by the cell in phosphorylation. Biotin is also suspected to play a stabilization role in blood sugar levels by biotin-dependent enzymes acetyl Co A carboxylase and pyruvate carboxylase.

Pyridoxine (vitamin B6) is frequently found to be low in diabetics. Those with peripheral neuropathy are especially suspect. Pyridoxine is necessary for many metabolic processes.

Magnesium has shown in many studies to be low in the American diet. Some studies show 80-85% of women in the United States consume less than the conservative RDA for magnesium. Magnesium deficiency is suspect in insulin resistance and low magnesium has been associated with hypoglycemia.

Zinc is associated with pancreatic B cells synthesis of insulin and insulin binding to liver and adipose tissue cells. Many studies show the American diet to be deficient in the intake of zinc.

Ascorbic acid (vitamin C) has been found deficient in many diabetics. Volumes are written on C and its many uses.

Maganese is a cofactor in certain enzymes involved in intermediary metabolism of carbohydrates. The pancreas and the pituitary have greater amounts of manganese than other organs.

Vitamin B 12 and foliate are involved in different steps of carbohydrate metabolism and are found lacking in the American diet. Foliate is involved with gluconeogenisis as a cofactor with enzymes in the liver and the small intestine.

Vitamin B 1 is a factor in carbohydrate metabolism and Krebs Cycle. It was found to be helpful in synergistic action.

Selenium is an essential factor as are antioxidants and may be a factor in glucose levels. Depleted soils and artificial fertilizer are lacking such micro nutrients.

The doctor should watch people with low blood sugar that the vanadyl does not cause it to further decrease. We have not personally seen this happen in clinical applications. We have our patients take one Vana-Chrome twice a day during the two meals that contain the most carbohydrates.

Some of the patients tested had to reduce their insulin intake. Darkfield examination showed a marked reduction in circulating fats after the nutrient combination was ingested.

Fig. 1 shows darkfield blood before and after nutrient intake. Many different nutrient combinations have been tested this last year. Vana-Chrome was one of the more effective.

CONCLUSIONS

Applied kinesiology coupled with other examination findings e.g. darkfield microscopy can be very useful. These tools together with using current information available in a literature search have proved to be effective in the production of a very positive blood glucose regulating product.

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PRIMARY MUSCLE TECHNIQUE

Robert A. Rakowski, D.C.

ABSTRACT

The purpose of this paper is to help practitioners streamline their therapeutic efforts by determining the highest priority need(s) of the patient. The technique discussed is an integration of concepts presented by doctors George Goodheart, John Bandy, Walter Schmitt, and Rene Espy.

INTRODUCTION

Patients often present with a myriad of complaints. These may include fatigue, joint aches, constipation and/or diarrhea, headaches, dizziness, nausea, slow metabolism, et cetera. In Applied Kinesiology (AK) we have long recognized that these symptoms are frequently a cascade of reactions arising from a single primary source. Examples of "primary sources" include ileocecal valve dysfunction, adrenal stress syndrome and liver detoxification problems. This paper offers a protocol to determine the primary source for any presenting symptom picture.

DISCUSSION

The procedure to be utilized will be presented in both outline and flowchart form. Once the patients primary problem has been determined, efforts should focus on reducing specific mechanical, chemical and psychological stresses that effect that muscle or organ system.

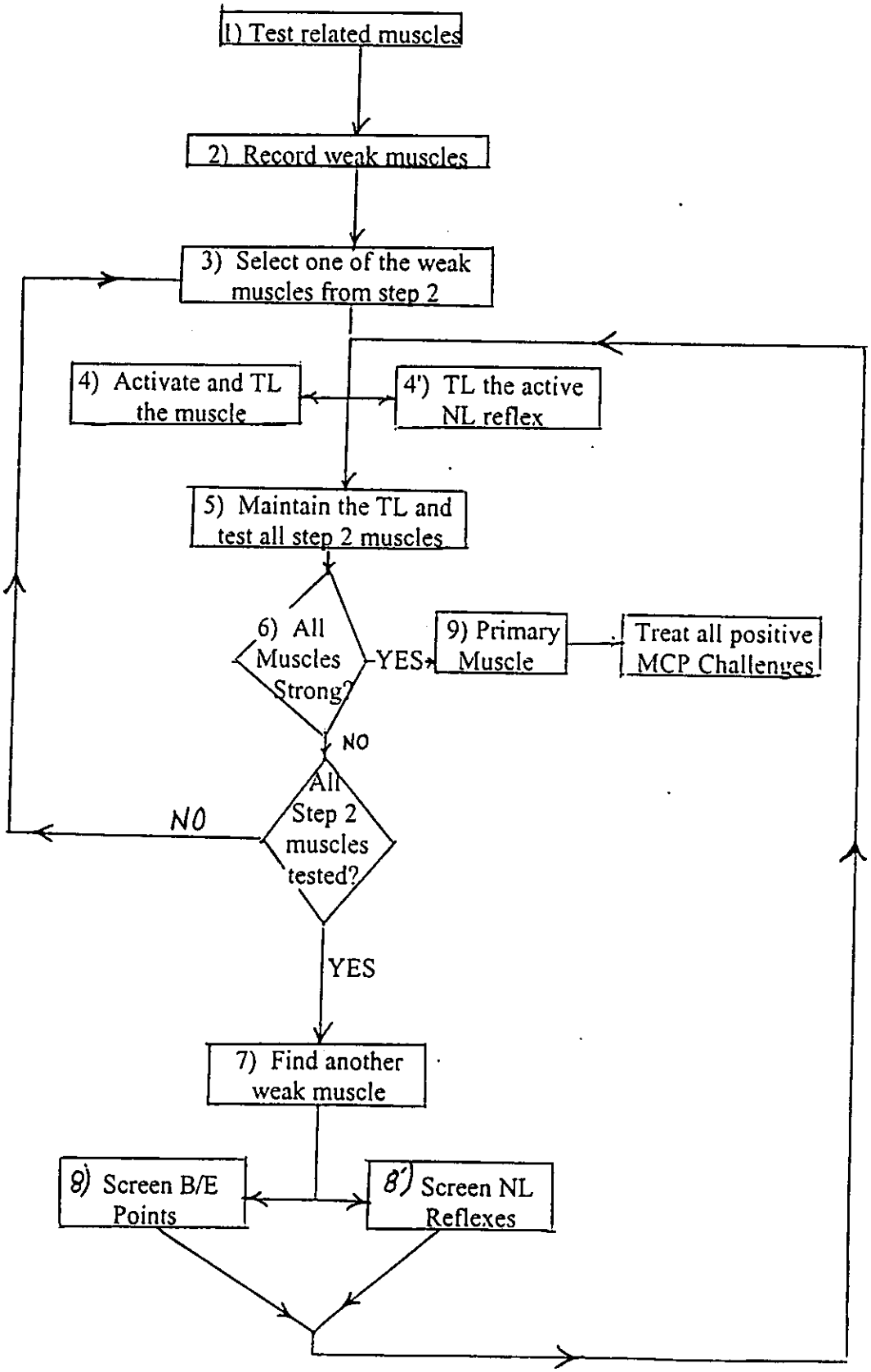
The following is an example of how this procedure can work for you and your patients. A patient presented in my office with acute low back pain of unknown origin. The low back examination revealed multiple weak muscles. After utilizing the procedure outlined below, the pectoralis major sternal (PMS) was determined to be the primary muscle. Further questioning of the patient revealed that he had started taking heavy doses of benadryl for poison ivy. After correcting all of the factors related to the PMS, all previously weak muscles tested strong and the patients pain level was greatly reduced. The procedure is as follows:

1. Test all muscles related to the patients primary complaint. This may include testing all muscles related to a joint or possibly muscles related to each organ system of the body. (Note: It may be beneficial to test the muscles bilaterally.)
2. Record all weak muscles.
3. Select the muscle that you believe to be related to the source of the problem. We will refer to this muscle as the primary muscle. (Note: The primary muscle will usually strengthen with therapy localization (TL) to it's organ neurolymphatic reflex.)
4. Activate and TL the suspected primary muscle or TL the active neurolymphatic reflex for that muscle.
5. Retest all of the previously weak muscles while maintaining the TL of step 4. (Note: TL to the activated primary muscle or the associated active neurolymphatic reflex will strengthen all of these muscles.) After you have determined the primary muscle, skip to step 9. Otherwise proceed with step 6.
6. Select another muscle recorded in step 2 and repeat steps 4 and 5. If none of the muscles recorded in step 2 are the primary muscle, proceed to step 7.
7. Find another weak muscle. It is helpful at this point to screen all of the neurolymphatic reflexes and beginning and ending acupoints to help locate the primary muscle.
8. Go to step 4.
9. This is the primary muscle. Evaluate and treat all positive chemical, structural and electromagnetic challenges.

CONCLUSION

There are numerous therapeutic options availble to the AK practitioner. The ultimate challenge is selectecting the most effective and appropriate treatment. The procedure discussed above will help to identify the patient's primary problem which will save time and effort in treating the total symptom picture.

Presenting Complaint



How to Quit Smoking
A Comprehensive Approach

Robert A. Rakowski, D.C.

ABSTRACT

This paper provides a brief overview of the strengths and weaknesses of various smoking cessation programs and introduces a comprehensive program that is assisted by applied kinesiology techniques.

INTRODUCTION

The contribution of cigarette smoking to disease is well summarized in a 1986 report published by the US. Department of Health and Human Services - "Today cigarette smoking is recognized as the single most preventable cause of death in our society, and the most important public health issue of our time."[1] The estimated annual excess mortality is about 350,000 - more than the total number of American lives lost in World War I, Korea, and Vietnam combined.[2] Most smokers recognize the harmful of effects of their habit and have at least a mild desire to quit. Additionally, there are a growing number of social and economic pressures encouraging smokers to quit. Many have tried a variety of quit smoking methods like "The Patch," hypnosis, and acupuncture. Others have tried to quit cold turkey. Each of these methods has its strenghts and weaknesses but, by themselves, none are a truly comprehensive approach. This paper will provide an overview of the strengths and weaknesses of each of these programs along with an introduction to a comprehensive approach that has helped numerous patients to "kick the habit."

DISCUSSION

A comprehensive approach to smoking cessation should consider mental, physical, and behavioral components. Mentioned first in this list is the mental component because this is, by far, the most important. Most people who quit smoking quit cold turkey. In other words, they just make up their minds to quit and do so. Other people have a strong desire to quit but suffer extreme physical withdrawal symptoms like sugar cravings and irritability. Rather than gain weight and suffer confrontational episodes with the people around them, they resume their smoking habit. These people will have a greater success rate when the physical withdrawal symptoms from nicotine are addressed. As for the behavioral component, it has often been said that only a habit can replace another habit. Preferably, individuals should replace their bad habits with good habits. Patients who have quit smoking must find a substitute for cigarettes, especially in the early stages.

Methods Utilized for Smoking Cessation: To follow is an overview of the most common methods utilized to aid in smoking cessation. These methods are to be compared to the comprehensive approach which includes mental, physical, and behavioral components.

Cold turkey: Most people who quit smoking make up their mind and just do it. This illustrates the importance and power of the mental aspect of quitting. Although this component is clearly the key to smoking cessation, these individuals would still benefit from a program that addresses the physical and behavior aspects of quitting.

- Mental component: Exclusive.
- Physical component: None.
- Behavior component: None.

The Patch: The patch is a new and popular product utilized to assist smoking cessation. A nicotine patch is applied to the patient's skin, and nicotine is

absorbed into the patient's body. This is analogous to smoking without lighting up. Over time, each patch is replaced with another containing lower and lower dosages of nicotine. Eventually the patient is weaned from nicotine. This approach has helped many people to quit smoking. However, since the patient is still absorbing nicotine, this procedure has numerous potential side effects. Additionally, it can be very dangerous for patients to smoke while wearing the patch.

- Mental component: Limited.
- Physical component: Primary - nicotine replacement.
- Behavioral component: None.

Hypnosis: Hypnosis has been used effectively as an aid for smoking cessation. The hypnotist provides subconscious suggestion to the patient to quit smoking.

- Mental component: Subconscious.
- Physical component: None.
- Behavioral component: Subconscious.

Acupuncture/Acupressure: Acupuncture has also proved beneficial as an aid to smoking cessation. Practitioners may chose to utilize needling techniques for 30 to 60 minutes over the course of 5 to 15 sessions, or longer lasting ear needles may be inserted for up to 7 days with repeated applications. (Note: This protocol was consistently stated by acupuncturists interviewed in the Houston TX area.)

- Mental component: Some with acu-aids or ear needles.
- Physical component: Primary - physical stimulation to replace nicotine.
- Behavioral component: None.

The Comprehensive Approach: People must first make up their mind to quit smoking (mental component). For this aspect we inform patients of the physical, social, and economic advantages to quitting.

- Physical: Smoking is recognized as the single most preventable cause of death in our society.1

Additionally, smoking is strongly associated with the following:

- Cancer - especially of the lung,
- Myocardial infarction,
- Emphysema,
- Chronic bronchitis,
- Atherosclerosis,
- Increased risk for spontaneous abortion, perinatal mortality, and reduced birthweight,
- Gastritis, and peptic ulcers,
- Discoloration of teeth and fingernails,[3]
- Offensive breath, and
- Smoke residue in clothes and hair.

Social: Recent studies have conclusively demonstrated that second hand smoke contains higher levels of carcinogens than "mainstream smoke." In response to public demand, many restaurants, airlines and office buildings are enforcing nonsmoking regulations.

Economic: The cost of a pack of cigarettes is currently about $2.00. Therefore, a 2 pack a day smoker will spend approximately $120 per month on cigarettes. If this same money were invested at a rate of 8 percent annually, over 30 years the patient would have a total investment of $178,843. Instead, this potential financial security is exchanged for a myriad of health problems.

All of these reasons can help patients to understand the necessity for smoking cessation. Additionally, it is often helpful to evaluate smokers for psychological reversal as described in Dr. Walther's Applied Kinesiology Synopsis.4 In cases of psychological reversal, a strong indicator muscle will weaken when the individual states, "I want to quit smoking." The indicator will remain strong when the patient states, "I want to continue smoking despite all of the detrimental effects to my health." The psychological reversal is treated by having the patient focus on the statement, "I want to quit smoking" while

tapping acu-point SI 1 or SI 3. The proper point, when therapy localized will eliminate the weakening observed with the positive statement.4 It is also beneficial to evaluate the small intestine meridians and neurolymphatic reflexes.

The physical component of the quit smoking program addresses the nicotine withdrawal symptoms. Nicotine is a powerful stimulant and affects, among other things, the smoker's adrenal glands. Withdrawal symptoms often include headaches, irritability, cravings for sweets, and in extreme cases, physiologic tremors. The comprehensive program addresses this problem by replacing nicotine with adrenal stimulants such as Siberian ginseng, gota kola, licorice, parsley (found in Nutriwest #1-AD formula), wild yam root, and/or possible adrenal tissue extract. Additionally, acupressure is utilized to provide stimulation to the adrenal glands. This adrenal stimulation greatly reduces, and in some cases, even eliminates the nicotine withdrawal symptoms. The kinesiologic procedure utilized to determine the most effective nutrient combination and acubead placement is as follows:

1. Have the patient therapy localize (TL) the adrenal neurolymphatic reflex (2 inches above and 1 inch lateral to the umbilicus).
2. If positive, treat the reflex until therapy localization is negated.
3. Stimulate bilaterally CX 7 (the sedation point of the CX meridian). This will cause positive therapy localization of the adrenal neurolymphatic reflex.
4. Maintain the therapy localization at the adrenal neurolymphatic reflex.
5. Utilize oral nutritional testing to find the nutrient(s) that will negate the positive TL.
6. While the patient maintains the adrenal neurolymphatic TL, use a blunt probe and 2 point therapy localize the ear until you find a place that negates the original TL. Mark this location. (I usually compress the tissue with the probe).
7. Apply an acubead to the ear location.
8. Proper placement of the acubead will negate the positive TL of the adrenal neurolymphatic even after the CX meridian is sedated.

The behavioral component of the program assumes that only a habit can replace another habit. It is suggested that patients chew gum or some other substances to provide some measure of oral stimulation. It is strongly advised that they not replace cigarettes with food. Patients are instructed to place their cigarette replacement (chewing gum et cetera) everywhere that they would habitually smoke. In the case of chewing gum, they should strategically place chewing gum by the phone, in the car, in the bathroom, on their desk et cetera. Many of my patients have taken up chewing cloves as a replacement for cigarettes.

CONCLUSION

Cigarette smoking is a major public health issue. Hundreds of thousands of illnesses and deaths are directly related to cigarette smoking. Most smokers recognize this but do not have the tools to overcome their addiction. The comprehensive approach addresses the mental, physical, and behavioral aspects of smoking cessation. Through this program, numerous patients have quit smoking. Additionally, many of these individuals have suffered little or no side effects.

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PYRAMIDAL DISTRIBUTION OF WEAKNESS

Michael D. Allen
DC, NMD, DAAPM, DACAN, DABCN, DIBAK
Chiropractic Neurologist

ABSTRACT

This paper deals with the physiological effects of a pyramidal distribution of weakness and how to evaluate it with the functional neurological techniques of applied kinesiology. Further, it details certain specific exercise techniques necessary to physiologically facilitate those areas of the neuraxis which have become hyperpolarized, thereby providing for the normalization of the physiological processes which bring the deafferentated areas back to threshold.

[Author's note: Those word(s) which appear in bold type may be found in the glossary at the end of the paper.]

INTRODUCTION

The key to understanding this paper is awareness that the entire human nervous system is receptor-based[1]. That is to say, *all* functions of human performance are as a result of the signals which arise from afferent input largely as a result of joint mechanoreceptor function.

We know that differences exist in membrane potential between the internal and external environments of a neuron. These differences can be observed in the laboratory as the resting membrane potential. But as a result of the complexities of human behavior and environmental stimulation, it is doubtful that such a resting state could exist in man. In fact, in man, there is always a constant flux of membrane potential differences which occur as a result of changes in spatial and **temporal summation**, pH, oxygen tissue saturation and temperature. This constant flux can be clinically seen as shifts toward either **depolarization** or hyperpolarization, or toward a more or less excited state. The point is that the function of the human nervous system is an integration of all those things which collectively result in potential differences across the neuronal membrane barrier. The integration of these effects is different at individual neurons, and at different times during human performance, due to the interrelationships of all those signals which come from the periphery to the central neuraxis and those which proceed from the central neuraxis to the periphery.

As a result, the description of the differences in membrane potential can be referred to as its ***central integrative state***. This term has profound clinical ramifications as the clinician can augment a multitude of factors which, in and of themselves, can change the functional state of a neuron and provide far reaching understanding of the nervous system's presentation, and indicate the proper therapeutic response.

An adequate afferent input brings the central integrative state of the neuraxis to a condition of functional readiness, thereby potentiating the motor response toward **sodium equilibrium potential**, or threshold. **Deafferentation**[2], on the other hand, drives the central integrative state of the neuraxis toward **potassium equilibrium potential**, or away from threshold, and results in a degenerative change in the functional status of its postsynaptic tissues which would otherwise effect a task other than the one they are producing as a result of that deafferentation. This allows the compromise that the controlling systems would have prevented. Hence the production of *the pyramidal distribution of weakness.*

DISCUSSION

The Afferent System

The main afferents to the somatosensory centers come from the graded, non-propagated action potentials which arise in the receptors in the organs of balance, the proprioceptors of the neck, and from the cerebellum (tonic labrynthine and tonic neck reflexes). They are organized in a rostral-caudal fashion, with the highest priority in the upper spine as opposed to the lower spine, the upper extremity as opposed to the lower extremity, and the upper part of the upper extremity as opposed to the lower part of the upper extremity.

Joint mechanoreceptors, particularly those of the axial skeleton, send afferent signals to the spinal cord via type Ia fibers. Their second order neurons terminate in the thalamus and cerebellum. And from the cerebellum, they ultimately reach the thalamus. The point is that *all* sensory afferents (except those of the sense of smell) ultimately terminate in the thalamus. From there, they project to the cerebrum as third order neurons via the corona radiata.

The somesthetic sensory cortex (areas 3, 1 and 2 of the postcentral gyrus of the frontal lobe) receives afferentation from the periphery in a somatotopically organized fashion such that the upper body components are represented rostral to those of the lower body, and projects its fibers to the motor cortex via engram cells, or cerebral interneuronal projections.

The sum of *all* the excitatory and inhibitory input to the sensory nervous system determines the central integrative state of the higher levels of the neuraxis. However, an increased level of excitatory stimulation does not necessarily mean an increased level of overall function if those excitatory stimuli excite inhibitory neurons. This can lead to an increased dampening of function with a decrease in overall excitability. The point is that any deafferentation of these tracts will tend to drive the postsynaptic neurons more toward potassium equilibrium potential, causing them to be less responsive to their afferent input. This creates a decreased ability to excite the inhibitory neurons, leading to consequential hyperactivity of those areas that would have otherwise been modulated by those inhibitory neurons.

The Efferent System

The motor cortex is divided into two separate divisions, the premotor (areas 4s and 6) and primary (area 4) motor areas. The premotor area lies just anterior to the primary motor area in the precentral gyrus. Very few premotor fibers project directly to the spinal cord via the pyramidal tract. Instead, most of the pyramidal fibers originate in areas 4s and 6, and cause more complex muscle movements, usually involving groups of muscles performing some specific task, rather than to individual muscles.

Pyramidal tract fibers originating in areas 4s and 6 do not initiate impulses to voluntary muscles, but act as inhibitors, suppressors, or "brakes" on the lower motor neurons and prevent them from over discharging when responding reflexively to sensory stimuli. If for some reason motor neurons are freed from their inhibitory or modulatory control, they discharge spontaneously in response to the segmental reflex stimuli to which they are usually dampened. This is due to an inhibition of the inhibitory controls. In other words, the nerves are *allowed* to do that which they are usually inhibited from doing.

To achieve their results, the premotor area mainly sends its signals into the primary motor cortex to excite multiple groups of muscles. Some of these signals pass directly to the motor cortex through subcortical nerve fibers. But the premotor cortex also has extensive connections with the basal ganglia and cerebellum, both of which transmit signals back by way of the thalamus to the motor cortex. Thus, the premotor cortex, the basal ganglia, the cerebellum, and the primary motor cortex constitute a complex overall system for voluntary control of muscle activity called the corticolenticulopallidostriatothalamocortical feedback loop.

A major share of the output signals from the basal ganglial system are inhibitory. Much of this inhibition feeds back to the

primary motor cortex to inhibit the pyramidal system itself, but still additional inhibitory signals pass into the brain stem to inhibit depolarization of the reticular formation and therefore the autonomic fibers, and vestibular nuclei. Therefore, release of these other motor neurons from the basal ganglial inhibition allows for an inhibition of the normal inhibitory function, or an excess excitatory motor response, leading to reflexogenic muscle spasm and autonomic concomitants.

The primary motor cortex receives the bulk of the input from its engram cells which are postsynaptic to the somatosensory cortex. From here, the primary motor cortex sends its fibers to the spinal cord and brain stem without synapse through the corticospinal (pyramidal) and corticobulbar (extrapyramidal) tracts, respectively.

The pyramidal tract is that neurological tract which has to do with fine voluntary and purposeful motor function. It is universally regarded as the descending pathway most concerned with voluntary, discrete, skilled movements of the distal extremities. It travels caudally to the pyramids of the medulla where approximately 80-90% of the fibers decussate to the contralateral side and become known as the crossed pyramidal or lateral corticospinal tract. The other 10-20% of the fibers remain ipsilateral and descend as the uncrossed (direct) pyramidal tract or anterior corticospinal tract. They terminate by synapsing with motoneurons in the anterior horn of the sinal cord.

The extrapyramidal system is much older than the pyramidal system. Its purpose is two-fold. First, it acts to dampen the strong facilitory influence of the pyramidal tract. Second, it modulates the autonomic functions of the intermediolateral cell column which takes its most rostral origin in the Edinger-Westphal nucleus of the third cranial nerve.

The extrapyramidal tract is also concerned to a great extent with protection. For example, when only the extrapyramidal system is functional, stimuli to the bottom of the feet causes a typical withdrawal protective type of reflex which is expressed by the upturned great toe and fanning of the other toes. But, when the pyramidal system is also fully functional, it suppresses the protective reflex and instead excites a higher order of motor function, including the normal effect of causing downward bending of the toes in response to sensory stimuli from the bottom of the feet. As we shall see, this becomes very important in the diagnosis of the pyramidal distribution of weakness.

Modulation

Specifically, the descending cascade of the extrapyramidal tract synapses in multiple areas: The basal ganglia, thalamus, the subthalamic nucleus of Luys, substantia nigra, red nucleus, reticular formation, pontine nuclei (and therefore cerebellar afferents and efferents through their homologous interneurons), inferior olive and spinal interneurons. The nucleus of Luys and substantia nigra send postsynaptic fibers to the red nucleus and reticular formation both ipsilaterally and contralaterally, while red nucleus and reticular formation send their terminal fibers to the cord. The red nucleus sends its efferents contralaterally to terminate on the ventral horn cells of the spinal cord. The reticular formation sends its efferent fibers to the cord both ipsilaterally and contralaterally. Because these fibers can arise from the thalamus via the hypothalamus without cortical tributaries, this tract is also known as the thalamohypothalamoreticulospinal tract, and its function is modulatory[3].

Considering the above, we find that the pyramidal and extrapyramidal tracts have almost direct communication with their anterior motoneurons of the cord for control of either individual muscles or small groups of muscles. This may be seen as purposeful motion or postural control, and is distinguished mainly in cervical segments with diminishing influence as it progresses caudally.

Deafferentation of the cerebral cortex can effect not only its somesthetic sense areas but also its motor responses. If that area is not brought to threshold, it is said to be driven more toward potassium equilibrium potential, adversely effecting its

ability to perform its requisite tasks resulting in dysfunction and the clinical symptoms of stroke antalgia, but without the tissue pathology seen concomitant with a vascular accident.

Not only do the skeletal muscles demonstrate the stroke antalgia in the form of a certain and specific postural gait, but also autonomic concomitants which are the same as, but not as a result of the patient having had a stroke. However, as a result of a hyperpolarization of those pathways which supply the vascular tissues, there can be a change in the blood supply to the cerebrum as a result of autonomic concomitants secondary to changes in the intermediolateral cell column.

Most lesions of the motor cortex, especially those caused by a stroke, involve both the motor cortex itself and deeper structures of the cerebrum, especially the basal ganglia. As we said above, if the basal ganglia is unable to perform its requisite functions of modulation there is a resultant production of myospasms. In fact, the greater the lesion to the basal ganglia, the greater the degree of spasm.

It is important to point out that the term "lesion" does not necessarily mean "damage". The signs and symptoms of a lesion are produced by any stimuli (or lack thereof) which causes the inability of the postsynaptic tissues to perform their function. In other words, a drive toward potassium equilibrium potential in a fiber which would have otherwise produced a postsynaptic excitatory response, or a drive toward sodium equilibrium in a fiber which produces a postsynaptic inhibitory response will result in the same drive away from threshold in its postsynaptic fiber. Likewise, a drive toward potassium equilibrium potential in a fiber which would have otherwise produced an inhibitory response will result in a drive toward sodium equilibrium potential in its postsynaptic neurons as a result of a lack of inhibition. The end result in each case is that the area involved is unable to perform its proper function, leading to a compromise in the central integrative state of its postsynaptic tissues. If this takes place throughout the neuraxis as a whole, it becomes compromised, causing it to be functionally dysfunctional.

The motor centers of the brain stem coordinate involuntary postural and positioning reflexes. When these centers have their requisite input of both excitatory and inhibitory stimuli, they are said to be at their functional threshold, and ready to respond adequately to peripheral stimuli, both internal and external.

Essentially, the function of the thalamohypothalamoreticulospinal tract is to inhibit the ipsilateral anterior muscles above T6, and those ipsilateral posterior muscles below T6. Any deafferentation of this tract will tend to drive its central integrative state toward potassium equilibrium potential and further from threshold, resulting in a concomitant hyperpolarization of its postsynaptic tissues — wherever they are. This creates an inhibition of its inhibitory effect, resulting in an increased tone of the anterior muscles above T6, and an increased tone in the posterior muscles below T6 on the side ipsilateral to the hyperpolarization. As a result, the patient will be unable to meet the demands of simple manual muscle tests such as that of the finger extensors and abductors, and dorsiflexors of the great toe, ipsilaterally. This is the pyramidal distribution of weakness. It is actually a misnomer. It is not a pyramidal distribution of weakness, but rather a hyperpolarization of the extrapyramidal tract which has lost its ability to perform its requisite inhibitory functions which modulate pyramidal influences. This has several structural ramifications which we will discuss.

The long term effects of the thalamohypothalamoreticulospinal hyperpolarization is a facilitation of those muscles which have lost their natural extrapyramidal inhibition. This leads to an increase in their muscle spindle function and, through monosynaptic facilitation of its alpha and gamma motoneurons, a consequential facilitation of that muscle. Further, through disynaptic postsynaptic inhibitory pathways, the antagonistic muscle becomes defacilitated. This drives the original muscle

further toward facilitation, setting up joint pathology with an increased injury potential. Additionally, these same nociceptive pathways are stimulated by **polyanionic glycosaminoglycans** which arise from the breakdown of joint cartilage. This nociception, together with other aberrant reflexes quicken the production of autonomic concomitants through the interneurons to the intermediolateral cell column. Further, through crossed extensor reflexes, the contralateral extremities respond in a comparable but converse manner simulating a postural or gait asymmetry. This leads to further nociception and resultant reflexogenic myospasms with a propagation of the pathology of the involved areas.

It is important to realize that the pyramidal distribution of weakness is not primarily one of thalamic hyperpolarization — although that is the origin of the thalamohypothalamoreticulospinal outflow — but rather one of cerebellar hyperpolarization as a result of joint mechanoreceptor deafferentation. The thalamic problems are secondary to the cerebellar dysfunction. The cerebellum receives its greatest afferent input from type Ia fibers including those from all joint mechanoreceptors and muscle spindles, both axial and peripheral.

Examples of the diagnostic tests for a pyramidal distribution of weakness are finger and great toe testing, physiologic blind spot evaluation, Rhomberg's test, finger-to-nose testing, and the evaluation of the ability to perform alternating movements in rapid, smooth and rhythmic succession such as quickly flipping the hands back and forth, and piano-type movements.

Finger/Great Toe Testing

One of the best clinical ways to investigate a patient for a pyramidal distribution of weakness is by evaluating the extensors and abductors of their fingers, as well as the dorsiflexors of their great toes. If these muscles are unable to meet the demands of manual muscle testing, it can be said that this patient has a high probability of displaying a pyramidal distribution of weakness which is named for the side of this functional dysfunction.

The finger extensors and abductors as well as the dorsiflexors of the great toe will be functionally inhibited on the ipsilateral side to the pyramidal distribution of weakness as a result of a lack of modulation from the extrapyramidal system upon the pyramidal system.

Have the patient stand facing you. Ask them to hold their hands out in front of them, with their palms down, and their fingers spread as far apart as possible. Gently but firmly use manual muscle testing procedures to evaluate the patient's ability to maintain this abduction by squeezing the index and little fingers together.

Next, have the patient adduct their fingers, with their hands and fingers in full extension, pointing upward. With the same discreet testing procedures, against your testing pressure, evaluate their ability to maintain this position.

Finally, have the patient sit with their feet hanging freely. Ask the patient to dorsiflex their great toes, and examine their ability to maintain that muscle strength by testing those muscles against resistance.

Any inability on the patient's part to resist the demands of manual muscle testing gives a strong indication of the existence of the pyramidal distribution of weakness on the side of these weaknesses. As a result of the thalamic hyperpolarization, there is an inhibition of the normal and ipsilateral modulatory effect of the thalamohypothalamoreticulospinal tract which normally leads to a dampening of the anterior muscles above T6 and the posterior muscles below T6, ipsilaterally. This results as an inhibition of the normal modulatory pathways, thereby allowing the facilitation of those muscles which would otherwise have been inhibited, resulting in a pyramidal distribution of weakness. This explains the patient's inability to maintain strength in their finger abductors and extensors, and ipsilateral dorsiflexors of the great toe.

Physiologic Blind Spot Evaluation

The corticobulbar fibers start out in company with the corticospinal tract and a few of the fibers continue along with the corticospinal system through the pyramids of the medulla. However, as described above, the majority of fibers quickly take a divergent route at the level of the midbrain.

If the extrapyramidal tracts influence the thalamus, and if the thalamus is brought to threshold as a result of all afferents except those of the sense of smell, and if the thalamus is further stimulated as a result of the corticospinal outflow to the thalamus, then the central integrative state of thalamic afferentation can be represented by the functional level of all those systems which bring the thalamus to threshold. One such area is that of vision, since the visual afferents of the optic nerve first terminate in the contralateral lateral geniculate body of the thalamus.

Should the central integrative state of the thalamus and therefore the lateral geniculate body be driven more toward potassium equilibrium potential, then the ability to perceive visual input will be decreased. As a result, there is a very high probability that the physiologic blind spot will be larger on the contralateral side.

To evaluate the physiologic blind spot, have the patient stand against a vertical surface such as a door or a wall. Hold a piece of paper (8 1/2" X 11") lengthwise against that surface, and have the patient put their nose on the other end of the paper.

Next, while the patient maintains that distance (11") from the surface, place the paper flat against that surface and place a small dot in the center of the paper. Have the patient cover their left eye with their left hand. Next ask them to focus their gaze on the small dot throughout the test. Their eye should not wander from that spot. With a sharp pencil, slowly move the pencil toward the right and ask the patient to tell you when the *tip* of the pencil disappears. When it does, place a dot, and continue laterally until the patient recognizes the tip of the pencil returns. Place another dot. Next, return to the center of that area of physiological blindness and move the pencil upward until the patient again notices the tip of the pencil. Place another dot. Finally, go back to the center of the area and do the same thing, moving your pencil toward the bottom of the paper. Place a dot when the patient notices the tip of the pencil. The four dots can now be connected and disrepresents the physiologic blind spot for that eye.

Now do the same thing for the left eye. Have the patient maintain the same distance from the paper. Have them cover the right eye with their right hand, and proceed as above, but in the opposite direction.

A person with a hyperpolarized thalamus will show a difference in size from one physiologic blind spot to the other. The side of the larger physiologic blind spot represents the side opposite to that of the hyperpolarized thalamus (since the optic nerve decussates at the optic chiasma). And, it gives a pictorial representation of the degree of input from all areas that have synaptic contact with that thalamus.

Autonomic Concomitants

Those postsynaptic projections of the thalamohypothalamoreticulospinal tract ultimately make contact with the intermediolateral cell column. Since the intermediolateral cell column is responsible for the disynaptic postsynaptic inhibition of nociceptive reflexogenic efferents in the anterior horn via its interneurons, the hyperpolarization of this area also explains the production of the autonomic concomitants.

A pyramidal distribution of weakness can be accompanied by several autonomic concomitants. The corticothalamohypothalamoreticulospinal pathway constitutes an extrapyramidal pathway by which motor regions of the cortex can act on the spinal motor apparatus. The physiological role of the projections from sensory regions of the cerebral cortex to reticulospinal neurons is less certain, but such pathways may be involved in the regulation of sensory input to the spinal cord through reticular-evoked presynaptic inhibition of spinal affer-

ent fibers, or through postsynaptic inhibition of spinal sensory interneurons. At the same time, this same pathway conveys autonomic modulation from the Edinger-Westphal nucleus of the third cranial nerve at its most rostral extent through intermediolateral cell column which terminates in the sacral portions of the cord at S2-4 to influence among other things, dilation of the pupils, respiration, circulation, sweating, shivering, etc.

Posture and Gait

Skeletal motor neurons are commonly referred to as the "final common pathway", because they integrate all central nervous system activity controlling a given muscle — from spindle afferent fibers, spinal interneurons involved in spinal reflexes, brainstem nuclei and cortical pyramidal cells. The function of the reflexes is to influence the neuronal signal, or in other words, modulate the interplay of one area with another. This can only happen, if you recall, *if* the reflex is brought to threshold. If it has been driven more toward potassium equilibrium potential (or away from threshold), then the modulatory control cannot occur resulting in nociceptive reflexogenic efferents as a consequence of deafferentation.

When a person takes a step from one foot to the other, it requires a stereotyped postural adjustment to be set in motion. This consists of a shift of body weight from one side to the other, and from back to front where the weight is born principally by the nonreaching foot and managed through the motion of the contralateral hand.

Try to imagine the impact on functional status if reflexes are not brought to threshold. This in itself represents a deafferentation of some presynaptic area which has been driven toward potassium equilibrium potential, resulting in the hyperpolarization of a function that should be modulating its postsynaptic stimuli. As a result, that function is freely allowed to do that which it should be modulated to perform.

EXERCISES

A patient who demonstrates a pyramidal distribution of weakness needs to be treated not only with specialized fast-stretch coupled[4] chiropractic[5] manual manipulative reductive techniques, but also certain specific exercises in order to facilitate the specific cerebellar hyperpolarization and its resultant thalamic drive toward potassium equilibrium potential, *and* to avoid further facilitation of the contralateral side of the cerebellum which has already been driven toward sodium equilibrium, and thereby depolarized.

In order to cause unilateral facilitation of the hyperpolarized thalamus contralateral to the cerebellar deafferentation secondary to a deafferentation of the joint mechanoreceptors, the patient must do only those exercises which encourages facilitation of: 1) The anterior muscles of the upper extremity on the side contralateral to the pyramidal distribution of weakness, 2) The posterior muscles of the upper extremity on the side ipsilateral to the pyramidal distribution of weakness, 3) The facilitation of those anterior muscles of the lower extremity on the side ipsilateral to the pyramidal distribution of weakness, and 4) The facilitation of those posterior muscles of the lower extremity on the side contralateral to the pyramidal distribution of weakness.

For example: A patient with a hyperpolarization of the right side of the cerebellum will present with a left pyramidal distribution of weakness as a result of the thalamic hyperpolarization. This results in a right foot forward gait pattern with the left arm forward and the head turned toward the right.

The patient's structural compromise is a result of a left thalamic hyperpolarization secondary to a decreased central integrative state of the right cerebellum and a zygapophyseal joint mechanoreceptor deafferentation, with a consequential and concomitant increase in the size of their right physiologic blind spot.

In the left pyramidal distribution of weakness, the muscles of a *neurologically* neutral posture will be functionally facilitated and functionally inhibited consistent with the right foot forward gait even though the patient may be physically standing in the *anatomically* neutral

position. As a result, each of the joints connected by these muscles will be drawn toward the facilitation and limited through the opposite direction, creating further deafferentation. It is important to keep in mind that we are discussing a neurologic posture, not necessarily a structurally visible variant from a plumb line (although that may be present also).

The left pyramidal distribution of weakness is best treated by exercising the right anterior and left posterior neck muscles, the extensors of the left upper extremity, flexors of the right upper extremity, anterior thigh and dorsiflexors of the great toe on the left lower extremity, and the posterior thigh and plantar flexors of the right lower extremity.

Further, the patient *should do no* flexion with the left side of their neck, or extension with the right side of their neck; no flexion of the left upper extremity; no extension of the right upper extremity; no extension of the left posterior thigh muscles or plantar flexors of its great toe; no flexion of the anterior thigh muscles of the right lower extremity or dorsiflexors of its great toe. *To do these exercises would facilitate the left cerebellum and consequently the right thalamus and encourage the pyramidal distribution of weakness, causing further exaggeration of the patient's physiologic blind spot and dysfunctional gait mechanisms.*

The patient can do some simple exercises at home. It is essentially a modification of the cross-crawl exercises, but with a specificity determined by the side of pyramidal weakness. In the case of a left pyramidal distribution of weakness, have the patient lie on their back with both their right leg and left arm flexed. This represents the right foot forward gait. Tell the patient that you want them to do *five things at once*: 1) extend the left arm, 2) flex the right arm, 3) extend the right leg, 4) flex the left leg, and 5) turn the head to the left. (The opposite procedure works for a right pyramidal distribution of weakness.) These should each be done slowly with purposeful movement.

The return to the start posture is as important as the exercise itself. Once the exercises have been done, the patient is to return to the original posture by moving each of the five areas one by one, but there is no specific order. If the patient were to return to the start posture all at once, they would be potentiating the contralateral thalamus and cerebellum, and allow the hyperpolarization to persist.

Explain to the patient that the muscles consistent with that gait pattern are each functionally facilitated and inhibited consistent with that posture. Whereas, the patient may be able to stand in the anatomical position, the neurological posture is one of "stroke antalgia".

TREATMENT

The best way to treat the pyramidal distribution of weakness is to direct a *fast stretch coupled chiropractic manual manipulative adjustive thrust* toward those joints demonstrating a resistance to movement on the side contralateral to the hyperpolarization of the thalamohypothalamoreticulospinal tract, or ipsilateral to the side of cerebellar deafferentation. A slow stretch directed toward those same joints will only serve to further stabilize the already fixated joints through the stimulation of the Ia afferents from muscle spindles, and lead to an increased degenerative potential with the consequential production of polyanionic glycosaminoglycans and nociception.

Faulty rib mechanics further contribute to the cerebellar and thalamic hyperpolarization through these same tracts. This leads to the thalamohypothalamoreticulospinal dysfunction, and a clinical state of hypoxia, which may result in changes in cellular stability with resultant free radical production, as well as changes in systemic pH, and other homeostatic instabilities.

Consequently, the patient should also receive supplemental oxygen therapy[6] to provide the substrate necessary to rebuild mitochondrial electron acceptors to facilitate cellular membrane replacement. Further, certain specific nutrients should be provided to enhance the metabolic processes, produce ATP, allow

for more purposeful protein replication and provide for the ability to scavage for free radicals.

It is advisable for the patient to utilize a Cybex Upper Body Ergometer (UBE) in order to provide the isokinetic activity necessary to facilitate the spinocerebellar and cuneocerebellar afferentation to the cerebellar centers, thereby stimulating the proper supra-spinal modulatory events necessary to increase bony co-adaptation and stabilize the series elastic elements of the paraspinal system.

After stabilization of the aerobic base and once the pyramidal distribution of weakness has been resolved, the patient will also benefit from an aerobic exercise program directed toward rebuilding the series elastic elements of the paraspinal system. This is necessary in order to rehabilitate the muscle necessary for the development of endurance activity, and to provide for a greater quantum of bony co-adaptation and the resultant zygapophyseal afferentation.

If the pyramidal distribution of weakness does not resolve in a reasonable period of time, this patient would probably benefit from the application of a full body cast, shoulder to hips, including one thigh. This is necessary in order to isolate the side of the pyramidal distribution of weakness and cause a state of selective atrophy in those muscles which have become functionally facilitated. In the case of a left pyramidal distribution of weakness, the cast should be applied with the isolation of the left lower extremity at the thigh. It is advisable for the patient to use the UBE at the same time as the full body cast.

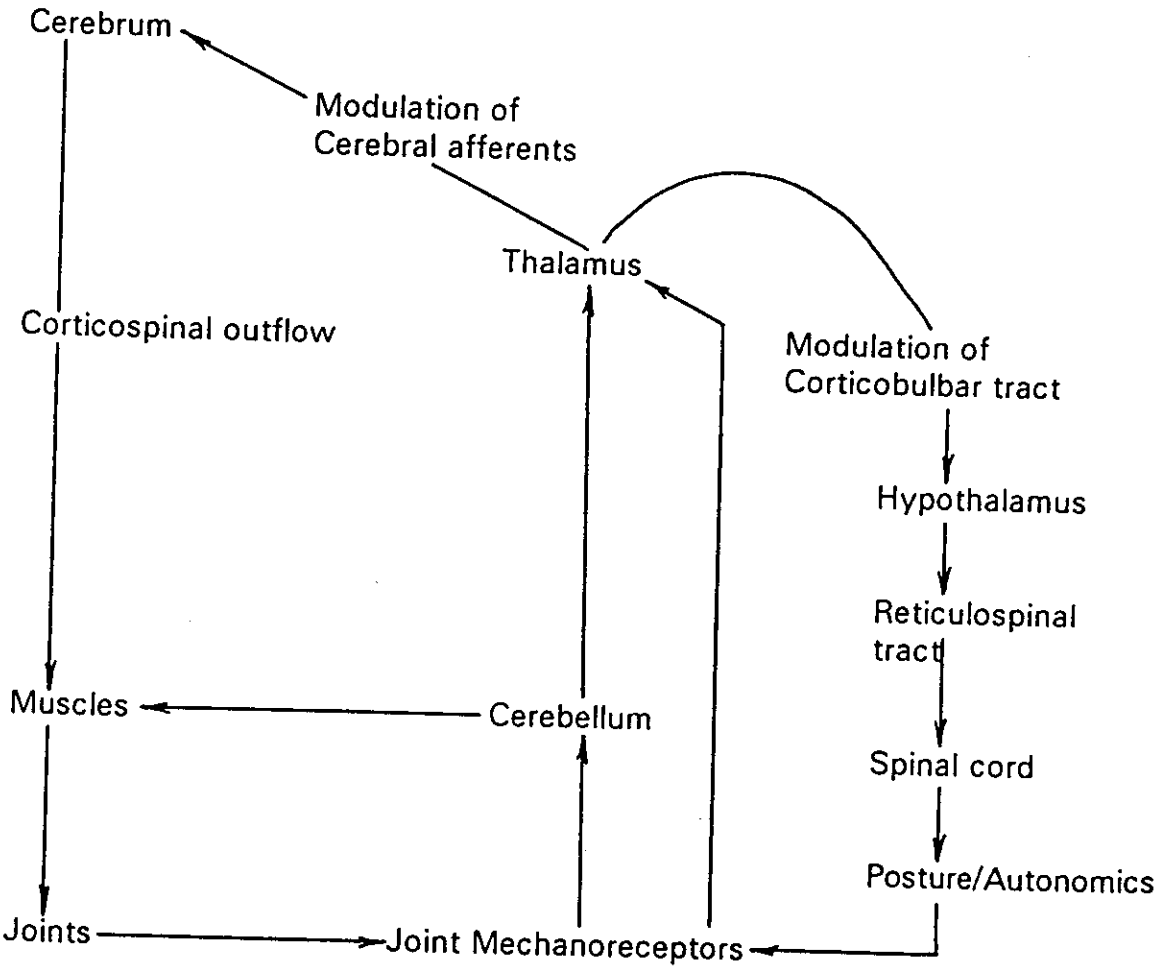
CONCLUSION

The simple elimination of one or two of the symptoms of a pyramidal distribution of weakness is not enough to say the treatment was a success. Because the human nervous system is multimodal, the elimination of one set of symptoms may cause the exacervation of another set of symptoms which are either related or unrelated to the first set of symptoms.

The pyramidal distribution of weakness is a very common finding in the great majority of chiropractic patients. It can be responsible for many of the most common symptoms ranging from mild autonomic dysfunctions to the most bizarre cases of structural compromise that respond slowly at best. Routinely, this kind of patient is the one who is told they have a "disc" pathology, and is referred out for surgery. When the procedures discussed in this paper are applied in an appropriate manner, these cases respond predictably.

The mark of successful treatment is a functional resolution of each of the examination findings mentioned above, and an increase in the central integrative state of the neuraxis as a whole. Reexamination should show a strengthening of the finger extensors and abductors and the dorsiflexors of the great toe, a balancing of the physiologic blind spots, a normalization of the neurological posture, a steadying of the cerebellar functions as well as the elimination of the autonomic concomitants and pain.

SCHEMATIC OF NEURONAL FLOW



SUGGESTED EXERCISES
Left Pyramidal Distribution of Weakness
Right Foot Forward Gait

	WEIGHT TRAINING	SLOW STRETCHES
RIGHT UPPER EXTREMITY (Above T6)	SCM	SCM
	Pectoralis	Pectoralis
	Ant. Deltoid	Ant. Deltoid
	Biceps	**Biceps**
	Internal rotators	Internal rotators
	Wrist flexors	Wrist flexors
	Finger adductors	Finger adductors
LEFT UPPER EXTREMITY (Above T6)	Upper trapezius	Upper trapezius
	Triceps	**Triceps**
	Rhomboids	Rhomboids
	Latissimus dorsi	Latissimus dorsi
	Teres minor	Teres Minor
	Wrist extensors	Wrist extensors
	Finger abductors	Finger abductors
RIGHT LOWER EXTREMITY (Below T6)	**Hamstrings**	**Hamstrings**
	Piriformis	Piriformis
	Abductors	Abductors
	TFL	TFL
	Foot plantar flexors	Foot plantar flexors
LEFT LOWER EXTREMITY (Below T6)	Adductors	Adductors
	Quadriceps	**Quadriceps**
	Abdominals	Abdominals
	Foot dorsiflexors	Foot dorsiflexors

(NOTE: The **bold** print signifies those muscles with which most people are most familiar)

SUGGESTED EXERCISES
Right Pyramidal Distribution of Weakness
Left Foot Forward Gait

	WEIGHT TRAINING	SLOW STRETCHES
LEFT UPPER EXTREMITY (Above T6)	SCM Pectoralis Ant. Deltoid **Biceps** Internal rotators Wrist flexors Finger adductors	SCM Pectoralis Ant. Deltoid **Biceps** Internal rotators Wrist flexors Finger adductors
RIGHT UPPER EXTREMITY (Above T6)	Upper trapezius **Triceps** Rhomboids Latissimus dorsi Teres minor Wrist extensors Finger abductors	Upper trapezius **Triceps** Rhomboids Latissimus dorsi Teres Minor Wrist extensors Finger abductors
LEFT LOWER EXTREMITY (Below T6)	**Hamstrings** Piriformis Abductors TFL Foot plantar flexors	**Hamstrings** Piriformis Abductors TFL Foot plantar flexors
RIGHT LOWER EXTREMITY (Below T6)	Adductors **Quadriceps** Abdominals Foot dorsiflexors	Adductors **Quadriceps** Abdominals Foot dorsiflexors

(NOTE: The bold print signifies those muscles with which most people are most familiar)

DEFINITIONS

1. Pyramidal distribution of weakness: Increased facilitation of the anterior muscles above T6, and an increased facilitation of the posterior muscles below T6; there may be symptoms of autonomic concomitants.
2. Hyperpolarized: A tissue whose functional state is depressed from threshold.
3. Central integrative state: The state of function of a tissue which is derived as a result of the sum of all excitatory and inhibitory input to that tissue.
4. Neuraxis: The nervous system from the peripheral nerves to the cerebrum.
5. Deafferentation: A decrease in the number of afferent signals being sent to a specific area as a result of a limited range of motion.
6. Potassium equilibrium potential: An increase in intracellular potassium with a consequential drive away from threshold; a state of depressed function.
7. Spatial summation: The sum of all the afferent input from the surrounding neuronal activity.
8. Temporal summation: The sum of all the afferent input from the surrounding neuronal activity within a given period of time.
9. Depolarization: A tissue whose functional state is driven more toward threshold and is in an increased state of readiness to respond.
10. Nociceptive reflexogenic efferents: The motor response to pain which results in myospasm and increased joint destabilization.
11. Polyanionic glycosaminoglycans: Specific biochemicals which arise as a result of the breakdown of joint cartilage.
12. Sodium equilibrium potential: An increase in intracellular sodium with a consequential drive toward threshold; a state of increased function.
13. Series elastic elements: The muscle(s), bones, nerves and blood vessels which compose a joint.

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THE CLINICAL ASPECTS OF VERTEBRAL COUPLING

Michael D. Allen
DC, NMD, DAAPM, DACAN, DABCN, DIBAK
Chiropractic Neurologist

ABSTRACT

The clinical aspects of vertebral coupling are important in order to understand the concept of functional afferentation of the neuraxis and the clinical manifestations of deafferentation. This paper deals with the neurological implications of both coupling and dyscoupling from both a physiological and neurological perspective.

INTRODUCTION

Plain radiographs are generally used to diagnose and/or evaluate the extent of major or overt osseous disorders such as congenital anomalies, canal stenosis, fractures, dislocations or erosive arthritis. However, they can also be used to examine a solitary segment of the full range of spinal motion.

When radiologically examining the cervical and lumbar spines from a functional perspective, it is clinically prudent to perform a nine view cervical and/or seven view lumbar series. In the cervical spine, this should consist of the standard seven view series, but also include right and left lateral bending views. The lumbar series should contain the five standard views as well as right and left lateral bending. The rational for these procedures will follow.

DISCUSSION

Cartesian Nomenclature

Any individual vertebra or a group of vertebrae can move through three cardinal ranges of motion designated by the Greek symbol theta ("Θ"), which means, "rotation". This system is named after its originator, Rene Descartes, and is called the "Cartesian" system. It represents a universally recognized coordinate system of nomenclature. It uses three intersecting coordinate planes along the "X", "Y" and "Z" axes to specify a directional position each relative to the others.

The Cartesian system is represented by two horizontal planes, and one vertical plane, all of which run perpendicular to each other. The X and Z axes both run horizontally. The X axis goes from left to right, while the Z axis goes from posterior to anterior. The vertical, or Y axis, goes from inferior to superior. (See figure 1)

This same coordinate system can be used in space travel as well as in describing body movement. Physiologically, if rotatory movement takes place around the X axis in a positive (+) direction, it is considered to be a flexion-type movement. Conversely, movement in a negative (-) direction would be extension. Therefore, when viewed from the right, flexion of the cervical spine is termed +ΘX axial rotation. The movement of the cervical spine around a -ΘX axial rotation is considered to be extension.

The same idea applies to movement around the Z axis. When viewed from behind, a +ΘZ axial rotation is considered to be a right lateral bending, whereas a -ΘZ axial rotation is a left lateral bending — or a clockwise and counter-clockwise range of motion of the vertebra or group of vertebrae, respectively.

If rotatory movement takes place around the Y axis in a positive (+) direction — as viewed from the feet — it is a clockwise movement, and is designated +ΘY axial torque. A -ΘY axial torque is a counter-clockwise movement around the Y axis.

Translatory movements are seen along the same axes but not preceded by the Greek symbol "Θ". They are simply in a positive (+) or negative (-) direction, along the path of its designation.

A +X translation is movement from left to right, and -X translation is movement from right to left. Following the same mode of thinking, a +Z translation is motion from posterior to anterior, and a -Z translation is from anterior to posterior. Finally, a +Y translation is a movement from caudal to rostral, while a -Y translation is a movement from rostral to caudal.

Application of these principles can be tricky at first, but with continuous use it becomes second nature. Cartesian nomenclature is very specific relative to other methods of designating vertebral position, the direction of movement and/or the limitation of that movement. It does not require the understanding of any specific technique in order to realize the position of vertebral subluxation. Likewise, the direction of manipulation can be readily understood by anyone using any adjustive technique.

Coupling

The human neuraxis is designed to receive information from the internal and external environment, and respond to it. The uptake of this information, in this case with regards to the internal environment, is through the joint mechanoreceptors

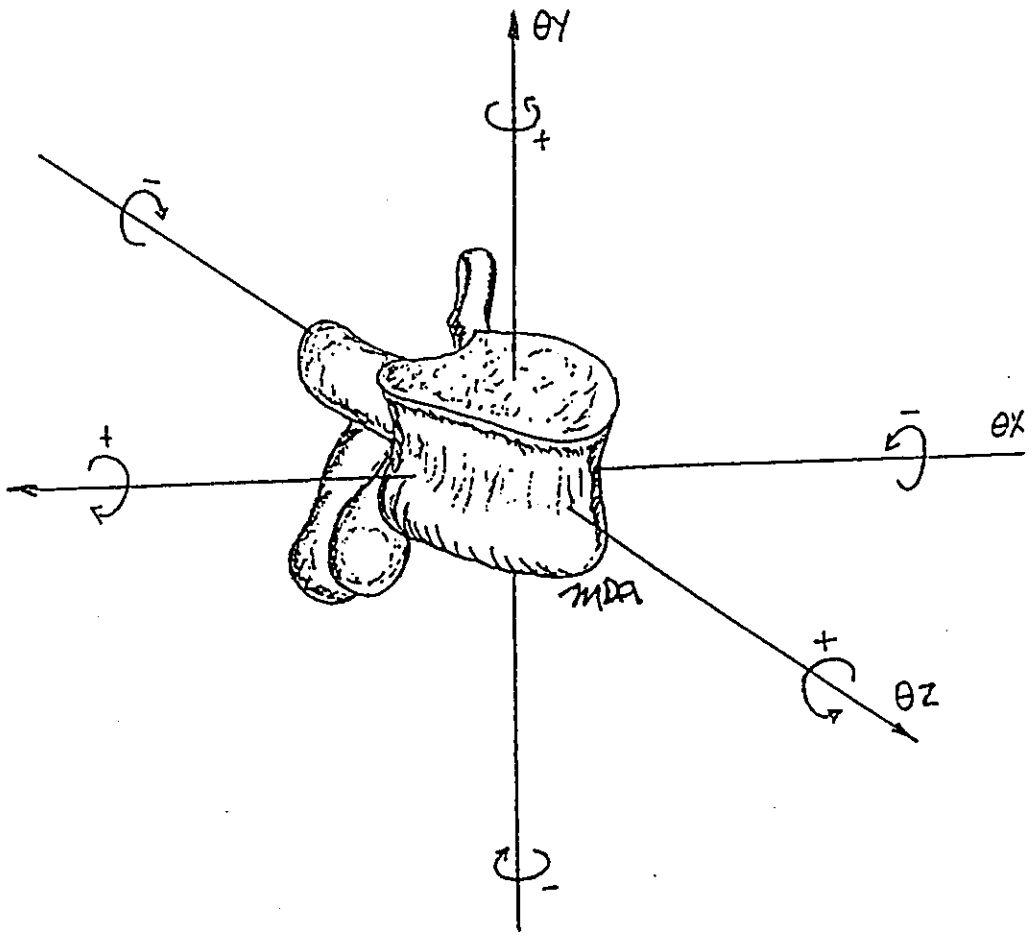


Figure 1

which are stimulated through the maneuvers of the musculoskeletal system. This provides a means for biomechanical feedback. Fundamental to this process are the zygapophyseal joint mechanoreceptors (ie, proprioceptors) whose graded, non-propagated stimulation should ideally summate both spatially and temporally in order to reach threshold, and excite their associated axons and generate a propagaged, non-graded afferent signal along their large diameter (type Ia) axons in order to bring the rostral neuraxis to its functional state.

The term "coupling" refers to the normal and physiologic action of a vertebra or group of vertebrae around or along any, or a combination of the three cardinal axes of movement. Another term for the same process is the "Instantaneous Axis of Rotation" (or "IAR"). It has to do with the movement of each individual vertebra, as well as one vertebra relative to its immediate neighbors.

Cervical Coupling

Normally, for example, when laterally bending to the right, the spinous processes of the cervical spine must couple toward the convexity of the curve. When laterally bending to the left, the converse happens; they should couple to the opposite side.

Coupling produces compression of the facets on the concave portion of the movement and expansion of those facets on the convex portion of the movement, causing different types of excitation of the zygapophyseal joint mechanoreceptors on each side, driving them toward sodium equilibrium potential, or excitation. This causes the afferentation of the higher centers whose tissues lie at the termination of each afferent segment of its neurologic tract, including the arousal of post-synaptic potentials with a concomitant facilitation of muscle function commensurate with that motion, and the resultant afferentation of higher neurologic centers.

Lumbar Coupling

Because of the prioritized rostral-to-caudal orientation of the neuraxis, the process of cervical spinal coupling is much more important than that of the lumbar spine, but the latter cannot be discounted. The former sets a precedence for the latter, but the latter must be able to function independent from the former. When the lumbar spine laterally bends to the right for example, the spinous processes must couple toward the concavity of the curve. Conversely, when the lateral bend is to the left side, the same thing must happen; the spinous processes must couple toward the concavity of the curve. This is opposite from that coupling seen in the cervical spine.

Purposeful Movement

Purposeful joint movement requires the coordinated action of many muscles. Even the relatively simple act of flexing the head on the neck requires the coordinated behavior of dozens of muscles. Motor coordination is the process of linking the contraction and relaxation of many independent muscles so they can act in concert and manage a single movement.

However, if the vertebral segments demonstrate a dyscoupled motion, it causes those facets on the concavity of the curve to respond as if they were undergoing expansion whereas those on the convexity of the curve to respond as if they were undergoing compression. Although the joint mechanoreceptors still function as they were designed, they are not brought to threshold. This causes a relative decrease in the number of excitatory postsynaptic potentials and an inability to summate either spatially or temporally. As a result, there is a relative increase in inhibitory postsynaptic potentials which lead to compromised function and the hyperpolarization of higher centers. In effect, the dyscoupling displays to the higher centers that the muscles on the concavity of the curve are relaxing while those on the convexity of the curve are contracting, resulting in a dysfunctional afferentation of the higher centers, driving them toward potassium equilibrium potential (inhibition) with the production of an aberrant population of postsynaptic potentials and the resultant hyperpolarization of those postsynaptic tissues. This is clinically known as "deafferentation".

Deafferentation is the result of compromised joint movement.

This leads to biomechanical deficits and the breakdown of polyanionic glycosaminoglycans of joint cartilage, depolarizing nociceptive type III and IV afferent fibers found in the intertransverse ligaments, the collateral ligaments of the appendicular skeleton, and lattice-like plexuses or free nerve endings of various articular tissues respectively, and resulting in nociceptive reflexogenic myospasms and concomitant painful experiences with a compensatory paucity of the modulatory effect of the higher centers on the alpha motoneurons in the anterior horn cells of the spinal cord. As well, the dysfunctional modulatory mechanisms lead to the presence of autonomic concomitants through the inhibition of the disynaptic postsynaptic inhibitory pathways through the intermediolateral cell column.

No matter its location, dyscoupling indicates the high probability of reflexogenic myospasm, and the resultant deafferentation of the neuraxis. Myospasms generate uncoupled signals to the supraspinal centers, causing the muscles to function contrary to their intended purpose, leading to further deafferentation which arises as a result of those myospasms. The normal vertebral movement is restricted and the spinous processes remain fixed in their position, prohibiting coupled motion. What we are saying is that dyscoupled movement mimics movement to the uncoupled side, causing a relative expansion of the facets on the concavity of the curve with a concomitant relative compression of the facets on the convexity of the curve. It is as if the spine neurologically laterally bent to the right when it physically went to the left. A completely dysfunctional movement!

The brain responds to dyscoupled movement by causing the muscles to do a motion which is consistent with the deafferentation. But as a result of the pathological movement, the motor response is unnatural to that which should physiologically occur. That is, the muscles which should be contracting on the inside of the lateral bend become relaxed, and those on the outside of the curve contract whey they should be relaxed. This leads to the breakdown of joint cartilage leading to further joint deafferentation and pain. The pain is a result of the noxious stimulation of free nerve endings, and all the neurological responses consistent with that stimulation.

There are several chemicals which cause noxious stimulation to free nerve endings. Among them are histamine, polyanionic glycosaminoglycans, lactic acid, bradykinins, prostaglandin E, and 5-hydroxytryptamine. No matter the source, if these chemicals find their way to nervous tissue, the result is nociception and its conscious and unconscious ramifications and resultant myospasms due to an inhibition of postsynaptic inhibition in the dorsal horn and/or inhibition of disynaptic postsynaptic inhibition from the interneuron from the intermediolateral cell column.

The quality of movement of each vertebral segment can be defined by evaluating the IAR of each spinal segment in question. Generally, symptomatic patients can be characterized as having a wider than normal distribution of their IAR's. The suggestion is that patients with spinal pain exhibit abnormal patterns of vertebral movement which could be detected by determining their IAR's both in the cervical and lumbar regions.

A substantial proportion of patients with spinal pain do exhibit abnormal IAR, but not necessarily does it relate to the area of perceived pain. This occurs in the context of patients in whom plain radiography was otherwise normal. A large proportion, although not all patients with spinal pain, exhibit abnormal IAR of spinal joints in areas remote to that of their pain.

Notwithstanding the lack of segmental specificity of abnormal IAR in the diagnosis of spinal pain, it is nonetheless striking that a substantial proportion of patients with spinal pain do exhibit abnormal IAR. This abnormality does not reveal the source of the patient's pain, but it does, however, correlate with the presence of pain. Pain is a completely personal experience and cannot be considered directly proportional to the amount of tissue destruction. Therefore, pain should

more correctly be thought of as a consequence of deafferentation rather than tissue damage.

Foremost, this relationship indicates that an abnormal IAR constitutes an objective marker for the presence of pain. An abnormal IAR indicates a biomechanical disturbance that occasionally occurs in normal persons, but occurs significantly more frequently in patients with pain. Nociceptive myospasm has the capacity to reduce the vertebral range of motion and to alter the IAR; this is in fact a common denominator.

However, the abnormal IAR is not the only cause of deafferentation. It signals the inability to inhibit nociception and/or pain as a result of a decreased afferent signal from the large diameter axons which arise from joint mechanoreceptors. Nociceptive reflexogenic myospasms can also result from biomechanical deficits resulting in a biochemical failure.

Palpation of Coupling

Palpation of cervical and/or lumbar coupling is relatively easy. Have the patient lie supine. The +ΘY axial rotation of C7, for example, can be examined by pressing the right transverse process from posterior to anterior. This causes a +Z translation of that transverse process and mimics a -ΘZ axial rotation of C7 and the entire cervical spine, because that is the way the cervical spine moves with that type of maneuver. At the same time, press the spinous process of C7 from left to right in order to mimic +ΘY axial torque. This is the way the vertebra should move if it is coupling properly. Any limitation of this motion represents the high probability that the range of the motion that vertebra is limited through -ΘZ axial rotation and +ΘY axial torque, and that the functional movement is dyscoupled.

The procedure for the lumbar vertebra is similar. However, if the range of motion is limited through -ΘZ axial rotation, it will also be limited through -ΘY axial torque by virtue of the mechanism of its coupling. Remember, the lumbar vertebrae couple opposite those of the cervical spine.

TREATMENT

A dysfunctional IAR with concomitant nociception indicates the need for osseous manipulation in order to increase excitatory postsynaptic potentials and decrease inhibitory postsynaptic potentials both spatially and temporally to those areas of the neuraxis requiring a greater level of function. This is done via a *fast stretch coupled manual chiropractic reductive techniques directed toward those joints which demonstrate a resistance to motion.*

The fast stretch coupled reduction causes the inhibition of the nociceptive myospasms via the excitation of type Ib afferents from Golgi tendon organs, and a simultaneous stimulation of type I and II joint mechanoreceptors found in zygapophyseal joints, resulting in an increased probability of a spatially and temporally summated barrage of afferentation to the spinal cord, cerebellum, thalamus and cerebrum, and a concomitant presynaptic inhibition of nociceptive reflexogenic afferents in the dorsal horn of the spinal cord. Further, through disynaptic postsynaptic pathways, the same large diameter axon's synaptic connections to the intermediolateral cell column normalizes autonomic concomitants, and reestablishes the modulation of corticospinal and corticobulbar pathways via the alpha motoneurons of the ventral horn.

The treatment may also require the application of physical therapy, the use of oxygen, dietary changes, and other parts of a balanced treatment program. However, the intent of this paper is to deal with the structural ramifications of vertebral coupling. As a result, other treatment programs will not be addressed at this time, but they should not be overlooked when indicated.

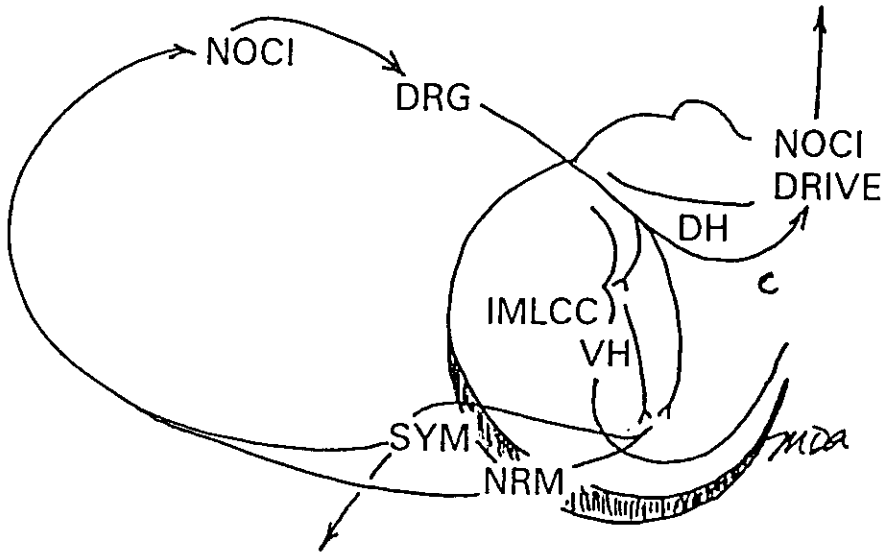
SUMMARY

The functional human vertebral column moves in a highly predictable manner. It does so as a result of the functional barrage of muscle and joint mechanoreceptors as well as via the afferentation of those other more rostral centers which make us human. It enables balance and stability, and the ability to inhibit nociception and/or pain. On the other hand, if deafferentation arises as a result of dyscoupled movement, for

example, degenerative changes and pain should be expected.

Dyscoupling and/or an abnormal IAR constitute objective markers for the presence of pain. They indicate a biomechanical disturbance that occurs in normal persons but occurs significantly more frequently in patients with pain of many kinds. Muscle spasm has the capacity to reduce the functional range of motion and to alter the IAR, resulting in further deafferentation and resultant reflexogenic myospasm. The cycle is viscious! This is common in all pain syndromes, and in the vast majority of cases can be corrected with *coupled* chiropractic manipulative techniques consistent with the normal IAR of its osseous structures with the intent to introduce the highest probability of afferentation of the neuraxis globally.

True health is the ability to maintain a functional nature in the face of adversity. There are many ways to confront these adversities. The most beneficial way to effect the functional status of the human nervous system is via the introduction of *coupled* spinal motion and the normalization of afferent signals to the most rostral centers of the neuraxis.



Key		
NOCI	=	Nociceptive input
DRG	=	Dorsal Root Ganglion
DH	=	Dorsal Horn
NOCI DRIVE	=	Nociceptive Drive
IMLCC	=	Intermediolateral Cell Column
VH	=	Ventral Horn
SYM	=	Sympathetic Outflow
NRM	=	Nociceptive Reflexogenic Myospasm

Figure 2

THE NEUROLOGIC IMPLICATIONS OF THE CHIROPRACTIC ADJUSTMENT

Michael D. Allen
DC, NMD, DAAPM, DACAN, DABCN, DIBAK
Chiropractic Neurologist

When a Doctor of Chiropractic adjusts a joint, any joint, certain populations of mechanoreceptor afferents are evoked. This is especially true when it comes to the zygapophyseal joints. Stimulating these areas through coupled manipulative techniques produces marvelous effects on the nervous system of the patient who is fortunate enough to be under chiropractic care. Pain, primary perceptual experiences, motor function, autonomic function, indeed the integrity of life and the patient's expression of humanism are effected by the functional state of the joint mechanoreceptors.

When looking at pain modalities, the effect of depolarization of joint mechanoreceptors results in an afferent stimulus at the dorsal horn onto apical internuncial neurons. These neurons send their axons to hyperpolarize, axoaxonally, on the primary neurons of the nociceptive system, causing them to be driven toward potassium equilibrium potential. The net result of this hyperpolarization is a decrease in the amount of bombardment of second order neurons at the basal spinal nucleus, thus decreasing the quantum of pain experienced by the patient in inverse proportion to the number of mechanoreceptors depolarized (i.e., the greater numbers of mechanoreceptors depolarized, the less the pain experienced by the patient). It must also be remembered that mechanoreceptor stimulation will concomitantly evoke thalamohypothalamoreticulospinal depolarization of the apical internuncial pool which will drive the central integrative state to postsynaptically inhibit nociception in the anterior horn through the interneuronal synapses.

Primary perceptual experience is dependent upon thalamic integrity, and thalamic integrity is itself dependent upon the integrity of the mechanoreceptors. Thus, a decrease in joint mechanoreceptor activity will cause a shift of the thalamic state of integration and consequently a cortical shift toward potassium equilibrium potential, bringing about a concomitant decrease in cortical activity. Therefore, increasing joint mechanoreceptor activity increases kinesthesis, joint position sense, postural relationships, the ability to think, remember, see, hear, etc. Indeed, it has a profound effect on the patient's ability to express their humanism.

Volitional motor activity is of course dependent upon cortical integrity, which is dependent upon thalamic depolarization to the cortex, which is largely a result of the mechanoreceptor afferent system. Even though the descending modulation through telencephalic (globus pallidus, caudate, putamen) and mesencephalic motor centers (red nucleus, reticular formation, substantia nigra, subthalamic nucleus of Lues) is beyond the scope of this brief monograph, suffice it to say that there are corticopallidostriatothalamocortical feedback loops comparing information to the existing central integrative state of the thalamus. It is by increasing mechanoreceptor afferentation that one can increase thalamic integrity, thereby increasing centripetal feedback to the globus pallidus and thus increase the central state of mesencephalic integration.

Other mechanoreceptor collaterals fire disynaptically to the cerebellum, upon which postural integrity is largely dependent through firing on the vestibular system. It's rostral pathways affect mesencephalic motor centers compensating for decreases in cortical volitional activity, and increasing bombardment of the alpha motoneurons and interneurons of the spinal cord at every level.

The autonomic nervous system is depolarized as a consequence of second order neuronal bombardment in reflexogenic pathways in the cord, as well as concomitant

extrapyramidal or thalamohypothalamoretic-ulospinal bombardment.

Since it is biomechanical integrity that gives us the highest populations of mechanoreceptor afferents, loss of biomechanical integrity, or aberrant biomechanical relationships will decrease mechanoreceptor afferents with resulting quantitative pathology or aberration in the patient's ability to alleviate pain, appreciate primary perceptual experience, and the ability to move and control vital functions. So, it is evident that our very humanism is dependent upon the integrity of mechanoreceptor function, a function that is best preserved by the well-trained Doctor of Chiropractic.

LOWER BACK PAIN IN PREGNANCY

Chiropractic Treatment and Results of 50 Cases

Victoria C. Arcadi, D.C.

ABSTRACT

Lower back pain is well known to all practitioners to be the most common complaint women have during pregnancy, and it can also be the most severe and debilitating. In this study, 50 pregnant women were treated with chiropractic adjustments and care for severe lower back pain. The gestation of the pregnancy in these women ranged from 2 weeks to 38 weeks and they were all treated for some degree of lower back pain. In all cases the cause of the back pain was sacroiliac joint dysfunction. All women were treated with specific chiropractic adjustments and adjunctive nutritional therapy. In all cases "lower back pain" was completely eliminated.

INTRODUCTION

In the treatment of a woman during pregnancy, one of the first complaints will be lower back pain. In fact, the majority of the population, believe that lower back pain is "normal" in pregnancy, and certainly the obstetricians constantly tell their pregnant patients that pain is, in fact normal, and there is no treatment except to deliver the baby. Then, the medical doctors say, the pain will go away.

The fact of the matter is that lower back pain is not only the most common but the most severe pain a woman can experience during her pregnancy. Studies have reported lower back pain in half of all pregnant women(1). Furthermore, pain always signals some sort of dysfunction or disharmony wherever the pain may be. In the case of lower back pain during pregnancy, the sacrum and how it relates to the articulation of the ilium is found to be the cause of the pain in the lower back. In a recent Swedish study, 52 of 79 pregnant women treated for low back pain, had a diagnosis of sacroiliac dysfunction. Seven of ten women treated by mobilization of the sacroiliac joints by orthopedic surgeons were totally relieved of all back pain(2). There were 862 women participating in the study, and seventy-eight percent of these women with low back pain had a sacroiliac dysfunction.

From the clinical experience of this author, not only are the sacral articulations involved, but the muscles which support the joint throughout the gluteal region are also involved. The severity of these straining muscles contribute largely to the intensity of the lower back pain and discomfort.

The purpose of this study is to describe a chiropractic treatment approach which shows that the most common cause of lower back pain in pregnancy is a sacroiliac joint dysfunction, with added emphasis with regard to the muscle strength and strain of these structures as well as the ligaments which support the joint, and defines a very effective treatment approach that effectively eliminates the pain utilizing Applied Kinesiology protocols.and procedures.

Methods

All 50 women were evaluated for general lower back pain of severe nature either bilateral or unilateral. A thorough examination including orthopedic, postural, and muscle testing was performed. The pain was described as localized in a generalized area of the lumbosacral area, the sacroiliac joint itself as well as in the belly of the gluteus medius muscle. There was pain to palpation in these areas mentioned as well as the piriformis muscle, unilateral or bilateral, and also in any or all of the ligaments involved in the sacroiliac articulation, specifically the posterior sacroiliac lig-

ament, sacrotuberous ligament, and the iliolumbar liagament. The muscle tests revealed possible weaknesses in one or more of the following muscles: the hamstrings, piriformis, and gluteus maximus muscles, unilaterally or bilaterally. In all 50 women, however, there was a weakness on specific muscle testing, in this study utilizing Applied Kinesiology procedures, of the gluteus medius muscle either unilaterally or bilaterally. The piriformis muscle and gluteus maximus were in many of the cases found weak unilaterally or bilaterally.

The back pain was severe in nature in all women and in some ambulation was difficult without pain. The pain was not described as gradual or sudden onset, but simply there. There was intermediate neuralgia down the sciatic nerve Grade 1 to sometimes Grade 3. Sitting was difficult as well as sleeping. Getting up from a sitting position especially induced pain in most cases.

Treatment methods utilized chiropractic care with adjunct nutritional therapy and Applied Kinesiology muscle balancing. Evaluation of the sacroiliac joints by palpation and muscle testing procedures revealed an instability of the sacrum. The weak muscles were strengthened utilizing Applied Kinesiology neurolymphatic procedures and muscle spindle/Golgi tendon therapy and/or fascial technique. The sacrum in all cases was adjusted in either the side posture position using Gonstead and/or Diversified techniques, or in the prone position. In all cases the sacrum was found to have two listings involving two moves to adequately adjust the sacrum. The listings were as follows: #1- Left inferior and posterior sacral segment 4, adjusted in an anterior superior vector with the contact hand on the left S4, and #2- Right posterior sacral segment 2, adjusted straight posterior with pisiform contact on right S2.

With multiple weaknesses or bilateral Gluteus Medius weaknesses, Vitamin E supplementation was implement in accordance with Applied Kinesiology protocols, using a water based food supplement dosages ranging from 30I.U. to as much as 1500 I.U. in difficult cases, taken at many different times in the day.

In all cases adrenal insufficiency was evaluated utilizing Applied Kinesiology diagnostic procedures.

RESULTS

In all the women treated with chiropractic adjustments, muscle balancing, and adjunctive nutritional therapies, in 100% of the cases, back pain was totally eliminated. The women after only the first adjustment of the sacrum, had an improvement of at least 75% to total relief from pain. In all cases the entire spine was evaluated and treated at each visit. In addition, the related muscles were evaluated, especially the gluteal muscles, specifically the gluteus medius, treated directly and all nutritional supplementation was addressed.

The women were treated initially one to two times a week for two weeks. After the severe pain was eliminated, the women were seen on a regular basis one time a week for at least two weeks, depending on the length of the pregnancy. If close to term the women were seen at least one time a week until delivery. Otherwise, since pregnancy and the growth of the baby is so unpredictable, with ligament laxity being a factor in every pregnancy, the women were seen as they needed care, or felt any discomfort whatsoever.

In nine years of clinical practice treating pregnant women, there have been only a very few cases in which adjustments to the sacrum and proper support to the gluteal muscles, did not relieve the lower back pain.

CONCLUSIONS

Pregnant women all over the world suffer from the terrible discomfort common in pregnancy of lower back pain. The sacral nerves innervate the uterus and with the sacrotuberous and the posterior sacroiliac ligament involved in laxicity due to the hormones in pregnancy, the sacrum becomes very unstable thus affecting the sacroiliac joint, making the whole lower back and pelvic region unstable and continues to stay that way until the baby is born. In the medical approach these women with severe back pain are sent away because there is no available treatment for them. There cannot be radiographic films taken in pregnancy, there can be no surgery, no drugs, and no shots. One of the only consistently effective treatments available is the non-invasive and safe approach which has over and over from clinical experience has been shown to work is chiropractic adjustments and care. The sacrum is evaluated and adjusted by whatever technique used, and the lower back pain, at least in this study, has been greatly relieved or completely eliminated. There is more nerve supply to the uterus, hence, the fetus, and the nervous system is put back to homeostasis.

The adjustment improved the outcomes of mother and baby in all cases. The benefits with properly adjusting a pregnant woman are instantaneous and no patient appreciates the special care from chiropractic as much as a pregnant woman. She usually feels better just walking through the office door.

The method of treatment shown in this study can be practiced by all chiropractors, and it is the opinion of the author that all chiropractors must be knowledgeable in adjusting and treating pregnant women.

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LOOKING FOR THE TRIGGER

Michael Lebowitz, D.C.

ABSTRACT

Biochemical and/or environmental triggers are often the reason for recurrent subluxations, muscle weaknesses, and neurolymphatic reflexes. Procedures for diagnosing and correcting these are discussed.

INTRODUCTION

Most of us have performed procedures such as correcting a patients pelvic biomechanics, and then had the patient walk around for a few minutes only to have the problem recur. This clues us in to the fact that we should check for foot and gait problems. Often when these are corrected, walking will not trigger your formerly positive finding to recur.

Triggers in general are much more common than most of us realize. In fact almost all recurring problems have triggers that contribute to the recurrence. I have observed that in many problems of recurring subluxations, muscle weakness, neurolymphatic reflexes, etc., the trigger is often a food, nutrient, or environmental culprit.

DISCUSSION

I do not know about the rest of you, but if I have a patient with neck or low back pain (not due to a recent trauma that is fairly severe), I do not expect to keep adjusting the same vertebra or working on the same muscle weakness more than one to three times. I always though seem to have some patients whose problems keep recurring and my corrections hold just very temporarily. In many of these patients there is a systemic problem causing recurrence such as inflammation secondary to dysbiosis (1), or a sensitivity, or nutrient deficiency.

In some patients even when these are corrected, the problem still recurs. I have found that most of these patients have biochemical or allergic factors that "blow out" only specific circuits (vertebrae, muscles, neurolymphatics). When we correct these problems (recurrent subluxation, muscle weakness, or neurolymphatic reflex) while the patient is exposed to the triggers (there can be more than one), not only do we get a more permanent correction, but often other chronic symptoms disappear. Two short case histories will illustrate.

1. Marcia, a thirty year old woman, had recurrent neck pain and subluxations that dozens of trips to another D.C. did not correct. Our careful muscle work and adjusting held for three to four days at best. On her third visit we recorrected everything. Marcia did not test positive for dysbiosis, nutrient deficiencies, or systemic food sensitivities.

   After the corrections we had her therapy localize the vertebra we had just corrected and the weak upper trapezius. We had her insalivate certain foods (I did this so to avoid this paper getting two stars). When she tasted garlic the subluxation and upper trapezius once again weakened. We told her to avoid garlic and sent her home (we hadn't perfected the technique yet). Marcia's neck problem held longer than usual but a week later it recurred again. The night before she had by mistake eaten a bean dish with garlic. On her return visit we had the usual findings. We corrected them and again garlic brought them back. This time we recorrected them while she tasted garlic and she has had no problems since.

2. Micah, a forty year old man, had recurrent headaches, sensitivity to odors, as well as dermatitis. We have worked with him for quite awhile with much success - correcting his chronic fatigue and brain fog (by correcting dysbiosis), but

the headaches and dermatitis made only a little progress. On almost every visit the adrenal and/or liver neurolymphatics test positive. Glandulars, vitamins, minerals, diet, structural work, etc., have not corrected these reflexes for more than a few days at most. He has not strengthened on any supplements for many months despite some of his symptoms being consistent with nutrient deficiencies. On this visit we corrected the neurolymphatics and then had him insalivate different substances to see which brought back the positive neurolymphatic findings. Dairy, onion, cauliflower, pepper, multivitamins (Basic Nutrients IV), B vitamins, and essential fatty acids (Omega Plus) all brought back the adrenal neurolymphatic. Wheat, corn, and buckwheat brought back the liver neurolymphatic. One by one we had him taste the foods while we treated the appropriate reflexes (what we treated were the set points for those organs (2)).

We had him avoid the triggers for three days then on reintroduction, had him retreat the appropriate set points. Since then he has had a dramatic lessening of symptoms and no recurrence of the adrenal or liver neurolymphatics. It appears that all these months that Micah did not test for supplementation, it was actually due to a sensitivity to supplementation. After doing the correction while insalivating the mentioned supplements, he started testing as needing them (they strengthened a weak G-2 muscle). Taking these started healing the dermatitis and weak organs. In the past, trying to take them had increased his symptoms due to his sensitivity to them.

We have performed this technique on many patients and have found foods, chemicals, metals, and supplements as frequent triggers for recurrent problems. Some problems can have twenty or more triggers and it may take a few visits to find them all (they do not always all show on the initial screening). The proper time to perform the procedure is after systemic sensitivities and dysbiosis have been corrected. The results have been quite gratifying.

CONCLUSION

Most recurrent findings have triggers that if found and corrected with the above mentioned procedures can resolve on a more permanent basis.

Summary of Procedures

1. If a patient has a recurrent subluxation, muscle weakness, or neurolymphatic reflex, first correct dysbiosis (l), food sensitivities, and nutrient deficiencies as these will often correct it.

2. If a patient still has a recurrent finding, correct it (adjustment, rubbing a neurolymphatic, etc.). Have the patient therapy localize the corrected area. It should test negative. Have the patient insalivate different positive triggers: foods, vitamins, etc. and see which items bring back a positive therapy localization. Chemicals and metals and EMFís can also be positive triggers.

3. While tasting the positive substance, recorrect the recurring finding. If it is a neurolymphatic, also treat the corresponding B&E point (2) by tapping or laser.

4 Avoid the substance for 72 hours. Have the patient retreat the area on reintroduction of the trigger (not necessary for a subluxation).

5. If a vitamin was the trigger, it is often necessary to supplement with it after the correction (it will now strengthen a weak G-2 muscle).

6. If the recurring finding still recurs, look for more triggers. They occasionally unlayer over a few visits.

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TREATING THE SEVERELY SENSITIVE ENVIRONMENTALLY ILL PATIENT

Michael Lebowitz, D.C.

ABSTRACT

In general there is an order of treatment when approaching the chronically sensitive patient to achieve optimal results. This idea is discussed.

INTRODUCTION

The severely sensitive environmentally ill patient is often the most challenging patient we have. These patients often have scores of symptoms effecting the nervous system, digestive system, endocrine system, and almost any other system. There can be literally hundreds of triggers- various foods, chemicals, metals, EMF's, etc., etc. For many of them we are their only hope. Where to begin on these patients and what type of sequence to follow can be hard to deduce because the volume of findings is overwhelming. Over the years of specializing in treating these patients a pattern of treatment has emerged that appears to bring optimum results.

DISCUSSION

If someone was throwing rocks at your house-you would have to initiate a strategy to correct the situation. Step one would be to get rid of the person throwing the rocks. Step two would be to repair the damage, and step three would be to rebuild. It is the same with the environmentally sensitive patient. There are three general steps to helping these patients. There can be some overlap of steps one and two simultaneously- and some of steps two and three simultaneously, but basically you need to stay in order. Just like it would be fruitless to try to repair a window that was presently still being bombarded by rocks; it would be equally fruitless to attempt to repair a liver still bombarded by mycotoxins, endotoxins, and xenobiotics. First we need to correct the problems before initiating repair.

This paper will not attempt to go into treatment procedures as seminar videos (1) and papers (2) are available to address these, but the purpose is to give a general overview and order of treatment as well as a few complimentary procedures to consider.

STEP 1

CORRECT DYSBIOSIS:

Dysbiosis (3) is often the main cause for these patients' symptoms. The mycotoxins and endotoxins are hepatotoxic as well as causing leaky gut syndrome, altered endocrine hormone levels, etc., etc. They also deplete nutrient levels quickly. I have found over the years that correcting dysbiosis either will correct many of the patients symptoms or will allow you to successfully move on and correct other issues that previously did not respond. Because of the connection between food sensitivities and dysbiosis, these should be addressed also. At this stage of treatment some patients can successfully handle vitamin and mineral supplements to correct deficiencies and some cannot.

STEP 2

Once dysbiosis and food sensitivities are corrected, it is appropriate to have the patient undergo detoxification procedures. To undergo detoxification before this, while the patient still has mycotoxin and endotoxin producing organisms and toxic reactions to sensitive foods yields little if any results in most cases. Detoxification can take many forms and should be patient specific. Possible detoxification procedures to consider include the following (remember do not start detox procedures until dysbiosis is corrected):

a) Sauna- sauna detoxification if done properly can be a lifesaver; if done improperly it can overload an already overloaded detox system and damage the patient. L. Ron Hubbard's book (4) gives good background

information. We have found that nutrient needs are very specific and can change hour to hour. It is beyond the scope of this paper to teach proper protocol.

b) Juice fasting, with daily water enemas has been a very effective form of detoxification. Again, proper procedures are talked about in our seminar videos.

c) Coffee enemas can be very effective. Read Sherry Rogers book (5) for details. We have found that in most patients any more than ten or twelve spaced at an every other day frequency is often too many.

d) Manual Lymph Drainage- the Vodder technique is very effective in detox and very gentle. We can supply names of MLD practitioners in your area.

e) Herbal Products- such as S.A.T. (6) or Medibulk (6) can aid detox but usually need to be done in addition to some of the above.

While dysbiosis can usually be corrected in 1-2 months, detoxification can take from weeks to years depending on the level of stored xenobiotics.

Many of these patients are metal toxic and these procedures will also assist in lowering their "load". If amalgam removal is necessary, the detox stage is the correct time to do it. Exact timing and procedures can be critical to prevent potentially severe side effects. Again see our videos for details. Detox is often necessary before and after dental procedures.

STEP 3

Rebuilding - Now that steps one and two are accomplished (at least in theory), the patientsí damaged body is ready to rebuild. Sometimes this will happen spontaneously at this stage or sometimes it needs our help.

Nutrient supplementation is critical now though often the procedures in our "Trigger" paper (7) need to be performed first so the patient can both tolerate and assimilate nutrients. Good basic A.K. is also essential. Lifestyle is critical to rebuilding: a healthy diet well suited to that person, exposure to only a minimal amount of environmental toxins, staying dysbiosis free, a maintenance dose of detoxification procedures, adequate exercise, sunshine, and rest are all critical.

CONCLUSION

There is a logical order of treatment when you have severely sensitive environmentally ill patients. Following these with some intelligent flexibility is necessary for obtaining optimum results.

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THREE VARIATIONS OF MANUAL MUSCLE TESTING

Walter H. Schmitt, Jr., D.C., DIBAK, DABCN

ABSTRACT

Manual muscle testing may be refined to three variations, the performance and clinical significance of which are discussed. The three types of testing are: G-1 (doctor started testing with eccentric contraction), G-2 submax (patient started submaximal concentric contraction followed by eccentric contraction,) and G-2 (patient started testing beginning with a near maximum isometric contraction of the muscle followed by eccentric contraction.) G-1 weakness is related clinically to spinal cord level reflex phenomena; G-2 submax is related to nociceptive withdrawal activity, immune system involvement, and visual motor activity; and G-2 weakness is related to the influence of other suprasegmental descending pathways. The usefulness of the three variations in determining the most useful AK pain relief techniques for a particular patient is reviewed.

INTRODUCTION

Manual muscle testing texts vary widely according to the type of contraction (eg., eccentric or concentric) to be measured, the timing and force of the tester, the timing and force of the patient's resistance to the movement, and other factors which will be discussed in this paper.

In 1964 Goodheart introduced the concept of using manual muscle testing for the identification of functional neurological problems. (1)(2) The professional investigation of this discipline has evolved to a clinical understanding that the outcomes of appropriately performed manual muscle tests may be employed to evaluate motor and sensory pathways throughout the neuraxis. Any neural system which has motor connections, either locally or by ascending or descending pathways, has the potential for functional evaluation using a somewhat more sophisticated system of manual muscle testing than is used in traditional neurological or impairment exams. This includes the possibilities of evaluating not only somatic sensory systems, but such central systems as visual motor activity (eg., accommodation reflexes), vestibular reflexes (eg., tonic labyrinthine reflexes), reticulospinal (autonomic) pathways, emotional (psychosomatic) pathways, and so on. By evaluating olfactory and gustatory stimuli, we have the tools for not only neurological evaluation, but neurochemical evaluation as well.

DISCUSSION

Observations have led to the discernment of three distinct methods of performing a manual muscle test. Clinically speaking, these are: 1) doctor started testing (also called G-1 testing), 2) patient started testing with submaximum contraction (called G-2 submax), and 3) patient started testing with maximum contraction (called G-2). There are a number of important factors including timing, amount of force by the tester, line of drive of the test, etc. which are part of the art of manual muscle testing. The most significant factor in extracting clinical information from a muscle test response is who (doctor or patient) starts the test and how much force is applied in which direction (i.e., eccentric, concentric, or isometric.)

1) DOCTOR STARTED TESTING ("G-1 TESTING")

Doctor started testing is an eccentric test of the muscle. The tester positions the patient for the test, makes contact with the patient, and tells the patient to resist his force in the appropriate direction. Then the tester pushes against the line of pull of

the muscle while the patient attempts to eccentrically contract the muscle. The outcome of the muscle test is usually graded "strong response" or "weak response" depending on the response of the patient to catch up with the doctor's force and "lock" the muscle in a static position. This is the type of manual muscle testing employed by Goodheart and most clinicians using applied kinesiology.

2) PATIENT STARTED SUB-MAXIMUM TESTING ("G-2 SUBMAX TESTING")

Patient started testing with submaximum contraction is a concentric followed by eccentric test. The patient is instructed to push against the tester's hand in the direction of the muscle pull. Immediately upon sensing the initial few degrees of concentric contraction, the tester then pushes against the patient's force in the direction of eccentric contraction. The patient's ability to "lock" against the additional force is graded "strong response" or "weak response."

3) PATIENT STARTED TESTING TO MAXIMUM ("G-2 TESTING")

In patient started testing with maximum contraction, the patient is instructed to push against the tester's hand while the tester holds the position as firmly as possible. When the tester observes that the patient has reached a maximum isometric force, the tester then pushes with increased force in the direction of eccentric contraction. It the patient is able to hold the position against the additional eccentrically applied force, the test is graded "strong response." If the patient is unable to hold the position, the test is graded as "weak response." It is obvious that a certain amount of skill is necessary to perform these tests and discriminate between the outcomes.

CLINICAL CORRELATIONS

Clinical observations first reported in 1986 (3) suggest that G-1 type testing be used for general screening and most AK testing. To elucidate the nature and source of the muscle weakness, G-2 submax and G-2 type testing is used. The amplification of muscle testing diagnosis using all three types of testing is of great clinical value in choosing the appropriate type of therapy to normalize the muscle imbalances.

G-1 weakness only is associated with problems arising from spinal level problems. These include subluxations, neurolymphatic reflexes, acupuncture tonification and sedation points, origin - insertion problems, and so on.

G-2 submax patterns (usually but not necessarily accompanied by G-1 and G-2 weakness patterns) are associated with withdrawal (flexor reflex afferent) responses resulting from tissue injury and nociceptor stimulation. G-2 submax patterns may also be associated with visual motor activity including accommodation and near-to-far reflexes and some extraocular activities.

G-2 submax patterns are also associated with immunological patterns such as allergy, hypersensitivity, and some infections. Differentiation of the source of a G-2 submax weakness is accomplished by TLing the areas associated with the immune system organs, that is, the thymus and the spleen. The thymus is TLed at the upper and lower sternum and the spleen at its neurolymphatic reflex area in the lateral left 7th intercostal space. If one or more of these "3 immune circuits" TL and negate the G-2 submax (and G-1 and G-2) weakness(es), then the problem is thought to be of immune origin. If TL to these 3 immune circuit areas is negative, investigation is directed toward identifying an area of tissue injury as the source of the weakness.

G-2 weakness patterns appear to be associated with suprasegmental sources of inhibition which are carried by descending pathways other than those associated with G-2 submax. The sources of these suprasegmental problems include cranial and TMJ related faults, emotional stress patterns and other cortical techniques, acupuncture head point ("B and E" points) techniques, "holographic" techniques, autonomic imbalances of sympathetic and parasympathetic nervous systems, and biochemical imbalances which may be monitored be the hypothalamus. These patterns usually are accompanied by a G-1 weakness, but such is not a hard rule.

Both functional and pathological (eg. multiple sclerosis, amyotrophic lateral sclerosis) patterns of upper motor neurons have been associated with G-2 only weakness patterns.

PAIN RELIEF TECHNIQUES & THE THREE TYPES OF WEAKNESS

The type(s) of weakness are of great value in directing the doctor to the most appropriate pain relief techniques for the patient's specific pain problem. When only a G-1 weakness is present, tonification point technique and/or spinal manipulation are the therapies of choice.

When G-2 weakness patterns are present, one or more or the following techniques will be successful: set point technique (4), cortical awareness techniques (5), holographic techniques (eg., right brain / left brain), and visceral referred pain techniques (6).

When G-2 submax patterns are present and TL to the 3 immune circuits is negative, the techniques of choice are injury recall technique (7), nociceptor stimulation - blocking technique (8), or cranial dural referred pain technique (9).

The type of muscle testing weakness patterns has been an excellent guide in choosing which pain control technique will be most effective for each patient. Tonification point tapping has produced fabulous results but has often failed. When failure is present, the weak indicator muscle may demonstrate a strong response to G-1 testing, but depending on the source of weakness, there will remain a G-2 and/or a G-2 submax weakness. What relief is achieved, it any, will be short-lived.

Nociceptor afferents affect alpha motorneurons (a-MN) through interneurons. These nociceptor reflexes operate through the a-MN bypassing the gamma motorneurons (g-MN) and the gamma loop. Mechanoreceptor reflex pathways operate through interneurons via the g-MNs and the gamma loop system. (10) This may explain the more complex weakness response patterns (i.e., G-1, G-2, and G-2 submax present simultaneously) associated with nociceptor stimulation type techniques.

CONCLUSIONS

Differentiating between weakness response patterns is an integral part of performing the functional neurological examination which is the basis for AK. The diagnostic information derived from identifying the type of weakness responses helps to more quickly identify the source of the patient's problems as well as direct therapeutic choices. The diagnostic advantage of using three types of muscle testing hastens diagnostic conclusions and speeds the care and recovery of the patient.

THREE TYPES OF MUSCLE TESTING

1. DOCTOR STARTED (ECCENTRIC)
   "G-1" GENERAL SCREENING

2. PATIENT STARTED SUBMAXIMAL (CONCENTRIC - ECCENTRIC)
   "G-2 SUBMAX"
   PAIN PATHWAY (INJURY RECALL TECHNIQUE)
   IMMUNE SYSTEM CHALLENGE TECHNIQUE
   VISUAL MOTOR FUNCTIONAL TESTING

3. PATIENT STARTED TO MAXIMUM (ISOMETRIC - ECCENTRIC)
   "G-2" SUPRASPINAL PROBLEMS

PAIN RELIEF TECHNIQUES & THREE TYPES OF MUSCLE TESTING

"G-1" - DOCTOR STARTED TESTING

THIS TYPE OF WEAKNESS IS ASSOCIATED WITH SPINAL LEVEL PROBLEMS SUCH AS:

1. TONIFICATION POINT TECHNIQUE
2. SIMPLE SPINAL SUBLUXATIONS

"G-2 SUBMAX" - PATIENT STARTED SUBMAXIMAL TESTING

THIS TYPE OF WEAKNESS IS ASSOCIATED WITH PAIN TYPE CONDITIONS IN WHICH YOU MIGHT USE:

1. NOCICEPTOR STIMULATION - BLOCKING ("NSB") TECHNIQUE
2. INJURY RECALL TECHNIQUE
3. CRANIAL DURAL REFERRED PAIN APPROACH

"G-2" - PATIENT STARTED TO MAXIMUM TESTING

THIS TYPE OF WEAKNESS IS ASSOCIATED WITH PROBLEMS ORIGINATING AT SUPRASPINAL LEVELS SUCH AS:

1. SET POINT TECHNIQUE
2. VISCERAL REFERRED PAIN TECHNIQUES (AUTONOMIC INVOLVEMENT)
3. CORTICAL AWARENESS TECHNIQUES
4. "HOLOGRAPHIC" TECHNIQUES

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TMJ & INTRINSIC SPINAL MUSCLE BALANCE

Walter H. Schmitt, Jr., D.C., DIBAK, DABCN

ABSTRACT

There is a relationship between mandibular protrusion, intrinsic spinal relaxation, and glycine. There is a similar relationship between mandibular retraction (retrusion), intrinsic spinal flexion and norepinephrine. Each mandibular position, spinal pattern, and neurotransmitter may strengthen a weak muscle. When a weak muscle strengthens by any of the three factors, each of the other two factors will also strengthen. All three factors will cause a positive therapy localization to one TMJ. The entire pattern may be negated by flexion of the atlanto-occipital joint (injury recall technique) performed simultaneously with any one of the three factors.

INTRODUCTION

An important pattern has been observed which demonstrates a relationship between mandibular protrusion and retraction, intrinsic spinal patterns, and the neurotransmitters glycine (GLY) and norepinephrine (NE). This pattern seems to be related to recurrent spinal subluxations. In many patients weak muscles have been found to strengthen to AK testing on protrusion or retraction (sometimes called retrusion) of the mandible. The muscle testing response to mandibular protrusion, relaxation (extension) of the spine, and insalivation of GLY all parallel each other. Likewise, a strengthening response by a weak muscle to mandibular retraction parallels the response to tensing (flexion) of the spine and insalivation of NE. When one of these three factors strengthens a weak muscle, so will the other two create a strengthening effect.

DISCUSSION

The spinal pattern seems to be related to intrinsic spinal muscle activity rather than gross paraspinal and postural muscle activity. Although the intrinsic spinal muscles are reputed to be beyond conscious control, these patterns appear to affect the intrinsic spinal muscles as a part of on overall neurological pattern of programmed motion.

When protrusion of the mandible strengthens a weak muscle, the supine patient is asked to relax the spine, releasing all tension of the muscles and letting it sink down into the table. This pattern of spinal relaxation also strengthens the weak muscle.

When retraction of the mandible strengthens a weak muscle, the supine patient is asked to tense the entire spine forward but to do so without raising off the table. This activity will cause a slight flexion pattern which recruits the intrinsic muscles without perceptible spinal flexion from the occiput to the pelvis. When retraction strengthens, this pattern of spinal tensing / flexion will also strengthen the weak muscle.

Trial and error testing with various neurotransmitter (NT) substances revealed that the protrusion / spinal relaxation pattern strengthening was accompanied by a similar strengthening response with insalivation with GLY. GLY is the major inhibitory NT in the spinal cord and is reputed to be the NT for the Renshaw cells which inhibit alpha motorneurons.

The mandibular retraction / intrinsic spinal flexion pattern strengthening is accompanied by a strengthening response with insalivation of a 1% NE solution. (1) This spinal flexion is similar to a sympathetic nervous system reaction, like when a scared cat arches its back.

The strengthening responses seen by each pattern of three factors is misleading. The strengthening responses are always accompanied by a weakening of a strong indicator muscle with therapy localization (TL) to one TMJ. In other words, if protrusion, relaxing the spine, and oral GLY each strengthen a weak muscle, then each will also cause a positive TL to a TMJ. We have called this a "durned if you do - durned if you don't" pattern which is present in many AK techniques which seem at first to help a patient by causing a strengthening response to a weak muscle, but are later to adversely affect some other system.

This entire pattern may be corrected by performing atlanto-occipital flexion (injury recall technique - IRT) with the patient TLing the involved TMJ while maintaining the mandibular protrusion or the spinal relaxation or the oral GLY.

The other strengthening pattern of mandibular retraction, spinal tensing, and oral NE will also accompanied by a positive TL to one TMJ. This pattern may be corrected similarly by atlanto-occipital flexion (IRT) with the patient TLing the involved TMJ while maintaining the mandibular retraction or spinal tensing or oral NE.

Both patterns (protrusion et al and retraction et al) may be present in the same patient. When this is the case, it is common for one pattern to not show up until the other is corrected. Although when both patterns are present, usually each is associated with a separate TMJ, both patterns have been observed to affect the same TMJ in some patients.

CONCLUSIONS

Following these corrections, there will be no strengthening response from the mandibular position, spinal pattern, or NT. There will often be an increase in some distant range of motion such as hip abduction. It also appears that recurrent subluxation patterns in the spine will not recur as in the past. The recurrence rate for these mandible / spinal / NT patterns has been quite low. One or two treatments have usually eliminated the pattern on subsequent examinations.

SUMMARY OF PROCEDURES

Testing a weak muscle, check for strengthening with mandibular:
1. Protrusion
2. Retraction

If protrusion strengthens:
The following will strengthen a weak muscle:
Relaxing spinal muscles (relaxing spine into table)
Glycine
One TMJ will TL with protrusion only.

If retraction strengthens:
The following will strengthen a weak muscle:
Tensing spinal muscles (fight or flee response)
Norepinephrine
One TMJ will TL with retraction only.

Correction:
Treat TMJ with protrusion or retraction by neck flexion IRT

REFERENCE

1. Available from Intertech, 170 S. Mountainway Drive, #110, Orem, Utah 84058. Phone (801) 226-6992

PRE & POST GANGLIONIC TECHNIQUE
Revisted

Sheldon C. Deal, D.C., NMD

ABSTRACT

This paper looks into a technique that was introduced in 1976. This technique gives us many insights as to how our multidimensional bodies operate The paper includes many updates that have occurred since 1976 which particularly helps us understand the bodies response to color and certain emotions.

HISTORY

This technique was originally called the Chakra Technique. The name was changed to the Pre and Post Ganglia in an effort to comply with more standard terminology. In the 1976 research manual

(1)Dr. Goodheart describes how Shafica Karagula in her book "Breakthrough To Creativity" (2) identified the whirling vortices of energy that extend from the body at various locations.

Chakra is a sanskrit word that means wheel or disc. Various authors have identified these vortices of energy and named them as different Chakras. C. W. Leadbetter (3), David Tansley (4) and Alice Bailey (5) are some better known examples of this.

Here is a list of Chakras and their Plexi:

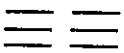
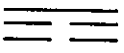
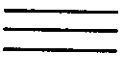
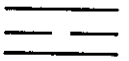
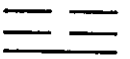
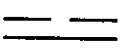
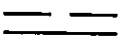
ROOT OR COCCYGEAL	=	PUBOCOCCYGEAL PLEXUS
SACRAL	=	SPLENIC PLEXUS
SOLAR PLEXUS	=	SOLAR PLEXUS
HEART	=	CARDIAC PLEXUS
THROAT	=	PULMONARY PLEXUS
BROW OR THIRD EYE	=	PHARYNGEAL PLEXUS
CROWN	=	CERVICAL PLEXUS

Goodheart found that the Pre Ganglionic areas along the spine would therapy localize and that a second hand on the Post Ganglionic areas on the front part of the body, which correspond to the alarm points would cancel out the first therapy localization.(6) Thus a two handed contact by either patient or the doctor was the treatment of choice to balance these energy centers.

UPDATE

I found the above method could be confused with a subluxation, an organ problem or an acupuncture problem. One way to tell what you found is that a chakra would therapy localize an inch or two above the body, whereas the other examples mentioned would not. So now I have a method of differentiation. I also found a short cut for checking all the chakras at one time, was to have the doctor touch SP-21 on the left. If this did not show then checking the individual chakras was not necessary and only if it did show, do you need to check them individually. This is in contrast with SP-21 on the right which shows a need to cross crawl. If the patient touches either SP-21 and it changes your indicator then that indicates a blood chemistry problem.(7)

A Chakra indicator also tells us what color or what emotion the patient could benefit by here is a chart listing some of the characteristics of each chakra.

CHAKRA	COLOR	EMOTION	NOTE	I CHING
ROOT	RED	I AM GROUNDED IN MY BEING I AM SECURE IN WHO I AM	C	
SACRAL	ORANGE	MY SEXUAL EXPRESSION IS FULFILLED I REPRODUCE WITH EASE	D	
SOLAR PLEXUS	YELLOW	I ACKNOWLEDGE MY GUT FEELING I AM IN TOUCH WITH MY UNIVERSE	E	
HEART	GREEN	I KNOW AND UNDERSTAND LOVE I ACCEPT LOVE FROM OTHERS	F#	
THROAT	BLUE	I CAN SPEAK FREELY I CAN EXPRESS MYSELF	G#	
BROW	INDIGO	I CAN SEE CLEARLY I COMPREHEND WHAT IS HAPPENING	A	
CROWN	VIOLET	I AM AT ONE WITH UNIVERSAL ENERGY I AM OPEN TO ALL KNOWLEDGE	B	

It can be demonstrated that the chakras can be affected and or fixed by any one of the methods indicated in the above chart. you may chose what suits your patient best or you may simply use a two handed contact to jump start the chakra. This is done by placing one hand on the front on the body and one hand on the back of the body and held until you can feel a equal pulsation under each hand A exception would be the brow chakra which requires one hand over the left eye and the crown chakra which requires one hand over the right eye.

The physical body is the densest component of many interactive energy fields. Each of these fields or higher dimensional bodies is connected to the physical cellular structure through a complex network of energy threads This integral web of life energies allows the higher vibrational forces to manifest in the physical body through their guiding effects upon the patterns of cellular growth and upon the unfolding of human consciousness.

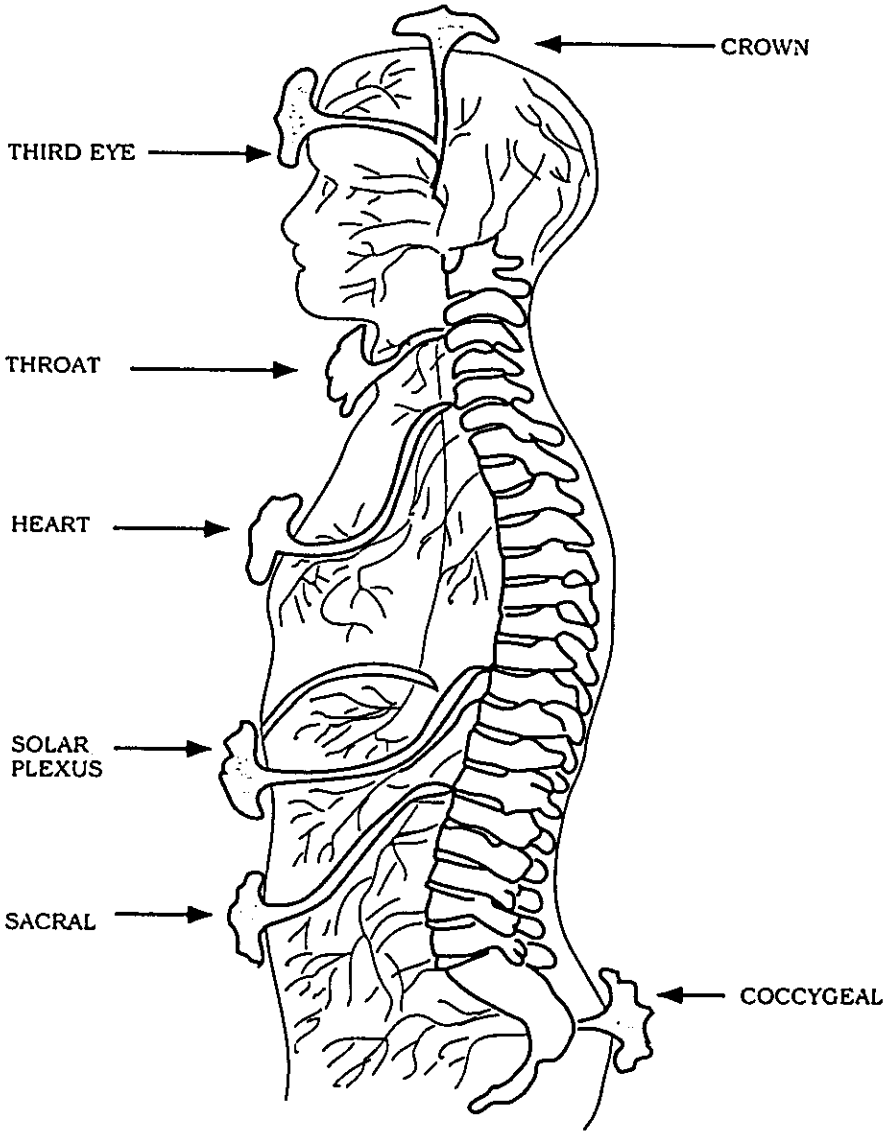
The multidimensional network allows energy of varying vibrational characteristics to flow into the body and influence behavior at both the cellular and organismic level. In order that incoming subtle energies are properly integrated into the cellular matrix, they must first pass through specialized step down transformers.

These unique centers, known as chakras, process vibrational energy of specific frequencies.

CONCLUSION

In quantum physics it is thought that mankind is made up of various energy fields that represent a multidimensional body. The Different chakras represent a connection between different levels. A lesion present at the chakra level can lead to untold problems in the nervous system and the organ systems. To find and correct a chakra lesion is a very worth while piece of the jigsaw puzzle.

The Seven Chakras
&
Autonomic Nerve Plexuses



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7. 1979 Workshop Procedure Manual, G. J. Goodheart, Self Published

SHEARING VS. COMPACTION TYPE INJURIES

David W. Leaf, D.C., DIBAK

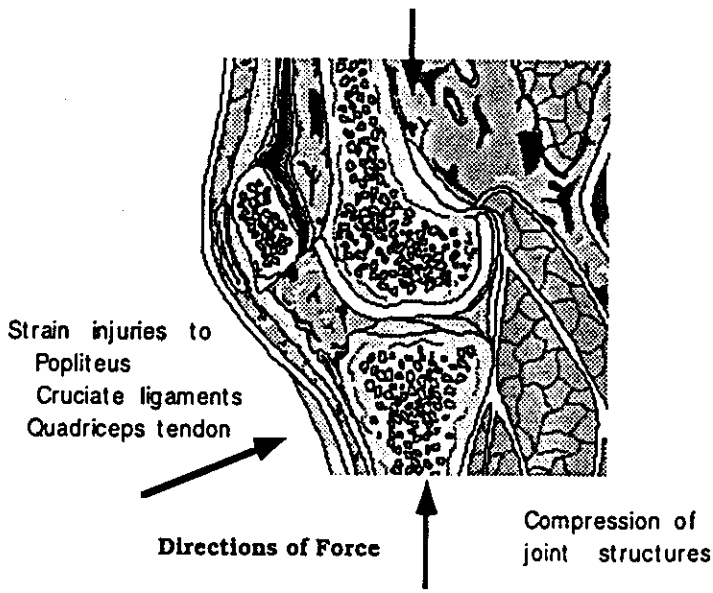
ABSTRACT

This paper discusses shearing and compression types injuries to joints, and proposes a treatment for multiple muscle weakness patterns found in a compressed joint. A discussion of a nutritional approach to ligament pain is also covered.

This past year, a patient presented himself for treatment who had fallen off of a bicycle. The history revealed that he had broken the fall with his right arm extended. The symptoms involved the whole arm with marked weakness of the muscles of the shoulder. Testing revealed weakness of the deltoid, supraspinatus, infraspinatus, teres minor and teres major muscles. The subscapularis was strong and internal rotation f the shoulder was markedly reduced at 40 degrees. There was difficulty in increasing the strength of the weak muscles using our normal treatment procedures. The thought occurred to me that when you jam your finger, the instinctive thing to do is to traction the joint. I placed the arm in the relative position that the arm was in during the fall and tractioned the joint. Immediately the weak muscles strengthened. This was the first patient that I used this procedure on.

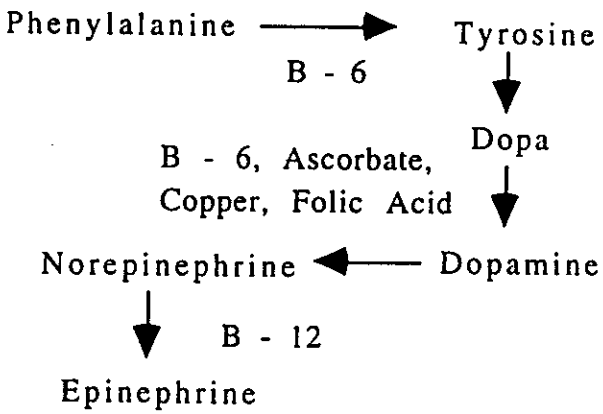
Traumatic injuries tend to occur in two types. The first is a shearing type of injury. These are the most common. They occur when the joints and related structures are strained and twisted causing injury to muscles, ligaments, skin and the proprioceptors of the joints. The classical examples of these types of injuries would be a strained ankle, a whiplash injury to the cervical spine or the person who bends over to pick up an object and feels a snapping in their back. These types of injuries require treatment to all of the injured structures for rapid recovery. Consequently, joint mobilization (manipulation), muscle, ligament and skin proprioceptive procedures and needed. The second type of injury is the compaction type. In this injury, the person falls to the ground and breaks the fall with the arm, holds the steering wheel with straight arms at impact, or jumps down and lands with locked knees. In these cases, there is little to no tearing of tissues and swelling, if present, is limited to the joint capsule.

In the shearing injury, you will find the trilogy of weak muscle, a synergistic muscle that exhibits tenderness to palpation and the need for strain counterstrain and an antagonist to the weak muscle that tests for the need of the Travell fascial procedures. These findings form the basis of the treatment that is needed.

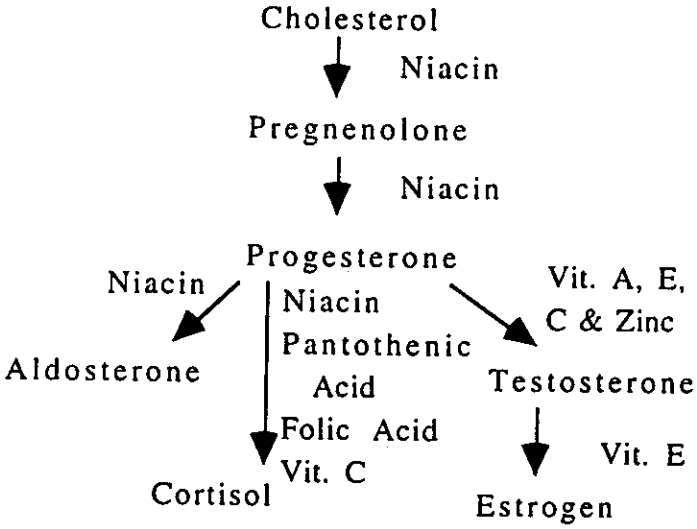


When the weakness pattern or the initial injury strains the ligaments, residual pain over the ligaments will be found. Following the concepts of Deutsch that there is a relationship between the ligaments and the adrenal glands, residual pain over the ligaments has been tested for response to adrenal type nutritional support. In general, if the ligament is involved with pronation or internal rotation of the joint, the pain pattern will diminish on the insalivation of nutrients designed to support the adrenal medulla. If the ligament is involved with supporting supination or external rotation of the joint, nutritional support for the adrenal cortex will reduce the pain.

In an in office study, 50 patients with pain over the lateral epicondyle associated with weakness of the pronator teres were tested for decrease in pain following chewing of a nutritional supplement designed to increase production of the tyrosine based hormones (Metagenics Energetics). In 46 of the cases, the patient's reported a dramatic decrease, over 60% reduction, in palpable pain. To ensure equal pressure for palpation, the Metrecom was used to measure the pressure of palpation. Initial pressures used to elicit the pain were used for the subsequent trials.



Twenty five patients with a history of eversion sprains with palpable pain over the peroneus tertius tendon were tested in a similar pattern. The nutrient combination chosen was Cortico B5-B6 from Metagenics. In this case, 22 cases showed the same reduction in pain to an equal stimulus.



In a review of 100 cases of shearing type injuries, 68 percent needed to be treated for proprioceptive imbalances of the skin.

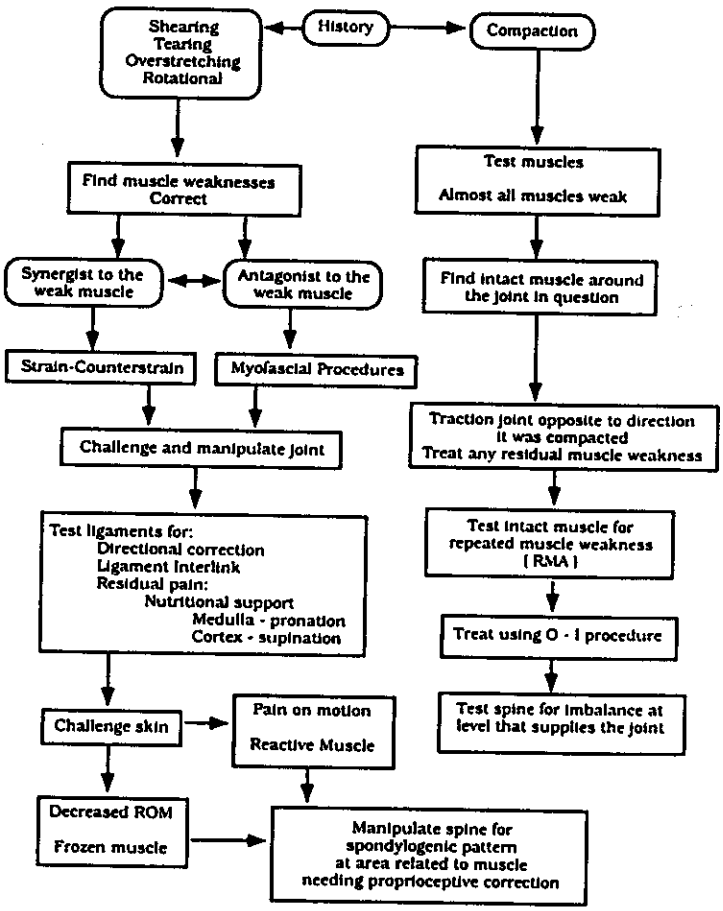
Depending upon the severity of the injury and the length of time before initiation of treatment, the patient will adapt to their injury and require treatment for muscle incoordination. These coordination problems may involve synergistic muscles or antagonist muscles. Imbalances in the coordination of the firing of synergistic muscles result in pain and the weakening of specific muscles under activation. The patient complains of pain or ache while using the extremity. Limitation in active range of motion indicates an imbalance in the strength of contraction of the prime mover to properly inhibit the antagonist of the prime mover. In all cases, the spine must be checked for involvement at the level of the innervation of any muscle that is treated for a proprioceptive imbalance and at the level that supplies the joint that has been traumatized.

In the compaction type of injury, the presenting findings are marked weakness of almost all muscles surrounding the joint. One or two muscles will test strong. Further testing will show that the "intact" muscle will weaken to repeated muscle activation. Hypothetically, the continued contraction of this muscle strains the attachments of the muscle so that upon repeated use of the muscle through its normal range of activity will result in a weakening effect. Treatment is then directed to the origin and insertion of this muscle. The pain normally associated with this type of treatment can be dramatically reduced by placing the involved muscle in its shortened position while performing a circular massage over the myotendinous areas.

In the compaction type of injury, the trauma is directed mostly to the joint itself. The stress effects mechanoreceptors and nociceptors in the joint structures. Biedert, Stauffer and Friederich investigated the occurrence of free nerve endings in the knee joint. They found that the density of type IV free nerve endings (nociceptors) was highest in the medial and lateral retinacula and the patellar ligaments. They concluded that the presence of these proprioceptive fibers is important in the active control of the patella and the rotation of the tibia and that injury to the mechanoreceptors found inside the joint capsule, especially the anterior cruciate ligament, results in knee instability. Repeated joint distraction apparently normalizes the afferent discharge of these articular receptors reestablishing the normal tonus of the muscles.

Goodheart wrote in 1994 about the repeated muscle activation procedure, expanding upon the observation of Leaf that a way to find hidden weakness patterns was to have the patient actively use the extremity ten times. Goodheart then developed a treatment protocol for treating this induced weakness pattern. Finding these muscle weakness patterns was not always easy. The observation that these patterns will always be found when the injury has occurred due to a compression of the joint has dramatically speeded up the recovery of these patient's.

The following treatment protocol is suggested for the two types of injuries.



In summary, there appear to be two different types of injuries frequently encountered in the office setting. One is of a shearing - tearing action that injuries multiple structures. Each of the structures requires prompt and efficacious treatment to speed recovery. The type of injuries sustained in a compression - compaction of a joint appear to be mostly to the internal structures within the joint and to the free nerve endings and mechanoreceptors of that joint. These types of injuries exhibit a common finding of multiple muscle weakness that responds to linear traction applied to the joint. In these cases, the remaining intact muscle responds to the repeated action technique developed by Goodheart in 1994.

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QUALITY MUSCLE TESTING

Wolfgang Gerz, M.D., DIBAK

The single most determining factor in AK, the key to whether we examine the patient in a sensible way, is a good muscle test. Unfortunately, from the beginning of his work until today, George Goodheart has never defined the AK muscle test , so that all his students and the body of ICAK could have had one - and the same - starting point.

Therefore, a multitude of totally different ways of muscle testing are actually applied in the various groups that work "kinesiologically": "Touch for Health", "Clinical Kinesiology", "Kinesiology" etc., but - unfortunately - also ICAK. Goodheart personally says (1):

"I taught myself to test muscles in the early 1960's, probably 1962 or 1963. The fundamental principles in Manual Muscle Testing by Kendall and Kendall were my guide.

I have not found that Doctor initiated or patient initiated muscle tests are clinically significant per se - careful instruction for the patient is necessary.

The "patient initiated weakness" on testing or the "Doctor initiated weakness" on testing is a further refinement in eventual therapeutic diagnosis. The former for dural involvement, the latter for supra spinal involvement.

The use of muscle testing is an art, but Kendall describes it well." .

Comment

1. George Goodheart's statement creates more uncertainty than less, because the third paragraph contradicts the second.
2. Although I sent George the English edition of the new book "Matrix System and Matrix Regulation" by Pischinger and Heine and also tried to inform the AK community about the importance of this Matrix System earlier (6), he still discusses the muscle testing only from the viewpoint of the nervous system.
3. The differentiation "patient initiated = dural" versus "doctor initiated = supraspinal" was first advocated by Wally Schmitt (7), who defined the so-called "Gamma 1"- and "Gamma 2"-Test.

   As modern neurology questions the existence of the "Gamma 1"- and "Gamma 2"-loops, he changed the names into "G 1" and "G 2" (8).
4. Modern Natural Medicine in Europe - based on the Matrix System or System of Ground Regulation according to Pischinger (5) - can no longer accept these definitions and differentiations, as there is no neuro-muscular interaction at all without participation of this Matrix System.

Walther writes (7): *"If a muscle is weak because of a subluxation, dysfunctioning neuromuscular spindle cell, cranial faults, or whatever, one cannot expect the nutrition associated with the muscle to strengthen it.*

Factors that influence Manual Muscle Testing: *Consistent timing is imperative in comparing one muscle test with another. The most crucial portion of applying pressure in a muscle test is at the beginning. Schmitt reports a difference when the action of the muscle being tested is initiated before or after the doctor's application of pressure. This is termed "doctor-induced" or "patient-induced" muscle testing.*

The routine method discussed here is for the doctor to provide resistance to the patient's isometric contraction with increasing pressure to take the patient's muscle into eccentric contraction; this is doctor-induced muscle testing. It is hypothesized that it tests the gamma I system. The patient-induced test is when the effort of the test is started by the patient concentrially contracting against the examiner, who tries to stop the test. This is hypothesized as testing the gamma II system. Most of the time patient-induced and doctor-induced muscle tests parallel each other."

Comment

I cannot agree with the last sentence; actually very often there is no correlation at all between the various muscle test versions. While the G1-Test gives all the advantages to the

examiner, a well-performed G 2-Test gives maximum advantage to the patient.

Goodheart, too, points out the difference in diagnostic information that can be obtained from the two different forms of muscle test (1).

Farkas (9) writes that, when examining a muscle for the effect of the North and South Pole of a strong magnet, the G 1-Test brings results exactly opposite to the G 2-Test. His conclusion is that the G 2-Test gives deeper and more useful information than the G 1-Test.

I think that the two most critical aspects of muscle testing are not mentioned at all in this description of Walther:

- the increase of speed of the examiner's pressure
- the level of force or better the amount of maximum contraction that the patient should or is allowed to reach

Let's follow George Goodheart's recommendation and read what Kendall & Kendall write (2):

"...Pressure must be applied gradually. The patient must be allowed to "set" his muscles against the examiner's pressure. The examiner cannot gauge the degree of strength unless pressure is applied gradually because slight pressure applied suddenly can "break" the pull of a muscle of any strength."

Walther (8) actually explains quite precisely how important the timing of the muscle test is, but unfortunately does not discuss the amount of force that the patient should be allowed to reach :

"In manual muscle testing, the muscle is required to adapt to the changing pressure of the examiner's force. This requires effective function of the gamma system adjusting the neuromuscular spindle cell, and proper interpretation of its afferent supply by the central nervous system.

The manual muscle test, as generally described, starts with the examiner asking the patient to resist as he applies force to the patient. With the examiner's application of force a sensation of muscle locking is perceived. With this perception, the examiner increases his testing pressure to overcome the patient's isometric contraction, taking the muscle into an eccentric contraction; that is, the muscle is lengthened by the examiner's pressure while the patient continues to attempt to stop the movement. It appears that a major factor in this type of test is the ability of the patient's nervous system to lock the muscle against the examiner's pressure, and to continue adapting the muscle to meet the changing demands of the examiner's test. Often the examiner perceives a muscle as weak because it is late in adapting to his changing pressure. If the examiner applies pressure very slowly, allowing additional time for the muscle to adapt to it, the muscle will be perceived as strong.

It is amazing that the time factor of the manual muscle test is not considered more thoroughly. As early as 1917, Ryan and Agnew (10) proposed that the product of force and time was the significant factor. Nicholas et al. (11) convincingly demonstrated the influence of time on the test. They state, "It has been generally assumed that manual muscle tests are tests of 'strength'; that is, of the force with which the patient resists the tester. Our data indicates that time required to move the limb through a certain range of motion multiplied by the average force of resistance applied during that range was the factor most highly correlated with the tester's perception of deficits in strength. "When a muscle is in voluntary isometric contraction, EMG reveals that additional muscle fibers contract at low forces; when the force increases, the rate of firing becomes the predominant mechanism to increase strength. Tension, velocity, and electrical activity are interdependent. ...This indicates the need of proper neurologic control for the muscle to meet the changing pressure demands of the manual muscle test."

Let us compare this description with what is actually happening in a Deltoid test between patient and examiner:

1. a) The patient is asked to bring the arm into the testing position of 90 degrees abduction and 90 degrees flexion in the elbow.
   b) The examiner now takes a soft broad contact over the elbow

   During this the patient has to hold the arm in the starting position; i.e. contract the deltoid and its synergists. Inability to even do this properly is definitely a

weakness, with or without pain and restriction to abduct to 90 degrees.

2. Now the actual interaction for the normal AK test between patient and examiner may start
:a) Patient and examiner increase their pressure:
the patient cranially
= towards abduction
the examiner caudally
= towards adduction.

The critical question now is:
Who - patient or examiner - contracts first and how hard and how rapid?

It is now time to define some terms:
Maximum force
= relative maximum force
= psychologically limited force:
"The amount of force that can be produced during voluntary maximum static muscular contraction which does not include the autonomous reserve - usually 30% in the average human being." (12)
This is usually called "strength".

Absolute maximum force
"The amount of force that can be produced during involuntary maximum static muscular contraction. It is impossible to develop absolute maximum force voluntarily; there must be some sort of additional stress (fright etc.) or Hypnosis or Doping. "(12)

Power :
= "The amount of dynamic force per time".(12)

I think that a good AK muscle test should examine the patient's ability to adapt to the changing pressure of the examiner by accessing some % of the reserves of the autonomous nervous system + Pischinger's Matrix system + Selye's system of adaptation - all of which are interrelated - involuntarily !

What are the possibilities of interaction between examiner and patient?

1. **The examiner pushes first.**
This examines the reaction time of the patient and his power (force per time!).
If the examiner only increases his pressure fast enough, he can always push the patient's arm down. It is therefore a matter of the examiner's intention and/or intuition, whether the muscle is perceived as "strong" or "weak".

2. **The patient contracts first.**
The question here is how hard he contracts and what is the examiner doing during that time?
Naturally, the patient can not be asked to contract with 20, 50 or 75% of his relative maximum strength. Therefore we are left with 3 options:

a) The patient pushes his arm up somehow, while the examiner tries to keep the test isometric by increasing his counterforce accordingly.
This is primarily a test for the reaction capacities of the examiner.

b) The patient pushes his arm up **somehow**, the examiner pushes against - thus trying to keep the test isometric - and then increases his pressure **somehow.**
This is only a variation or actually a combination of 1. and 2a), as the two most critical questions remain undefined: how hard is the contraction of the patient, and how quickly and how hard does the examiner develop his additional force?

c) The patient is given all the time to contract the muscle as hard as he can; he can therefore reach his maximum relative strength. (= P max.). The examiner increases his pressure parallel to the patient's increase, but in the opposite direction, thus keeping the test isometric. When he feels that the patient has reached P max., he carefully (slowly and gently) increases his pressure by some more percent, while the patient is asked to keep pushing as hard as possible.

DISCUSSION

Option 2c)
is the most clearly defined and the one that gives maximum advantage to the patient.

2a)
is definitely less useful unless the patient is hardly able to keep his arm in the starting position.

and 2b)
give every advantage to the examiner. He can actually do with the patient's muscle whatever he wants: the "classic" Touch-For-Health" or "Kinesiology" test!

The sceptic but serious newcomer as well as the more advanced AKer who questions the quality of his own or somebody elses testing have no well defined guidelines how to really test a muscle.

In my opinion, this situation was and is unacceptable, and I have therefore tried for years to get George Goodheart to authorize one definition for an AK muscle test.

During the ICAK Summer Meeting in Philadelphia 1991we discussed this topic again and he asked me to write a description and definition of how to test a Deltoid.

With the help of my most important teacher in AK, David Leaf, D.C., the following description of a Deltoid Test was written and then authorized by George Goodheart:

Dr. George Goodheart, D.C., the founder of Applied Kinesiology, defines the art of muscle testing as follows, using the deltoid muscle as an example:

"I ask the patient to bring the arm into a position of 90° abduction with 90° flexion in the elbow. I then make sure that the patient understands the testing procedure, which consists of the patient pushing into a direction of further abduction as hard as he can against my own pressure. This pressure is applied with a broad soft contact of my hand resting over the distal humerus and the proximal forearm in a caudalward direction.

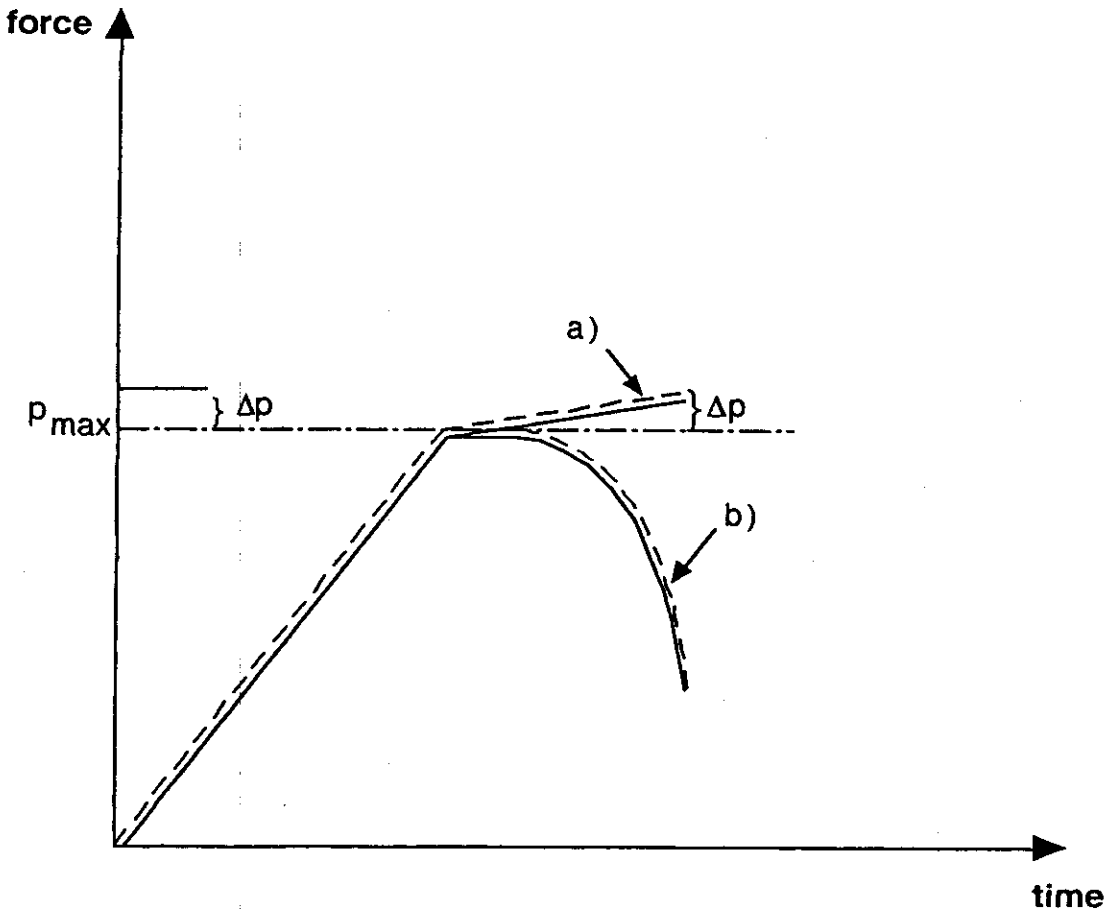
The whole muscle test is isometric; I feel how the patient builds up his maximum force and then add a little more pressure of 3 - 5 % for 1.5 - 2.5 seconds. "Strength" is defined as the patient's ability to resist the little extra pressure; "weakness" is defined as the inability to resist that extra pressure.

So, in essence, it is a patient started test that does not evaluate absolute muscle strength in pounds or kp's, but the patient's ability to perform a maximum isometric contraction plus resist my little extra pressure.

Unless for specific questions like aerobic/anaerobic problems the test shouldn't last longer than 2 - 3 seconds."

During the many years I have been studying, teaching and discussing AK - with beginners as well as advanced AKers - I have experienced again and again, how much a detailed and understandable definition for the AK Test is wanted.

Therefore, according to George's definition of a Deltoid Test, I have been suggesting the graphic in figure 1 and verbal description since 1992:



P max =		Relative maximum strength of the patient (= subjective maximum strength)
___	=	Patient´s pressure
-----	=	Examiner´s pressure
ΔP	=	2-3-4% extra pressure, started by examiner, gently and slowly increasing
a)	=	Patient´s muscle is strong, i.e. able to adequately adapt to the increase Δp exerted by the examiner ("locking in")
b)	=	Patient´s muscle is "weak", i.e. suddenly "collapsing". This shows inability to adapt to Δp

Recognizing P max and sensible testing show the examiner´s art of muscle testing.
The slight increase Δp, exerted by the examiner, and the patient´s reaction to it - (a) or (b) - are the critical steps of the AK muscle test
This description of a muscle test meets the following criteria:

1. The speed of the examiner's increase in force (power) plays hardly any role.
   This is a criterion that both Kendall & Kendall and Walther find essential.

2. As much as possible all advantages are given to the patient.
   - He contracts the muscle - after careful instruction - first and as hard as he can.
   - He does not have to react to the examiner's pressure, which would be unknown both as to the strength and power!
   - Therefore the patient does not have to pay attention to or take care of the examiner during the muscle test itself. The adaptation to the slight increase in the examiner's test pressure delta p at the end of the test should be **involuntarily!**
     It is the examiner's responsibility to sense the patient's P max.!

3. Thus it is primarily the patient or more precisely his neuromuscular adaptive capacity which is being examined. The ever-existing interaction between examiner and patient can be reduced as much as possible.
4. The examination is most individual:
   The examiner has to sense and adapt to the patient's personal P max.. This relates to his own strength as well as the testing position (including adequate leverage) and the speed and duration of the test.

This is quite opposite the typical G 1 or "Touch-For Health" or "Kinesiology" test where the patient is asked "to hold" or "resist", which really means he has to react to the unknown pressure of the examiner. As was pointed out earlier, most advantages in this type of test are with the examiner: he contracts first the patient can easily be overpowered both by the amount of force but especially by the unknown increase of speed (power), both of which remain undefined the patient can only reach P max if he has perfect reaction compared to the examiner's power.

Undoubtedly some practitioners and even lay people have tremendous success with this method. What this requires, however, is an almost radiaesthetic intuition to just adapt the testing pressure to the patient's power and strength.

Is this - adaptation to the patient's power and strength - not exactly the most essential requirement for the "classic" AK test as suggested above?

Why are there people who do not want to give maximum advantage to the patient in a clear and well-defined manner?

The answer may not be welcome for a number of people:
it is easier to cheat with the "G 1" = "TFH" = "Kinesiology" Test!

Or, in nicer words:
The doctor/patient interaction can be deliberately influenced by the examiner according to his own opinions and prejudices, and the patient can be impressed with a "pseudo - measurement" and be formed or manipulated towards any direction.

Those who want to do that should go ahead or continue to do so.
I think, however, that this would be exactly opposite the underlying idea of AK, which is to examine the patient as individually as possible, trying to understand his own personal adaptive and reactive capacities and functions.

Again, I want to stress that everybody can test as he likes. Also, I am not at all implying that anybody tests wrong voluntarily or knowingly projects too much of his own thoughts into the muscle test.

However, I want to remind you of the following statements by George Goodheart, the founder of our wonderful method Applied Kinesiology:

"Muscle testing is an art"

"You can only find what you know"

"There are many ways to test a muscle"

The reader and student of AK should now be able to choose the most appropriate form of muscle testing!

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LET'S ALL SPEAK ENGLISH, PART II

Wolfgang Gerz, M.D., DIBAK

Since the introduction of "the hypertonic muscle" into AK (1,2), many terms have been created and used, which have never been put together in one concept.

While writing this paper, I had a telephone conversation with Wally Schmitt, because I needed a reference from him. This was: "Let's all speak English" - and the more I thought about it, the more I liked this title and the idea behind Wally's article: to create a clear and simple terminology that we can all use to communicate in a better way.

If we accept that there are three different possible test results in a muscle test -weak, normotonic, hypertonic - (3, 4), we have to define these states in which a muscle can be found, first: see table 1.

Then we have to think about what may happen to a muscle, when **any type of Challenge** is positive:

- a weak muscle may become normotonic strong
- a weak muscle may become hypertonic strong
- a normotonic muscle may become weak
- a normotonic muscle may become hypertonic
- a hypertonic muscle may become weak
- a hypertonic muscle may become normotonic

That's it. Six possible reactions. Table 2 shows these graphically, with the abbreviations meaning:

s = strengthening of a weak muscle
w = weakening of a normotonic muscle
SC = Superchallenge = weakening of a hypertonic muscle
HC = Hypertonic Challenge = either a weak or a normotonic muscle becomes hypertonic
NC = Normotonic Challenge = a hypertonic muscle becomes normotonic

The "modern" patient, i.e. the one that is in an advanced stage of resistance according to Selye (5), may constantly shift between either one of these reaction modes (table 3).

What does this mean for us in Applied Kinesiology?

It means nothing else than the clear need to examine all patients who we either suspect to be hypertonic or to become hypertonic after a challenge in a different way from the usual procedure:
Every strong muscle has to be checked for normotonicity/hypertonicity after every single challenge!

TABLE 1
Normotonic Muscles

When a muscle has been identified as strong, a further differentiation must be used to determine if this muscle is hypertonic or normotonic. In AK, a normotonic muscle is defined as one which is strong, but is perceived as weakening when one of the following procedures is used:

a) TL to the sedation point of the meridian associated with that muscle, on the side of the muscle being tested.

b) "Running the meridian in reverse":
The associated meridian can be contacted and stroked lightly, but quickly, with the palmar aspect of the hand, in the direction from the highest point to the lowest point on the meridian. This should inhibit the related muscle for approximately 10 sec.

c) Spindle Cell Manipulation:
A contact is taken to the center of the muscle belly with two fingers approximately 5 to 10 cm apart in line with the muscle. Pressure is then applied sharply, 5 times, with both fingers toward each other (approximation of the muscle belly fibers). This should inhibit a muscle for up to 10 sec.

d) Either of the two poles of a strong, axially polarized magnet (minimum 2000 gauss), centrally placed upon the belly of the muscle.

Regarding the effects of the N and S pole of magnets on the strength of muscles, seemingly contradictory responses have been observed. Therefore, at this time, it is suggested that both poles be tested.

If none of the above methods causes weakening of the muscle, it is defined as hypertonic. It is recommended that at least two of the above procedures be used when evaluating the status of a muscle.

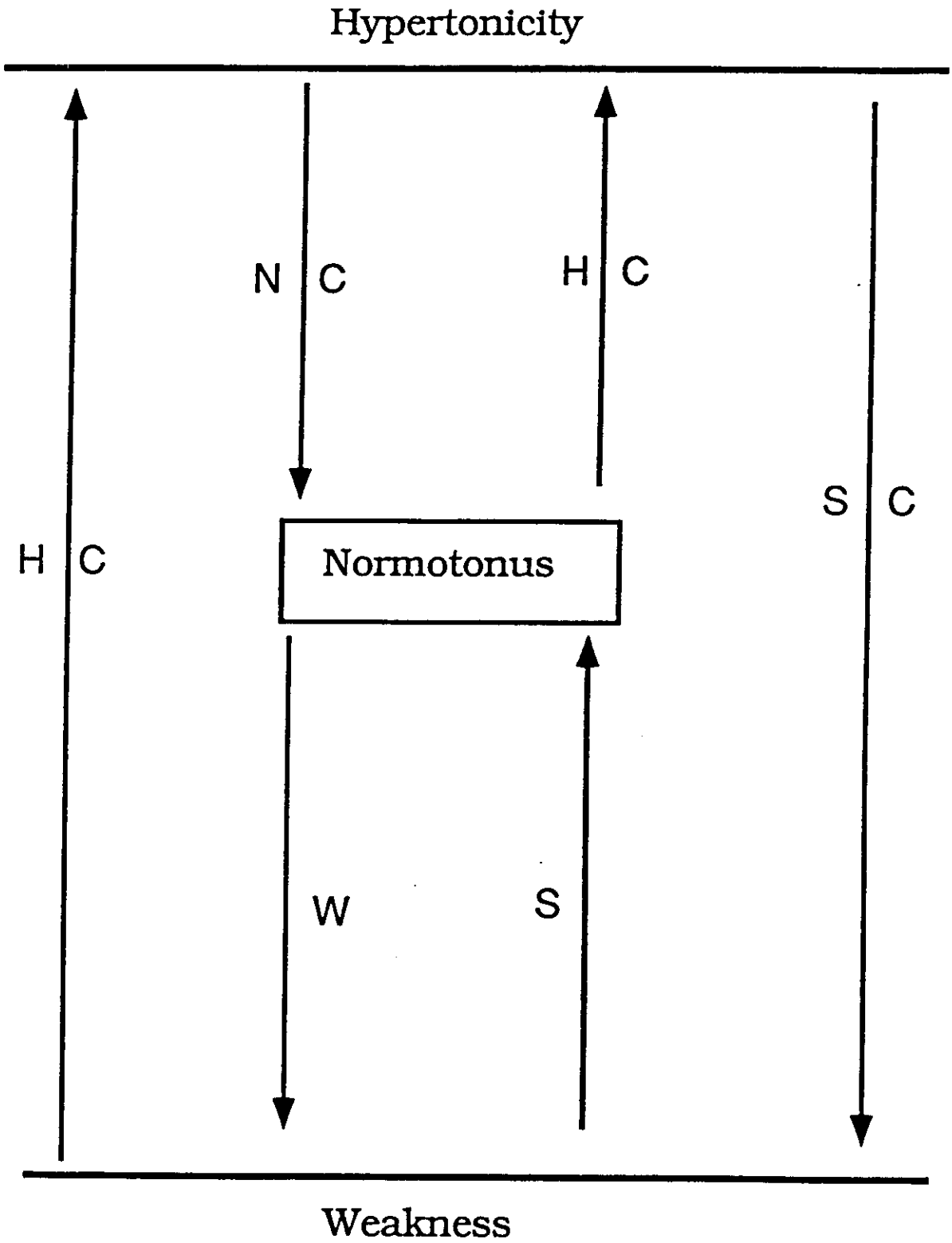
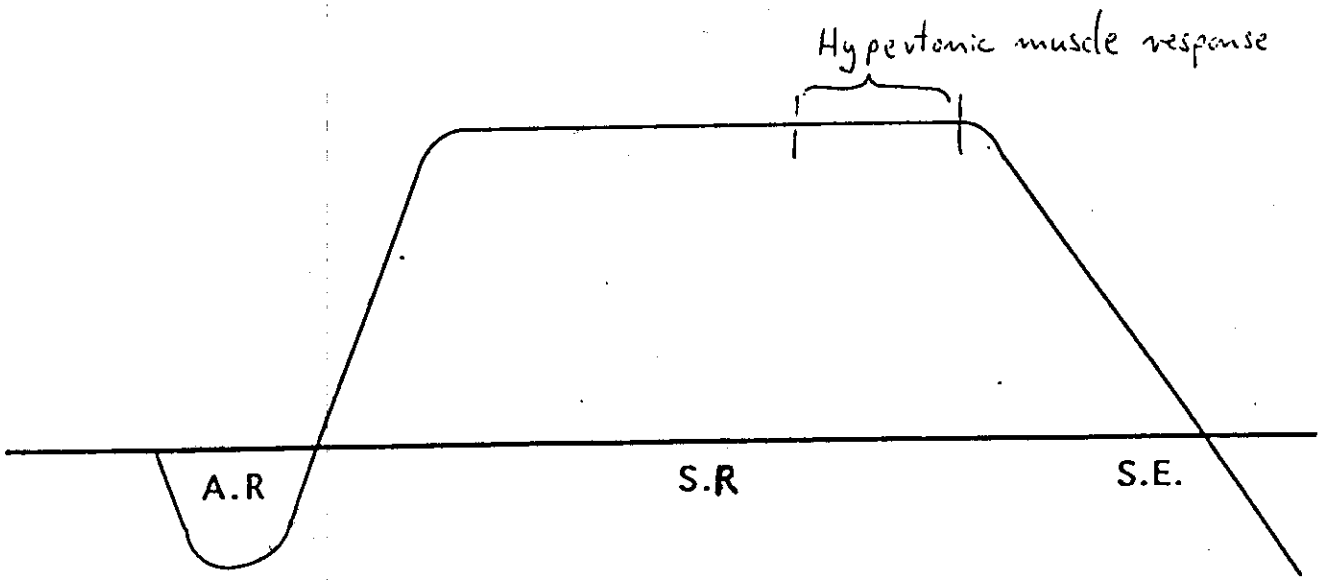


Table 2

The triphasic course of the stress response according to Selye



A.R. = Alarm Reaction
S.R. = Stage of Resistance
S.E. = Stage of Exhaustion

Table 3

This definition was agreed upon during the 1994 conference of ICAK-Europe in Monte Carlo in a meeting of the European Diplomates.

Some examples may show the significance:

a) a patient may have a strong left PMS and a weak right quad. An oral challenge with Candida antigen leaves the PMS strong, but also strengthens the weak Quad. Does that make clinical sense? Does it mean we have to feed the patient with Candida three times daily? No!! The explanation is most likely that both muscles have responded to the Challenge with hypertonicity (HC), which means an allergy-like, hyper-ergic reaction to the antigen and - as clinical consequence - a need for adequate treatment against Candida!!

b) Starting with a hypertonic indicator in a patient with a severe emotional problem, TL to the stomach neurovascular is performed. The muscle remains strong. Does this mean a negative TL?

No!! The indicator may have become normotonic through the TL and thus indicate a need for exactly that treatment!

c) Using AK with visceral osteopathy, we have found that mechanical challenges to organs (like an ICV, kidney or liver) also can produce a hypertonic response (HC). This means a contraindication for treatment according to this challenge! Spinal, pelvic and cranial challenges hardly ever produce a hypertonic challenge (HC).

On the other hand, especially the Total Compression Syndrome as described by Chris Smith (6) frequently is a Superchallenge (SC), when TLed bilaterally anywhere on the left and right side of the body, as Chris found out.

Another frequent mechanical SC is hard bite! This shows a need for further evaluation of the Stomatognathic System! In this case, we have often found that a corrective challenge according to Meersseman (7) or Gelb (8) may be the Normotonic Challenge (NC), i.e. : **putting the condyles of the mandible into the right position may make all muscles in the body normotonic!!**

d) A 40-year-old female patient comes to the office with a myriad of complaints, from allergies to headaches. Examination shows all muscles to be strong. Does that mean she is healthy? No!! Checking for hypertonicity reveals that all here muscles are hypertonic!

It is then tested, which remedies make here normotonic: Rescue Remedy, Pulsatilla D 200, Zinc, Adrenal Extract. When evaluating homeopathic or flower remedies, the most appropriate remedies will be those which make a hypertonic muscle normotonic.

You can only find them, however, if you test for hypertonicity/normotonicity!!

e) A patient with "fibromyalgia" responds to oral zinc with freedom of pain in a variety of muscles which were previously painful. Does that mean he has to be given zinc? No!!

After the strong muscles are tested for hypertonicity, they are found to have actually become hypertonic! Immediately, with the zinc still on the tongue, orthomolecular copper is tested and found to be NC!! Consequence: the patient receives a prescription for Copper (PE), 1 capsule t.i.d. .

A follow-up test a weak later brings the opposite result: now Zinc 30 (PE) is NC, while Copper is now weakening and therefore stopped as a medication.

Especially in rheumatoid diseases this antagonism of Zinc and Copper is the critical key to successful treament ! (9)

Similar antagonistic effects have to be looked for with Calcium and Magnesium, Omega 3 and Omega 6 oils, B 3 and B 6 etc. !

Table 4 shows a list of the most common test substances that we use in our office.

Pay attention to all possible reactions and their frequency in our General Practice.

CONCLUSION

Especially patients with immune system related disorders - allergies, intolerances, chronic fatigue, stress syndromes, dybiosis incl. Candida and parasite infections, rheumatoid diseases, MS, malignomas etc. - should be evaluated for hypertonicity/normotonicity, whenever strong muscles are found. This may reveal findings, which are highly significant for the understanding and consequently successful treatment, but would otherwise - if only evaluating for weak and strong - be overlooked.

A suggestion for terminology to document the findings is made: SC, HC, NC, w, s.

Comments and criticism are welcome and necessary, because we need to "all speak the same English" if we want to expand AK as it deserves.

Wolfgang Gerz, M.D.,
Diplomate ICAK
Sonnenlängstr. 2, 81369
München, Germany
FAX 49 - 89 - 781928

SCREENING CHEMIE

	w —> n	n —> w	h —> w (SC)	h —> n (NC)	w —> h (HC)	n —> h (HC)
Histamin D12	XXX	s	s	XXX	s	s
Histidin	X	XXX	XXX	s	XXX	XXX
PCCK (Chinin)	s	XXX	XXX	s	s	XXX
Zink	XXX	s	s	XXX	s	s
Kupfer (Copper)	XXX	s	s	XXX	s	s
Thymus TL	XXX	XXX	XXX	XXX	s	s
NSAR mix (Aspirin)	X	X	s	s	s	s
Magnesium	XXX	s	s	XXX	s	s
Calcium	XXX	s	s	XXX	s	s
Candida albicans/ tropicalis	s	XXX	XXX	s	XXX	XXX
Nystatin	XXX	X	s	XXX	X	X
Vit. B3 (Niacitol)	XXX	s	s	X	s	s
Cholin	XXX	s	s	X	s	s
Vit. B6 (P-5-P)	XXX	s	s	XXX	s	s
$NaHCO_3$	XXX	s	s	XXX	s	s
Silber-amalgam D6	XXX	s	s	XXX	s	s

w = weak = schwach
n = normoton
h = hyperton

NC = Normotoner Challenge
SC = Superchallenge
HC = Hypertoner Challenge

s = selten = seldom
X = "normal"
XXX = häufig = very often

Table 4

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DYSLEXIC & LEARNING DISABLED CHILDREN
An Examination Approach Using Applied Kinesiology

Jeff Farkas D.C., DIBAK

This paper will attempt to assist the health care practitioner determine the therapeutic priorities in the total care of children suffering from dyslexia and other forms of learning disability. Generally speaking, affected children need to receive various supports corresponding to each side of the "Triad of Health" in order to demonstrate marked and lasting improvement. The challenge for the therapist is to systematically assess the case in order to arrive at the elusive underlying cause(s) and organize the potential therapies to best serve the individual patient.

PATIENT HISTORY

Has the child always displayed the types of traits that are now in question: slow learning, lack of concentration, irrational behaviors and thought processes? Has the child always been fidgety, unresponsive or unable to follow directions?

or

did it begin after one of the following?:

	consider
Physical trauma:	Craniaosacral therapies, Homeopathy
Birth or subsequent trauma, perhaps followed by a period of immobilization:	Craniosacral therapies; gait patterning
Illness:	Nosode therapy (for pathogenic agent and medications)
Vaccinations:	Nosodes and constitutional Homeopathy
Receipt of new glasses, orthodontic braces, orthotics:	evaluate patient with and without appliance. Note effect on muscle strength, gait patterns, cranial and sacral findings, etc.
Emotional trauma:	Homeopathy, Bach Flower Remedies, Craniosacral therapies

If the child has had the symptom constellation lifelong, without the presence of a known precipitation factor from the list above, gait pattern therapies, Homeopathy, as well as Craniosacral therapies may facilitate improvement; however, it then becomes imperative to train our focus on some other factors:

Allergies and Intolerance

Food allergies, with a classic elevated IgE or IgG response can often, when left undiagnosed and untreated, produce myriad symptoms including those which lead to school and learning problems. True allergies are best determined through blood screening and RASP testing.

More insidious are the food sensitivities which many children suffer from. These produce a constant stress on the immune and nervous systems that can certainly interfere with a child's ability to concentrate, learn and perform. The constant stress serves as well to deplete the body of specific nutrients, primarily elemental zinc and Vit B6, with deficiencies further impairing the child's function.

Be sure to ask the mother about her breast-feeding history. Intestinal brush border closure and therefore, normal intestinal permeability, does not occur in babies until the 6th - 8th month of life. Cow's milk or cow's milk formulas given before that time may penetrate the too permeable intestinal wall introducing foreign proteins systemically resulting in the development of food allergies and sensitivities.

To screen for the presence of hidden sensitivities, a strong normotonic indicator should be retested after the insalivation of either histidine or pancreatic cholecystokinin (PCCK). If muscle retesting reveals either weak or hypertonic muscles, this strongly suggests the presence of food sensitivities and the need for a thorough food sensitivity test.

When the patient has many or all muscles hypertonic, we can test for the presence of a high-priority "Superchallenge" using these muscles as indicators (a Superchallenge is a challenge that can provoke a weakness in an otherwise hypertonic muscle). *If hypertonic muscles become weak to histidine or PCCK, the probability that the food sensitivity is a powerful factor in the child's dis-ease pattern is increased.*

Instead of using the above factors which act as "lesion" irritants, we can muscle test the following potential "anti-sensitivity" factors:

Histaminum D12
Zinc (citrate or picolinate)
Pyridoxyl -5 -Phosphate (activated B6)
Copper (picolinate or glycinate)
Calcium (citrate)

When introduction of one or more of the above (primarily the first 3) causes a weak muscle to strengthen, negation of a TL related to the immune system or digestive tract, or the normalizing of a previously tested hypertonic muscle, a complete food test should be undertaken and supplementation of the well-tested remedy should commence.

Food testing is performed by individually testing the muscle response of a normotonic indicator muscle to the oral insalivation of foods which the child eats regularly. Small amounts of food are placed, on-at-a-time, on the child's tongue and an indicator muscle is tested after the child has noted, by slightly biting or chewing, the food's taste. Chewing should be halted during the muscle test, as this may produce a muscle change in response to malocclusion. Both weak and hypertonic muscle responses indicate that the food just tested is not tolerated and must be avoided for a minimum of one month before the possibility of retesting and, perhaps, conservative reintroduction into the diet if the food is no longer offending.

In the case of food sensitivity testing using AK, as is true in all AK muscle testing protocols, the use of a surrogate is suggested in the case of children either too young or too unconcentrated to execute proper muscle testing.

A large percentage of food sensitivities are the expression of a general dysbiosis. This condition, defined as an imbalance of the normal intestinal tract floral milieu, may be caused by the presence of bacterial antigens, yeast (such as Candida albicans) or parasites. As upwards of 80% of immune active cells are found in tissues relating to digestion, the systemic stress to a child caused by the presence of a yeast, parasitic or bacterial proliferation is significant and certain to affect that child's ability to function in school. Examination of the child must include a screening for these factors, to be performed as follows:

The child is tested for reactivity to yeast antigens. Weakening or hypertonicity upon exposure indicates a mycotic overload and the need for the appropriate anti-mycotic therapy as well as a reduced carbohydrate, sugar-free diet. The correct anti-mycotic is chosen based upon that medicaments ability to negate the weakness caused by the previous exposure or it's ability to normalize a hypertonic muscle. Typically, therapy will begin with amphotericin followed by nystatin or a plant-based anti-candida formula (i.e. AC Formula @ from Pure Encapsulations).

Long-term therapy should also include lactobacillus cultures, fructooligosaccharides, E. coli and other flora found in the normal eusymbiotic intestinal tract.

Children who test well to the plant-based AP Formula @ from Pure Encapsulations (containing berberine and gentian), Artemesia annua or to Furamid @ may be harboring a parasite which will require therapy. In any condition of dysbiosis, a confirming stool analysis can help guide the therapist to the best treatment regimen. We have found the Comprehensive Digestive Analysis & Parasitology performed by Great Smokies Labs in North Carolina to be the most thorough and cost-effective. Objective measures, such as stool analysis also help parents to comprehend the presence and extent of the problem, thus guaranteeing increased patient compliance, so vital to the child's improvement.

It is necessary to remember that allergies and intolerances may be to air-borne substances as well - often with the same negative consequences for children. These include pollens, molds and 2nd hand cigarette smoke, all of which can be tested by asking the patient to inhale the substance and testing muscle response. Weakening or hypertonicity or a previously normotonic indicator muscle suggests intolerance and the need for avoidance. Administration of corresponding nosodes, as well as anti-oxidant agents which test well, can facilitate therapy.

Negation of a positive therapy localization, strengthening of weak muscles &/or normalization of hypertonic muscles when exposed to nosodes of chemical agents such as formaldehyde, toluol or lindane suggest a history of damaging exposure to the tested substance. Reactivity to low potencies (i.e. 6x) is usually associated with acute and current exposure. A good response to a higher potency suggests a prior exposure. The nosode can also be administered along with any other Orthomolecular and Phytotherapeutics that test well.

The possibilities include:

Vits. A,C,E
n-acetyl cysteine
Selenium
Zinc
Fe
Bioflavanoids
milk thistle

Note that the above list is comprised of substances with known anti-oxidant properties. These will test well in cases of cell damage, whether the cause is toxicity from chemical, heavy metals or dysbiosis.

Be sure to use pure orthomolecular and phytotherapeutic substances. Patients, particularly those with allergies and sensitivities, cannot afford to have their difficulties compounded by the intake of substances with fillers and tableting agents which may themselves not be tolerated.

Dental Problems

The first dental problem to rule out it that of a toxicity caused by silver amalgam fillings. These contain a mixture of known poisonous heavy metals. The presence of even 1 or two such fillings needs to be considered as a source for immune and nervouse system irritation for a learning disabled child. Normalization of a weak or hypertonic muscle when exposed to a homeopathic Amalgam D6 preparation suggests a heavy metal toxicity.

This can be objectively confirmed or conclusively ruled out through laboratory testing. The use of heavy metal chelaters such as DMPS and DMSA, in conjunction with mineral replacement therapy, can rid the body of the offending heavy metal. It is, of course, vital that all such fillings be replaced with a well tolerated substitue material.

It is worth mentioning here that the presence of various metals need to be considered and objectively examined. Lead, cadmium and nickel are other examples of heavy metals shown to cause symptoms that include learning disabilities in children.

Combinations of detoxifiers, mineral replacements, Homeopathics and anti-oxidantes all need to be incorporated in the treatment regimen.

Critical to treating learning disabled children is an orthopedic evaluation of the Temporomandibular Joint (TMJ) and dental occlusion. Experts concur that many dyslexic children present with the same constellation of symptoms and findings: right brain lateralization, forward stoop posture, foot pronation and a classic "overjet" bite. An examination of the teeth and oral cavity is therefore essential. Teeth should be evenly spaced and the midline of upper and lower teeth should correspond sagittaly. No excessive overbite should be present. Crooked, missing or extra teeth, as well as an obvious malocclusion need to by evaluated by a holistically oriented orthodontist for necessary corrections &/or appliances. Lack of obvious visual findings is no guarantee of absence of a relevant malocclusion. Using AK, this can be easily evaluated as follows:

A normotonic indicator muscle should be found and tested again after the patient performs each of the following:

action:	weakening or hypertonicity suggests:
TMJ motion w/o TL	muscle of mastication dysbalance; also consider lymphatic congestion or aerobic/anaerobic pathways disturbance and premature contact.
TL to TMJ (w/o motion)	malocclusion / disc pathology
TL to TMJ with jaw motion	malocclusion with imbalance of corresponding mastication muscles (i.e., Temporalis, Masseter, int. and ext. Pterygoideus, Buccinator, Digastric)

Differential diagnosis must be performed in cases of TL with jaw motion or muscle status change via TMJ motion w/o TL. the specific motion eliciting or negating positive TL indicates the specific muscle involved as well as nature of problem (i.e. Trigger point, muscle spindle cell, etc.)

Weak or hypertonic muscles can also be used as indicators. Change in muscle status after any of the above actions should also be considered a positive finding for a TMJ problem requiring appropriate analysis and treatment.

Malocclusions need to be evaluated and treated by a professional trained to do so. Any prescribed removable appliances, braces, excercises, etc. can be further evaluated by an experienced AK practitioner for material tolerance and therapeutic value. In addition, any treatment needs to complimented by repeated evaluation of the muscles of masticulation, as well as the craniosacral system in order to assure that the patient is progressing and adapting without muscle imbalances and structural, particularly cranial, faults.

The Craniosacral System

The standing child should be evaluated for general postural problems. Scolioses, plumb line deviations or observed uneven heights of eyes, ears, shoulders, hips, knees and arches are all indicative of structural imbalances either caused by, or reflected in, stress of the craniosacral system. This stress, with the ensuing tension in the dural membrane, can provide the impetus for changes in the skull and spine, disturbing the brain and nervous system housed within them. The presence of cranial faults (absent or impaired motion of individual cranial bones or sutures) can be screened for with AK as follows:

A normotonic indicator muscle should be retested after the patient performs each of the following:

action:	weakness or hypertonicity suggests:
2-hand TL (anywhere on the body on both sides of the midline)	Total Compression Syndrome
inspiration	expiration assist fault
expiration	inspiration assist fault
half inspiration	parietal descent
half expiration	temporal bulge
forced inspiration or expiration	Sphenobasilar respiratory fault
oral or nasal respiration only	glabellar fault
unilateral nasal respiration	universal (occipital) fault

Weak muscles may also be used as indicators. A change in muscle status after any of the above suggests a functional pathology of the craniosacral system requiring appropriate therapy.

Therapy localization of cranial bones and sutures also suggest the need for craniosacral therepy, as does abundance or weak or hypertonic muscle unilaterally. Bear in mind that, according to Dr. George Goodheart, recidivous cranial faults of any kind indicate the need for Zinc.

The cranium and pelvis, with the spinal cord, Dura and vertebrae in between, function and operate together; therefore, treatments of the cranium should always be done in conjunction with an evaluation of the spine and pelvis. Commonly, a pelvic category according to Major DeJarnette D.C. (the founder of Sacro Occipital Technique) will be found. This can be screened for as follows:

One handed TL to either sacroiliac joint (causing altered muscle status) - Category II

Cat II is said to be primarily a weight-bearing lesion that is found in conjunction with adrenal fatigue, TMJ and sutural lesions.

Two handed TL to both joints simultaneously (causing altered muscle status) - Category I

Cat I should be considered an expression of excess dural tension, associated with cranial vault disrelationships, long-term organic aberrations and chronic postural stress.

Positive findings for either of the above indicate the need for appropriate Sacrooccipital/Applied Kinesiology therapy.

Other common findings include TL to the sacrum, coccyx or individual vertebra. All suggests that structural stress plays a role in the patient's status and most, if not all, will benefit from an appropriate manual therapy in the course of their holistic treatment protocol.

We can slo conclude that the cranial/sacral system is stressed and in need of treatment when the following muscle weaknesses are found:

muscle:	**associated finding:**
Sartorius	Cat II
Gracilis	"
Hamstring	"
Hamstring - bilateral	sacral inspiratory assist
Gluteus (medius &/or maximus)	Cat I
Piriformis	Cat I (may be associated with any pelvic problem)
Quadratus lumborum	Cat 1
SCM - bilateral	internal frontal fault
Subscapularis - bilateral	sternal fixation

Podiatric problems

A major source of structural and, therefore, translated dural stress may be found in the poor gait dynamics associated with podiatric problems and ill-fitting shoes. Be on the lookout for fallen arches, misshapen Achilles tendon, hammer toes, club foot, etc. They all may indicate the need for orthotics or some other form of orthopedic assistance. The shoes themselves may also provide a hint of mechanical difficulties requiring muscle and/or orthopedic support. Shoes should demonstrate even wear and no signs of being overly tight-fitting.

There is an elegant AK screen which can be used to see if the patient has some form of gait disturbance. It is performed as follows:

The child is brought into a normal walking position; one leg forward, with 70% of weight on the forward foot. The following muscles *should test weak* due to normal gait facilitation and inhibition patterns: Lattisimus dorsi and Sternocleidomastoideus on the side opposite the forward leg and Upper Trapezius on the forward leg side.

Failure to exhibit this normal pattern reflects a disturbance wich can be repeatedly monitored to gauge the effectiveness of Orthotics, structural therapies as well as treatments designed to effect right/left brain lateralization.

The child needs to be evaluated for normal development of contralateral neurologic organization; interaction of flexors, extensors, abductors, etc. Children who have been immobilized due to birth defects or trauma often do not have the opportunity to freely develop the neurologic relationships that are responses, both physical and emotional. The knowledgeable AK'er can establish if the child has developed through the homolateral, contralateral and combination gait phases by testing muscle reactions to specific positions and movements.

Neurologic Disorganization:

This geveralized AK term, also widely reported as "switching", is a description used for those patients experiencing and exhibiting a lack of afferent and efferent organization. This may be in the form of dis-relationships between left and right, upper and lower, front and back, analytic and emotional, etc. All children who are dyslexic exhibit some form of disorganization. Testing for the particular form often provides a clue in deciding which direction of diagnosis and treatment needs to be pursued.

Treating certain reflex points with massage or laser is often a mechanism for bringing the patient into a better organized state. Pursuant therapies can then be better integrated by thepatient, regardless of their form.

Therapy localizations to the following points are general indicators for "switching":

Bilateral K27 : normal or cross-handed placement
GV 20
Simultaneous umbilicus with unilateral K27 (left hand umbilicus, right hand to right K27 and then vice versa)
Simultaneous umbilicus and L3 (referred to in AK as posterior umbilicus)
Umbilicus and coccyx
CV24 and GV 27 simultaneously

Care should be taken to test all scars for therapy localization. If found to be positive, possible therapies include laser, Neural Therapy and various energetic creams (i.e. Jonen Salbe, APM). The umbilicus alone may test positive and should be considered as the "primary scar" and treated accordingly. The local field disturbances caused by scars can detrimentally effect the entire structural, emotional and chemical regulation mechanism.

The "switching" points listed above often therapy localize as "Superchallenges"; so high in the diagnostic prioritization so as to produce a weakness in a previously hypertonic muscle. When this is the case, we can treat the positively TL'ed point with vigorous massage, repeated tapping or laser energy. This often will return the body to a normotonic state making further examination easier. It has also been suggested that switching parameters need always be examined because, by definition, the "switched" patient is in a pattern of aberrant afferent-efferent organization; therefore, information we receive by way of muscle testing will always be more reliable if we can first bring the patient into an organized state.

It is important to remember that the neurologic disorganization problem is that of a symptom "on display". It is vital that the examiner investigate the cause of the neurologic disorganization. Testing to see if the positive "switching" TL can be negated by some chemical, emotional or structural factor can provide a clue as to the source of the disorganization (ex: TL to bilateral K27 negated by Histaminum D12 suggest food or airborne sensitivities as a source of disorganization!)

As mentioned above, we can treat the entry point into the problem (the positive TL) by way of laser, tapping, accupuncture needles etc. but the treatment approach must include a holistic evaluation aimed at discovering, as well as treating, the emotional, chemical or structural stressor ultimately responsible.

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DIVISION III - COMMENTS ON PUBLISHED PAPERS

DR. GEORGE J. GOODHEART
RESEARCH REPORT