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A MESSAGE FROM THE CHAIRMAN

Dr. Philip Maffetone
Chairman, ICAK-U.S.A.

Research is one of ICAK's priorities. The sharing of a member's clinical observations in this publication is the launching of a research idea, and a step up a path where too few in the health community have ever gone. While not yet ready for the submission process mandated by scientific and medical journals, many of our members have clinical insights to explore. *The Proceedings* is one publication which bridges that wide gap, and we are fortunate to maintain and encourage its existence.

These published works by our members are often a first step in a process which improves our understanding of clinical methods and outcomes. It can also be part of a process which results in enhancing the material through further evaluation or peer input to publish in other journals. The mechanism is set up for just such a process, and we invite you to be part of it.

INTRODUCTION

This thirty-fifth collection of papers from members of the International College of Applied Kinesiology-U.S.A. contains 31 papers by 20 authors. The papers will be presented by the authors to the general membership at the Summer Meeting of ICAK-U.S.A. in San Francisco, California, June 23-25, 1993. 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 Winter 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. The third section 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.

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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, all centered at the top of the paper with a 3/4 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.

Hypotheses - 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.

(Instructions Cont.)

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 single-spaced on plain paper. No papers typed on office letterhead will be accepted. 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 o the paper must be a minimum of 3/4 inch, and the top and bottom margins must be a minimum of 3/4 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.

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., P.O. Box 905, Lawrence, KS 66044-0905, (913) 542-1801.

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

X

* Adverse mechanical tension in the meningeal membranes
* Meridian system imbalance
* Lymphatic and vascular impairment

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

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

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

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

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

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

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

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

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

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

When properly performed, applied kinesiology can provide valuable insights into physiologic dysfunctions; however, many individuals have developed methods that use muscle testing (and related procedures) in a manner inconsistent with the approach advocated by the International College of Applied Kinesiology-U.S.A. Clearly the utilization of muscle testing and other 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 Counil for review.

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DIVISION II - CRITICAL REVIEW

DIVISION III - COMMENTS ON PUBLISHED PAPERS

DR. GEORGE J. GOODHEART - RESEARCH REPORT

** Material in this paper does not conform with the ICAK Status Statement

DIVISION I - INFORMATIVE PAPERS

AK INTEGRATION OF THOMPSON, ACTIVATOR AND COX TECHNIQUES.

Joseph Angleitner, D.C.

ABSTRACT

AK provides a number of innovative ways to measure activities and functions throughout the body. Their readout is a muscle test, which has been shown to be an effective diagnostic tool. When addressing the structural components of a vertebral subluxation complex, a number of established chiropractic technical applications may be enhanced when applied within this context.

INTRODUCTION

Theoretically speaking, a practitioner is limited by his technique and excused by his philosophy; i.e. he is confined not only by his belief system, but to it. Instead of solely pursuing the criterion of protocol, Applied Kinesiology adds, among others, that of utility, arranging things in a way that provides practical, methodical and imaginative verifications, freeing one from the constraints of tradition. Discussed below are three procedures which fit nicely into this framework.

THOMPSON TERMINAL POINT TECHNIQUE:(1)

Discussion:

For practical purposes it may be said that the Deerfield Leg Check with cervical rotation amounts to a static positional challenge, utilizing a muscle test instead of a change in leg length as an indicator. This has been useful at times when a hidden cervical subluxation or disc is in question in the presence of advanced degeneration, ligament stretch reaction, or other such contraindication to manual force vs. resistance type of adjustment.

Procedure

1. A good place to start is with a functional unilateral or bilateral middle deltoid weakness.

2. Ask the patient to assume various positions, usually extension, sometimes with a slight amount of rotation, which might negate the weakness, after Bandy.(2)

3. Next, with the patient prone, perform a cervical syndrome leg check according to Thompson Technique for the side of the lesion; i.e. the side of SCM flexion.

4. Challenge for a specific vertebral level on the positive side.

5. When the vertebral level has been determined try to

position the patient's head on the cervical section of the table that most closely approximates the position that negated the weakness in the sitting position, by adjusting the headpiece into posterior translation and extension. The patient's head must be held in the appropriate degree of rotation with the stabilizing hand during the adjustment.

6. One light thrust is enough.

7. Ask the patient to be seated again and retest the indicator muscle.

ACTIVATOR METHODS: (3)

Discussion

A similar method of therapy localization may be applied in the adjustment of hidden problems in the cervical spine combining Dr. Thompson's Technique and Dr. Fuhr's Activator Methods. The positional challenges as taught by Activator Methods Seminars may be verified not only by a change in leg length, but by muscle testing, when a suitable table or surface is not available. In cases of diffuse upper thoracic spine or neck pain when no specific therapy localization or muscle weakness may be found, or in cases mentioned above, this combination of provocative testing has been found effective.

Procedure

1. First ask the patient to lay his / her head on one side and, instead of looking for a change in leg length, test the hamstring group, thus modifying the Activator Methods mid-thoracic challenge and the Thompson Cervical Syndrome challenge. The side of SCM flexion is the side of lesion.

2. Next, use specific provocative testing for C7, C5, C1/2 and occiput, after Fuhr.

3. When the level is found, an adjustment may be accomplished utilizing the soft tip of the Activator instrument during three phases of respiration, the drop section of the table, or any other suitable method convenient to the patient and doctor.

COX TECHNIQUE: (4)

Discussion

Flexion / Distraction is not only an extraordinary technique for the correction of lumbar disc lesions, but elements may be incorporated into a screening procedure for a hidden lumbar disc.

Procedure

1. After completing an AK protocol, including cranial and pelvic category correction, adjustment of vertebral subluxations, etc. a convenient way to check for a subclinical lumbar disc is simply to ask the prone patient to place both legs toward one edge of the table while placing the pelvis as close as possible near the other.

2. Test a hamstring group.

3. If functional weakness is elicited in either leg toward either side, repeat the test on a Cox table, again laterally flexing the patient's legs toward right and left, testing both legs.

4. If, for example, there is functional hamstring weakness toward right lateral flexion of the right or both hamstring groups, there is a likelihood that there is a right lateral disc involvement. If the left group weakens only and the right remains strong during right lateral flexion, there is a probable left medial disc involvement.

5. The same steps apply toward left lateral flexion.

6. Next ask the patient to therapy localize for the level of the lesion, after Goodheart. (5)

7. Apply five to ten movements of flexion / distraction, during the appropriate phase of respiration, toward the opposite side that produced functional hamstring weakness, utilizing a suitable point of contact, depending on medial or lateral lesion. The most efficient point of contact for a medial disc lesion has been with the heel of the hand over the superior spinous process, pressing slightly cephalad with the pisiform; and on the superior transverse process on the side of the lateral lesion, pressing cephalad with the pollicis muscle.

8. Retest both hamstring groups during right and left lateral flexion positional challenges. This procedure may have to be repeated and / or modified on subsequent visits before all indicators are negative.

CONCLUSION

Given the unique ability to measure function, the AK practice lends itself well to investigation. What is retained is usually what works. The above discussion is not to imply that the referenced chiropractic techniques, when accurately applied and practiced as taught, should be compared in any way to what has been described. Bits and pieces of these techniques were expedient to this author and were applied successfully during clinical situations on a daily basis. The patient base was drawn from this author's general practice, divided somewhat equally into the above three procedures over the past year, with a

greater than 90% success rate in terms of subjective and objective response. The procedures were most useful during acute phases of injury and subclinical situations, following correction of neurologic disorganization, cranial / sacral manipulative techniques and attention to related factors of the IVF, according to this examiner's knowledge, ability and understanding. Two patients remained symptomatic, although one of them demonstrated marked subjective improvement. Both demonstrated the presence of stenosis secondary to disc herniation as evidenced by magnetic resonance imaging. One of the patients had prior fusion of C5/6.

References:

1. Thompson Terminal Point Technique Seminars, c/o Life Chiropractic College, Post Graduate Studies, Marietta, Georgia.

2. Dr. John Bandy, November 11 - 12, 1989, Cervical and Lumbar Disc Seminar, Atlanta, Georgia.

3. Activator Methods, Inc. 3714 E Indian School Rd. Phoenix, Az. 85018

4. Cox, James M., Low Back Pain, Mechanism, Diagnosis, and Treatment (Baltimore, Hong Kong, London, Sydney, Williams and Wilkins, 1990)

5. Walther, David S. Applied Kinesiology, Synopsis, (Pueblo, Co., Systems DC, 1988)

CATEGORY ONE MUSCULATURE PROCEDURE

By
Harold J. Briks, D.C.
October 1992

ABSTRACT: The following paper reviews the musculature specifically involved in the Category I pelvic fault procedure. It includes the name of the muscles involved, appropriate palpation sites, classification into suprailiac crest (crest sign) and gluteal regions (dollar sign), associated cranial faults, associated subluxation patterns, and nutritional support to the involved musculature.

INTRODUCTION

The Category One pelvic lesion is one that is very frequently seen in an AK practice. There are specific musculature patterns observed and examined in determination of the Category One pelvic fault and its correction. These musculature patterns determine the type of Category One pelvic lesion (1), the corrective pelvic adjustment and its sequence (2), the vertebral subluxation regions of the spine associated with the specific Category One musculature pattern, the specific cranial faults associated with each pattern (1), and nutritional support of each involved pattern.

The Category One pelvic fault musculature pattern may be divided into the supriailiac crest region and gluteal region. These have been referred to by De Jarnette (1) as the crest and dollar signs respectively. This paper will maintain De Jarnette's nomenclature in referring to these musculature patterns.

A. THE CREST SIGN

1. Muscle involvements
   Sacrospinalis (2) (3)
   Quadratus Lumborum (2) (3)
   External abdominal oblique (2)
   Internal abdominal oblique (anterior and lateral divisions) (2)

   These muscles can be tested in either the prone, supine, or weightbearing positions. Correction involved treatment of the neurolymphatics, neurovasculars, meridian circuits, and proprioceptive technique. (2)

2. Palpation

   Lightly palpate the muscles of the iliac crest using the fingertips stroking the musculature region of the 3rd, 4th, and 5th lumbar from center laterally. The major crest sign will be tighter and broader on one side than the other. (1)

   Another method of palpation is to place the Doctor's thumbs on the right and left side of the iliac crest 3 inches from the spinous of L4 and probe the adjacent musculature medially and

anteriorly. The muscle that elicits a painful response is the major crest sign. (1) Double repeated challenging of the crest while testing the strong indicator muscle on either right or left and with the weakening of the indicator muscle can determine the region of the pelvic corrective thrust.

3. Therapy localization

The patient places one hand above the iliac crest and the other hand at the ipsilateral T12-L1 paraspinal musculature while in the prone or weight bearing position. Therefore the patient is contacting the skin above the ipsilateraly involved sacrospinalis, quadratus lumborum, and abdominal obliques, (2)(3) A strong indicator muscle will weaken. Appropriate neurolymphatic drainage, neurovascular stretch reflexes, meridan circuit, proprioceptive procedure and blocking should negate the positive therapy localization.

4. Associated cranial faults

Temporal bulge on the side of the weak crest (the flaccid and weak muscle) is usually involved. At times an internal or external rotation of the temporal cranial fault can be found. The doctor challenges the parieto-temporal suture above the center of the temporal squama with the thumb of one hand and the mastoid process with the thumb of the other hand in the opposite direction with approximately 2 lbs. of pressure. A strong indicator muscle will weaken. Treat the temporal opposite the challenge causing muscle weakness in the phase of respiration abolishing the weakness 7 to 10 times or until an accentuated motion is felt in the temporal upon full inspiration.

5. Associated subluxation pattern

The following subluxation patterns should only be localized and treated if a Category one crest muscle pattern exists: (1)(6)

Subluxations of the cervical spine
Subluxations of the lower thoracic spine (T6-T12)
Subluxations of the ilia
Subluxations of the ribs and extremeities

6. Associated nutritional support

Vitamin ACP/M by Nutri-West Labs, Inc. or Cataplex ACP by Standard Process Labs, Inc. is highly recommended as the nutritional support for the crest sign. (4) With the patient prone, the nutrient should be chewed and tasted while testing one of the previously weak crest sign muscles. Similarly upon probing the crest sign an exquisitely sensitive area will be elicited. The appropriate sensitive chewing and tasting of the nutritional support will eliminate the probing pain of the crest sign.

Vitamin E and Calcium nutrition can also be evaluated for the aformentioned testing criteria. They are secondary to the testing of ACP/M and are based upon the nutritional support for each of the muscles involved in the crest sign.

B. THE DOLLAR SIGN

1. Muscle involvements

Piriformis (1)(2)(3)
Gluteus maximus (1)(2)
Gluteus medius (1)(2)
Gluteus minimus (1)(2)
Gemellus superior (1)
Gemellus inferior (1)
Obturator Internus (1)

De Jarnette mentions that the above seven muscles actually comprise the dollar sign. (1)

These muscles can be tested in either the prone, supine, side posture, or weightbearing positions. Correction involves treatment of the neurolymphatics, neurovasculars, meridian circuits, and proprioceptive techniques. (2)

2. Palpation

The dollar sign is approximately three inches inferior to and two inches lateral to the PSIS in most adults. (1) It can be probed medially and anteriorly with 5 lbs. of pressure utilizing the doctors thumbs thereby palpating the entire rim and center of the sign. The palpation will detect an area approximately the size of a silver dollar, hence the name "dollar sign." A painful side is elicited. That is considered the major dollar sign. (1)

Another method is to utilize the finger tap technique to the center of the sign. With the doctors index fingers comfortably poised the wrists are "flicked" medially and anteriorly into the center of the dollar sign. One side will have much greater tension to the flick than the other side. This again will indicate the major dollar sign. (1)

3. Therapy localization

The patient places the fingertips of both hands on the center of the suspected dollar sign region or the other side. A previously strong indicator muscle (like the hamstrings) will weaken if there is an involved dollar sign. Again, this region is three inches inferior and two inches lateral to the PSIS.

In difficulat cases to diagnose the new therapy localization technique as proposed by Goodheart (5) can be utilized. The

patient uses an interdigitated spread finger pattern and places the palmer surface in contact with the dollar sign region. Again, a previously strong indicator muscle will weaken upon manual muscle testing. Appropriate neurolymphatic drainage, neurovascular stretch reflex, meridian circuits, proprioceptive procedure, and blocking should negate the positive therapy localization.

4. Associated Cranial Faults

The occiput is the major cranial fault associated with the dollar sign. (1) (6) The sphenobasilar inspiration assist should first be tested. (1) If this is not found, check the following in descending order of occurrence:

Cranial inspiration assist
Sphenobasilar torsion
Interosseous cranial fault
Lambdoidal suture cranial fault
Sphenobasilar expiration assist
Cranial expiration assist (2)

Correct what is found in the appropriate direction on the phase of respiration that strengthens a previously weak muscle.

5. Associated Subluxation Patterns

The following subluxation pattern should only be localized and treated if a Category One Dollar sign muscle pattern exists: (1) (6)

Subluxations of the upper thoracic spine (T1-T5)
Subluxations of the entire lumbar spine
Subluxations of the sacrum

6. Associated Nutritional Support

Cataplex E by Standard Process Labs, Inc. is highly recommended as the nutritional support for the dollar sign; raw Gonadal support is also recommended. (4) With the patient prone, the nutrient-should be chewed and tasted while testing one of the previously weak dollar sign muscles. Similarly, upon probing the dollar sign, an exquisitely sensitive area will be elicited. The appropriate chewing and tasting of the nutritional support will eliminate the probing pain of the dollar sign.

DISCUSSION

The dollar and crest sign diagnosis and therapeutic procedure are a small part of a much broader therapeutic regimen termed the Category One Pelvic Fault procedure. (1) (2) (3) The correction of the Category One Pelvic-Fault is totally dependant on the correction of the crest and dollar sign musculature. Knowledge of the specfic muscle pattern involvement is indispensable in untorquing the Pelvis (2) (3) and consequently the dura mater.

The pelvic torque fixated the synovial joint (or boot part) of the sacroiliac in an anterior direction and the opposing side is fixated posteriorly. (1) This causes a disruption of the entire meningeal system particularly of the dura mater and subsequently affects the neurology and physiology of the entire nervous system. The dollar and crest sign muscle pattern procedure corrects the loss of reciprocity of the right and left synovial sacroiliac joints with the pelvic torque. The dollar sign probably represents the involved sacral part of the sacroiliac synovial joint and the crest sign probably represents the iliac part of the sacroiliac synovial joint. (1) The dollar sign is indicative of a neural disturbance and the crest sign is indicative of a myological disturbance. (1) These disturbances are direct effects of the Category One dural torquing. Therefore, accurate and precise De Jarnette block placement and procedure is a necessary corollary to normalization of dural torquing that give rise to the crest and dollar sign musculature pattern.

It is interesting to note that one can determine the type of cranial fault involvement based upon crest and dollar sign musculature patterns. The cranial involvement is of course the cephalad firm, attachment region of the meningeal system especially the dura mater, and must be corrected in order to normalize the dural torque pattern. Utilizing the dollars and crests signs as indicators for the cranial involvements facilitates locating the precise cranial fault correction and facilitates rapid improvement of the Category One problem.

This author has not found that all patients presenting with a Category One can be corrected in one office visit. (2) In many patients repetition of blocking procedure is necessary to normalize pelvic function and dural torque problems. During these repetative Category One visits, the muscular pattern may change from a dollar sign to a crest sign to no sign. This is commensurate with De Jarnette's findings. (1)

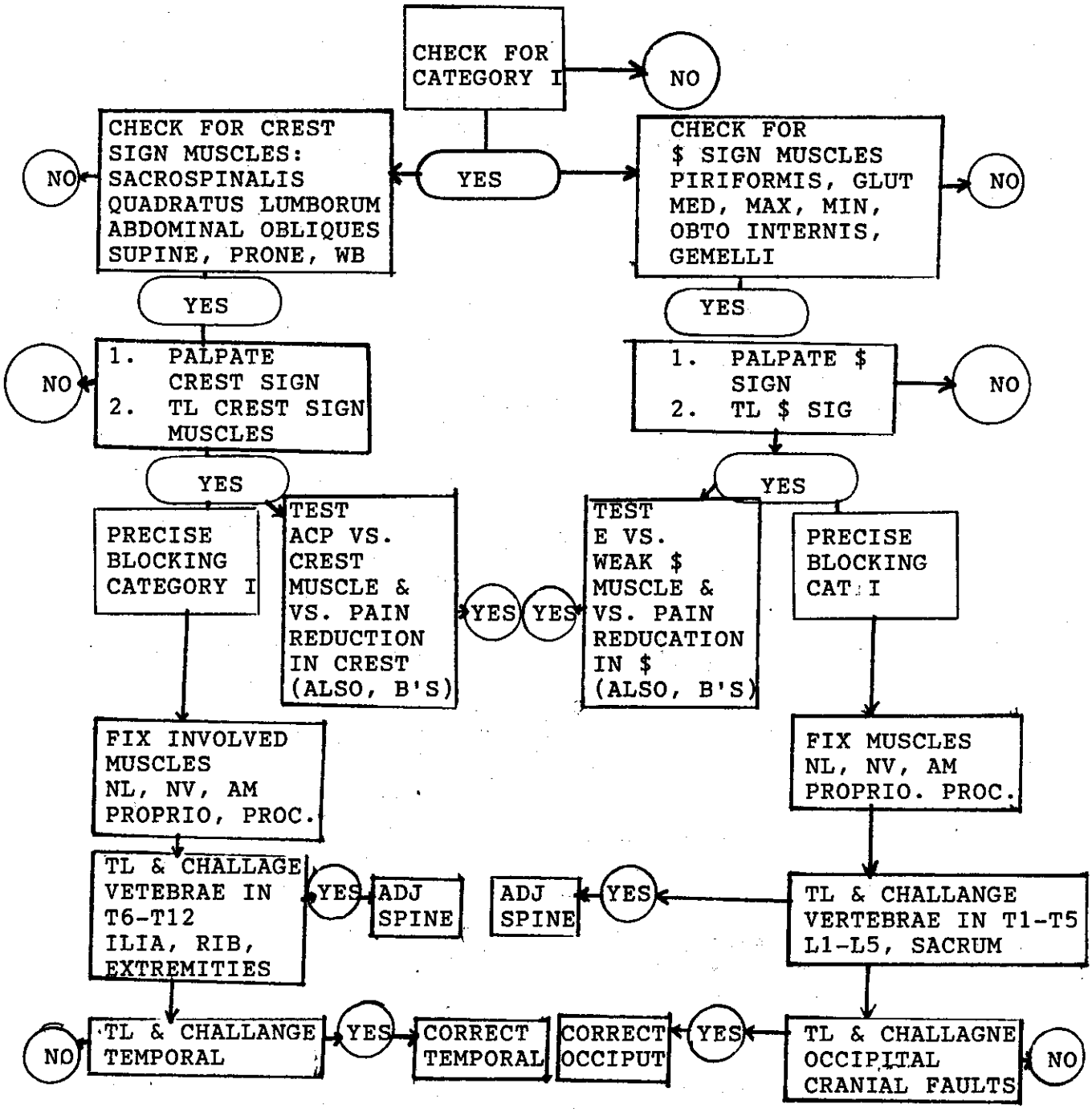
It is also important to note that even though the major nutritional supports for the dollar and crest signs are ACP and E respectively, the dural torque pattern associated with a Category One pelvic fault responds favorably to transformed form of B complex vitamins as recommended by Goodheart. (5) Each should be tested to aid Category One crest and dollar sign correction. Of course, the nutrient should be tested for its efficiency prior to structurally correcting the muscle pattern involvement.

CONCLUSIONS

The importance of diagnosing and treating the Category One musculature patterns is clear when we observe the central role these patterns have as both indicators and monitors of the entire status of the cerebrospinal fluid system. These musculature patterns, therefore, quintessentially represent the zero defect in the dura mater, deviations from that zero defect, and the body in health and dissease.

FLOW CHART FOR CREST AND DOLLAR SIGNS

Harold J. Briks, D.C.



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THE CATEGORY ONE VASOMOTOR SUBLUXATION

By
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October 1992

ABSTRACT: This paper reviews the diagnosis and treatment of the Category One Vasomotor subluxation, its importance in the treatment of the Category One Pelvic fault, suspected regions of the spine it may be found in, the technique suited to adjusting the spine, associated nutrition, and conditions that the Category One Vasomotor subluxation treatment can aid in correcting.

INTRODUCTION

The Category One pelvic fault procedure as proposed by De Jarnette (1) involves a very precise adjustment to the spine after termination of normal De Jarnette blocking procedures. The area of the spine that this specific technique is administered to is referred to as the vasomotor (VM) subluxation and subsequently the manipulative thrust to the VM subluxation is referred to as the vasomotor(VM) adjustment. This paper will discuss the procedures prior to locating and revealing the VM subluxation, visual observation of localization, palpation methods, therapy localization and challenge procedures, spinal adjusting procedures, and associated nutritional support for the VM adjustment. The discussion will review the essential importance of locating and adjusting the VM subluxation to complete the Category One Pelvic Fault treatment.

A. S.B.+ or S.B.-

After the Category one blocking procedure and specific repeated pelvic thrusts (1)(2)(3), the S.B.+ (sacral base positive) or S.B.- (sacral base negative) must be determined. (1) This is comparable to a sacral inspiration or sacral expiration assist. (2) The method of treatment however adds a few more pointers in refining the technique's procedure:

1. For non-Kyphotic dorsal spines, a sternal roll is placed underneath the patient's chest centered lenghtwise starting beneath the sternal notch.

2. After determination of S.B.+ or S.B.-, (1) sacral inspiratory or sacral expiratory assist respectively (2) utilizing the customary testing procedures, the De Jarnette blocks should be shifted from their Category One short leg position to both blocks resting underneath the ASIS's and pointing to the center of the symphysis pubis for a sacral inspiratory assist. The position of the blocks for the S.B.- or sacral expiratory technique is the same as Goodheart's for the sacral expiratory assist sacral angle technique.(3)

3. With the patient's arms encompassing the headpiece, the patient tractions his spine cephalad pulling on the headpiece with his hands while the doctor presses on the sacrum with the appropriate phase of respiration. For S.B.+ the patient tractions on inspira-

tion while the doctor presses the sacral apex, and both release on expiration. For S.B.- the patient tractions on expiration while the doctor presses the sacral base, and both release on inspiration. Five - 10 respiratory cycles are administered repeatedly in this fashion. (1)

B. LOCATING THE VASOMOTOR SUBLUXATION

1. Visual Observation

The aforementioned traction procedure causes and reveals the whitening (blanching) of the skin overlying one or more vertebral segments, T1-L5, expecially in the thoracic region. An entire segmental area will exhibit this whitening effect. (In black, dark, or yellow patients the skin reaction will blush at the site of the vasomotor subluxation.)

2. Palpation

Either on or within the blanched area a set of 3 vertebrae will be involved in the vasomotor subluxation. Utilizing the dorsal pad of the thumb the vertebral spinous process tip is moved in a cephalad direction. The probable vasomotor subluxation's spinous tip will not be easy to locate and will be approximated to the spinous tip of its immediately inferior vertebra and apposed against it. Exquisite sensitivity is usually how the patient will describe the palpation along with a stinging and burning sensation. (Let the patient know it will be sensitive, beforehand.)

3. Therapy Localization

The patient contacts the suspected VM subluxation at the spinous tip while the doctor tests a previously strong indicator muscle which weakens. The patient breaks the contact and then pulls the spinous he/she was touching caudally or cephalad. The previously strong indicator muscle will again weaken. Determine the phase of respiration that abolishes the weakness. This author has found that the indicator muscles weakens when the patient pulls the spinous tip in a caudal direction most of the time. The exception to the rule seems to be in patients exhibiting a dorsal kyphosis in which case there will be a positive therapy localization when the patient pulls the spinous tip cephalad.

4. Challenge

The doctor challenges the suspected VM subluxation in much the same way that palpation is accomplished. Cephalad pressure (8 lbs.) is exerted on the spinous tip by the doctor and a previously strong muscle will usually weaken (except for the kyphotic cases mentioned above.) Again, find the phase of respiration that abolishes the weakness.

C. The Vasomotor Pump

Prior to the VM adjustment it is recommended that the spinous process be pumped 5-10 times cephalad on the phase of respiration that abolishes the weakness. While the spinous process of the VM subluxation is being pumped with inspiration the doctor's thumb and third finger of the free hand grasps the skin overlying the lamina of the cervical vertebra associated with the VM subluxation. Moisture will be felt on the bilaterally grasped cervical segment while the VM subluxation is pumped. The VM subluxation is ready to be adjusted.

The following are the recommended grasped cervical vertebrae associated with the sequential VM subluxation:

C1	with	D1-D2	and	D10
C2	with	D3	and	D11-D12
C3	with	D4-D5	and	L1
C4	with	D6	and	L2
C5	with	D7	and	L3
C6	with	D8	and	L4
C7	with	D9	and	L5

D. The VM Adjustment

Adjust opposite to the therapy localization direction pulled by the patient or in the same direction challenged by the doctor using the phase of respiration that abolishes the weakness. This will usually be cephalad on completion of full expiration. Rarely, is it caudad (except for the kyphotic patient), and rarely does one adjust on the spinous process on full inspiration.

The doctor's fifth lateral metacarpo-phalangial joint is used as a lever to treat the VM subluxation. (1) Clinical experience has indicated that to be successful in releasing the VM subluxation, the doctor places his left hand flat on the spine, removes the slack of the skin 2-3 segments below the VM subluxation, and approaches the spinous process from the inferior direction superiorly. Once the skin slack is removed, the doctor maneuvers the left lateral metacarphalagial joint into the spinous process. His left wrist is reinforced with the free hand for bracing. The patient completes three full cycles of respiration and the doctor thrusts cephalad (not anteriorly). Frequently and osseous audible crepitus release can be heard.

E. Associated Nutritional Support for the V.M. adjustment

Ligaplex I by Standard Process Labs, chewed and tasted strengthens a weak indicator muscle after therapy localization to the spinous process of the VM subluxation. It also lessens or removes the exquisite sensitivity of the VM subluxation's spinous process upon palpation after being tasted and chewed.

DISCUSSION

The vasomotor subluxation and correction is an integral part of the entire Category One Pelvic fault correction. The site of this subluxation is related to a local dural torquing by the dentate ligaments within the spine. (1) This therefore, also discrupts the integrity of the spinal fluid system and may set the groundwork for recurring Category One pelvic faults. (1) There is also weakening of the interspinalis muscles at the site of the V.M. subluxation, and the V.M. adjustment frees minor vasomotor constrictions in the spinal muscles that reciprocate via their circulation with the substance of the spinal cord segments. (1) This vasomotor constriction is probably the cause of the blanching observed in the skin overlying the V.M. subluxation. It is interesting to note that the author has always clincially observed a hyperemic ruddish spot after the manipulative thrust to the V.M. subluxation indicating a freeing of the vasomotor constriction at the spot.

The exquisite sensitivity at the spinous tip of the V.M. subluxation is due to the attachment of both the interspinalis muscle and compressed interspinous ligament. The apposition and approximation of teh spinous process in the vasomotor subluxation has been termed by De Jarnette (1) as the "kissing" spinous processes. This may be a cause of both Baystrup's spinouses as well as severe anteriority subluxations.

The V.M. subluxation spinal pump is administered to begin the untorquing process of the dura and return the crebrospinal fluid pressure to normal at the site. (1) The patient may complain of severely exquisite sensitivity at the pumping site and should be forewarned of the possible tenderness. The patient should be reassured that after the V.M. adjustment the sensitivity will diminish by at least 90%. (This has been observed clinically by the author). The sternal roll is placed under the patient's chest to free the costoververtebral articulations by spreading the ribs and maintaining a proper thoracic curve.

Although De Jarnette indicates that any area between T1 through L5 could be the V.M. subluxation, (1) the author has found that of the last 170 patients that have exhibited V.M. subluxations, 34% were at T5, 18% were at T6, 12% were at T7, 12% were at T9, and 6% were at T8. Very rarely has the author found an V.M. subluxation above T4 or below T9.

The V.M. subluxation can also be found on line one occipital fiber analysis and trapezius fiber analysis as found in the S.O.T. procedures. (1) They are commensurate with the blanching reaction, therapy localization, and challenge to determine the V.M. subluxation.

The typical patient picture that requires the V.M. subluxation correction presents the following symptoms and signs all of which are corrected very successfully by the above procedures:

A. Pain in the thoracic spine that can be relieved only momentarily by squaring the shoulders backward in a "military attention."

B. Stomach disturbances and disorders.

C. Well endowed buxom women.

D. On viewing the spine from posterior to anterior on postural analysis, the thoracic spine has a region of "sinking forward" into the body giving rise to the commonly seen "Pottenger's saucer."

E. The beginning of a "Dowinger's hump" in the cervico-thoracic spine.

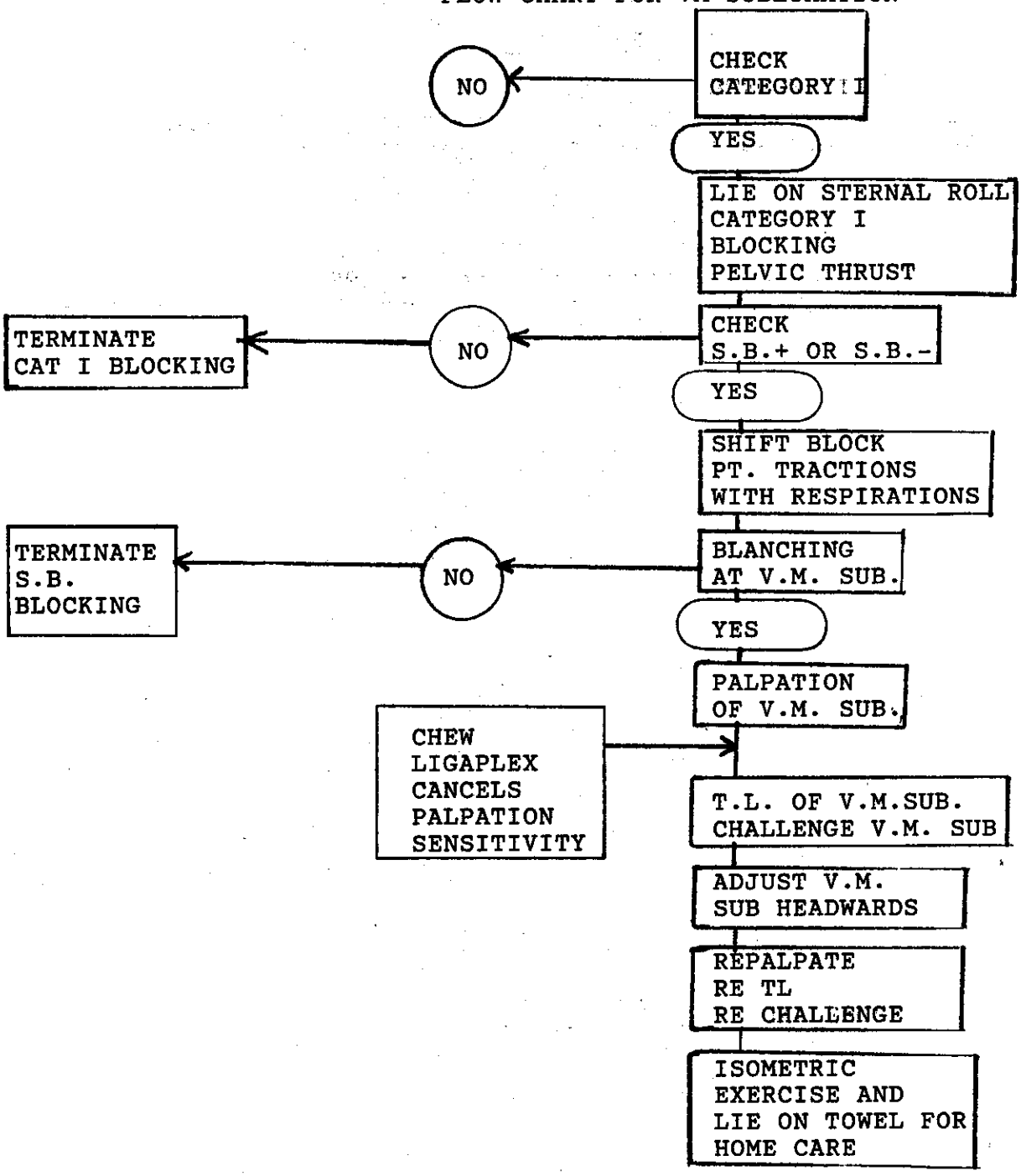
Remarkable changes occur rapidly after a few adjustments in the areas of the spine not only in the reversal of symptoms and signs but also in the structural presentation of the patient. They stand taller, they move better, their digestion improves, bras no longer hurt, and the "humplike" appearance of the cervicothoracic spine disappears.

Our center recommends that the patient lie on the floor with a sternal roll or rolled up bathtowel lengthwise for 5 minutes a day in order to support the VM correction and support the proper curve in the thoracic spine. We've also recommended that the patient do an isometric exercise of clasping the hands and pushing into the center of the chest as the shoulders are rolled forward.

CONCLUSIONS

The detection and correction of the vasomotor subluxation as described in this paper forms part and parcel of the Category One Pelvic fault correction procedure. Its inclusion in the AK repertoire and armementarium will help many patients very quickly and will prevent reoccurance and recidivism of the Category One. The structural changes the patient will feel and see will also be quick and remarkable. I highly recommend it to all Applied Kinesiologists as an important procedure for correction of the dural torque pattern and cerebrospinal fluid system.

FLOW CHART FOR VM SUBLUXATION



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APPLIED KINESIOLOGY MANAGEMENT OF DYSPHONIA

Cecilia A. Duffy, D.C.

ABSTRACT: This paper presents a case history of a 55 year old female with dysphonia. She lost her ability to sing soprano and was unable to increase her decibel volume (yell, talk or sing loudly). These symptoms were eliminated via applied kinesiology diagnosis and treatment which required correction of structural, chemical, and mental aspects. Following symptom abatement, the patient was recommended to a dentist for insertion of a partial plate to replace her missing right upper rear molars.

INTRODUCTION: Dorland's Illustrated Medical Dictionary defines dysphonia as "any impairment in voice; a difficulty in speaking" <1>. Dysphonia and aphonia ("loss of voice" <1>) occurs as a result of vertebrobasilar ischemia <2>, disease of the larynx, paralysis of the vocal cords, or hysteria <3>. This patient did not exhibit signs or symptoms consistent with these diagnoses.

DISCUSSION: A 55 year-old female complained of losing her ability to sing soprano, yell, and talk loudly. She stated that she first noted a sudden loss of the soprano range during choir practice, immediately following an unusually stressful day at work. Later, she also realized that she was unable to yell or raise her voice. She retained the ability to speak in normal tones and to sing baritone. Since the patient initially consulted me by phone, I referred her for examination by an otorhinolaryngologist to rule out laryngeal pathology which proved to be unremarkable. Speech therapy was recommended by the specialist, however, the patient chose chiropractic care. The patient's condition was strongly influenced by emotional factors due to her extensive involvement and leadership in community and church singing activities.

Pertinent physical examination revealed orthostatic hypotension (Ragland's sign) with seated blood pressure 148/80, standing blood pressure 140/80; pulse seated 76, standing 76; urinary chlorides (sodium) measured via Koensberg test elevated at 30 plus; pupil dilation to light; the right three molars on the superior dental arch were absent; and the uvula deviated left on elevation of the soft palate. Orthopedic examination of the cervical spine was unremarkable except for subjective complaint of tightness at the end of cervical range of motion.

The first four treatments revealed the following findings which were diagnosed and corrected via applied kinesiology: Positive challenge on jaw clenching without therapy localization of the temporomandibular joint (TMJ) which was negated by placement of a tongue depressor between the teeth. Cranial faults included: universal interosseous, right internal frontal, and left sphenobasilar expiration assist. Jugular decompression was performed to correct the uvular deviation. Hyoid muscle imbalances required neuromuscular spindle cell sedation of the left anterior digastric and bilateral geniohyoid and mylohyoid. The unopposed right lower molar teeth showed evidence of neurological tooth displacement necessitating buccal to lingual, rotary, and lingual to buccal adjustment upon inspiration (anterior to posterior respectively). Grade 4/5 weaknesses were found in the right upper trapezius, left sternocleidomastoid, and right sartorius muscles requiring various corrections via the five factors. Vertebral subluxations included a right lateral occiput, anterior C5, and left posterior T4. Vertebral fixations included upper three cervicals (C1-C2-C3) and mid-thoracic (T5-T6-T7). The pelvis was found in a Category I torque lesion along with a requirement for left limbic technique (fixation of the left first rib with the transverse process of C7). The right and left temporalis neuromuscular spindle cell required sedation <4> <5>. Due to the positive findings indicating functional hypoadrenia, the patient was placed on an adrenal nucleoprotein extract (Drenamin, Standard Process Laboratories) <6>. She was advised to remove processed foods from her diet. Cervical excercises were recommended <7>.

At the time of the fourth treatment there was no change in her ability to sing or raise her voice, however, she reported that her neck tightness was now intermittent and much less intense. During the two week interval between her fourth and fifth visit, she noted a gradual but complete recovery in her ability to sing soprano and raise her voice. Initially, the return of the singing voice was intermittent, and adversely affected by mental (as opposed to physical) stress. This occurred four weeks after her initial adjustment and prior to dental referral.

She was referred for dental consultation due to the absence of the right upper molars and a partial plate was fitted seven weeks following her first applied kinesiology treatment. The day after placement of the partial she received her sixth treatment. She reported normal singing with only episodic difficulty that was quickly rectified with relaxation and cervical excercises. There were no cranial or TMJ findings at that time.

Two weeks later, on her eighth visit, she reported no difficulties with singing or raising her voice. Follow-up was performed when she returned for an unrelated back injury 11 weeks after discharge for the dysphonia. She reported no exacerbations of the dysphonia at that time.

CONCLUSION: This case history presents successful, cost effective management of dysphonia utilizing applied kinesiology diagnosis and treatment. Pathology of the larynx was ruled out by ENT examination. There was no clinical manifestations of vertebrobasilar ischemia or hysteria. Specific structural corrections were examined and treated in the spine and pelvis, with special attention to the cranium, TMJ, and hyoid. There was diagnosable dysfunction in the TMJ via manual muscle testing without the use of therapy localization due to the absent molars, therefore, dental referral for placement of a partial was necessary to prevent recidivism. Absence of dentition is frequently associated with TMJ instability <5>. Lasting correction in such cases without a dental appliance is improbable. Cervical exercises were used predominantly as a conscious relaxation for the patient when she felt emotionally stressed. Reassurance and detailed explanation of procedures was necessary in this case due to the strong emotional component. Chemical corrections included nutritional support for the adrenal glands and dietary improvement.

This case of dysphonia appeared to involve structural, chemical, and emotional factors which combined to cause a functional rather than pathological problem that was easily diagnosed and fully responsive to applied kinesiology.

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C H I R O P R A C T I C C O S T E F F E C T I V E N E S S

I N

C A R P A L T U N N E L S Y N D R O M E

By
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January 1, 1992

ABSTRACT: This paper discusses the neglected factors contributing to the high frequency and recidivism of CTS surgery and suggests that the most cost effective approach to diagnosis and correction of the subluxation complex factors (SCF) (2) involved in CTS is Manual Muscle Testing (MMT) in accordance with published Applied Kinesiology (AK) protocol. AK addresses CTS in the wholistic manner necessary to insure correction of all involved factors including visceral and structural reflexes as discussed herein which, left uncorrected, contribute to the low success rate and poor cost effectiveness in CTS surgery. The recent discovery of the effects of rib cage movement hypothesized to (in effect) serve as an auxiliary pump of cerebrospinal fluid has greatly increased effectiveness in hand and wrist symptoms as demonstrated herein via case history. This paper suggests that INFORMED CONSENT of the CTS patient to surgery should include reference to the availablity of the cost-effective, conservative techniques described herein. Long term studies are necessary to establish differences in effectiveness between medical and chiropractic techniques in CTS>

Key Words: Carpal Tunnel Syndrome, chiropractic, Applied Kinesiology.

I N T R O D U C T I O N

Carpal Tunnel Syndrome (CTS) surgery ranks second highest in frequency and highest in repeat surgery in the USA, second only to the D&C which ranks number one. (1) The actual cause/effect relationships in carpal tunnel syndrome (CTS) and other upper limb complaints, such as: DeQuervain's syndrome (DQS), Pisiform/ Hamate Syndrome (PHS), and simple forearm, wrist and hand pain, are "incommensurable" with the current medical paradigm. This writer opines that such physiological dysfunctions cannot occur in the absence of "subluxations" (defined as interference with nerve energy flow). Subluxations can be corrected by simple, conservative, manipulative techniques. A high percentage of CTS symptoms may be related to visceral dysfunction. (2) This paper describes the SCF in CTS that, if corrected, may significantly decrease the number of patients presently requiring high cost, potentially dangerous surgery.

CHRONICITY is the key to the primary (local) or secondary (reflex) nature of CTS. Why should one joint injury resolve with eventual disappearance of pain and a similar injury result in chronic pain? The biomechanics of a primary CTS or simple problem caused by trauma (overuse, abuse or accident) localized to the wrist joint is well described in the medical literature which offers much information on how to "treat" the problem but lacks the clinically useful information on how to "fix" it. Therefore, a large percentage of the patients

receiving medical treatment only, may suffer chronicity from simple failure to: adjust the wrist joint, balance the musculature, and supply the nutritional assistance to assist the healing process. Many of the resulting surgeries may be preventable. Likewise, patients treated by traditional spine-only chiropractors who fail to consider the relationships of the joints to the viscera by way of the articular neurology that ties various well established reflexes (AK) to organ/muscle relationships via the interneuronal pool may not benefit as greatly as those treated in accordance with AK protocol. (4) This may also be true for those who receive spinal adjustments without receiving nutritional support to aid healing and help maintain the adjustment. (7)

Mechanically induced chronicity is due to reinjury from overuse and/or abuse which involves an admittedly subtle distinction. Overuse is seen in: carpentry, plumbing, electrical work and waitressing - abuse in: weight lifting, pushups, washing floors on hands and knees, using the heel of the hand as a hammer etc. most of which require long term hyperextension of a wrist joint under pressure. Other vectors in chronicity, especially the failure of acute episodes to quickly respond to average measures, strongly suggests inclusion of the nutritional and reflex factors.

AK quickly identifies the source and nature of the involvement e.g., weakness observed on MMT of thumb muscles indicating median nerve involvement may respond to Therapy Localization to the Cervical Spine. If nerve supply to muscles affecting the wrist joint are compromised at the Cervical spine little benefit can be obtained from performing surgery at the wrist joint! Adjusting and taping the wrist joint and applying physical therapy without correcting the cervical spine would be just as fruitless. As experience in the treatment of CTS accumulates, one finds that occasionally other lesser known techniques of AK are necessary to eliminate symptoms by correcting causes. Correction of some of these more unusual relationships are often necessary to achieve lasting correction in the CTS. Common examples of often overlooked causes of symptom recidivism are; ileocecal valve reflex pain, requirement for Ligament Interlink Technique, the Radial/Ulnar bypass adjustment and hypothyroidism. (4)

Of the last fifteen patients examined by this writer complaining of forearm wrist and hand pain, twelve reported no trauma or overuse. All twelve showed evidence of ileocecal valve involvement and functional hypoadrenia. All twelve suffered pain on manual muscle testing (MMT) of the wrist extensors which was immediately decreased by performing the MMT during Therapy Localization (TL) (3) of the Ileocecal Valve (ICV). All twelve showed startling increase in this MMT strength and seven of them experienced a reduction or total absence of pain during MMT following simple manual stimulation of the ICV Neurolymphatic reflex points.

C A S E #1

A 49 year old male Chiropractor was seen 6-29-91 suffering muscle weakness, pain and swelling of the right wrist and hand with approximately 80% loss of ROM in the ability to approximate the thumb

and little finger. Previous history revealed minor surgery earlier that year (2/91) for trigger thumb on the affected side. Medical (neurological) diagnosis based on EMG studies etc. in early June, resulted in diagnosis of CTS and surgery was recommended.

Physical trauma was not involved. The patient first noticed the symptoms (in early June) upon awakening following a normal nights sleep. The patient also complained of poor sleep habits and a feeling of being "wired" (restless). Due to the wrist and hand pain and weakness, the patient was unable to perform his duties as a Chiropractor.

Postural examination of the patient (thin, wiry, alert male) showed severe head and shoulder tilt, arm and hand position of Teres Minor weakness on the right (Thyroid indicator), knee rotation and medial longitudinal arch (foot) inferiority suggesting Sartorius weakness (Adrenal indicator) and inferior navicular bone. Posteriority of the shoulder on one side and buttock on the opposite side suggested the presence of pelvic torque. The first MMT examination showed evidence of pain and weakness on MMT of the wrist extensors momentarily negated by/during therapy localization to the ileocecal valve. MMT of the wrist extensors lend themselves to quick assessment of the reflexes involved and give immediate proof of the necessity for their correction.

AK corrections included: ICV dysfunction; muscle weaknesses associated with the hypoadrenia and thyroid dysfunction (via "Then and Now" meridian technique); pelvic balancing (category one); a typical chiropractic adjustment was delivered to the following: dorsal spine (T7/T4); 5th Cervical left anterior; posteriorities of the Capitate, Pisiform/Hamate; posteriority of the proximal end of the radius. Mobilization and adjustment of the wrist and elbow was initially accompanied by much crepitus. This usually decreases in followup treatment as tissues normalize and begin to retain their normal anatomical positions. Following these corrections the patient showed normal MMT on the wrist extensors on the involved (right) side without pain. The patient was then able to oppose the thumb and little finger and to resist firm MMT pressure to separate.

Case #1 did fine for several days, reporting more restful sleep and a reduction in restlessness and feeling of being "wired". He then suffered a relapse of symptoms. When seen the second time (12 days later), the patient complained of numbness and weakness rather than pain (produced edematously) and was unable to initiate movement in the direction of opposition of the thumb and little finger. At that examination the patient showed evidence of thyroid dysfunction (via AK diagnosis) which required attention to thyroid function. "Pituitary Drive" technique and treatment to the (thyroid related) Teres Minor muscles (fascial flushing, NL and NV) immediately eliminated all of the symptoms and findings on MMT (the numbness in the hand and weakness of the thumb and little finger) and elevated (axillary) body temperature to normal, indicating improved thyroid function in response to the specific treatment. The patient then demonstrated full function of thumb and little finger with no numbness.

It is significant that the inability to oppose the thumb and little finger improved from zero to full function, including full return of sensation in the hand, immediately after AK diagnosed Chiropractic corrections to improve thyroid function. No other treatment was necessary on this visit.

Two days later the patient telephoned (The patient lived six hours away by auto) in the evening to report severe swelling, pain and weakness and stated that his hand was hot and red. His surgeon (trigger thumb) advised elevation and ice packs. I advised him to get to an emergency room immediately, suggesting that it sounded like a fulminating infection that probably needed to be drained. The emergency room doctor also did not see the need for other than antiinflammatories etc. The patient insisted on further attention so the base of the thumb was opened surgically with the release of a large amount of pus. The infection was then treated medically with IV antibiotics.

Increasing thyroid function encourges an underlying infection to blossom forth. I have seen this quite often, especially in patients with "quiet" dental abscesses which never quite blossom until the thyroid function is normalized. It is unfortunate that such infections are treated by antibiotics when intravenous vitamin C has been proved (in hospital) to be effective in curing infections such as Diptheria, Hemolytic Streptococcus, Staphylococcus and many other "infectious" conditions such as Pneumonia, within hours. (3)

Differential MMT of the thumb, little finger, flexors of the wrist and hand and other muscles can be accomplished in the difficult case requiring treatment to selected reflex points. Inexperienced muscle testors find this difficult to demonstrate due to errors inpositioning, line of drive and the natural tendency for the doctor to overpower the patient during diagnostic muscle testing. A high degree of skill is required for assessing the minimal differences in muscle strength when pain is present upon testing therefore inexperienced doctors are advised to treat suspected reflex areas of involvement when differentiation is too difficult to assess. Nerve entrapment at the Carpal Tunnel will cause weakness of the thumb muscles while entrapment or stretch at the Hamate will cause weakness at the little finger. Most carpal bones are found posteriorly displaced and affect thumb and/or little finger muscle strength. Difficulty in differentiation is also confounded by the crossover of Ulnar nerve branches to the thumb side. Careful palpation of the nerves near the elbow joint is useful in determining exact areas of dysfunction especially in long standing problems with adaptive mechanisms and multiple entrapments.

Difficult patients are checked in "PLUS" technique positions to insure elimination of all dural torque. (5) Following normalization of "PLUS" findings the patient is then asked to walk to determine if factors interfering with normal "PLUS" findings are recidivistic. If for example walking causes a return of a positive "PLUS" finding the patient is asked to taste E-POISE (Standard Process Labs) which usually negates the positive "PLUS". The patient is then placed on three capsules of E-POISE per day (one per meal) until rechecked at

the next office visit. Depending upon the intensity of the pain and response to treatment, the patient is seen from one day to one month later. Four to five days are required for the ICV reflex pain to subside.

A good clinical indicator of ICV involvement in CTS is the patient suffering pain of high intensity unrelieved by pain medication. This is always a good clinical indicator of ICV involvement regardless of pain location or characteristics other than intensity. ICV induced pain is disabling - the patient is unable to perform usual tasks. In many bonafide injuries due to trauma, an overriding ICV involvement often interferes with the usual response to analgesics, reducing or completely eliminating their effects. Since sudden fright or shock causes flaccidity of the ICV, emotional dysfunction (6) (frontal bone reflexes) should always be ruled out in trauma cases or when pain fails to respond to analgesics.

When seen at the first office visit, the patient's pain and swelling and loss of function was due mainly to the reflex effects from the ICV. The exact mechanisms are unknown however clinical responses are predictable. Surprisingly, patients with forearm, wrist and hand pain and measurable dysfunction (via MMT) respond immediately when the ICV is involved and corrected.

Many doctors are unaware that thyroid dysfunction produces a myxedematous condition at the carpal tunnel, fewer are aware of the corrective action necessary to correct the condition. The obese patient with bilateral CTS who is awakened at night with numbness and tingling with no history of trauma is usually suffering from a myxedematous swelling and a vitamin B6 deficiency - if there is pain also - the chance of an overriding ICV dysfunction is increased. The relationships involved are totally incommensurable with current medical thinking.

The percentage of involvement of ICV dysfunction in the SCF, producing diverse symptoms, is very high and while correction of the ICV dysfunction is not a panacea or universal involvment, it certainly should be ruled out as a contributing factor in every patient visit, regardless of symptoms.

The routine adjustment used by this writer in adjusting the wrist joint is to adjust the capitate from P to A by placing the treating doctor's thumbs in the middle of the posterior aspect of the wrist joint and exert firm pressure while "jostling" the wrist joint to and fro and side to side which usually produces considerable crepitus. This is followed by a whipping adjustment of the Capitate anteriorly under the adjusting thumbs TOWARDS the palm with sharp slight extension of the wrist joint. The doctor's thumbs are then slid over to the ulnar side and contact is taken above the Pisiform/Hamate area and the same adjusting motions are repeated. Some doctors have been observed attempting to adjust the wrist joint in exactly the opposite direction, a questionable maneuver. Use of an objective "measuring stick" to gauge the effects of the adjustment is helpful in this regard. Proof of the success of the proper direction of adjustment is the immediate, demonstrable return of function (strength) of

opposition of thumb and little finger on MMT. Adjustment of the Pisiform/Hamate improves little finger strength. Adjustment of the Capitate and the distal ends of the radius/ulnar may improve thumb and/or little finger opposition strength. Heavy origin/insertion (grinding) pressures over the attachments of the Pronator Quadratus and ligaments of the wrist joint and "Golgi Tendon Technique" facilitate the improvement. The final adjustment is a traction/snap/ squeeze to bring the distal ends of the radius together. The wrist should then be taped while the patient holds the fingers spread with the wrist joint in a slightly flexed position to avoid compression that interferes with circulation. There is an occasional need to perform trigger point therapy on contraction induced weakness (strain/coounter-strain) of the small muscles of the hand, particularly at the thumb.

The thumb is reflexly affected symptomatically by Liver and Gallbladder dysfunction which may contribute overriding factors that may require attention in the relief of forearm, wrist and hand pain.

THE CHIROPRACTIC DIAGNOSES OF CARPAL TUNNEL SYNDROME

The Chiropractic oriented diagnoses evidencing the multifactorial approach in the described case above would be the following:

1. Teres Minor Stretch Weakness (thyroid), Ileocecal Valve flaccidity. sartorius weakness (adrenal). (indications of the major visceral corrections necessary)
2. Major pelvic imbalance affecting dorsal and cervical spine.
3. Medial Posterior proximal Ulnar, posterior Capitate and Pisiform. (This indicates the direction of corrective thrust necessary)
4. Posteriority of the radius at the elbow joint. (This indicates the direction of corrective thrust for the Radius.)
5. Inspiration assist cranial fault bilateral. (This indicates the need for the Pituitary Drive Technique.)
6. Left Anterior displacement of the fifth Cervical Vertebrae. (This indicates the level and direction of corrective thrust)

To avoid tautology, the diagnosis should contain within it, the corrections necessary. If a patient reports pain in the leg and the doctor's diagnosis is sciatica - we have nothing more than a tautology in this writer's opinion. The doctor simply calls the pain in the leg - sciatica. There is no indication in this diagnosis of which of the many different factors involved in the production of a sciatica is responsible for its presence. For a Chiropractor to use this type of diagnosis is to mimic the medical model. The proper diagnosis in sciatica would be for example, right flexion displacement of L4 on L5 with wedging to the left, along with a description of muscle imbalances and subluxations in the pelvis. This indicates, within the diagnosis, what the corrective action should be. Diagnosis is like asking the right question. In order to formulate the proper question, all of the facts must be known, full knowledge provides the proper question. Once the right question is asked, the answer is there, you cannot ask the proper question without knowing the answer!

C A S E # 2
(rib pump technique requirement)

This 48 year old assembly line auto worker described the pain in his left wrist and hand by pointing to his left anterior wrist joint and drawing a line to the mid palm. He also complained of long term pain in the left knee. The veins of the right leg showed numerous varicosities. He mentioned a history of kidney stones. To test the rib pump effects, the usual adjustment was rendered with the exception of rib pump technique. The patient felt relief of all symptoms following the first asjustment which succeeded in increasing pain tolerance to blood pressure cuff on the right leg to 200 mm from 150 mm and eliminating pain on testing of the left wrist extensors. Pelvis was corrected for category one and left anterior tibial reactivity was reset by pressing together on the muscle spindles. This overactivity was inhibiting the left sartorius, causing the long standing knee pain. The patient was counseled on kidney stones and his dietary and placed on a prophylactic dose of B6 and magnesium. Upon return, eight days later, the patient claimed that his knee pain was gone but there was no change in the left wrist symptoms which returned a couple days after the treatment. At this visit the wrist extensors were tested and found to be normal. The patient was then tested while laying on his left side and the wrist extensors weakened. This was negated by TL to the rib pump correction point at the fourth intercostal space on the left. Strain/counterstrain correction was then made of the rib pump reflex point and the wrist extensors no longer weakened in the left side lying position. Side lying testing is sometimes necessary to demonstrate a weakness and is effective in demonstrating effects of body positioning, especially for patients who cannot sleep on one side or the other. Routinely, the muscles are simply tested in normal fashion and when found weak in the clear are tested against TL to the rib pump point etc. The patient was seen two weeks later and reported that his left wrist pain was gone, in spite of the fact that he was forced to perform on several different jobs requiring heavy lifting and repetitive activity. My experience is beginning to suggest that much of the previous recidivism in carpal tunnel syndrome attributed to other factors was in fact due to failure to correct the rib pump fault. The reader is strongly advised to learn and use rib pump technique, especially in the treatment of athletes. In the treatment of several college baseball pitchers I have found the rib pump technique essential in eliminating elbow pain previously attributed to overuse.

D I S C U S S I O N

MOST FREQUENT CORRECTIONS MADE IN CTS

The rib pump technique, recently discovered by Goodheart, appears to be frequently involved in problems at the wrist joint and present experience suggests that it may ultimately be one of the more frequently observed factors in CTS. With this exception, the following list is provided suggesting the order of frequency of occurrence.

1. Posterior Capitate correction to anterior.
2. Posterior Pisiform/Hamate correction to anterior.

3. Separation of the Distal radius/ulnar joint, approximation correction.
4. Ileocecal Valve Neurolymphatic points, heavy rotary massage.
5. Adrenal Neurolymphatic points, heavy rotary massage.
6. Anteriority of C5 or C6 Cervical vertebra, adjust A to P.
7. Wrist, hand and elbow muscle balancing.
8. Pituitary drive technique (thyroid), pump mastoids forward on inspiration for four minutes or until body temperature normalizes. (98.0F in the axilla)
9. Teres Minor fascial flush technique (thyroid) until body temperature normalizes. (98.0F in the axilla)
10. Sartorius/Gracilic muscle balancing (adrenal).
11. Vitamin B6 deficiency.
12. Radial/Ulnar Bypass. (usually to bring ulnar distally and radius superiorly).
13. Pronator Teres reactivity and strain/counterstrain activity.
14. Brachioradialis variable dysfunction, especially accompanied by insomnia, benefitted by tryptophan.

MOST FREQUENT CAUSES OF EXACERBATIONS IN CTS

Regardless of the initial onset of CTS, whether it be traumatic or non-traumatic, most exacerbations in this writers practise are brought on by the following listed in order of frequency:

1. Ingestion of Popcorn - due to the large number of Amish patients seen by this writer whose favorite snack food is popcorn. Statistically, this is the most common nutritional factor in recidivism due to its deleterious effects on the ICV. Seeds and nuts are also provocative foods in this regard. The neurologically silent wrist joint is often "lit up" reflexly by ingestion of seeds, nuts or popcorn which cause reflux at the ICV. The patient is asymptomatic for long periods of time and suddenly wakes up with CTS symptoms with no trauma - failure to recognize ICV dysfunction in this patient can result in unnecessary surgery. The "lighting up" is thought by this writer to involve two major sources: 1. neurological connections based upon structure relating the wrist joint via the interneuronal pool connecting dermatomes, myotomes and sclerotomes and 2. a chemical factor involving absorption of inflammatory factors, particularly at the terminal ileum which exacerbates and adds to edematous conditions at the carpal tunnel.

2. Milk. The reader is invited to investigate the copious peer reviewed literature on the subject of milk intolerance and its production of gas in the Cecum resulting in anatomical distortions allowing ICV reflux.

3. Overuse via various carpentry, plumbing, electrical and assembly line work habits. (palm pressure with the wrist extended)

4. Abuse of physical and/or chemical nature. Using the palm of the hand as a hammer etc. or overindulgence in coffee, nicotine, or drugs which cause glandular and visceral problems reflexing into the wrist joint.

RECOMMENDED NUTRITIONAL SUPPORT

The author recommends the following nutritional supplementation provided by Biotics Research Corp. Houston, Texas 77326 or biochemical equivalents. "Nutritional Protocols" (7) is an excellent reference source for the use of these nutrients.

Primary support, especially in edema related numbness:

B6 PHOSPHATE 2 tablets t.i.d.
BIO-B-100 1 or 2 tablets t.i.d.
B100MG-ZYME 2 tablets t.i.d.
ZN-ZYME Forte 1 tablet t.i.d. for one week then
CU-ZYME 1/day.

Optional Synergists

OSTEO-B PLUS 1 tablet t.i.d.
CHONDROPLUS 2 tablets t.i.d.
HYDROZYME 2 tablets per meal

With low thyroid (avoid milk, ground meat and fluoridated water)

THYROSTIM 1-3 tablets t.i.d.

With ileocecal valve (avoid seeds, nuts and popcorn)

CHLOROCAPS 1 am 1 pm
CYTOZYME AD 1-2 tablets t.i.d.

C O N C L U S I O N

In this author's experience chronicity in CTS symptomatology is of multifactorial nature. Even when initially produced by trauma, elimination of symptoms often requires attention to glandular (thyroid/adrenal) function, bowel function (Ileocecal valve reflux), spinal mechanics, (cervical, dorsal and pelvic balance) and the joints of the lower extremities, all of which are reflexly linked to the upper limb on a visceral or gait related basis. For example: a visceral disturbance in the gall bladder/liver may neurologically "light up" the distal segments in the upper limb by spillover, setting up the chronicity; hypothyroid (myxedema) would serve to reduce metabolism, lowering body temperature and decreasing oxygenated blood supply to the area, both of which will exacerbate pain.

Attention to the nutritional status of the CTS patient is frequently necessary to achieve lasting success. This author expects future studies to confirm the importance of the dietary factor in CTS chronicity due to chemically provoked neurological dysfunction.

Occasionally, older debilitated patients suffering recidivism require a year or more to completely recover from the symptoms of CTS and require up to ten or fifteen adjustments (AK) over that period of time due to occasional exacerbations which seem to be caused by overuse, abuse, dietary indiscretions etc.

In the public interest long term controlled studies should be performed under hospital or university settings to properly gauge the cost effectiveness of chiropractic care in this very frequent and high cost surgery.

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CHIROPRACTIC COST EFFECTIVENESS IN GLAUCOMA

By
Daniel H. Duffy, D.C.
1-1-93

ABSTRACT: An increase in the horizontal visual field of an adult male glaucoma patient was noted following cranial bone corrections based upon diagnosis via Manual Muscle Testing (MMT) using Applied Kinesiology protocol.

INTRODUCTION

A brief review of facts clinically relevant to acute onset glaucoma suggest that: all eye pain should be considered glaucomatous until proven otherwise; the visual field (confrontation field test) and fundus (cupping of the disc) should be examined immediately; a diffuse corneal (steamy) haze may be present; vision may be markedly blurred; pain is usually severe; and the pupil may be moderately dilated, fixed and unresponsive to light. (1)

This author previously demonstrated, (by changes in ocular pressures demonstrated via non contact tonometer) immediately before and after treatment, the effectiveness of chiropractic in reducing pathological ocular pressures in a chronic glaucoma patient. (2)

Expertise in MMT supplies the doctor with a readily available, accurate, noninvasive, inexpensive, diagnostically pertinent meter with which to observe the effects of chiropractic adjustments on neurophysiological function. (3) It is no longer necessary to wait hours, days or weeks to observe the results of manipulation. MMT demonstrates the neurological consequences of the adjustment which can then be assessed for their physiological results, proved by previously measured parameters such as, ocular pressure, visual acuity, field of vision etc. Measurements of such pertinent parameters before and after each treatment prove or disprove the effectiveness of the adjustment. In the case of the glaucoma patient small areas of the visual field can be quickly and simply documented by the visual field confrontation test. (4) The eyeball can be palpated/applanated for reduction in pressure (2) and simple visual acuity and pain reduction can be assessed.

CASE HISTORY

An adult male complaining of right eye and facial pain had been seen by a dentist, family doctor and ENT specialist without relief. Examination revealed evidence of glaucoma, loss of vision in the right half of the visual field of the right eye, right TMJ dysfunction, internal rotation of the right frontal bone (without the usual flexor weakness) and right posteriority of the second cervical vertebrae. Related findings were: torque lesion of the pelvis diagnosed as a category one with right sacroiliac lesion, subluxation complex factors involving the fourth and seventh dorsal vertebra, fixation subluxation

factor involving the seventh cervical and first rib head on the left, grade 4/5 weakness of the right upper trapezius and left sternocleidomastoideus - all corrected via AK protocol. The second and third measurements of the visual field (see Fig.1) were made during the second office visit just before and after treatment. A 5.5 centimeter increase in the visual field was noted and symptoms (above) were relieved. The patient, an itinerant worker, was not seen again. Long term benefits of chiropractic care in glaucoma are unknown. What is known and has been demonstrated herein and elswhere (2) is that visual acuity, visual field and ocular pressure can be beneficially influenced by a single chiropractic adjustment.

D I S C U S S I O N

Ocular hypertension can be a frequent cause of insidious visual loss since most patients have open angle glaucoma which is asymptomatic (the only clue may be frequent changes in glasses). Occasionally the initial symptoms go unrecognized. While open angle glaucoma is painless, angle-closure glaucoma usually produces pain, burning or halos and occurs only with closure of a preexisting narrow anterior chamber angle found in the elderly (due to physiologic lens enlargement) hyperopes, and Asians. (1) This may be aggravated by exposure to darkness (sitting in a darkened theater), increased adrenal stimulation (epinephrine dilates the pupil) or anticholinergics all of which stimulate pupillary dilation further closing the angle. (1) Simple palpation of the eyeball which reveals pain and increased pressure calls for measurement of the eye field. Any patient with evidence of glaucoma in the chiropractor's office should be adjusted and then immediately referred to a medical opthamologist.

Eyeball pressures can usually be palpably and measurably affected by cranial manipulation, temperomandibular joint balancing, cervical spine adustments, and other factors. Increase in ocular tension is almost always caused by impaired outflow rather than excessive secretion. Impairment of outflow due to blockage by inflammatory debris or adhesions (uveitis), RBC's, tumors etc. at the anterior chamber angle can be exacurbated by the effect of simple structural faults adding to an already compromised situation. Many cases seen by this writer show tilting of the sphenoid (more often found elevated on the left with left eye protrusion compared to the right) which requires balancing of the external pterygoids and other cranial and cervical corrections. This imbalance probably interferes with central retinal vein drainage and in the acute case may require prolonged and sometimes slightly modified "respiratory assist pumping" of the cranium to achieve desired results in pain reduction and visual acuity and field. Evidence of adrenal dysfunction is also a frequent finding in this author's experience an often necessitates AK treatment to balance functional hypo and hyper adrenalism. Correction of patient behavioral patterns responsible for the adrenal imbalances is also frequently necessary. Responsibility for the glaucoma patient should be shared by chiropractors and MD's. Optometrists are frequently available for fast, inexpensive checking of ocular pressures before and after treatment by the chiropractor, once the patient has been identified and is being properly controlled.

C O N C L U S I O N

Contrary to popular opinion, an unknown percentage of glaucoma related visual losses may be preventable/correctable by chiropractic care if such treatment is not delayed. Whenever possible patients should be helped to avoid the use of medications such as topical beta blockers which can cause systemic side effects and oral medications which can cause malaise and anorexia and lead to symptoms suggestive of metastatic cancer. (1) A glaucoma prevention program consisting of early detection, correction and prevention of the structural/ nutritional factors in glaucoma related blindness via low cost chiropractic care could be expected to result in considerable cost reduction for the surgical, medical, and other tax supported services for the blind. Chiropractic treatment of functional disturbances involving vision provide instant results which are immediately measurable by the doctor and experienced by the patient as reduction in eye pain and increased visual acuity. The case reported herein received medical and chiropractic attention concurrently. The response displayed in Fig. 1 was obtained after the patient had been medicated for approximately one week. The second and third measurements were accomplished on the same office visit. Prompt crisis medical techniques can spare vision otherwise lost permanently and all patients with head, face or eye pain should be screened for proper visual fields. Failure to receive prompt corrections of cranial bones, neck flexors/extensors and other mechanical relatives of head, face and eye pain may also result in permanent blindness. The least benefit obtainable via chiropractic conservative measures is relief of head, face and eye pain even in those cases where vision has already been permanently lost or greatly reduced and the patient is a steady user of oral and topical medications. Chronic (medically treated) glaucoma patients regularly treated by this author are frequently relieved of intermittent headache, face and eye pain caused by the normal stress and strain of daily life in a patient with angle-closure glaucoma. Most of these patients needlessly suffer chronic headache, eye and face pain that is often relieved by a single chiropractic adjustment based upon AK diagnosis and this author's experience indicates that, while all glaucoma patients need chiropractic care, an unknown number may respond fully to chiropractic care alone. In the interests of cost effectiveness and particularly in the public interest regarding effective, noninvasive, side effect free health care - long term controlled studies are necessary to properly gauge effects of chiropractic care in this area. Informed consent of the glaucoma patient should include advice as to the possible beneficial effects of chiropractic care.

N U T R I T I O N A L R E C O M M E N D A T I O N S

Based upon nutritional history, blood chemistries, physical diagnosis and MMT (AK protocol) any or all of the following Biotics nutritional compounds (or their equivalents) may be necessary:

EYE 2 tabs t.i.d.; MG-ZYME 2 tabs t.i.d.; BIOPROTECT 2 caps t.i.d.; BIOFLAVANOIDS 1 cap q.i.d.; BIO-C PLUS 2 tabs t.i.d.; BILBERRY HERB 1-2 tabs/caps t.i.d. (5)

C H I R O P R A C T I C

C O S T E F F E C T I V E N E S S

I N

S I A L A D E N I T I S

By
Daniel H. Duffy, D.C.

ABSTRACT An adult female nursing student complained of intermittent swelling of the jaw area associated with chewing, eating and the production of saliva for two weeks which finally resulted in permanent swelling and pain over the entire side of her head, face and jaw. This uninsured patient was advised that medical diagnosis, not including treatment, would be $247.00. She responded to a single chiropractic treatment at a cost of $30.00, representing cost effectiveness of over 800%. Corrective therapy was diagnosed via manual muscle testing (MMT) based upon Applied Kinesiology (AK)(1) protocol. The patient continues to do well for almost a year following the single chiropractic treatment.

Key words: Sialadenitis, Chiropractic, cost effectiveness.

I N T R O D U C T I O N

Medical diagnosis of this patient was sialadenitis, an inflammation without pyogenic involvement. The patient showed no evidence of bacterial infection and claimed that her condition had been present for two or three weeks. She stated that the swelling of her jaw was: intermittent initially, would subside in a "short period of time", and was brought on by eating and drinking. For the few days just prior to chiropractic treatment, the swelling had remained constant and pain was beginning to envelope the entire ipsilateral side of the cranium, especially the ear neck and jaw.

D I S C U S S I O N

Postural examination revealed severe head tilt which is frequently accompanied by temperomandibular joint (TMJ) dysfunction. The following subluxation factors were diagnosed and corrected:

1. Lymphatic blockage to the right upper trapezius corrected by vigorous massage of reflex control points at the right posterior arch of the atlas/local occipital area and right intertubercular groove, (this correction resulted in a 50% reduction of the head tilt).
2. displacement of the occiput on the atlas and left rotational posteriority of the second cervical vertebra.
3. stretch weakness of the sternocleidomastoideus.
4. overactivity in the right stomach meridian of acupuncture.
5. overactivity in the right external pterygoid secondary to grade 4\5 weakness of the left external pterygoid. Overactivity in the closer muscles: masseter, buccinator, temporalis on the left.

6. Subluxation complex involving the entire pelvis as a unit with sacroiliac lesion on the side of involvement of the parotid.
7. Subluxation complex involving displacement of the seventh dorsal vertebrae and associated dysfunction of the fourth dorsal.
8. Positive therapy localization of the right temporal bone of the cranium just above the ear, a cranial subluxation factor known as a "temporal bulge" or "cranial bulge" which has been found to be associated with bilateral pectoralis muscles weakness (grade 4/5) which responds to half an inspiration and/or a source of lingual HCl.

The patient's severe head tilt upon initial examination was caused by a grade 4\5 weakness of the right upper trapezius and displacement of the occiput on the atlas, both diagnosed via MMT. The postural effect of these structural faults can cause a stretch of the Vagus nerve which can result in parasympathetic dysfunction.

The patient was seen two years previously complaining of pain and stiffness of the neck and general malaise. These symptoms were diagnosed via manual muscle testing (MMT) using AK protocol. The patient was pleasantly surprised at the efficacy of chiropractic and sought care for her son who was suffering from allergies. The son likewise responded rapidly to chiropractic care and continues to do well. It was the response to these apparently non-chiropractic conditions that prompted the patient to seek help for her sialadenitis.

Following correction of all subluxation factors listed above, the patient gargled a mouth full of Phosfood (Standard Process Labs) and sucked on a Betaine Hydrochloride tablet to provoke salivation. The parotid area was then manually drained to force out the solidifications and "gravel" and the patient experienced immediate relief of all symptoms.

In two previous cases: a sizable stone was manually retrieved from the sublingual duct (Bartholin's d.) of an adult male with anterior neck swelling, pain and discomfort; a sizable stone was retrieved from the parotid duct of a postmenopausal female, both of these patients responded to a single treatment without recidivism for seven and fifteen years respectively. My office staff reminds me of several other such cases which have long since passed from my memory. We estimate that over a period of twenty years successful intervention in such blockages has been performed an estimated six or seven times, not an insignificant amount considering the average knowledge amongst lay persons concerning the true scope of chiropractic care.

In further consideration of Sialadenitis (2): calculus formation is more common in Wharton's (larger and more radiopaque) duct than in Stensen's (smaller and radiolucent) duct; in Wharton's duct they may be palpable in the anterior floor of the mouth; In Stensen's in the buccal area alongside the second molar; the most common bacterial infection (S. aureus) involves the parotid and submandibular glands and is treated with IV antibiotics, hot compresses and sialogogues; ultrasound or CT scan may be helpful in establishing diagnosis; failure to remove the obstruction requires excision of the gland parenchyma; usually even recurrent sialadenitis of childhood resolves.

C O N C L U S I O N

An unknown percentage of Sialadenitis patients respond to conservative manipulative techique. Teaching hospitals associated with medical colleges should request chiropractic assistance to conduct studies to determine the full potential of chiropractic in this area. Savings in health care costs could be considerable while at the same time sparing patients further potential harm from unnecessary drug and surgical intervention.

Note that the 800% cost effectiveness of chiropractic care reported herein did not include the cost of medical treatment (or possible surgical intervention) to CORRECT the condition but to simply DIAGNOSE it. Savings compared to medical treatment, which minimally includes medication (possibly IV), sialogogues and hot compressess, may result in savings up to 1500% of more and If medical intervention failed, making surgery necessary, the savings would be substantially increased even further.

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NAUSEA OF PREGNANCY RESPONDS TO CHIROPRACTIC CARE

By
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March 1, 1992

ABSTRACT: A 30 year old in her first pregnancy was immediately relieved of a seven week long bout of nausea and vomiting following manipulative corrections diagnosed via Applied Kinesiology Technique (AKT).

Key words: Nausea, pregnancy, Chiropractic, Applied Kinesiology.

INTRODUCTION

A thirty year old female suffering nausea and vomiting for seven weeks of pregnancy resulting in steady weight loss and much physical and mental debilitation, was immediately relieved of her symptoms by Chiropractic manipulation diagnosed via AKT. (1) Although the patient was familiar with Chiropractic care and had previously been completely relieved of a life long allergic condition requiring regular injections by a medical allergy specialist, she was initially guided in her approach to her pregnancy related problems by her Medical Gynecologist who was unaware of the effectiveness of Chiropractic care in patients of this type. Therefore she failed to consider Chiropractic care until she became desperate. The patient had been free of all allergy medications for two years prior to onset of pregnancy and relative to her nausea, had been advised by her MD to "tough it out".

CASE HISTORY

Findings diagnosed via AKT:

1. Flaccidity of the ileocecal valve.(ICV)
2. Subluxation complex involving malposition of the Occiput, and lack of proper lymphatic flow in the Right Upper Trapezius and Left Sternocleidomastoideus.
3. Subluxation complex involving the dorsal spine.
4. Pelvic instability caused by a subluxation complex involving the sacroiliac joint and right piriformis muscle.
5. Subluxation complex involving lymphatic drainage of the Sartorius muscle, including Whole Adrenal Gland-negated positive therapy localization (PTL) of the Adrenal Neurolymphatic points. The source of Whole Adrenal Gland was Biotics. (3)

Following correction of the ICV and the adjustment of the occiput the patient remarked that her nausea was gone. The remaining subluxations were then corrected and the patient continued to do well until normal delivery. She was seen once a month throughout the remainder of her pregnancy and remarked that towards the end of the month she would feel a slight return of symptoms which would abate with each Chiropractic adjustment. The patient usually required pelvic balancing and spinal adjustments diagnosed and corrected in accordance with published AKT.

DISCUSSION

Subluxations resulting in flaccidity of the ICV or what is commonly called by some Chiropractors, "The Open Ileocecal Valve" allow reflux of cecal contents into the terminal ileum. Reflux of toxins disturbs conditions in the terminal ileum. The presumption is that the toxins are then absorbed and carried about the body and especially in the absence or deficiency of proper amounts of antioxidants, cause widespread symptomatology which is usually medically misdiagnosed and therefore mistreated. The misdiagnosis is due largely to the incommensurability of the concept of ileocecal valve induced symptoms with current medical models. The situation is further complicated by academic resistance towards acceptance of the new diagnostic tools made available by recent AKT discoveries involving subluxation factors. (4) A subluxation complex including Occipital displacement can cause Vagal stimulation which increases gastric secretion of histamine resulting in pain, nausea and vomiting. (2)

Some patients suffering nausea of pregnancy require vitamins B6, C and K. ICV patients are often in need of supplemental vitamin K. A new effective source of Vitamin K has been found produced by Biotics Inc., in their Chlorocaps (3). The patient is instructed to dump half of the capsule on the tongue in the AM and PM. ICV positive therapy localization (PTL) (1) was found to respond to lingual tasting of Biotics Chlorophyll in 16 of 18 patients tested. The Biotics Whole Adrenal which is taken from bovine fetus prior to being contaminated by environmental poisons in feeds etc. has been found effective in every clinical trial of muscle testing against PTL and sartorius or gracilis weakness.

C O N C L U S I O N

While many women suffer the nausea of pregnancy, few seek chiropractic care for relief. The most common problems in such nausea are associated with ileocecal valve dysfunction and subluxations affecting vagal nerve function. Because of the need for optimal nutrition in pregnancy, necessary for fetal development, more widespread publicity on the effectiveness of chiropractic in this regard should be made available. Informed consent of the patient suffering nausea of pregnancy should include mention of the use of chiropractic care.

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Update... René Espy, D.C. and Nancy McBride, D.C.

A CONTINUING STUDY IN THE CORRECTION OF PATHOLOGICALLY WEAK MUSCLES WITH THE APPLICATION OF SPECIFIC MUSCLE FREQUENCY

by

Dr. René Espy and Dr. Nancy McBride

ABSTRACT

This paper is a continuum in the study presented at the 1992-93 annual meeting of the ICAK-USA. In the previous study we spoke of the possibilities of frequency definition as a potential correction for certain pathological deterioration. In the time elapsed since the previous paper was submitted we have discovered that not only is a base wave and carrier wave necessary but optimum muscle response requires a specific intensity and wave form. The authors contend that Applied Kinesiology muscle testing and evaluating techniques deals only with one frequency which in our clinical practice has not been sufficient to correct the type of pathologic conditions described herein. At the time of our presentation in 1992, we believed that we had found a way to access previously unaccessible muscles. With further study we found that a specific pattern of frequencies is needed to hologramically correct major pathological muscle patterns. With the outcome of this study, it is hopeful that the Applied Kinesiologist will have the objective tools to qualitatively and quantitatively measure specific muscle testing procedures and thereby be able to include a greater number of patients within their already broad scope of practice.

INTRODUCTION

If one takes an objective look at the body and realizes its comparisons to the electronic computer one might see that the evolutionary model of "survival of the fittest" is a basis for the comparison between the brain - body complex and the electronic computer. By studying the neuron and its feedback system one realizes the thousands of inputs it must coordinate for balanced function. One can then appreciate the possibilities of functions available to the individual impulse. 1.

With the continued study of those patients with extremely poor prognosis and seeing results that appear almost impossible it is the contention of the authors that the problem is not in the inability of the body to perform a specific function. Rather it may be that the impulse has taken the wrong direction or that the transmission of impulses is inhibited by delay lines that slow down transmission or completely inhibit function. Most of the time this is not due to pathology but rather misguided signals.

When the point of reference for any function in the body is interrupted, distorted or disturbed in any way its correlations are inhibited because the body has begun its system of adaptation. In the case of paralysis, for instance, one might ask if the issue is one of pathologic damage or is the body's normal inhibitory mechanism activated such that the normal frequency impulses cannot reach their destination.

At the 1992 annual ICAK-USA meeting we demonstrated, on a patient who had been a childhood victim of Polio, using an electro-acuscope instrument, that a specific weak muscle could be isolated and when a frequency specific for that muscle was applied to the body the muscle would respond in strength.

With continued research, we found that while we could apply the frequency and elicit a desired result that that instrument proved inadequate. We then approached an engineer, Duane Hall, who has designed many instruments for Dynatronics, a Salt Lake City, Utah company which develops diagnostic and therapeutic instruments primarily for the Chiropractic profession. With Duane's engineering expertise we were able to design an instrument that not only holds its frequency but by oscilloscopic testing maintains that frequency within 1/100th of a Hz.

The instrument used is the Dynatron 200. It is an instrument approved by the FDA and both the hardware and software have also been upgraded to our specifications. We also have the goniometer prototype, pressure gauge and computer program to analyze the pounds of pressure used in testing a muscle.

Updating the information from the previous year, we found it necessary to use a basic frequency, a carrier wave, specific intensity and specific wave form. Without these four components it is not possible

to achieve a lasting result. Numerous instruments have been tested and the upgraded Dynatron 200 is the only one able to hold the frequency at the specific requirements and to give an output that is measurable by the most sophisticated measuring equipment.

If we look at the brain - body complex we realize its various functions have thousands of possibilities. We must also realize that when the brain is affected by some aspect of change, disease or trauma, that all of our mental processes, emotions, sensations, perceptions, feelings, intellectual processes, memories and states of consciousness are also changed instantaneously.

DISCUSSION

The brain-body has a hierarchical system that is organized in such a manner that each level of the system is organized in itself and also within the system as a whole.

The higher brain levels deal with a few complex and powerful processing elements and define goals and priorities while the lower levels have a multitude of relatively simple processing elements doing similar jobs. It appears as though the brain-body has three major functions: one, to make trivial decisions the higher levels don't have to be concerned with; two, to deal with complex problems and three to deal with the survival of the system as a whole.

It is interesting to note that the higher levels control the lower by inhibiting their actions except as desired or needed. This is a phenomena that was designed by an intelligence greater than us that sets up a system so that if the higher system is damaged, the lower more primitive units or lower levels which are normally inhibited, are released to function. Thus vital functions are not eliminated and survival is permitted.

The lowest level of the central nervous system is the spinal cord and it is the major route of input and output for the brain. Most of the time the sensory input from the body and most of the output to the muscles passes through the spinal cord. This sets up an intelligent terminal for both input and output of information.

One of the important aspects of brain-body function is memory. We have no real proof of the location of memory but in observation of patients it is the contention of the authors that every cell contains slots for memory and is therefore memory is distributed throughout the system.

The brain and body have the capacity to store memories of facts and events. It is the contention of the authors that trauma can interfere with the body's normal processing channels. Trauma in any form can release memories that interfere with normal function and could create inappropriate behavior. Ibid.

PROCEDURE

Due to the fact that the cases being presented are so unique, we will explain each individually instead of following the normal scientific protocol.

Case 1
Richard was diagnosed as having Multiple Sclerosis ten years ago. He has a history of double vision, significant loss of bladder control, walks with a cane and frequently falls due to instability and lack of muscle control.

Case 2
Mark was diagnosed as a paraplegic after falling off a horse and twisting his spine, injuring his spinal column. Mark's prognosis was negative. No hope was given for any possibility of ever using his lower limbs.

Case 3
Mary was walking with her husband when suddenly her left leg became weak and she was unable to continue. After a period of 13 years the weakness progressed until she was forced to use a molded urethane brace to support her lower leg.

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Case 4
Sylvia was diagnosed with fibromyositis. She had no strength in either lower extremity. Her walking was very labored and she was unable to walk up stairs without pulling herself up with a railing.

Case 5
Steve was diagnosed with Muscular Dystrophy 23 years ago. He had typical muscle wasting and walks with molded braces on both legs.

Case 6
Anita has severe osteoporosis with 44% bone density loss. She has sustained multiple rib fractures from minor physical insult.

The significant areas checked according to the parameters of the study are the muscles of the body depending upon the affected area. These muscle tests are those taken from the Muscle manuals published by the authors and are dependent upon the affected areas of the patients. 2. (See muscle manuals).

MATERIALS AND METHODS

Each patient was examined kinesiologically to determine the extent of muscle weakness. The examination included orthopedic and nueological testing, however for this study emphasizes specific muscle testing. The instrument used for muscle evaluation if the Dynatron 200 that has beenadapted and upgraded according to the specifications of the authors. The subjects were patients with serious conditions that had not responded to connventional medicine and had presented themselves to this office for care. The muscles tested are in the specific interrelations charts published in the June 1992-93 Proceedings. We have included more charts than the myomere charts as patients with other problems may require specific frequencies from charts other than myomeres.

EXAMINATION

Upon examination, Richard was found to have the following weak muscles : All the muscles of the abdomen, bilaterally, all the muscles of the low back weak bilaterally, all the muscles of the ilium weak on the left, all the muscles of the pelvis weak bilaterally, all the thigh muscles on the left weak, all the muscles of the calf and ankle weak on the left. The point of reference for Richard was the L 4 myomere. Refer to the interrelationship sheets for L4 myomere. 3

Upon examination, Mark was found to have absolutely no strength in any muscle below the navel. The point of reference for Mark was the L 3 myomere. Refer to the interrelationship sheets for L3 myomere. 3

Upon examination, Mary was found to have weakness of all of the muscles related to the L 4 myomere. Refer to the interrelationship sheets for L4 myomere. 3

Upon examination, Sylvia was found to have weakness of all the muscles of the lower extremity bilaterally. She also was unable to flex her arms above a 20° angle and had weakness of the majority of the shoulder/arm muscles. The point of reference for Sylvia was the Cranial Nerve 5 myomere. Refer to the interrelationship sheets for Cranial Nerve V myomere. 3

Upon examination of Anita the primary display area of the body was the ovary. The hormones produced by the ovary are, as you know, a vital component of calcium metabolism. Anita was found to have the following weak muscles: All of the muscles relating to the Ovary. Refer to the interrelationship sheets for Ovary.

Upon examination, Steve was found to have weakness in multiple areas of the body. The primary point of reference was the C 7 myomere. All of the muscles related to the C 7 myomere were also weak. Refer to the interrelationship sheets for L4 myomere. 3

TREATMENT PHASE

The patients were lying supine on the treatment table and electrodes were placed on the forehead with an electrolyte gel. The instrument was set at a specific basic Hz., plus the carrier wave for the specific point

of reference and the specific intensity of the specified wave form. The previously tested weak muscles were retested and the results recorded.

Note: The more severe the pathology the greater is the length of time needed for applying the frequency and the more complex the treatment becomes. As noted earlier in this discussion when trauma affects the body numerous adaptive processes are activated, therefore normalization requires reintegration in a specific order. It is interesting to observe that even though a body has suffered severe trauma once the "channel" has been opened the body can elicit normal function.

FINDINGS

When the specific frequency for the individual muscles was applied no change in strength was noted. When the frequency for the specific point of reference (organ, myomere, vertebral level, etc.) was applied it was noted that the previously tested weak muscles now tested strong.

CONCLUSION

The specific frequency of each muscle is stored in memory even in the presence of pathology or severe trauma. The fact that when the correct frequency is applied to a weak muscle and the muscle responds within seconds implies that the frequency does not have to be relearned but rather the pathway must be reconnected. As stated earlier, the multiplicity of functions and variables extends into the thousands and proper messages are implicit in proper function. Treatment of a muscle that is pathologically impaired requires the recongition of its specific frequency in order for the five IVF of Applied Kinesiology to be accessed. It was a very frustrating procedure to determine which frequency was affecting a muscle. Through many trial and error testing procedures and many failures in determining frequency, once the concept of factors affecting a mucle having direct frequency correlations to many areas of the same muscle the concept of muscle frequency became more stable. It was an amazing phenomonena to the authors to discover that, not only does each muscle have a specific frequency, it also correlates numerous frequencies depending upon the function and need for use of each muscle.

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MUSCLE RELATIONSHIPS

ACUPUNCTURE INDEX/NUMERICAL

(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals of Dr. Beardall.)

(052) XA 3	Occipitalis
(058) Si 3	Levator Palpebrae, Superior Division
(060) Tw 4	Orbicularis Oculi, Superior Division
(062) H 7	Orbicularis Oculi, Inferior Division
(070) K 2	Depressor Septi
(098) B 6	Orbicularis Oris, Upper Division
(100) G 2	Orbicularis Oris, Inferior Division
(102) Li 19	Buccinator
(110) B 56	Temporalis, Parietal Division
(112) Tw 10	Temporalis, Occipital Division
(114) G 39	Masseter, Superficial Division
(118) G 38	Masseter, Deep Division
(120) Si 5	Pterygoid Internal Medialis, Sphenoid Division
(122) Lv 4	Pterygoid Internal Medialis, Palatine Division
(124) St 38	Pterygoid External Lateralis, Upper Div.-Disc
(126) St 41	Pterygoid External Lateralis, Lower Division
(130) G 1	Rectus Superior Bulbi
(132) Si 14	Rectus Inferior Bulbi
(134) G 38	Rectus Medialis Bulbi
(136) Sp 5	Rectus Lateralis Bulbi
(138) G 28	Obliquus Superior Bulbi
(140) Lu 4	Obliquus Inferior Bulbi
(270) B 58	Upper Trapezius, Scapular Division
(272) St 10	Upper Trapezius, Clavicular Division
(274) Cx 2	Sternocleidomastoid, Sternal Division
(276) Lv 6	Sternocleidomastoid, Clavicular Division
(278) Sp 10	Scalenus Anterior
(282) Cx 9	Scalenus Medius
(284) Tw 12	Scalenus Posterior
(286) Cv 17	Platysma, Anterior Division
(288) St 45	Platysma, Posterior Division
(290) K 25	Digastric, Anterior Belly
(292) K 17	Digastric, Posterior Belly
(294) Cv 15	Stylohyoid
(296) Li 7	Mylohyoid
(298) H 8	Geniohyoid
(300) Li 8	Sternohyoid
(302) Li 13	Sternothyroid
(304) G 33	Thyrohyoid
(306) H 7	Omohyoid
(308) XL 3	Longus Coli, Vertical Division
(314) Tw 3	Longus Capitis
(322) Tw 16	Splenius Capitis, Mastoid Division
(326) Lv 7	Splenius Cervicis
(332) K 9	Semispinalis Capitis
(334) Tw 16	Semispinalis Cervicis
(346) G 40	Obliquus Capitis Superior
(350) St 37	Interspinalis (Cervical)
(362) St 44	Cricoarytenoideus Lateralis
(370) Sp 4	Thyroarytenoideus
(380) Tw 2	Constrictor Pharyngeus Medius
(390) Gv 22	Trapezius, Middle Division
(392) Tw 10	Trapezius, Lower Division

(394) Li 15	Levator Scapula, Superior Division
(396) Li 14	Levator Scapula, Inferior Division
(398) Lv 8	Rhomboid Minor
(400) St 36	Rhomboid Major
(402) St 33	Serratus Anterior, Superior Division
(404) Tw 6	Serratus Anterior, Inferior Division
(406) G 21	Pectoralis Minor, Superior Division
(408) St 28	Pectoralis Minor, Inferior Division
(410) Li 13	Subclavius, Clavicular Division
(412) H 7	Subclavius, Scapular Division
(414) Tw 15	Latissimus Dorsi, Thoracic Division
(416) Lu 5	Latissimus Dorsi, Lumbar Division
(418) Tw 3	Latissimus Dorsi, Iliac Division
(420) St 43	Supraspinatus, Spine Division
(422) B 60	Supraspinatus, Fossa Division
(424) Cx 1	Infraspinatus, Superior Division
(426) Tw 5	Infraspinatus, Middle Division
(428) Lu 6	Infraspinatus, Inferior Division
(430) Li 4	Teres Minor
(432) K 11	Teres Major, Superior Division
(434) St 40	Teres Major, Inferior Division
(436) Tw 9	Subscapularis, Superior Division
(438) Tw 3	Subscapularis, Third Division
(440) Cx 4	Subscapularis, Second Division
(442) Tw 5	Subscapularis, Inferior Division
(444) Lv 10	Coracobrachialis, Coracoid Division
(446) B 62	Coracobrachialis, Septal Division
(448) Lu 8	Deltoid, Posterior, Medial Division
(450) Lv 6	Deltoid, Posterior, Lateral Division
(452) Lv 3	Deltoid, Middle, Posterior Division
(454) Cx 3	Deltoid, Middle, Anterior Division
(456) G 31	Deltoid, Anterior, Scapular Division
(458) G 43	Deltoid, Anterior, Clavicular Division
(460) G 30	Pectoralis Major, Clavicular Division
(462) Sp 4	Pectoralis Major, Sternal Division
(464) Cx 7	Pectoralis Major, Costal Division
(466) Sp 9	Biceps Brachii Longhead
(468) Tw 8	Biceps Brachii Shorthead
(470) B 50	Triceps, Longhead
(472) G 39	Triceps, Lateral Head
(474) Li 2	Triceps, Medial Head
(476) Lv 11	Articularis Cubiti
(478) Tw 9	Brachialis
(480) St 32	Brachioradialis, Humeral Division
(482) Tw 14	Brachioradialis, Septal Division
(484) B 64	Pronator Teres, Humeral Division
(486) St 14	Pronator Teres, Ulnar Division
(488) Li 9	Anconeus, Olecranon Division
(490) St 40	Anconeus, Ulnar Division
(492) K 3	Supinator, Radial Division
(494) Tw 6	Supinator, Ulnar Division
(496) Li 6	Pronator Quadratus, Proximal Division
(498) St 18	Pronator Quadratus, Distal Division
(500) Si 7	Palmaris Longus
(502) B 66	Flexor Carpi Radialis, Abductor Division
(504) H 2	Flexor Carpi Radialis, Flexor Division
(506) Sp 9	Flexor Carpi Ulnaris, Flexor Division

(508) Lu 11	Flexor Carpi Ulnaris, Adductor Division
(510) K 24	Extensor Carpi Ulnaris, Adductor Division
(512) G 30	Extensor Carpi Ulnaris, Extensor Division
(514) St 43	Extensor Carpi Radialis Brevis
(516) B 2	Extensor Carpi Radialis Longus, Ext Division
(518) K 6	Extensor Carpi Radialis Longus, Abductor Division
(520) Lu 3	Extensor Pollicis Longus, Ulnar Division
(522) Lv 6	Extensor Pollicis Longus, Septal Division
(524) K 2	Extensor Pollicis Brevis, Radial Division
(526) St 24	Extensor Pollicis Brevis, Septal Division
(528) K 9	Flexor Pollicis Longus
(530) Lu 9	Abductor Pollicis Longus, Ulnar Division
(532) Tw 4	Abductor Pollicis Longus, Radial Division
(534) B 51	Flexor Pollicis Brevis
(536) Lu 8	Interossei Pollicis (Palmaris First)
(538) H 6	Opponens Pollicis, Flexor Division
(540) B 51	Opponens Pollicis, Abductor Division
(542) G 35	Abductor Pollicis Brevis
(544) Sp 5	Adductor Pollicis Transversus
(546) Tw 15	Adductor Pollicis Obliquus
(548) Cx 7	Extensor Digitorum Communis Manus, Medial Division
(550) Tw 9	Extensor Digitorum Communis Manus, Lateral Division
(552) St 37	Extensor Digiti Minimi Manus
(554) Li 13	Extensor Indicis Proprius
(556) G 40	Flexor Digitorum Superficialis, Medial Division
(558) Li 8	Flexor Digitorum Superficialis, Lateral Division
(560) Li 15	Flexor Digitorum Profundus Manus, Medial Division
(562) Si 1	Flexor Digitorum Profundus Manus, Lateral Division
(564) K 25	Interossei Dorsales Manus, Fourth
(566) St 39	Interossei Dorsales Manus, Third
(568) Si 8	Interossei Dorsales Manus, Second
(570) Lv 9	Interossei Dorsales Manus, First
(572) K 8	Lumbricales Manus, Fourth Division
(574) Lv 3	Lumbricales Manus, Third
(576) Tw 17	Lumbricales Manus, Second
(578) Gv 23	Lumbricales Manus, First
(580) G 22	Flexor Digiti Minimi Brevis, Manus
(582) H 4	Abductor Digiti Minimi Manus, Flexor Division
(584) Si 7	Abductor Digiti Minimi Manus, Abductor Division
(586) St 18	Opponens Digiti Minimi Manus, Abductor Division
(588) Cx 3	Opponens Digiti Minimi Manus, Flexor Division
(590) Tw 14	Interossei Palmaris, Fourth
(592) K 1	Interossei Palmaris, Third
(594) Sp 13	Interossei Palmaris, Second
(596) Cv 9	Palmaris Brevis
(610) G 31	Spinalis Thoracis, Lumbar Division
(612) B 62	Spinalis Thoracis, Thoracic Division
(614) H 5	Longissimus Thoracis, Superior Division
(618) St 32	Longissimus Thoracis, Inferior Division
(642) G 34	Levator Costorum, Inferior Division
(648) Lv 4	Serratus Posterior, Superior Division
(652) Lu 7	Serratus Posterior, Inferior Division
(656) RG 30/LSp 21	Diaphragm, Right Lumbar Division
(662) LG 30/RSp 21	Diaphragm, Left Lumbar Division
(690) Sp 11	Pyramidalis
(692) Li 14	Obliquus Externus Abdominis, Anterior Division
(694) Lv 4	Obliquus Externus Abdominis, Lateral Division

(696) Si 8	Obliquus Internus Abdominis, Anterior Division
(698) Cx 8	Obliquus Internus Abdominis, Lateral Division
(700) Cx 4	Rectus Abdominis, First Division
(702) Li 14	Rectus Abdominis, Second Division
(704) Tw 12	Rectus Abdominis, Third Division
(706) G 31	Rectus Abdominis, Fourth Div., Medialis
(708) Li 9	Rectus Abdominis, Fourth Div., Lateralis
(710) Lu 10	Iliacus
(712) G 32	Iliacus Minor
(714) Si 5	Transverse Abdominis, Upper Division
(718) H 4	Transverse Abdominis, Lower Division
(722) G 37	Psoas Major, Lumbar Division
(724) Lv 4	Psoas Major, Thoracic Division
(726) Gv 2	Psoas Major, Diaphragmatic Division
(728) St 29	Psoas Minor
(730) St 38	Quadratus Lumborum, Costal Division
(732) Sp 8	Quadratus Lumborum, Lumbar Division
(734) Li 15	Multifidus, Lumbosacral Division
(736) Si 9	Iliocostalis Lumborum
(738) Tw 1	Longissimus Lumborum
(740) Lv 5	Coccygeus, Sacral Division
(742) Li 10	Coccygeus, Coccyx Division
(744) Gv 26	Pubococcygeus
(746) G 41	Iliococcygeus
(752) K 7	Cremaster
(780) St 39	Gluteus Medius, Posterior Division
(782) Cx 2	Gluteus Medius, Middle Division
(784) Lu 11	Gluteus Medius, Anterior Division
(786) Sp 3	Gluteus Minimus, Anterior Division
(788) Li 1	Gluteus Minimus, Posterior Division
(790) G 34	Tensor Fascia Lata, Anterior Division
(792) Lu 1	Tensor Fascia Lata, Posterior Division
(794) Tw 7	Rectus Femoris, Reflected Head
(796) Lu 6	Rectus Femoris, Straight Head
(798) St 38	Pectineus
(800)L St 41	Adductor Brevis (Left)
(800)R St 41	Adductor Brevis (Right)
(804) Si 6	Adductor Longus, Inferior Division
(806) B 65	Adductor Longus, Superior Division
(808) St 32	Gracilis
(810) Si 9	Sartorius
(812) Tw 13	Obturator Externus
(814) Lu 11	Quadratus Femoris
(816) K 5	Vastus Medialis, Upper Division
(818) Li 7	Vastus Medialis, Middle Division
(820) Sp 12	Vastus Medialis, Lower Division
(822) Li 5	Obturator Internus
(824) Lu 10	Biceps Femoris, Shorthead
(826) Lu 5	Biceps Femoris, Longhead, Fibular Division
(828) Li 11	Biceps Femoris, Longhead, Tibial Division
(830) Li 8	Vastus Lateralis, Superior Division
(832) K 4	Vastus Lateralis, Middle Division
(834) Tw 4	Vastus Lateralis, Lower Division
(836) Tw 14	Vastus Intermedius, Medial Division
(838) Sp 1	Vastus Intermedius, Lateral Division
(840) B 63	Articularis Genu
(842) XL 2	Adductor Magnus, Vertical Division

(844) Li 3	Adductor Magnus, Oblique Division
(846) G 44	Adductor Magnus, Transverse Division
(848) St 41	Gluteus Maximus, Iliac Division
(850) H 1	Gluteus Maximus, Sacral Division
(852) B 13	Gluteus Maximus, Coccygeal Division
(854) G 30	Semitendinosus
(856) Sp 8	Semimembranosus, Tibial Division
(858) B 25	Semimembranosus, Popliteal Division
(860) Sp 8	Piriformis
(862) Li 17	Gemellus Inferior
(864) Si 2	Gemellus Superior
(870) G 32	Popliteus
(872) Tw 11	Gastrocnemius, Medial Division
(874) Li 6	Gastrocnemius, Lateral Division
(876) Tw 13	Plantaris
(878) Cx 8	Soleus Medial Division
(880) Si 12	Soleus, Lateral Division
(882) Li 8	Tibialis Posterior, Tibial Division
(884) H 3	Tibialis Posterior, Fibular Division
(886) H 2	Peroneus Longus, Cuneiform Division
(888) Li 7	Peroneus Longus, Metatarsal Division
(890) G 32	Peroneus Brevis, Fibular Division
(892) B 67	Peroneus Brevis, Septal Division
(894) Tw 9	Peroneus Tertius
(896) Sp 6	Tibialis Anterior, Supinator Division
(898) Lu 4	Tibialis Anterior, Dorsiflexor Division
(900) H 9	Flexor Hallucis Longus, Tibial Division
(902) B 58	Flexor Hallucis Longus, Fibular Division
(904) Lv 1	Extensor Hallucis Longus, Interosseous Division
(906) Si 8	Extensor Hallucis Longus, Fibular Division
(908) Si 11	Flexor Digitorum Longus, Medial Division
(910) G 27	Flexor Digitorum Longus, Lateral Division
(912) H 6	Extensor Digitorum Longus, Medial Division
(914) B 27	Extensor Digitorum Longus, Lateral Division
(920) K 10	Adductor Hallucis, Superior Division
(922) Lu 2	Adductor Hallucis, Inferior Division
(924) Cv 7	Flexor Hallucis Brevis, First Cuneiform Division
(926) H 8	Flexor Hallucis Brevis, Tendonal Division
(928) Sp 3	Flexor Hallucis Brevis, Third Cuneiform Division
(930) Tw 10	Flexor Hallucis Brevis, Cuboid Division
(932) B 51	Abductor Hallucis Oblique Head, Peroneus Division
(934) Cv 5	Abductor Hallucis Oblique Head, Metatarsal Division
(936) Tw 12	Abductor Hallucis Transverse Head, Medial Division
(938) Si 4	Abductor Hallucis Transverse Head, Lateral Division
(940) B 43	Extensor Hallucis Brevis
(942) G 33	Quadratus Plantae, Medial Division
(944) G 42	Quadratus Plantae, Lateral Division
(946) Cx 6	Flexor Digitorum Brevis, Medial Division
(948) Li 5	Flexor Digitorum Brevis, Lateral Division
(950) B 63	Flexor Digitus Pedis, Second
(952) Li 4	Flexor Digitus Pedis, Third
(954) Lv 9	Flexor Digitus Pedis, Fourth
(956) Lv 6	Flexor Digitus Pedis, Fifth
(958) B 65	Adductor Digitus Pedis, Second
(960) Si 7	Adductor Digitus Pedis, Third
(962) Li 20	Adductor Digitus Pedis, Fourth
(964) H 8	Adductor Digitus Pedis, Fifth

(966) B 2	Abductor Digitus Pedis, Second
(968) Tw 11	Abductor Digitus Pedis, Third
(970) G 43	Abductor Digitus Pedis, Fourth
(972) Lv 2	Abductor Digitus Pedis, Fifth
(974) St 33	Abductor Digiti Minimi Pedis
(976) Cx 8	Extensor Digitorum Brevis

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B 2 Abductor Digitus Pedis, Second (966)
B 2 Extensor Carpi Radialis Longus, Ext Division (516)
B 6 Orbicularis Oris, Upper Division (098)
B 13 Gluteus Maximus, Coccygeal Division (852)
B 25 Semimembranosus, Popliteal Division (858)
B 27 Extensor Digitorum Longus, Lateral Division (914)
B 43 Extensor Hallucis Brevis (940)
B 50 Triceps, Longhead(470)
B 51 Abductor Hallucis Oblique Head, Peroneus Division (932)
B 51 Flexor Pollicis Brevis (534)
B 51 Opponens Pollicis, Abductor Division (540)
B 56 Temporalis, Parietal Division (110)
B 58 Flexor Hallucis Longus, Fibular Division (902)
B 58 Upper Trapezius, Scapular Division (270)
B 60 Supraspinatus, Fossa Division (422)
B 62 Coracobrachialis, Septal Division (446)
B 62 Spinalis Thoracis, Thoracic Division (612)
B 63 Articularis Genu (840)
B 63 Flexor Digitus Pedis, Second (950)
B 64 Pronator Teres, Humeral Division (484)
B 65 Adductor Digitus Pedis, Second (958)
B 65 Adductor Longus, Superior Division (806)
B 66 Flexor Carpi Radialis, Abductor Division (502)
B 67 Peroneus Brevis, Septal Division (892)

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Cv 5 Abductor Hallucis Oblique Head, Metatarsal Division (934)
Cv 7 Flexor Hallucis Brevis, First Cuneiform Division (924)
Cv 9 Palmaris Brevis (596)
Cv 15 Stylohyoid (294)
Cv 17 Platysma, Anterior Division (286)

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Cx 2 Gluteus Medius, Middle Division (782)
Cx 2 Sternocleidomastoid, Sternal Division (274)
Cx 3 Deltoid, Middle, Anterior Division (454)
Cx 3 Opponens Digiti Minimi Manus, Flexor Division (588)
Cx 4 Rectus Abdominis, First Division (700)
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Cx 6 Flexor Digitorum Brevis, Medial Division (946)
Cx 7 Extensor Digitorum Communis Manus, Medial Division (548)
Cx 7 Pectoralis Major, Costal Division (464)
Cx 8 Extensor Digitorum Brevis (976)
Cx 8 Obliquus Internus Abdominis, Lateral Division (698)
Cx 8 Soleus Medial Division (878)
Cx 9 Scalenus Medius (282)

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G 1 Rectus Superior Bulbi (130)
G 2 Orbicularis Oris, Inferior Division (100)
G 21 Pectoralis Minor, Superior Division (406)
G 22 Flexor Digiti Minimi Brevis, Manus (580)
G 27 Flexor Digitorum Longus, Lateral Division (910)
G 28 Obliquus Superior Bulbi (138)
G 30 Extensor Carpi Ulnaris, Extensor Division (512)
G 30 Pectoralis Major, Clavicular Division (460)
G 30 Semitendinosus (854)
G 31 Deltoid, Anterior, Scapular Division (456)
G 31 Rectus Abdominis, Fourth Div., Medialis (706)
G 31 Spinalis Thoracis, Lumbar Division (610)
G 32 Iliacus Minor (712)
G 32 Peroneus Brevis, Fibular Division (890)
G 32 Popliteus (870)
G 33 Quadratus Plantae, Medial Division (942)
G 33 Thyrohyoid (304)
G 34 Levator Costorum, Inferior Division (642)
G 34 Tensor Fascia Lata, Anterior Division (790)
G 35 Abductor Pollicis Brevis (542)
G 37 Psoas Major, Lumbar Division (722)
G 38 Masseter, Deep Division (118)
G 38 Rectus Medialis Bulbi (134)
G 39 Masseter, Superficial Division (114)
G 39 Triceps, Lateral Head(472)
G 40 Flexor Digitorum Superficialis, Medial Division (556)
G 40 Obliquus Capitis Superior (346)
G 41 Iliococcygeus (746)
G 42 Quadratus Plantae, Lateral Division (944)
G 43 Abductor Digitus Pedis, Fourth(970)
G 43 Deltoid, Anterior, Clavicular Division (458)
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Gv 22 Trapezius, Middle Division (390)
Gv 23 Lumbricales Manus, First (578)
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H 2 Flexor Carpi Radialis, Flexor Division (504)
H 2 Peroneus Longus, Cuneiform Division (886)
H 3 Tibialis Posterior, Fibular Division (884)
H 4 Abductor Digiti Minimi Manus, Flexor Division (582)
H 4 Transverse Abdominis, Lower Division (718)
H 5 Longissimus Thoracis, Superior Division (614)
H 6 Extensor Digitorum Longus, Medial Division (912)
H 6 Opponens Pollicis, Flexor Division (538)
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H 7 Orbicularis Oculi, Inferior Division (062)
H 7 Subclavius, Scapular Division (412)
H 8 Adductor Digitus Pedis, Fifth (964)
H 8 Flexor Hallucis Brevis, Tendonal Division (926)

H 8 Geniohyoid (298)
H 9 Flexor Hallucis Longus, Tibial Division (900)

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K 1 Interossei Palmaris, Third (592)
K 2 Depressor Septi (070)
K 2 Extensor Pollicis Brevis, Radial Division (524)
K 3 Supinator, Radial Division (492)
K 4 Vastus Lateralis, Middle Division (832)
K 5 Vastus Medialis, Upper Division (816)
K 6 Extensor Carpi Radialis Longus, Abductor Division (518)
K 7 Cremaster (752)
K 8 Lumbricales Manus, Fourth Division (572)
K 9 Flexor Pollicis Longus (528)
K 9 Semispinalis Capitis (332)
K 10 Adductor Hallucis, Superior Division (920)
K 11 Teres Major, Superior Division (432)
K 17 Digastric, Posterior Belly (292)
K 24 Extensor Carpi Ulnaris, Adductor Division (510)
K 25 Digastric, Anterior Belly (290)
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Li 1 Gluteus Minimus, Posterior Division (788)
Li 2 Triceps, Medial Head(474)
Li 3 Adductor Magnus, Oblique Division (844)
Li 4 Flexor Digitus Pedis, Third (952)
Li 4 Teres Minor (430)
Li 5 Flexor Digitorum Brevis, Lateral Division (948)
Li 5 Obturator Internus (822)
Li 6 Gastrocnemius, Lateral Division (874)
Li 6 Pronator Quadratus, Proximal Division (496)
Li 7 Mylohyoid (296)
Li 7 Peroneus Longus, Metatarsal Division (888)
Li 7 Vastus Medialis, Middle Division (818)
Li 8 Flexor Digitorum Superficialis, Lateral Division (558)
Li 8 Sternohyoid (300)
Li 8 Tibialis Posterior, Tibial Division (882)
Li 8 Vastus Lateralis, Superior Division (830)
Li 9 Anconeus, Olecranon Division (488)
Li 9 Rectus Abdominis, Fourth Div., Lateralis (708)
Li 10 Coccygeus, Coccyx Division (742)
Li 11 Biceps Femoris, Longhead, Tibial Division (828)
Li 13 Extensor Indicis Proprius (554)
Li 13 Sternothyroid (302)
Li 13 Subclavius, Clavicular Division (410)
Li 14 Levator Scapula, Inferior Division (396)
Li 14 Obliquus Externus Abdominis, Anterior Division (692)
Li 14 Rectus Abdominis, Second Division (702)
Li 15 Flexor Digitorum Profundus Manus, Medial Division (560)
Li 15 Levator Scapula, Superior Division (394)
Li 15 Multifidus, Lumbosacral Division (734)
Li 17 Gemellus Inferior (862)
Li 19 Buccinator (102)
Li 20 Adductor Digitus Pedis, Fourth(962)

LUNG MERIDIAN

Lu 1 Tensor Fascia Lata, Posterior Division (792)
Lu 2 Adductor Hallucis, Inferior Division (922)
Lu 3 Extensor Pollicis Longus, Ulnar Division (520)
Lu 4 Obliquus Inferior Bulbi (140)
Lu 4 Tibialis Anterior, Dorsiflexor Division (898)
Lu 5 Biceps Femoris, Longhead, Fibular Division (826)
Lu 5 Latissimus Dorsi, Lumbar Division (416)
Lu 6 Infraspinatus, Inferior Division (428)
Lu 6 Rectus Femoris, Straight Head (796)
Lu 7 Serratus Posterior, Inferior Division (652)
Lu 8 Deltoid, Posterior, Medial Division (448)
Lu 8 Interossei Pollicis (Palmaris First)(536)
Lu 9 Abductor Pollicis Longus, Ulnar Division (530)
Lu 10 Biceps Femoris, Shorthead(824)
Lu 10 Iliacus (710)
Lu 11 Flexor Carpi Ulnaris, Adductor Division (508)
Lu 11 Gluteus Medius, Anterior Division (784)
Lu 11 Quadratus Femoris (814)

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Lv 1 Extensor Hallucis Longus, Interosseous Division (904)
Lv 2 Abductor Digitus Pedis, Fifth (972)
Lv 3 Deltoid, Middle, Posterior Division (452)
Lv 3 Lumbricales Manus, Third (574)
Lv 4 Obliquus Externus Abdominis, Lateral Division (694)
Lv 4 Psoas Major, Thoracic Division (724)
Lv 4 Pterygoid Internal Medialis, Palatine Division (122)
Lv 4 Serratus Posterior, Superior Division (648)
Lv 5 Coccygeus, Sacral Division (740)
Lv 6 Deltoid, Posterior, Lateral Division (450)
Lv 6 Extensor Pollicis Longus, Septal Division (522)
Lv 6 Flexor Digitus Pedis, Fifth (956)
Lv 6 Sternocleidomastoid, Clavicular Division (276)
Lv 7 Splenius Cervicis (326)
Lv 8 Rhomboid Minor (398)
Lv 9 Flexor Digitus Pedis, Fourth (954)
Lv 9 Interossei Dorsales Manus, First (570)
Lv 10 Coracobrachialis, Coracoid Division (444)
Lv 11 Articularis Cubiti (476)

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Si 1 Flexor Digitorum Profundus Manus, Lateral Division (562)
Si 2 Gemellus Superior (864)
Si 3 Levator Palpebrae, Superior Division (058)
Si 4 Abductor Hallucis Transverse Head, Lateral Division (938)
Si 5 Pterygoid Internal Medialis, Sphenoid Division (120)
Si 5 Transverse Abdominis, Upper Division (714)
Si 6 Adductor Longus, Inferior Division (804)
Si 7 Abductor Digiti Minimi Manus, Abductor Division (584)
Si 7 Adductor Digitus Pedis, Third (960)
Si 7 Palmaris Longus (500)
Si 8 Extensor Hallucis Longus, Fibular Division (906)
Si 8 Interossei Dorsales Manus, Second (568)

Si 8 Obliquus Internus Abdominis, Anterior Division (696)
Si 9 Iliocostalis Lumborum (736)
Si 9 Sartorius (810)
Si 11 Flexor Digitorum Longus, Medial Division (908)
Si 12 Soleus, Lateral Division (880)
Si 14 Rectus Inferior Bulbi (132)

SPLEEN MERIDIAN

Sp 1 Vastus Intermedius, Lateral Division (838)
Sp 3 Flexor Hallucis Brevis, Third Cuneiform Division (928)
Sp 3 Gluteus Minimus, Anterior Division (786)
Sp 4 Pectoralis Major, Sternal Division(462)
Sp 4 Thyroarytenoideus (370)
Sp 5 Adductor Pollicis Transversus (544)
Sp 5 Rectus Lateralis Bulbi (136)
Sp 6 Tibialis Anterior, Supinator Division (896)
Sp 8 Piriformis (860)
Sp 8 Quadratus Lumborum, Lumbar Division (732)
Sp 8 Semimembranosus, Tibial Division (856)
Sp 9 Biceps Brachii Longhead(466)
Sp 9 Flexor Carpi Ulnaris, Flexor Division (506)
Sp 10 Scalenus Anterior (278)
Sp 11 Pyramidalis (690)
Sp 12 Vastus Medialis, Lower Division (820)
Sp 13 Interossei Palmaris, Second (594)

STOMACH MERIDIAN

St 10 Upper Trapezius, Clavicular Division (272)
St 14 Pronator Teres, Ulnar Division (486)
St 18 Opponens Digiti Minimi Manus, Abductor Division (586)
St 18 Pronator Quadratus, Distal Division(498)
St 24 Extensor Pollicis Brevis, Septal Division (526)
St 28 Pectoralis Minor, Inferior Division (408)
St 29 Psoas Minor (728)
St 32 Brachioradialis, Humeral Division(480)
St 32 Gracilis (808)
St 32 Longissimus Thoracis, Inferior Division (618)
St 33 Abductor Digiti Minimi Pedis (974)
St 33 Serratus Anterior, Superior Division (402)
St 36 Rhomboid Major (400)
St 37 Extensor Digiti Minimi Manus (552)
St 37 Interspinalis (Cervical) (350)
St 38 Pectineus (798)
St 38 Pterygoid External Lateralis, Upper Div.-Disc (124)
St 38 Quadratus Lumborum, Costal Division (730)
St 39 Gluteus Medius, Posterior Division (780)
St 39 Interossei Dorsales Manus, Third (566)
St 40 Anconeus, Ulnar Division (490)
St 40 Teres Major, Inferior Division (434)
St 41 Gluteus Maximus, Iliac Division (848)
St 41 Pterygoid External Lateralis, Lower Division (126)
L St 41 Adductor Brevis (Left)(800)
R St 41 Adductor Brevis (Right)(800)
St 43 Extensor Carpi Radialis Brevis (514)
St 43 Supraspinatus, Spine Division (420)

St 44 Cricoarytenoideus Lateralis (362)
St 45 Platysma, Posterior Division (288)

TRIPLE WARMER MERIDIAN

Tw 1 Longissimus Lumborum (738)
Tw 2 Constrictor Pharyngeus Medius (380)
Tw 3 Latissimus Dorsi, Iliac Division (418)
Tw 3 Longus Capitis (314)
Tw 3 Subscapularis, Third Division (438)
Tw 4 Abductor Pollicis Longus, Radial Division (532)
Tw 4 Orbicularis Oculi, Superior Division (060)
Tw 4 Vastus Lateralis, Lower Division (834)
Tw 5 Infraspinatus, Middle Division (426)
Tw 5 Subscapularis, Inferior Division (442)
Tw 6 Serratus Anterior, Inferior Division (404)
Tw 6 Supinator, Ulnar Division (494)
Tw 7 Rectus Femoris, Reflected Head (794)
Tw 8 Biceps Brachii Shorthead (468)
Tw 9 Brachialis (478)
Tw 9 Extensor Digitorum Communis Manus, Lateral Division (550)
Tw 9 Peroneus Tertius (894)
Tw 9 Subscapularis, Superior Division (436)
Tw 10 Flexor Hallucis Brevis, Cuboid Division (930)
Tw 10 Temporalis, Occipital Division (112)
Tw 10 Trapezius, Lower Division (392)
Tw 11 Abductor Digitus Pedis, Third (968)
Tw 11 Gastrocnemius, Medial Division (872)
Tw 12 Abductor Hallucis Transverse Head, Medial Division (936)
Tw 12 Rectus Abdominis, Third Division (704)
Tw 12 Scalenus Posterior (284)
Tw 13 Obturator Externus (812)
Tw 13 Plantaris (876)
Tw 14 Brachioradialis, Septal Division (482)
Tw 14 Interossei Palmaris, Fourth (590)
Tw 14 Vastus Intermedius, Medial Division (836)
Tw 15 Adductor Pollicis Obliquus (546)
Tw 15 Latissimus Dorsi, Thoracic Division (414)
Tw 16 Semispinalis Cervicis (334)
Tw 16 Splenius Capitis, Mastoid Division (322)
Tw 17 Lumbricales Manus, Second (576)

XA MERIDIAN

XA 3 Occipitalis (052)

XL MERIDIAN

XL 2 Adductor Magnus, Vertical Division (842)
XL 3 Longus Coli, Vertical Division (308)

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LG 30/RSp 21 Diaphragm, Left Lumbar Division (662)
RG 30/LSp 21 Diaphragm, Right Lumbar Division (656)

CRANIAL INDEX/NUMERICAL

(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals of Dr. Beardall.)

(052) Styloid	Occipitalis
(058) Occiput, Lateral	Levator Palpebrae, Superior Division
(060) Mandible	Orbicularis Oculi, Superior Division
(062) Occiput, Universal	Orbicularis Oculi, Inferior Division
(070) Frontal, External	Depressor Septi
(098) Parietal, Bulge	Orbicularis Oris, Upper Division
(100) Sphenoid	Orbicularis Oris, Inferior Division
(102) Frontal, External	Buccinator
(110) Parietal, Bulge	Temporalis, Parietal Division
(112) Vomer	Temporalis, Occipital Division
(114) Mandible	Masseter, Superficial Division
(118) Vomer	Masseter, Deep Division
(120) Lacrimal	Pterygoid Internal Medialis Sphenoid Division
(122) Occiput, Universal	Pterygoid Internal Medialis, Palatine Division
(124) Occiput, Universal	Pterygoid External Lateralis, Upper Div.-Disc
(126) Palatine	Pterygoid External Lateralis, Lower Division
(130) Lacrimal	Rectus Superior Bulbi
(132) Temporal, External	Rectus Inferior Bulbi
(134) TMJ A-P	Rectus Medialis Bulbi
(136) Frontal, Internal	Rectus Lateralis Bulbi
(138) Mandible	Obliquus Superior Bulbi
(140) Parietal, Bulge	Obliquus Inferior Bulbi
(270) Occiput, Lateral	Upper Trapezius, Scapular Division
(272) Maxillary, M-L	Upper Trapezius, Clavicular Division
(274) Occiput, Lateral	Sternocleidomastoid, Sternal Division
(276) Glabella	Sternocleidomastoid, Clavicular Division
(278) Parietal, Bulge	Scalenus Anterior
(282) Occiput, Universal	Scalenus Medius
(284) Ethmoid	Scalenus Posterior
(286) Glabella	Platysma, Anterior Division
(288) Nasal	Platysma, Posterior Division
(290) Glabella	Digastric, Anterior Belly
(292) Ethmoid	Digastric, Posterior Belly
(294) Styloid	Stylohyoid
(296) Frontal, External	Mylohyoid
(298) Vomer	Geniohyoid
(300) Temporal, Internal	Sternohyoid
(302) TMJ A-P	Sternothyroid
(304) Sphenoid	Thyrohyoid
(306) Maxillary A-P	Omohyoid
(308) Vomer	Longus Coli, Vertical Division
(314) Temporal, Internal	Longus Capitis
(322) Temporal, External	Splenius Capitis, Mastoid Division
(326) Occiput, Lateral	Splenius Cervicis
(332) Temporal, External	Semispinalis Capitis
(334) Mandible	Semispinalis Cervicis
(346) Occiput, Lateral	Obliquus Capitis Superior Division
(350) Parietal, Bulge	Interspinalis (Cervical)
(362) Palatine	Cricoarytenoideus Lateralis
(370) Temporal, Internal	Thyroarytenoideus
(380) TMJ M-L	Constrictor Pharyngeus Medius
(390) Frontal, Internal	Trapezius, Middle Division
(392) Parietal, Bulge	Trapezius, Lower Division

(394) Occiput, Lateral	Levator Scapula, Superior Division
(396) Temporal, External	Levator Scapula, Inferior Division
(398) Frontal, Internal	Rhomboid Minor
(400) Vomer	Rhomboid Major
(402) Parietal, Bulge	Serratus Anterior, Superior Division
(404) Maxillary M-L	Serratus Anterior, Inferior Division
(406) TMJ A-P	Pectoralis Minor, Superior Division
(408) Temporal, External	Pectoralis Minor, Inferior Division
(410) Temporal, Internal	Subclavius, Clavicular Division
(412) Sphenoid	Subclavius, Scapular Division
(414) Sphenoid	Latissimus Dorsi, Thoracic Division
(416) Sphenoid	Latissimus Dorsi, Lumbar Division
(418) TMJ A-P	Latissimus Dorsi, Iliac Division
(420) Mandible	Supraspinatus, Spine Division
(422) Ethmoid	Supraspinatus, Fossa Division
(424) Nasal	Infraspinatus, Superior Division
(426) TMJ A-P	Infraspinatus, Middle Division
(428) Frontal, External	Infraspinatus, Inferior Division
(430) Nasal	Teres Minor
(432) Occiput, Lateral	Teres Major, Superior Division
(434) Frontal, Internal	Teres Major, Inferior Division
(436) Maxillary M-L	Subscapularis, Superior Division
(438) Temporal, Internal	Subscapularis, Third Division
(440) Palatine	Subscapularis, Second Division
(442) Maxillary M-L	Subscapularis, Inferior Division
(444) Styloid	Coracobrachialis, Coracoid Division
(446) Vomer	Coracobrachialis, Septal Division
(448) Nasal	Deltoid, Posterior, Medial Division
(450) Maxillary A-P	Deltoid, Posterior, Lateral Division
(452) Sphenoid	Deltoid, Middle, Posterior Division
(454) Sphenoid	Deltoid, Middle, Anterior Division
(456) Sphenoid	Deltoid, Anterior, Scapular Division
(458) Sphenoid	Deltoid, Anterior, Clavicular Division
(460) Parietal, Bulge	Pectoralis Major, Clavicular Division
(462) Frontal, External	Pectoralis Major, Sternal Division
(464) Temporal, External	Pectoralis Major, Costal Division
(466) Styloid	Biceps Brachii Longhead
(468) TMJ A-P	Biceps Brachii Shorthead
(470) Lacrimal	Triceps, Longhead
(472) TMJ M-L	Triceps, Lateral Head
(474) Mandible	Triceps, Medial Head
(476) Palatine	Articularis Cubiti
(478) Temporal, Internal	Brachialis
(480) Maxillary A-P	Brachioradialis, Humeral Division
(482) Temporal, External	Brachioradialis, Septal Division
(484) Temporal, Internal	Pronator Teres, Humeral Division
(486) Maxillary M-L	Pronator Teres, Ulnar Division
(488) Styloid	Anconeus, Olecranon Division
(490) Sphenoid	Anconeus, Ulnar Division
(492) Frontal, External	Supinator, Radial Division
(494) TMJ A-P	Supinator, Ulnar Division
(496) Frontal, Internal	Pronator Quadratus, Proximal Division
(498) Frontal, Internal	Pronator Quadratus, Distal Division
(500) Parietal, Bulge	Palmaris Longus
(502) Mandible	Flexor Carpi Radialis, Abductor Division
(504) Maxillary M-L	Flexor Carpi Radialis, Flexor Division
(506) Sphenoid	Flexor Carpi Ulnaris, Flexor Division

(508) Temporal, Internal	Flexor Carpi Ulnaris, Adductor Division
(510) Mandible	Extensor Carpi Ulnaris, Adductor Division
(512) Temporal, Internal	Extensor Carpi Ulnaris, Extensor Division
(514) Maxillary M-L	Extensor Carpi Radialis Brevis
(516) Parietal Descent	Extensor Carpi Radialis Longus, Ext Division
(518) Temporal, External	Extensor Carpi Radialis Longus, Abductor Division
(520) Maxillary A-P	Extensor Pollicis Longus, Ulnar Division
(522) Sphenoid	Extensor Pollicis Longus, Septal Division
(524) Vomer	Extensor Pollicis Brevis, Radial Division
(526) Parietal, Bulge	Extensor Pollicis Brevis, Septal Division
(528) Occiput, Universal	Flexor Pollicis Longus
(530) Maxillary (M-L)	Abductor Pollicis Longus, Ulnar Division
(532) Vomer	Abductor Pollicis Longus, Radial Division
(534) Frontal, External	Flexor Pollicis Brevis, Superior Div
(536) Parietal, Bulge	Interossei Pollicis (Palmaris First)
(538) Palatine	Opponens Pollicis, Flexor Division
(540) Occiput, Universal	Opponens Pollicis, Abductor Division
(542) Parietal, Bulge	Abductor Pollicis Brevis
(544) Ethmoid	Adductor Pollicis Transversus
(546) Sphenoid	Adductor Pollicis Obliquus
(548) Glabella	Extensor Digitorum Communis Manus, Medial Division
(550) Parietal, Bulge	Extensor Digitorum Communis Manus, Lateral Division
(552) Lacrimal	Extensor Digiti Minimi Manus
(554) Temporal, External	Extensor Indicis Proprius
(556) Vomer	Flexor Digitorum Superficialis, Medial Division
(558) Glabella	Flexor Digitorum Superficialis, Lateral Division
(560) Palatine	Flexor Digitorum Profundus Manus, Medial Division
(562) Styloid	Flexor Digitorum Profundus Manus, Lateral Division
(564) Frontal, External	Interossei Dorsales Manus, Fourth
(566) Maxillary M-L	Interossei Dorsales Manus, Third
(568) Maxillary M-L	Interossei Dorsales Manus, Second
(570) Sphenoid	Interossei Dorsales Manus, First
(572) Ethmoid	Lumbricales Manus, Fourth Division
(574) Frontal, External	Lumbricales Manus, Third
(576) TMJ A-P	Lumbricales Manus, Second
(578) TMJ A-P	Lumbricales Manus, First
(580) Ethmoid	Flexor Digiti Minimi Brevis, Manus
(582) Ethmoid	Abductor Digiti Minimi Manus, Flexor Division
(584) Inferior Conchae	Abductor Digiti Minimi Manus, Abductor Division
(586) Inferior Conchae	Opponens Digiti Minimi Manus, Abductor Division
(588) Parietal, Bulge	Opponens Digiti Minimi Manus, Flexor Division
(590) Occiput, Lateral	Interossei Palmaris, Fourth
(592) Occiput, Lateral	Interossei Palmaris, Third
(594) Vomer	Interossei Palmaris, Second
(596) Parietal, Bulge	Palmaris Brevis
(610) Mandible	Spinalis Thoracis, Lumbar Division
(612) TMJ A-P	Spinalis Thoracis, Thoracic Division
(614) Temporal, Internal	Longissimus Thoracis, Superior Division
(618) Ethmoid	Longissimus Thoracis, Inferior Division
(642) Vomer	Levator Costorum, Inferior Division
(648) Frontal, Internal	Serratus Posterior, Superior Division
(652) Frontal, External	Serratus Posterior, Inferior Division
(656) Maxillary, M-L	Diaphragm, Right Lumbar Division
(662) Maxillary, M-L	Diaphragm, Left Lumbar Division
(690) Vomer	Pyramidalis
(692) Occiput, Universal	Obliquus Externus Abdominis, Anterior Division
(694) Parietal, Bulge	Obliquus Externus Abdominis, Lateral Division

(696) Ethmoid	Obliquus Internus Abdominis, Anterior Division
(698) Frontal, Internal	Obliquus Internus Abdominis, Lateral Division
(700) Temporal, Internal	Rectus Abdominis, First Division
(702) Sphenoid	Rectus Abdominis, Second Division
(704) Sphenoid	Rectus Abdominis, Third Division
(706) Frontal, Internal	Rectus Abdominis, Fourth Div., Medialis
(708) Sphenoid	Rectus Abdominis, Fourth Div., Lateralis
(710) Occiput, Universal	Iliacus
(712) Frontal, Internal	Iliacus Minor
(714) Parietal, Descent	Transverse Abdominis, Upper Division
(718) Parietal, Bulge	Transverse Abdominis, Lower Division
(722) Occiput, Lateral	Psoas Major, Lumbar Division
(724) Occiput, Universal	Psoas Major, Thoracic Division
(726) Maxillary M-L	Psoas Major, Diaphragmatic Division
(728) Maxillary M-L	Psoas Minor
(730) Vomer	Quadratus Lumborum, Costal Division
(732) Palatine	Quadratus Lumborum, Lumbar Division
(734) Styloid	Multifidus, Lumbosacral Division
(736) Palatine	Iliocostalis Lumborum
(738) Maxillary M-L	Longissimus Lumborum
(740) Palatine	Coccygeus, Sacral Division
(742) Maxillary M-L	Coccygeus, Coccyx Division
(744) Occiput, Universal	Pubococcygeus
(746) Lacrimal	Iliococcygeus
(752) Occiput, Universal	Cremaster
(780) Maxillary M-L	Gluteus Medius, Posterior Division
(782) Parietal Descent	Gluteus Medius, Middle Division
(784) Occiput, Lateral	Gluteus Medius, Anterior Division
(786) Temporal, Internal	Gluteus Minimus, Anterior Division
(788) Maxillary M-L	Gluteus Minimus, Posterior Division
(790) Parietal, Bulge	Tensor Fascia Lata, Anterior Division
(792) Parietal, Bulge	Tensor Fascia Lata, Posterior Division
(794) Occiput, Lateral	Rectus Femoris, Reflected Head
(796) TMJ M-L	Rectus Femoris, Straight Head
(798) Maxillary M-L	Pectineus
(800L) Ethmoid	Adductor Brevis (Left)
(800R) Ethmoid	Adductor Brevis (Right)
(804) Parietal Descent	Adductor Longus, Inferior Division
(806) Maxillary A-P	Adductor Longus, Superior Division
(808) Frontal, External	Gracilis
(810) Frontal, External	Sartorius
(812) Temporal, Internal	Obturator Externus
(814) Styloid	Quadratus Femoris
(816) Glabella	Vastus Medialis, Upper Division
(818) Glabella	Vastus Medialis, Middle Division
(820) Styloid	Vastus Medialis, Lower Division
(822) Vomer	Obturator Internus
(824) Zygoma	Biceps Femoris, Shorthead
(826) Occiput, Universal	Biceps Femoris, Longhead, Fibular Division
(828) Temporal, External	Biceps Femoris, Longhead, Tibial Division
(830) Parietal, Bulge	Vastus Lateralis, Superior Division
(832) TMJ A-P	Vastus Lateralis, Middle Division
(834) Parietal, Bulge	Vastus Lateralis, Lower Division
(836) Lacrimal	Vastus Intermedius, Medial Division
(838) Parietal Descent	Vastus Intermedius, Lateral Division
(840) Occiput, Lateral	Articularis Genu
(842) Vomer	Adductor Magnus, Vertical Division

(844) TMJ A-P	Adductor Magnus, Oblique Division
(846) Occiput, Universal	Adductor Magnus, Transverse Division
(848) Inferior Conchae	Gluteus Maximus, Iliac Division
(850) Nasal	Gluteus Maximus, Sacral Division
(852) Palatine	Gluteus Maximus, Coccygeal Division
(854) Frontal, External	Semitendinosus
(856) Sphenoid	Semimembranosus, Tibial Division
(858) TMJ M-L	Semimembranosus, Popliteal Division
(860) Occiput, Lateral	Piriformis
(862) Occiput, Lateral	Gemellus Inferior
(864) Zygoma	Gemellus Superior
(870) Temporal, Internal	Popliteus
(872) Occiput, Universal	Gastrocnemius, Medial Division
(874) Occiput, Lateral	Gastrocnemius, Lateral Division
(876) Zygoma	Plantaris
(878) Frontal, External	Soleus Medial Division
(880) Frontal, External	Soleus, Lateral Division
(882) Parietal, Bulge	Tibialis Posterior, Tibial Division
(884) Sphenoid	Tibialis Posterior, Fibular Division
(886) Ethmoid	Peroneus Longus, Cuneiform Division
(888) Parietal, Bulge	Peroneus Longus, Metatarsal Division
(890) Parietal, Bulge	Peroneus Brevis, Fibular Division
(892) Temporal, Internal	Peroneus Brevis, Septal Division
(894) Temporal, Internal	Peroneus Tertius
(896) Frontal, External	Tibialis Anterior, Supinator Division
(898) Frontal, External	Tibialis Anterior, Dorsiflexor Division
(900) Vomer	Flexor Hallucis Longus, Tibial Division
(902) Inferior Conchae	Flexor Hallucis Longus, Fibular Division
(904) Mandible	Extensor Hallucis Longus, Interosseous Division
(906) Palatine	Extensor Hallucis Longus, Fibular Division
(908) Maxillary M-L	Flexor Digitorum Longus, Medial Division
(910) Parietal, Bulge	Flexor Digitorum Longus, Lateral Division
(912) Vomer	Extensor Digitorum Longus, Medial Division
(914) Occiput, Universal	Extensor Digitorum Longus, Lateral Division
(920) Ethmoid	Adductor Hallucis, Superior Division
(922) Frontal, Internal	Adductor Hallucis, Inferior Division
(924) Lacrimal	Flexor Hallucis Brevis, First Cuneiform Division
(926) Frontal, External	Flexor Hallucis Brevis, Tendonal Division
(928) TMJ A-P	Flexor Hallucis Brevis, Third Cuneiform Division
(930) Palatine	Flexor Hallucis Brevis, Cuboid Division
(932) Nasal	Abductor Hallucis Oblique Head, Peroneus Division
(934) Occiput, Lateral	Abductor Hallucis Oblique Head, Metatarsal Division
(936) Mandible	Abductor Hallucis Transverse Head, Medial Division
(938) Styloid	Abductor Hallucis Transverse Head, Lateral Division
(940) Temporal, External	Extensor Hallucis Brevis
(942) Styloid	Quadratus Plantae, Medial Division
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(946) Ethmoid	Flexor Digitorum Brevis, Medial Division
(948) Sphenoid	Flexor Digitorum Brevis, Lateral Division
(950) Inferior Conchae	Flexor Digitus Pedis, Second
(952) Nasal	Flexor Digitus Pedis, Third
(954) Ethmoid	Flexor Digitus Pedis, Fourth
(956) Mandible	Flexor Digitus Pedis, Fifth
(958) Occiput, Universal	Adductor Digitus Pedis, Second
(960) TMJ M-L	Adductor Digitus Pedis, Third
(962) Styloid	Adductor Digitus Pedis, Fourth
(964) Temporal, External	Adductor Digitus Pedis, Fifth

(966) Vomer	Abductor Digitus Pedis, Second
(968) Mandible	Abductor Digitus Pedis, Third
(970) Lacrimal	Abductor Digitus Pedis, Fourth
(972) Maxillary A-P	Abductor Digitus Pedis, Fifth
(974) Occiput, Lateral	Abductor Digiti Minimi Pedis
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(482) Cuboid, Lateral	Brachioradialis, Septal Division
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(810) Navicular	Sartorius
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(814) Distal Phalanx Great Toe	Quadratus Femoris
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(818) Fourth Metatarsal	Vastus Medialis, Middle Division
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Orbicularis Oris, Inferior Division (100)
Rectus Abdominis, Fourth Div., Lateralis (708)
Rectus Abdominis, Second Division (702)
Rectus Abdominis, Third Division (704)
Semimembranosus, Tibial Division (856)
Subclavius, Scapular Division (412)
Thyrohyoid (304)
Tibialis Posterior, Fibular Division (884)

THIRD CUNEIFORM, MEDIAL

Adductor Magnus, Oblique Division (844)
Biceps Brachii Shorthead (468)
Flexor Hallucis Brevis, Third Cuneiform Division (928)
Infraspinatus, Middle Division (426)
Latissimus Dorsi, Iliac Division (418)
Lumbricales Manus, First (578)
Lumbricales Manus, Second (576)
Pectoralis Minor, Superior Division (406)
Rectus Medialis Bulbi (134)
Spinalis Thoracis, Thoracic Division (612)
Sternothyroid (302)
Supinator, Ulnar Division (494)
Vastus Lateralis, Middle Division (832)

THIRD METATARSAL

Abductor Pollicis Longus, Ulnar Division (530)
Coccygeus, Coccyx Division (742)
Diaphragm, Left Lumbar Division (662)
Diaphragm, Right Lumbar Division (656)
Extensor Carpi Radialis Brevis (514)
Flexor Carpi Radialis, Flexor Division (504)
Flexor Digitorum Longus, Medial Division (908)
Gluteus Medius, Posterior Division (780)
Gluteus Minimus, Posterior Division (788)
Interossei Dorsales Manus, Second (568)
Interossei Dorsales Manus, Third (566)

Longissimus Lumborum (738)
Pectineus (798)
Pronator Teres, Ulnar Division (486)
Psoas Major, Diaphragmatic Division (726)
Psoas Minor (728)
Serratus Anterior, Inferior Division (404)
Subscapularis, Inferior Division (442)
Subscapularis, Superior Division (436)
Upper Trapezius, Clavicular Division (272)

ORGAN/TISSUE INDEX/NUMERICAL #1

(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals of Dr. Beardall.)

(052) Gallbladder Duct
(058) Colon (Transverse)
(060) Thyroid
(062) Thymus
(070) Ovary/Testicle
(098) Liver
(100) Anterior Pituitary
(102) Ovary/Testicle
(110) Pancreas (Sugar)
(112) Pineal
(114) Adrenal
(118) Posterior Pituitary
(120) Thyroid
(122) Thymus
(124) Pineal
(126) Ovary/Testicle
(130) Kidney
(132) Stomach (Special Cells)
(134) Liver
(136) Pancreas (Sugar)
(138) Liver
(140) Liver
(270) Colon (Ascending/Descending)
(272) Thymus
(274) Pancreas (Sugar)
(276) Lymphatics of Submandibular
(278) Ovary/Testicle
(282) Lymphatics of Submandibular
(284) Kidney
(286) Pancreas (Sugar)
(288) Lymphatics of Rectum
(290) Pancreatic Duct
(292) Gallbladder (Arteries)
(294) Thyroid
(296) Bladder
(298) Thyroid
(300) Gallbladder Duct
(302) Kidney
(304) Thymus
(306) Spleen
(308) Ovary/Testicle
(314) Anterior Pituitary
(322) Anterior Pituitary
(326) Adrenal
(332) Spleen
(334) Salivary Gland (Sublingual)
(346) Spleen
(350) Colon (Ascending/Descending)
(362) Pancreatic Duct
(370) Uterus-Prostate (Digestive Portion)
(380) Liver
(390) Posterior Pituitary
(392) Bladder

Occipitalis
Levator Palpebrae, Superior Division
Orbicularis Oculi, Superior Division
Orbicularis Oculi, Inferior Division
Depressor Septi
Orbicularis Oris, Upper Division
Orbicularis Oris, Inferior Division
Buccinator
Temporalis, Parietal Division
Temporalis, Occipital Division
Masseter, Superficial Division
Masseter, Deep Division
Pterygoid Internal Medialis, Sphenoid Division
Pterygoid Internal Medialis, Palatine Division
Pterygoid External Lateralis, Upper Div.-Disc
Pterygoid External Lateralis, Lower Division
Rectus Superior Bulbi
Rectus Inferior Bulbi
Rectus Medialis Bulbi
Rectus Lateralis Bulbi
Obliquus Superior Bulbi
Obliquus Inferior Bulbi
Upper Trapezius, Scapular Division
Upper Trapezius, Clavicular Division
Sternocleidomastoid, Sternal Division
Sternocleidomastoid, Clavicular Division
Scalenus Anterior
Scalenus Medius
Scalenus Posterior
Platysma, Anterior Division
Platysma, Posterior Division
Digastric, Anterior Belly
Digastric, Posterior Belly
Stylohyoid
Mylohyoid
Geniohyoid
Sternohyoid
Sternothyroid
Thyrohyoid
Omohyoid
Longus Coli, Vertical Division
Longus Capitis
Splenius Capitis, Mastoid Division
Splenius Cervicis
Semispinalis Capitis
Semispinalis Cervicis
Obliquus Capitis Superior
Interspinalis (Cervical)
Cricoarytenoideus Lateralis
Thyroarytenoideus
Constrictor Pharyngeus Medius
Trapezius, Middle Division
Trapezius, Lower Division

(394) Sinus (Frontal)	Levator Scapula, Superior Division
(396) Parathyroid	Levator Scapula, Inferior Division
(398) Posterior Pituitary	Rhomboid Minor
(400) Liver	Rhomboid Major
(402) Lung	Serratus Anterior, Superior Division
(404) Thymus	Serratus Anterior, Inferior Division
(406) Lung	Pectoralis Minor, Superior Division
(408) Kidney	Pectoralis Minor, Inferior Division
(410) Ear (External)	Subclavius, Clavicular Division
(412) Posterior Pituitary	Subclavius, Scapular Division
(414) Lymphatics of Ileum	Latissimus Dorsi, Thoracic Division
(416) Parotid Gland	Latissimus Dorsi, Lumbar Division
(418) Urethra (Membranous Portion)	Latissimus Dorsi, Iliac Division
(420) Thymus	Supraspinatus, Spine Division
(422) Anterior Pituitary	Supraspinatus, Fossa Division
(424) Thyroid	Infraspinatus, Superior Division
(426) Eye	Infraspinatus, Middle Division
(428) Thymus	Infraspinatus, Inferior Division
(430) Thyroid	Teres Minor
(432) Pineal	Teres Major, Superior Division
(434) Vagina/Penis	Teres Major, Inferior Division
(436) Heart	Subscapularis, Superior Division
(438) Sinus (Sphenoid)	Subscapularis, Third Division
(440) Esophagus	Subscapularis, Second Division
(442) Pancreatic Duct	Subscapularis, Inferior Division
(444) Kidney	Coracobrachialis, Coracoid Division
(446) Bladder	Coracobrachialis, Septal Division
(448) Spleen	Deltoid, Posterior, Medial Division
(450) Eye	Deltoid, Posterior, Lateral Division
(452) Spleen	Deltoid, Middle, Posterior Division
(454) Lung	Deltoid, Middle, Anterior Division
(456) Lung	Deltoid, Anterior, Scapular Division
(458) Stomach	Deltoid, Anterior, Clavicular Division
(460) Uterus/Prostate	Pectoralis Major, Clavicular Division
(462) Mammary	Pectoralis Major, Sternal Division
(464) Anterior Pituitary	Pectoralis Major, Costal Division
(466) Kidney	Biceps Brachii Longhead
(468) Ovary/Testicle	Biceps Brachii Shorthead
(470) Duodenum (Superior Portion)	Triceps, Longhead
(472) Salivary Gland (Sublingual)	Triceps, Lateral Head
(474) Thyroid	Triceps, Medial Head
(476) Bladder	Articularis Cubiti
(478) Tonsil	Brachialis
(480) Ileum (Peyer's Patches)	Brachioradialis, Humeral Division
(482) Kidney	Brachioradialis, Septal Division
(484) Adrenal	Pronator Teres, Humeral Division
(486) Kidney	Pronator Teres, Ulnar Division
(488) Bladder	Anconeus, Olecranon Division
(490) Pancreatic Duct	Anconeus, Ulnar Division
(492) Ovary/Testicle	Supinator, Radial Division
(494) Uterus/Prostate	Supinator, Ulnar Division
(496) Stomach (Fundus)	Pronator Quadratus, Proximal Division
(498) Sinus (Maxillary)	Pronator Quadratus, Distal Division
(500) Kidney	Palmaris Longus
(502) Gallbladder	Flexor Carpi Radialis, Abductor Division
(504) Thyroid	Flexor Carpi Radialis, Flexor Division
(506) Ovary/Testicle	Flexor Carpi Ulnaris, Flexor Division

(508) Uterus/Prostate	Flexor Carpi Ulnaris, Adductor Division
(510) Colon (Appendix)	Extensor Carpi Ulnaris, Adductor Division
(512) Kidney	Extensor Carpi Ulnaris, Extensor Division
(514) Duodenum (Special Cells)	Extensor Carpi Radialis Brevis
(516) Stomach (Pyloric Antrum)	Extensor Carpi Radialis Longus, Ext Division
(518) Pancreatic Duct	Extensor Carpi Radialis Longus, Abductor Division
(520) Anterior Pituitary	Extensor Pollicis Longus, Ulnar Division
(522) Lymphatics of Thoracic Duct	Extensor Pollicis Longus, Septal Division
(524) Gallbladder Duct	Extensor Pollicis Brevis, Radial Division
(526) Adenoid	Extensor Pollicis Brevis, Septal Division
(528) Pancreatic Duct	Flexor Pollicis Longus
(530) Lymphatics of Face and Neck	Abductor Pollicis Longus, Ulnar Division
(532) Pancreas (Sugar)	Abductor Pollicis Longus, Radial Division
(534) Adrenal (Cortex)	Flexor Pollicis Brevis
(536) Bladder	Interossei Pollicis (Palmaris First)
(538) Posterior Pituitary	Opponens Pollicis, Flexor Division
(540) Pharynx	Opponens Pollicis, Abductor Division
(542) Pancreas (Sugar)	Abductor Pollicis Brevis
(544) Heart	Adductor Pollicis Transversus
(546) Anterior Pituitary	Adductor Pollicis Obliquus
(548) Ovary/Testicle	Extensor Digitorum Communis Manus, Medial Division
(550) Pancreas (Sugar)	Extensor Digitorum Communis Manus, Lateral Division
(552) Lung	Extensor Digiti Minimi Manus
(554) Pancreas (Sugar)	Extensor Indicis Proprius
(556) Pancreas (Sugar)	Flexor Digitorum Superficialis, Medial Division
(558) Tonsil	Flexor Digitorum Superficialis, Lateral Division
(560) Bladder	Flexor Digitorum Profundus Manus, Medial Division
(562) Jejunum	Flexor Digitorum Profundus Manus, Lateral Division
(564) Heart	Interossei Dorsales Manus, Fourth
(566) Pancreatic Duct	Interossei Dorsales Manus, Third
(568) Spleen	Interossei Dorsales Manus, Second
(570) Anterior Pituitary	Interossei Dorsales Manus, First
(572) Nose	Lumbricales Manus, Fourth Division
(574) Parathyroid	Lumbricales Manus, Third
(576) Parathyroid	Lumbricales Manus, Second
(578) Posterior Pituitary	Lumbricales Manus, First
(580) Pancreas (Sugar)	Flexor Digiti Minimi Brevis, Manus
(582) Lymphatics of Colon	Abductor Digiti Minimi Manus, Flexor Division
(584) Ileum (Special Cells)	Abductor Digiti Minimi Manus, Abductor Division
(586) Jejunum	Opponens Digiti Minimi Manus, Abductor Division
(588) Pancreas (Protein)	Opponens Digiti Minimi Manus, Flexor Division
(590) Spleen	Interossei Palmaris, Fourth
(592) Ovary/Testicle	Interossei Palmaris, Third
(594) Ileum (Special Cells)	Interossei Palmaris, Second
(596) Gallbladder	Palmaris Brevis
(610) Heart	Spinalis Thoracis, Lumbar Division
(612) Pineal	Spinalis Thoracis, Thoracic Division
(614) Stomach	Longissimus Thoracis, Superior Division
(618) Adrenal	Longissimus Thoracis, Inferior Division
(642) Gallbladder	Levator Costorum, Inferior Division
(648) Bladder	Serratus Posterior, Superior Division
(652) Nose	Serratus Posterior, Inferior Division
(656) Lymph. of Jej./Lymph.Leg Ing Nodes	Diaphragm, Right Lumbar Division
(662) Lymph.Spleen/Lymph.Leg Ing Nodes	Diaphragm, Left Lumbar Division
(690) Bladder	Pyramidalis
(692) Colon (Ascending/Descending)	Obliquus Externus Abdominis, Anterior Division
(694) Lymphatics of Colon	Obliquus Externus Abdominis, Lateral Division

(696) Colon (Hepatic & Splenic Flexure)	Obliquus Internus Abdominis, Anterior Division
(698) Tonsil	Obliquus Internus Abdominis, Lateral Division
(700) Adrenal	Rectus Abdominis, First Division
(702) Duodenum (Horizontal Portion)	Rectus Abdominis, Second Division
(704) Ileum	Rectus Abdominis, Third Division
(706) Duodenum (Ascending Portion)	Rectus Abdominis, Fourth Div., Medialis
(708) Vagina/Penis	Rectus Abdominis, Fourth Div., Lateralis
(710) Colon (Ascending/Descending)	Iliacus
(712) Pancreas (Sugar)	Iliacus Minor
(714) Salivary Gland (Sublingual)	Transverse Abdominis, Upper Division
(718) Thymus	Transverse Abdominis, Lower Division
(722) Kidney	Psoas Major, Lumbar Division
(724) Kidney	Psoas Major, Thoracic Division
(726) Adrenal	Psoas Major, Diaphragmatic Division
(728) Lymphatics of Stomach	Psoas Minor
(730) Liver	Quadratus Lumborum, Costal Division
(732) Gallbladder	Quadratus Lumborum, Lumbar Division
(734) Ovary/Testicle	Multifidus, Lumbosacral Division
(736) Lung	Iliocostalis Lumborum
(738) Jejunum	Longissimus Lumborum
(740) Ileum	Coccygeus, Sacral Division
(742) Stomach (Pyloric Canal))	Coccygeus, Coccyx Division
(744) Lung	Pubococcygeus
(746) Thyroid	Iliococcygeus
(752) Spleen	Cremaster
(780) Gallbladder Duct	Gluteus Medius, Posterior Division
(782) Uterus/Prostate	Gluteus Medius, Middle Division
(784) Lung	Gluteus Medius, Anterior Division
(786) Pancreas (Sugar)	Gluteus Minimus, Anterior Division
(788) Pancreatic Duct	Gluteus Minimus, Posterior Division
(790) Colon (Ascending/Descending)	Tensor Fascia Lata, Anterior Division
(792) Thyroid	Tensor Fascia Lata, Posterior Division
(794) Thyroid	Rectus Femoris, Reflected Head
(796) Jejunum	Rectus Femoris, Straight Head
(798) Colon (Transverse)	Pectineus
(800 L) Duodenum	Adductor Brevis (Left)
(800 R) Gallbladder Duct (Amp. of Vater)	Adductor Brevis (Right)
(804) Ileum	Adductor Longus, Inferior Division
(806) Liver	Adductor Longus, Superior Division
(808) Adrenal	Gracilis
(810) Adrenal	Sartorius
(812) Heart	Obturator Externus
(814) Ovary/Testicle	Quadratus Femoris
(816) Lung	Vastus Medialis, Upper Division
(818) Adrenal	Vastus Medialis, Middle Division
(820) Posterior Pituitary	Vastus Medialis, Lower Division
(822) Lung	Obturator Internus
(824) Colon (Ascending/Descending)	Biceps Femoris, Shorthead
(826) Colon (Ascending/Descending)	Biceps Femoris, Longhead, Fibular Division
(828) Uterus-Prostate (Digestive Portion)	Biceps Femoris, Longhead, Tibial Division
(830) Thyroid	Vastus Lateralis, Superior Division
(832) Pancreatic Duct	Vastus Lateralis, Middle Division
(834) Ileum	Vastus Lateralis, Lower Division
(836) Ileum	Vastus Intermedius, Medial Division
(838) Tonsil	Vastus Intermedius, Lateral Division
(840) Ovary/Testicle	Articularis Genu
(842) Uterus/Prostate (Broad Ligament)	Adductor Magnus, Vertical Division

(844) Anterior Pituitary	Adductor Magnus, Oblique Division
(846) Nose	Adductor Magnus, Transverse Division
(848) Pancreas (Sugar)	Gluteus Maximus, Iliac Division
(850) Posterior Pituitary	Gluteus Maximus, Sacral Division
(852) Bladder	Gluteus Maximus, Coccygeal Division
(854) Posterior Pituitary	Semitendinosus
(856) Lymphatics of Submandibular	Semimembranosus, Tibial Division
(858) Ileum (Peyer's Patches)	Semimembranosus, Popliteal Division
(860) Uterus/Prostate (Digestive Portion)	Piriformis
(862) Lung	Gemellus Inferior
(864) Heart	Gemellus Superior
(870) Vagina/Penis	Popliteus
(872) Duodenum	Gastrocnemius, Medial Division
(874) Colon (Ascending/Descending)	Gastrocnemius, Lateral Division
(876) Jejunum	Plantaris
(878) Adrenal	Soleus Medial Division
(880) Colon (Rectum)	Soleus, Lateral Division
(882) Ileum	Tibialis Posterior, Tibial Division
(884) Heart	Tibialis Posterior, Fibular Division
(886) Bladder	Peroneus Longus, Cuneiform Division
(888) Parathyroid	Peroneus Longus, Metatarsal Division
(890) Lung	Peroneus Brevis, Fibular Division
(892) Bladder	Peroneus Brevis, Septal Division
(894) Lung	Peroneus Tertius
(896) Jejunum	Tibialis Anterior, Supinator Division
(898) Spleen	Tibialis Anterior, Dorsiflexor Division
(900) Liver	Flexor Hallucis Longus, Tibial Division
(902) Esophagus, Abdominal Portion	Flexor Hallucis Longus, Fibular Division
(904) Thymus	Extensor Hallucis Longus, Interosseous Division
(906) Urethra	Extensor Hallucis Longus, Fibular Division
(908) Anterior Pituitary	Flexor Digitorum Longus, Medial Division
(910) Anterior Pituitary	Flexor Digitorum Longus, Lateral Division
(912) Uterus/Prostate	Extensor Digitorum Longus, Medial Division
(914) Thyroid	Extensor Digitorum Longus, Lateral Division
(920) Anterior Pituitary	Adductor Hallucis, Superior Division
(922) Heart	Adductor Hallucis, Inferior Division
(924) Pineal	Flexor Hallucis Brevis, First Cuneiform Division
(926) Tonsil	Flexor Hallucis Brevis, Tendonal Division
(928) Salivary Gland (Submandibular)	Flexor Hallucis Brevis, Third Cuneiform Division
(930) Jejunum	Flexor Hallucis Brevis, Cuboid Division
(932) Stomach	Abductor Hallucis Oblique Head, Peroneus Division
(934) Nose	Abductor Hallucis Oblique Head, Metatarsal Division
(936) Ileum	Abductor Hallucis Transverse Head, Medial Division
(938) Lymphatics of Jejunum	Abductor Hallucis Transverse Head, Lateral Division
(940) Pancreatic Duct	Extensor Hallucis Brevis
(942) Stomach (Special Cells)	Quadratus Plantae, Medial Division
(944) Vagina/Penis	Quadratus Plantae, Lateral Division
(946) Ovary/Testicle	Flexor Digitorum Brevis, Medial Division
(948) Colon (Ascending/Descending)	Flexor Digitorum Brevis, Lateral Division
(950) Lymphatics of Ileum	Flexor Digitus Pedis, Second
(952) Ear (External)	Flexor Digitus Pedis, Third
(954) Thymus	Flexor Digitus Pedis, Fourth
(956) Liver	Flexor Digitus Pedis, Fifth
(958) Pancreatic Duct	Adductor Digitus Pedis, Second
(960) Bladder	Adductor Digitus Pedis, Third
(962) Salivary Gland (Submandibular)	Adductor Digitus Pedis, Fourth
(964) Sinus (Nasal)	Adductor Digitus Pedis, Fifth

(966) Spleen
(968) Adrenal
(970) Uterus/Prostate
(972) Nose
(974) Nose
(976) Thymus

Abductor Digitus Pedis, Second
Abductor Digitus Pedis, Third
Abductor Digitus Pedis, Fourth
Abductor Digitus Pedis, Fifth
Abductor Digiti Minimi Pedis
Extensor Digitorum Brevis

ORGAN/TISSUE INDEX/NUMERICAL #2

(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals of Dr. Beardall.)

(052) Thyroid
(058) Vagina/Penis
(060) Ear (Internal)
(062) Pancreatic Duct
(070) Parathyroid
(098) Gallbladder(Arteries)
(100) Ovary/Testicle
(102) Colon (Transverse)
(110) Pharynx
(112) Pancreatic Duct
(114) Lung (Bronchiole)
(118) Thyroid
(120) Posterior Pituitary
(122) Tonsil
(124) Ovary/Testicle
(126) Parathyroid
(130) Stomach (Special Cells)
(132) Lymphatics of Gallbladder
(134) Gallbladder Duct
(136) Urethra
(138) Lymphatics of Gallbladder Duct
(140) Lymphatics of Jejunum
(270) Eye
(272) Ear (Internal)
(274) Gallbladder
(276) Larynx
(278) Bladder
(282) Gallbladder
(284) Urethra
(286) Duodenum (Descending Portion)
(288) Pancreatic Duct
(290) Bladder
(292) Stomach (Veins)
(294) Ureter
(296) Tonsil
(298) Eye
(300) Uterus/Prostate
(302) Adrenal
(304) Nose
(306) Eye
(308) Jejunum
(314) Uterus/Prostate
(322) Pancreas (Protein)
(326) Heart
(332) Vagina/Penis
(334) Uterus/Prostate
(346) Lung
(350) Stomach (Fundus)
(362) Parotid Gland
(370) Pancreas (Protein)
(380) Pharynx
(390) Ear (Internal)
(392) Kidney

Occipitalis
Levator Palpebrae, Superior Division
Orbicularis Oculi, Superior Division
Orbicularis Oculi, Inferior Division
Depressor Septi
Orbicularis Oris, Upper Division
Orbicularis Oris, Inferior Division
Buccinator
Temporalis, Parietal Division
Temporalis, Occipital Division
Masseter, Superficial Division
Masseter, Deep Division
Pterygoid Internal Medialis, Sphenoid Division
Pterygoid Internal Medialis, Palatine Division
Pterygoid External Lateralis, Upper Div.-Disc
Pterygoid External Lateralis, Lower Division
Rectus Superior Bulbi
Rectus Inferior Bulbi
Rectus Medialis Bulbi
Rectus Lateralis Bulbi
Obliquus Superior Bulbi
Obliquus Inferior Bulbi
Upper Trapezius, Scapular Division
Upper Trapezius, Clavicular Division
Sternocleidomastoid, Sternal Division
Sternocleidomastoid, Clavicular Division
Scalenus Anterior
Scalenus Medius
Scalenus Posterior
Platysma, Anterior Division
Platysma, Posterior Division
Digastric, Anterior Belly
Digastric, Posterior Belly
Stylohyoid
Mylohyoid
Geniohyoid
Sternohyoid
Sternothyroid
Thyrohyoid
Omohyoid
Longus Coli, Vertical Division
Longus Capitis
Splenius Capitis, Mastoid Division
Splenius Cervicis
Semispinalis Capitis
Semispinalis Cervicis
Obliquus Capitis Superior
Interspinalis (Cervical)
Cricoarytenoideus Lateralis
Thyroarytenoideus
Constrictor Pharyngeus Medius
Trapezius, Middle Division
Trapezius, Lower Division

(394) Spleen	Levator Scapula, Superior Division
(396) Parotid Gland	Levator Scapula, Inferior Division
(398) Liver	Rhomboid Minor
(400) Mammary	Rhomboid Major
(402) Ovary/Testicle	Serratus Anterior, Superior Division
(404) Lung	Serratus Anterior, Inferior Division
(406) Heart	Pectoralis Minor, Superior Division
(408) Lung	Pectoralis Minor, Inferior Division
(410) Pancreatic Duct	Subclavius, Clavicular Division
(412) Liver	Subclavius, Scapular Division
(414) Pancreas (Sugar)	Latissimus Dorsi, Thoracic Division
(416) Lymphatics of Duodenum	Latissimus Dorsi, Lumbar Division
(418) Colon (Transverse)	Latissimus Dorsi, Iliac Division
(420) Sinus (Maxillary)	Supraspinatus, Spine Division
(422) Esophagus	Supraspinatus, Fossa Division
(424) Lymphatics of Lung	Infraspinatus, Superior Division
(426) Gallbladder	Infraspinatus, Middle Division
(428) Gallbladder Duct	Infraspinatus, Inferior Division
(430) Ovary/Testicle	Teres Minor
(432) Ear (External)	Teres Major, Superior Division
(434) Lung	Teres Major, Inferior Division
(436) Posterior Pituitary	Subscapularis, Superior Division
(438) Thyroid	Subscapularis, Third Division
(440) Heart	Subscapularis, Second Division
(442) Uterus/Prostate	Subscapularis, Inferior Division
(444) Nose	Coracobrachialis, Coracoid Division
(446) Lung	Coracobrachialis, Septal Division
(448) Tonsil	Deltoid, Posterior, Medial Division
(450) Colon (Ascending/Descending)	Deltoid, Posterior, Lateral Division
(452) Lung	Deltoid, Middle, Posterior Division
(454) Gallbladder	Deltoid, Middle, Anterior Division
(456) Adrenal	Deltoid, Anterior, Scapular Division
(458) Lung	Deltoid, Anterior, Clavicular Division
(460) Heart	Pectoralis Major, Clavicular Division
(462) Liver	Pectoralis Major, Sternal Division
(464) Vagina/Penis	Pectoralis Major, Costal Division
(466) Spleen	Biceps Brachii Longhead
(468) Colon (Sigmoid)	Biceps Brachii Shorthead
(470) Eustachian Tube	Triceps, Longhead
(472) Gallbladder	Triceps, Lateral Head
(474) Uterus/Prostate	Triceps, Medial Head
(476) Parotid Gland	Articularis Cubiti
(478) Jejunum	Brachialis
(480) Ovary/Testicle	Brachioradialis, Humeral Division
(482) Lung	Brachioradialis, Septal Division
(484) Vagina/Penis	Pronator Teres, Humeral Division
(486) Posterior Pituitary	Pronator Teres, Ulnar Division
(488) Heart	Anconeus, Olecranon Division
(490) Lymphatics of Gallbladder	Anconeus, Ulnar Division
(492) Ear (Internal)	Supinator, Radial Division
(494) Liver	Supinator, Ulnar Division
(496) Eustachian Tube	Pronator Quadratus, Proximal Division
(498) Kidney	Pronator Quadratus, Distal Division
(500) Lymphatics of Throat	Palmaris Longus
(502) Esophagus	Flexor Carpi Radialis, Abductor Division
(504) Adrenal	Flexor Carpi Radialis, Flexor Division
(506) Ileum	Flexor Carpi Ulnaris, Flexor Division

(508) Nose	Flexor Carpi Ulnaris, Adductor Division (508)
(510) Stomach (Pyloric Vestibule)	Extensor Carpi Ulnaris, Adductor Division
(512) Uterus/Prostate	Extensor Carpi Ulnaris, Extensor Division
(514) Nose	Extensor Carpi Radialis Brevis
(516) Kidney	Extensor Carpi Radialis Longus, Ext Division
(518) Liver	Extensor Carpi Radialis Longus, Abd. Division
(520) Gallbladder Duct	Extensor Pollicis Longus, Ulnar Division
(522) Ovary/Testicle	Extensor Pollicis Longus, Septal Division
(524) Liver	Extensor Pollicis Brevis, Radial Division
(526) Pancreatic Duct	Extensor Pollicis Brevis, Septal Division
(528) Stomach (Pyloric Gland)	Flexor Pollicis Longus
(530) Nose	Abductor Pollicis Longus, Ulnar Division
(532) Ovary/Testicle (Fall. Tube/Sem.Vesicle)	Abductor Pollicis Longus, Radial Division
(534) Adrenal (Medulla)	Flexor Pollicis Brevis
(536) Sinus (Nasal)	Interossei Pollicis (Palmaris First)
(538) Ovary/Testicle	Opponens Pollicis, Flexor Division
(540) Pineal	Opponens Pollicis, Abductor Division
(542) Gallbladder Duct	Abductor Pollicis Brevis
(544) Ileum	Adductor Pollicis Transversus
(546) Lymphatics of Colon	Adductor Pollicis Obliquus
(548) Pharynx	Extensor Dig. Communis Manus, Medial Division
(550) Spleen	Extensor Dig. Communis Manus, Lateral Division
(552) Posterior Pituitary	Extensor Digiti Minimi Manus
(554) Kidney	Extensor Indicis Proprius
(556) Lymphatics of Jejunum	Flexor Digitorum Superficialis, Medial Division
(558) Anterior Pituitary	Flexor Digitorum Superficialis, Lateral Division
(560) Spleen	Flexor Digitorum Profundus Manus, Medial Division
(562) Liver	Flexor Digitorum Profundus Manus, Lateral Division
(564) Thymus	Interossei Dorsales Manus, Fourth
(566) Esophagus	Interossei Dorsales Manus, Third
(568) Sinus (Frontal)	Interossei Dorsales Manus, Second
(570) Bladder	Interossei Dorsales Manus, First
(572) Lymphatics of Colon	Lumbricales Manus, Fourth Division
(574) Lymphatics of Rectum (Anal Canal)	Lumbricales Manus, Third
(576) Ovary/Testicle (Fall. Tube/Sem. Vesicle)	Lumbricales Manus, Second
(578) Liver	Lumbricales Manus, First
(580) Lymph. of Gallbladder Duct	Flexor Digiti Minimi Brevis, Manus
(582) Adrenal	Abductor Digiti Minimi Manus, Flexor Division
(584) Lung (Bronchiole)	Abductor Digiti Minimi Manus, Abductor Division
(586) Sinus (Maxillary)	Opponens Digiti Minimi Manus, Abductor Division
(588) Uterus/Prostate	Opponens Digiti Minimi Manus, Flexor Division
(590) Larynx	Interossei Palmaris, Fourth
(592) Pharynx	Interossei Palmaris, Third
(594) Salivary Gland (Sublingual)	Interossei Palmaris, Second
(596) Salivary Gland (Sublingual)	Palmaris Brevis
(610) Bladder	Spinalis Thoracis, Lumbar Division
(612) Heart	Spinalis Thoracis, Thoracic Division
(614) Pancreatic Duct	Longissimus Thoracis, Superior Division
(618) Mammary	Longissimus Thoracis, Inferior Division
(642) Colon (Sigmoid)	Levator Costorum, Inferior Division
(648) Lymphatics of Lung	Serratus Posterior, Superior Division
(652) Jejunum	Serratus Posterior, Inferior Division
(656) Heart	Diaphragm, Right Lumbar Division
(662) Lymphatics of Pancreas	Diaphragm, Left Lumbar Division
(690) Nose	Pyramidalis
(692) Sinus (Frontal)	Obliquus Externus Abdominis, Anterior Division
(694) Sinus (Sphenoid)	Obliquus Externus Abdominis, Lateral Division

(696) Eye	Obliquus Internus Abdominis, Anterior Division
(698) Uterus/Prostate	Obliquus Internus Abdominis, Lateral Division
(700) Colon (Ascending/Descending)	Rectus Abdominis, First Division
(702) Salivary Gland (Sublingual)	Rectus Abdominis, Second Division
(704) Eye	Rectus Abdominis, Third Division
(706) Esophagus	Rectus Abdominis, Fourth Div., Medialis
(708) Bladder	Rectus Abdominis, Fourth Div., Lateralis
(710) Lymphatics of Jejunum	Iliacus
(712) Kidney	Iliacus Minor
(714) Posterior Pituitary	Transverse Abdominis, Upper Division
(718) Duodenum (Descending Portion)	Abdominis, Lower Division
(722) Colon (Rectum)	Psoas Major, Lumbar Division
(724) Mammary	Psoas Major, Thoracic Division
(726) Bladder	Psoas Major, Diaphragmatic Division
(728) Posterior Pituitary	Psoas Minor
(730) Colon (Ascending/Descending)	Quadratus Lumborum, Costal Division
(732) Tonsil	Quadratus Lumborum, Lumbar Division
(734) Colon (Sigmoid)	Multifidus, Lumbosacral Division
(736) Stomach (Pyloric Valve)	Iliocostalis Lumborum
(738) Eye	Longissimus Lumborum
(740) Pancreatic Duct	Coccygeus, Sacral Division
(742) Thymus	Coccygeus, Coccyx Division
(744) Heart	Pubococcygeus
(746) Uterus/Prostate	Ileococcygeus
(752) Pancreatic Duct	Cremaster
(780) Vagina/Penis	Gluteus Medius, Posterior Division
(782) Mammary	Gluteus Medius, Middle Division
(784) Ovary/Testicle	Gluteus Medius, Anterior Division
(786) Lung	Gluteus Minimus, Anterior Division
(788) Vagina/Penis	Gluteus Minimus, Posterior Division
(790) Eustachian Tube	Tensor Fascia Lata, Anterior Division
(792) Vagina/Penis	Tensor Fascia Lata, Posterior Division
(794) Liver	Rectus Femoris, Reflected Head
(796) Lymphatics of Ileum	Rectus Femoris, Straight Head
(798) Pancreatic Duct	Pectineus
(800 L) Left Eye	Adductor Brevis (Left)
(800 R) Right Eye	Adductor Brevis (Right)
(804) Vagina/Penis	Adductor Longus, Inferior Division
(806) Ovary/Testicle	Adductor Longus, Superior Division
(808) Uterus/Prostate	Gracilis
(810) Lymphatics of Ileum	Sartorius
(812) Spleen	Obturator Externus
(814) Nose	Quadratus Femoris
(816) Duodenum (Special Cells)	Vastus Medialis, Upper Division
(818) Gallbladder	Vastus Medialis, Middle Division
(820) Ileum	Vastus Medialis, Lower Division
(822) Larynx	Obturator Internus
(824) Eustachian Tube	Biceps Femoris, Shorthead
(826) Spleen	Biceps Femoris, Longhead, Fibular Division
(828) Salivary Gland (Sublingual)	Biceps Femoris, Longhead, Tibial Division
(830) Thymus	Vastus Lateralis, Superior Division
(832) Salivary Gland (Submandibular)	Vastus Lateralis, Middle Division
(834) Pancreatic Duct	Vastus Lateralis, Lower Division
(836) Parotid Gland	Vastus Intermedius, Medial Division
(838) Salivary Gland (Sublingual)	Vastus Intermedius, Lateral Division
(840) Adrenal	Articularis Genu
(842) Bladder	Adductor Magnus, Vertical Division

(844) Uterus/Prostate (Broad Ligament)
(846) Lymphatics of Thyroid
(848) Pancreatic Duct
(850) Uterus/Prostate (Digestive Portion)
(852) Colon (Rectum)
(854) Adrenal
(856) Adrenal
(858) Pancreatic Duct
(860) Ovary/Testicle
(862) Uterus/Prostate
(864) Ovary/Testicle
(870) Gallbladder
(872) Kidney
(874) Liver
(876) Lymphatics of Tonsil
(878) Anterior Pituitary
(880) Liver
(882) Pancreas (Protein)
(884) Ovary/Testicle
(886) Duodenum
(888) Gallbladder
(890) Urethra (Membranous Portion)
(892) Nose
(894) Bladder
(896) Urethra
(898) Ovary/Testicle
(900) Anterior Pituitary
(902) Kidney
(904) Anterior Pituitary
(906) Heart (Left Ventricle)
(908) Stomach
(910) Kidney
(912) Heart
(914) Posterior Pituitary
(920) Uterus/Prostate (Digestive Portion)
(922) Liver
(924) Lymphatics of Stomach
(926) Pancreatic Duct
(928) Lung
(930) Lung
(932) Adenoid (Palatine)
(934) Lung
(936) Ovary/Testicle
(938) Colon (Rectum)
(940) Jejunum
(942) Salivary Gland (Sublingual)
(944) Larynx
(946) Larynx
(948) Salivary Gland (Sublingual)
(950) Sinus (Maxillary)
(952) Kidney
(954) Vagina/Penis
(956) Sinus (Maxillary)
(958) Eye
(960) Spleen
(962) Eye
(964) Tonsil

Adductor Magnus, Oblique Division
Adductor Magnus, Transverse Division
Gluteus Maximus, Iliac Division
Gluteus Maximus, Sacral Division
Gluteus Maximus, Coccygeal Division
Semitendinosus
Semimembranosus, Tibial Division
Semimembranosus, Popliteal Division
Piriformis
Gemellus Inferior
Gemellus Superior
Popliteus
Gastrocnemius, Medial Division
Gastrocnemius, Lateral Division
Plantaris
Soleus Medial Division
Soleus, Lateral Division
Tibialis Posterior, Tibial Division
Tibialis Posterior, Fibular Division
Peroneus Longus, Cuneiform Division
Peroneus Longus, Metatarsal Division
Peroneus Brevis, Fibular Division
Peroneus Brevis, Septal Division
Peroneus Tertius
Tibialis Anterior, Supinator Division
Tibialis Anterior, Dorsiflexor Division
Flexor Hallucis Longus, Tibial Division
Flexor Hallucis Longus, Fibular Division
Extensor Hallucis Longus, Interosseous Division
Extensor Hallucis Longus, Fibular Division
Flexor Digitorum Longus, Medial Division
Flexor Digitorum Longus, Lateral Division
Extensor Digitorum Longus, Medial Division
Extensor Digitorum Longus, Lateral Division
Adductor Hallucis, Superior Division
Adductor Hallucis, Inferior Division
Flexor Hallucis Brevis, First Cuneiform Division
Flexor Hallucis Brevis, Tendonal Division
Flexor Hallucis Brevis, Third Cuneiform Division
Flexor Hallucis Brevis, Cuboid Division
Abductor Hallucis Oblique Head, Peroneus Division
Abductor Hallucis Oblique Head, Metatarsal Division
Abductor Hallucis Transverse Head, Medial Division
Abductor Hallucis Transverse Head, Lateral Division
Extensor Hallucis Brevis
Quadratus Plantae, Medial Division
Quadratus Plantae, Lateral Division
Flexor Digitorum Brevis, Medial Division
Flexor Digitorum Brevis, Lateral Division
Flexor Digitus Pedis, Second
Flexor Digitus Pedis, Third
Flexor Digitus Pedis, Fourth
Flexor Digitus Pedis, Fifth
Adductor Digitus Pedis, Second
Adductor Digitus Pedis, Third
Adductor Digitus Pedis, Fourth
Adductor Digitus Pedis, Fifth

(966) Thymus
(968) Ureter
(970) Thyroid
(972) Lung
(974) Posterior Pituitary
(976) Tonsil

Abductor Digitus Pedis, Second
Abductor Digitus Pedis, Third
Abductor Digitus Pedis, Fourth
Abductor Digitus Pedis, Fifth
Abductor Digiti Minimi Pedis
Extensor Digitorum Brevis

ADENOID

Abductor Hallucis Oblique Head, Peroneus Division (932)
Extensor Pollicis Brevis, Septal Division (526)

ADRENAL

Abductor Digiti Minimi Manus, Flexor Division (582)
Abductor Digitus Pedis, Third (968)
Articularis Genu (840)
Deltoid, Anterior, Scapular Division (456)
Flexor Carpi Radialis, Flexor Division (504)
Flexor Pollicis Brevis (534)
Flexor Pollicis Brevis (534)
Gracilis (808)
Longissimus Thoracis, Inferior Division (618)
Masseter, Superficial Division (114)
Pronator Teres, Humeral Division (484)
Psoas Major, Diaphragmatic Division(726)
Rectus Abdominis, First Division (700)
Sartorius (810)
Semimembranosus, Tibial Division (856)
Semitendinosus (854)
Soleus Medial Division (878)
Splenius Cervicis (326)
Sternothyroid (302)
Vastus Medialis, Middle Division(818)

BLADDER

Adductor Digitus Pedis, Third (960)
Adductor Magnus, Vertical Division (842)
Anconeus, Olecranon Division(488)
Articularis Cubiti (476)
Coracobrachialis, Septal Division (446)
Digastric, Anterior Belly (290)
Flexor Digitorum Profundus Manus, Medial Division (560)
Gluteus Maximus, Coccygeal Division (852)
Interossei Dorsales Manus, First (570)
Interossei Pollicis (Palmaris First) (536)
Mylohyoid (296)
Peroneus Brevis, Septal Division(892)
Peroneus Longus, Cuneiform Division(886)
Peroneus Tertius (894)
Psoas Major, Diaphragmatic Division (726)
Pyramidalis (690)
Rectus Abdominis, Fourth Div., Lateralis (708)
Scalenus Anterior (278)
Serratus Posterior, Superior Division (648)
Spinalis Thoracis, Lumbar Division (610)
Trapezius, Lower Division (392)

COLON (APPENDIX)

Extensor Carpi Ulnaris, Adductor Division(510)

COLON (ASCENDING/DESCENDING)

Biceps Femoris, Longhead, Fibular Division (826)
Biceps Femoris, Shorthead(824)
Deltoid, Posterior, Lateral Division (450)
Flexor Digitorum Brevis, Lateral Division(948)
Gastrocnemius, Lateral Division (874)
Iliacus (710)
Interspinalis (Cervical) (350)
Obliquus Externus Abdominis, Anterior Division(692)
Quadratus Lumborum, Costal Division (730)
Rectus Abdominis, First Division (700)
Tensor Fascia Lata, Anterior Division(790)
Upper Trapezius, Scapular Division(270)

COLON (HEPATIC & SPLENIC FLEXURE)

Obliquus Internus Abdominis, Anterior Division(696)

COLON (RECTUM)

Abductor Hallucis Transverse Head, Lateral Division (938)
Gluteus Maximus, Coccygeal Division (852)
Psoas Major, Lumbar Division (722)
Soleus, Lateral Division(880)

COLON (SIGMOID)

Biceps Brachii Shorthead (468)
Levator Costorum, Inferior Division (642)
Multifidus, Lumbosacral Division (734)

COLON (TRANSVERSE)

Buccinator (102)
Latissimus Dorsi, Iliac Division (418)
Levator Palpebrae, Superior Division (058)
Pectineus(798)

DUODENUM (ASCENDING PORTION)

Rectus Abdominis, Fourth Div., Medialis (706)

DUODENUM (DESCENDING PORTION)

Abdominis, Lower Division (718)
Platysma, Anterior Division (286)

DUODENUM (HORIZONTAL PORTION)

Rectus Abdominis, Second Division (702)

DUODENUM (SPECIAL CELLS)

Extensor Carpi Radialis Brevis(514)
Vastus Medialis, Upper Division (816)

DUODENUM (SUPERIOR PORTION)

Triceps, Longhead(470)

DUODENUM

Adductor Brevis (Left) (800 L)
Gastrocnemius, Medial Division(872)
Peroneus Longus, Cuneiform Division (886)

EAR (EXTERNAL)

Flexor Digitus Pedis, Third (952)
Subclavius, Clavicular Division(410)
Teres Major, Superior Division (432)

EAR (INTERNAL)

Orbicularis Oculi, Superior Division (060)
Supinator, Radial Division (492)
Trapezius, Middle Division (390)
Upper Trapezius, Clavicular Division (272)

ESOPHAGUS

Flexor Carpi Radialis, Abductor Division (502)
Flexor Hallucis Longus, Fibular Division(902)
Interossei Dorsales Manus, Third (566)
Rectus Abdominis, Fourth Div., Medialis (706)
Subscapularis, Second Division(440)
Supraspinatus, Fossa Division (422)

EUSTACHIAN TUBE

Biceps Femoris, Shorthead(824)
Pronator Quadratus, Proximal Division (496)
Tensor Fascia Lata, Anterior Division (790)
Triceps, Longhead(470)

EYE

Adductor Brevis (Left) (800 L)
Adductor Brevis (Right) (800 R)
Adductor Digitus Pedis, Fourth (962)
Adductor Digitus Pedis, Second (958)
Deltoid, Posterior, Lateral Division(450)
Geniohyoid (298)
Infraspinatus, Middle Division(426)
Longissimus Lumborum (738)
Obliquus Internus Abdominis, Anterior Division (696)
Omohyoid (306)
Rectus Abdominis, Third Division (704)
Upper Trapezius, Scapular Division (270)

GALLBLADDER (ARTERIES)

Digastric, Posterior Belly (292)
Orbicularis Oris, Upper Division (098)

GALLBLADDER

Deltoid, Middle, Anterior Division (454)
Flexor Carpi Radialis, Abductor Division(502)
Infraspinatus, Middle Division (426)
Levator Costorum, Inferior Division (642)
Palmaris Brevis (596)
Peroneus Longus, Metatarsal Division (888)
Popliteus (870)
Quadratus Lumborum, Lumbar Division(732)
Scalenus Medius (282)
Sternocleidomastoid, Sternal Division (274)
Triceps, Lateral Head(472)
Vastus Medialis, Middle Division (818)

GALLBLADDER DUCT

Abductor Pollicis Brevis (542)
Adductor Brevis (Right) (800 R)
Extensor Pollicis Brevis, Radial Division(524)
Extensor Pollicis Longus, Ulnar Division (520)
Gluteus Medius, Posterior Division(780)
Infraspinatus, Inferior Division (428)
Occipitalis (052)
Rectus Medialis Bulbi (134)
Sternohyoid (300)

HEART

Adductor Hallucis, Inferior Division (922)
Adductor Pollicis Transversus (544)
Anconeus, Olecranon Division (488)
Diaphragm, Right Lumbar Division (656)
Extensor Digitorum Longus, Medial Division (912)
Extensor Hallucis Longus, Fibular Division (906)
Gemellus Superior (864)
Interossei Dorsales Manus, Fourth (564)
Obturator Externus (812)
Pectoralis Major, Clavicular Division (460)
Pectoralis Minor, Superior Division (406)
Pubococcygeus (744)
Spinalis Thoracis, Lumbar Division (610)
Spinalis Thoracis, Thoracic Division (612)
Splenius Cervicis (326)
Subscapularis, Second Division (440)
Subscapularis, Superior Division(436)
Tibialis Posterior, Fibular Division(884)

ILEUM

Abductor Hallucis Transverse Head, Medial Division (936)
Adductor Longus, Inferior Division(804)
Adductor Pollicis Transversus (544)
Coccygeus, Sacral Division(740)
Flexor Carpi Ulnaris, Flexor Division (506)
Rectus Abdominis, Third Division(704)
Tibialis Posterior, Tibial Division(882)
Vastus Intermedius, Medial Division(836)

Vastus Lateralis, Lower Division (834)
Vastus Medialis, Lower Division (820)

ILEUM (PEYER'S PATCHES)

Brachioradialis, Humeral Division(480)
Semimembranosus, Popliteal Division (858)

ILEUM (SPECIAL CELLS)

Abductor Digiti Minimi Manus, Abductor Division (584)
Interossei Palmaris, Second (594)

JEJUNUM

Brachialis (478)
Extensor Hallucis Brevis (940)
Flexor Digitorum Profundus Manus, Lateral Division (562)
Flexor Hallucis Brevis, Cuboid Division (930)
Longissimus Lumborum(738)
Longus Coli, Vertical Division (308)
Opponens Digiti Minimi Manus, Abductor Division (586)
Plantaris (876)
Rectus Femoris, Straight Head(796)
Serratus Posterior, Inferior Division (652)
Tibialis Anterior, Supinator Division(896)

KIDNEY

Biceps Brachii Longhead(466)
Brachioradialis, Septal Division(482)
Coracobrachialis, Coracoid Division(444)
Extensor Carpi Radialis Longus, Ext Division (516)
Extensor Carpi Ulnaris, Extensor Division(512)
Extensor Indicis Proprius (554)
Flexor Digitorum Longus, Lateral Division (910)
Flexor Digitus Pedis, Third (952)
Flexor Hallucis Longus, Fibular Division (902)
Gastrocnemius, Medial Division (872)
Iliacus Minor (712)
Palmaris Longus(500)
Pectoralis Minor, Inferior Division(408)
Pronator Quadratus, Distal Division (498)
Pronator Teres, Ulnar Division(486)
Psoas Major, Lumbar Division(722)
Psoas Major, Thoracic Division(724)
Rectus Superior Bulbi (130)
Scalenus Posterior (284)
Sternothyroid (302)
Trapezius, Lower Division (392)

LARYNX

Flexor Digitorum Brevis, Medial Division (946)
Interossei Palmaris, Fourth (590)
Obturator Internus (822)
Quadratus Plantae, Lateral Division (944)
Sternocleidomastoid, Clavicular Division (276)

LIVER

Adductor Hallucis, Inferior Division (922)
Adductor Longus, Superior Division(806)
Constrictor Pharyngeus Medius (380)
Extensor Carpi Radialis Longus, Abd. Division (518)
Extensor Pollicis Brevis, Radial Division (524)
Flexor Digitorum Profundus Manus, Lateral Division (562)
Flexor Digitus Pedis, Fifth (956)
Flexor Hallucis Longus, Tibial Division(900)
Gastrocnemius, Lateral Division (874)
Lumbricales Manus, First (578)
Obliquus Inferior Bulbi (140)
Obliquus Superior Bulbi (138)
Orbicularis Oris, Upper Division(098)
Pectoralis Major, Sternal Division (462)
Quadratus Lumborum, Costal Division(730)
Rectus Femoris, Reflected Head (794)
Rectus Medialis Bulbi (134)
Rhomboid Major (400)
Rhomboid Minor (398)
Soleus, Lateral Division (880)
Subclavius, Scapular Division (412)
Supinator, Ulnar Division (494)

LUNG

Abductor Digiti Minimi Manus, Abductor Division (584)
Abductor Digitus Pedis, Fifth (972)
Abductor Hallucis Oblique Head, Metatarsal Division (934)
Brachioradialis, Septal Division (482)
Coracobrachialis, Septal Division (446)
Deltoid, Anterior, Clavicular Division (458)
Deltoid, Anterior, Scapular Division(456)
Deltoid, Middle, Anterior Division(454)
Deltoid, Middle, Posterior Division (452)
Extensor Digiti Minimi Manus (552)
Flexor Hallucis Brevis, Cuboid Division (930)
Flexor Hallucis Brevis, Third Cuneiform Division (928)
Gemellus Inferior (862)
Gluteus Medius, Anterior Division(784)
Gluteus Minimus, Anterior Division (786)
Iliocostalis Lumborum (736)
Masseter, Superficial Division (114)
Obliquus Capitis Superior (346)
Obturator Internus (822)
Pectoralis Minor, Inferior Division (408)
Pectoralis Minor, Superior Division(406)
Peroneus Brevis, Fibular Division(890)
Peroneus Tertius (894)
Pubococcygeus (744)
Serratus Anterior, Inferior Division (404)
Serratus Anterior, Superior Division (402)
Teres Major, Inferior Division (434)
Vastus Medialis, Upper Division (816)

LYMPHATICS OF THE COLON

Abductor Digiti Minimi Manus, Flexor Division (582)
Adductor Pollicis Obliquus (546)
Lumbricales Manus, Fourth Division (572)
Obliquus Externus Abdominis, Lateral Division (694)

LYMPHATICS OF THE DUODENUM

Latissimus Dorsi, Lumbar Division (416)

LYMPHATICS OF THE FACE AND NECK

Abductor Pollicis Longus, Ulnar Division (530)

LYMPHATICS OF THE GALLBLADDER

Anconeus, Ulnar Division (490)
Rectus Inferior Bulbi (132)

LYMPHATICS OF THE GALLBLADDER DUCT

Flexor Digiti Minimi Brevis, Manus (580)
Obliquus Superior Bulbi (138)

LYMPHATICS OF THE ILEUM

Flexor Digitus Pedis, Second (950)
Latissimus Dorsi, Thoracic Division(414)
Rectus Femoris, Straight Head (796)
Sartorius (810)

LYMPHATICS OF THE JEJUNUM

Abductor Hallucis Transverse Head, Lateral Di vision (938)
Diaphragm, Right Lumbar Division (656)
Flexor Digitorum Superficialis, Medial Division (556)
Iliacus (710)
Obliquus Inferior Bulbi (140)

LYMPHATICS OF LEG AND INGUINAL NODES

Diaphragm, Left Lumbar Division (662)
Diaphragm, Right Lumbar Division (656)

LYMPHATICS OF THE LUNG

Infraspinatus, Superior Division (424)
Serratus Posterior, Superior Division (648)

LYMPHATICS OF THE PANCREAS

Diaphragm, Left Lumbar Division (662)

LYMPHATICS OF THE RECTUM

Lumbricales Manus, Third (574)
Platysma, Posterior Division(288)

LYMPHATICS OF THE SPLEEN

Diaphragm, Left Lumbar Division (662)

LYMPHATICS OF THE STOMACH

Flexor Hallucis Brevis, First Cuneiform Division (924)
Psoas Minor (728)

LYMPHATICS OF THE SUBMANDIBULAR

Scalenus Medius (282)
Semimembranosus, Tibial Division(856)
Sternocleidomastoid, Clavicular Division(276)

LYMPHATICS OF THE THORACIC DUCT

Extensor Pollicis Longus, Septal Division(522)

LYMPHATICS OF THE THROAT

Palmaris Longus (500)

LYMPHATICS OF THE THYROID

Adductor Magnus, Transverse Division (846)

LYMPHATICS OF THE TONSIL

Plantaris (876)

MAMMARY

Gluteus Medius, Middle Division (782)
Longissimus Thoracis, Inferior Division (618)
Pectoralis Major, Sternal Division(462)
Psoas Major, Thoracic Division (724)
Rhomboid Major (400)

NOSE

Abductor Digiti Minimi Pedis (974)
Abductor Digitus Pedis, Fifth (972)
Abductor Hallucis Oblique Head, Metatarsal Division (934)
Abductor Pollicis Longus, Ulnar Division (530)
Adductor Magnus, Transverse Division (846)
Coracobrachialis, Coracoid Division (444)
Extensor Carpi Radialis Brevis (514)
Flexor Carpi Ulnaris, Adductor Division (508)
Lumbricales Manus, Fourth Division(572)
Peroneus Brevis, Septal Division (892)
Pyramidalis (690)
Quadratus Femoris (814)
Serratus Posterior, Inferior Division (652)
Thyrohyoid (304)

OVARY/TESTICLE

Abductor Hallucis Transverse Head, Medial Division (936)
Abductor Pollicis Longus, Radial Division (532)
Adductor Longus, Superior Division (806)
Articularis Genu (840)
Biceps Brachii Shorthead (468)
Brachioradialis, Humeral Division (480)
Buccinator (102)
Depressor Septi (070)
Extensor Digitorum Communis Manus, Medial Division (548)
Extensor Pollicis Longus, Septal Division (522)
Flexor Carpi Ulnaris, Flexor Division (506)
Flexor Digitorum Brevis, Medial Division (946)
Gemellus Superior (864)
Gluteus Medius, Anterior Division (784)
Interossei Palmaris, Third (592)
Longus Coli, Vertical Division (308)
Lumbricales Manus, Second (576)
Multifidus, Lumbosacral Division(734)
Opponens Pollicis, Flexor Division (538)
Orbicularis Oris, Inferior Division (100)
Piriformis (860)
Pterygoid External Lateralis, Lower Division(126)
Pterygoid External Lateralis, Upper Div.-Disc (124)
Quadratus Femoris(814)
Scalenus Anterior (278)
Serratus Anterior, Superior Division (402)
Supinator, Radial Division (492)
Teres Minor (430)
Tibialis Anterior, Dorsiflexor Division (898)
Tibialis Posterior, Fibular Division (884)

PANCREAS (PROTEIN)

Opponens Digiti Minimi Manus, Flexor Division(588)
Splenius Capitis, Mastoid Division (322)
Thyroarytenoideus (370)
Tibialis Posterior, Tibial Division (882)

PANCREAS (SUGAR)

Abductor Pollicis Brevis (542)
Abductor Pollicis Longus, Radial Division(532)
Extensor Digitorum Communis Manus, Lateral Division (550)
Extensor Indicis Proprius (554)
Flexor Digiti Minimi Brevis, Manus (580)
Flexor Digitorum Superficialis, Medial Division (556)
Gluteus Maximus, Iliac Division(848)
Gluteus Minimus, Anterior Division (786)
Iliacus Minor (712)
Latissimus Dorsi, Thoracic Division (414)
Platysma, Anterior Division(286)
Rectus Lateralis Bulbi (136)
Sternocleidomastoid, Sternal Division(274)
Temporalis, Parietal Division(110)

PANCREATIC DUCT

Adductor Digitus Pedis, Second (958)
Anconeus, Ulnar Division(490)
Coccygeus, Sacral Division (740)
Cremaster (752)
Cricoarytenoideus Lateralis (362)
Digastric, Anterior Belly (290)
Extensor Carpi Radialis Longus, Abductor Division (518)
Extensor Hallucis Brevis(940)
Extensor Pollicis Brevis, Septal Division (526)
Flexor Hallucis Brevis, Tendonal Division (926)
Flexor Pollicis Longus (528)
Gluteus Maximus, Iliac Division (848)
Gluteus Minimus, Posterior Division(788)
Interossei Dorsales Manus, Third (566)
Longissimus Thoracis, Superior Division (614)
Orbicularis Oculi, Inferior Division (062)
Pectineus (798)
Platysma, Posterior Division (288)
Semimembranosus, Popliteal Division (858)
Subclavius, Clavicular Division (410)
Subscapularis, Inferior Division(442)
Temporalis, Occipital Division (112)
Vastus Lateralis, Lower Division (834)
Vastus Lateralis, Middle Division(832)

PARATHYROID

Depressor Septi (070)
Levator Scapula, Inferior Division(396)
Lumbricales Manus, Second (576)
Lumbricales Manus, Third (574)
Peroneus Longus, Metatarsal Division(888)
Pterygoid External Lateralis, Lower Division (126)

PAROTID GLAND

Articularis Cubiti (476)
Cricoarytenoideus Lateralis (362)
Latissimus Dorsi, Lumbar Division(416)
Levator Scapula, Inferior Division (396)
Vastus Intermedius, Medial Division (836)

PHARYNX

Constrictor Pharyngeus Medius (380)
Extensor Dig. Communis Manus, Medial Division (548)
Interossei Palmaris, Third (592)
Opponens Pollicis, Abductor Division(540)
Temporalis, Parietal Division (110)

PINEAL

Flexor Hallucis Brevis, First Cuneiform Division (924)
Opponens Pollicis, Abductor Division (540)
Pterygoid External Lateralis, Upper Div.-Disc (124)
Spinalis Thoracis, Thoracic Division (612)

Temporalis, Occipital Division(112)
Teres Major, Superior Division(432)

PITUITARY, ANTERIOR

Adductor Hallucis, Superior Division(920)
Adductor Magnus, Oblique Division (844)
Adductor Pollicis Obliquus (546)
Extensor Hallucis Longus, Interosseous Division (904)
Extensor Pollicis Longus, Ulnar Division (520)
Flexor Digitorum Longus, Lateral Division(910)
Flexor Digitorum Longus, Medial Division (908)
Flexor Digitorum Superficialis, Lateral Division (558)
Flexor Hallucis Longus, Tibial Division (900)
Interossei Dorsales Manus, First (570)
Longus Capitis (314)
Orbicularis Oris, Inferior Division(100)
Pectoralis Major, Costal Division(464)
Soleus Medial Division (878)
Splenius Capitis, Mastoid Division (322)
Supraspinatus, Fossa Division (422)

PITUITARY, POSTERIOR

Abductor Digiti Minimi Pedis (974)
Extensor Digiti Minimi Manus (552)
Extensor Digitorum Longus, Lateral Division (914)
Gluteus Maximus, Sacral Division(850)
Lumbricales Manus, First (578)
Masseter, Deep Division(118)
Opponens Pollicis, Flexor Division (538)
Pronator Teres, Ulnar Division (486)
Psoas Minor (728)
Pterygoid Internal Medialis, Sphenoid Division (120)
Rhomboid Minor (398)
Semitendinosus (854)
Subclavius, Scapular Division(412)
Subscapularis, Superior Division (436)
Transverse Abdominis, Upper Division (714)
Trapezius, Middle Division (390)
Vastus Medialis, Lower Division(820)

SALIVARY GLAND (SUBLINGUAL)

Biceps Femoris, Longhead, Tibial Division (828)
Flexor Digitorum Brevis, Lateral Division (948)
Interossei Palmaris, Second (594)
Palmaris Brevis (596)
Quadratus Plantae, Medial Division (942)
Rectus Abdominis, Second Division (702)
Semispinalis Cervicis (334)
Transverse Abdominis, Upper Division(714)
Triceps, Lateral Head(472)
Vastus Intermedius, Lateral Division (838)

SALIVARY GLAND (SUBMANDIBULAR)

Adductor Digitus Pedis, Fourth (962)
Flexor Hallucis Brevis, Third Cuneiform Division (928)
Vastus Lateralis, Middle Division (832)

SINUS (FRONTAL)

Interossei Dorsales Manus, Second (568)
Levator Scapula, Superior Division(394)
Obliquus Externus Abdominis, Anterior Division (692)

SINUS (MAXILLARY)

Flexor Digitus Pedis, Fifth (956)
Flexor Digitus Pedis, Second (950)
Opponens Digiti Minimi Manus, Abductor Division (586)
Pronator Quadratus, Distal Division(498)
Supraspinatus, Spine Division (420)

SINUS (NASAL)

Adductor Digitus Pedis, Fifth (964)
Interossei Pollicis (Palmaris First) (536)

SINUS (SPHENOID)

Obliquus Externus Abdominis, Lateral Division (694)
Subscapularis, Third Division(438)

SPLEEN

Abductor Digitus Pedis, Second (966)
Adductor Digitus Pedis, Third (960)
Biceps Brachii Longhead(466)
Biceps Femoris, Longhead, Fibular Division (826)
Cremaster (752)
Deltoid, Middle, Posterior Division(452)
Deltoid, Posterior, Medial Division(448)
Extensor Dig. Communis Manus, Lateral Division (550)
Flexor Digitorum Profundus Manus, Medial Division (560)
Interossei Dorsales Manus, Second (568)
Interossei Palmaris, Fourth (590)
Levator Scapula, Superior Division (394)
Obliquus Capitis Superior (346)
Obturator Externus (812)
Omohyoid (306)
Semispinalis Capitis (332)
Tibialis Anterior, Dorsiflexor Division(898)

STOMACH

Abductor Hallucis Oblique Head, Peroneus Division (932)
Deltoid, Anterior, Clavicular Division(458)
Flexor Digitorum Longus, Medial Division (908)
Longissimus Thoracis, Superior Division (614)

STOMACH (FUNDUS)

Interspinalis (Cervical) (350)

STOMACH (PYLORIC ANTRUM)

Extensor Carpi Radialis Longus, Ext Division (516)

STOMACH (PYLORIC CANAL)

Coccygeus, Coccyx Division(742)

STOMACH (PYLORIC GLAND)

Flexor Pollicis Longus (528)

STOMACH (PYLORIC VALVE)

Iliocostalis Lumborum (736)

STOMACH (PYLORIC VESTIBULE)

Extensor Carpi Ulnaris, Adductor Division (510)

STOMACH (SPECIAL CELLS)

Quadratus Plantae, Medial Division(942)
Rectus Inferior Bulbi (132)
Rectus Superior Bulbi (130)

STOMACH (VEINS)

Digastric, Posterior Belly (292)

THYMUS

Abductor Digitus Pedis, Second (966)
Coccygeus, Coccyx Division (742)
Extensor Digitorum Brevis (976)
Extensor Hallucis Longus, Interosseous Division (904)
Flexor Digitus Pedis, Fourth (954)
Infraspinatus, Inferior Division(428)
Interossei Dorsales Manus, Fourth (564)
Orbicularis Oculi, Inferior Division (062)
Pterygoid Internal Medialis, Palatine Division(122)
Serratus Anterior, Inferior Division(404)
Supraspinatus, Spine Division(420)
Thyrohyoid (304)
Transverse Abdominis, Lower Division (718)
Upper Trapezius, Clavicular Division(272)
Vastus Lateralis, Superior Division (830)

THYROID

Abductor Digitus Pedis, Fourth (970)
Extensor Digitorum Longus, Lateral Division (914)
Flexor Carpi Radialis, Flexor Division (504)
Geniohyoid (298)
Iliococcygeus (746)

Infraspinatus, Superior Division (424)
Masseter, Deep Division (118)
Occipitalis (052)
Orbicularis Oculi, Superior Division (060)
Pterygoid Internal Medialis, Sphenoid Division (120)
Rectus Femoris, Reflected Head(794)
Stylohyoid (294)
Subscapularis, Third Division (438)
Tensor Fascia Lata, Posterior Division(792)
Teres Minor (430)
Triceps, Medial Head(474)
Vastus Lateralis, Superior Division(830)

TONSIL

Adductor Digitus Pedis, Fifth (964)
Brachialis (478)
Deltoid, Posterior, Medial Division (448)
Extensor Digitorum Brevis (976)
Flexor Digitorum Superficialis, Lateral Division (558)
Flexor Hallucis Brevis, Tendonal Division (926)
Mylohyoid (296)
Obliquus Internus Abdominis, Lateral Division(698)
Pterygoid Internal Medialis, Palatine Division (122)
Quadratus Lumborum, Lumbar Division (732)
Vastus Intermedius, Lateral Division(838)

URETER

Abductor Digitus Pedis, Third (968)
Stylohyoid (294)

URETHRA (MEMBRANOUS PORTION)

Latissimus Dorsi, Iliac Division(418)

URETHRA

Extensor Hallucis Longus, Fibular Division (906)
Peroneus Brevis, Fibular Division (890)
Rectus Lateralis Bulbi (136)
Scalenus Posterior (284)
Tibialis Anterior, Supinator Division (896)

UTERUS/PROSTATE (DIGESTIVE PORTION)

Adductor Magnus, Vertical Division (842)
Biceps Femoris, Longhead, Tibial Division(828)
Piriformis (860)
Thyroarytenoideus (370)

UTERUS/PROSTATE

Abductor Digitus Pedis, Fourth (970)
Adductor Hallucis, Superior Division (920)
Adductor Magnus, Oblique Division (844)
Extensor Carpi Ulnaris, Extensor Division (512)
Extensor Digitorum Longus, Medial Division(912)
Flexor Carpi Ulnaris, Adductor Division (508)

Gemellus Inferior (862)
Gluteus Maximus, Sacral Division (850)
Gluteus Medius, Middle Division(782)
Gracilis (808)
Ileococcygeus (746)
Longus Capitis (314)
Obliquus Internus Abdominis, Lateral Division (698)
Opponens Digiti Minimi Manus, Flexor Divisi on (588)
Pectoralis Major, Clavicular Division(460)
Semispinalis Cervicis (334)
Sternohyoid (300)
Subscapularis, Inferior Division (442)
Supinator, Ulnar Division(494)
Triceps, Medial Head(474)

VAGINA/PENIS

Adductor Longus, Inferior Division (804)
Flexor Digitus Pedis, Fourth (954)
Gluteus Medius, Posterior Division (780)
Gluteus Minimus, Posterior Division (788)
Levator Palpebrae, Superior Division (058)
Pectoralis Major, Costal Division (464)
Popliteus (870)
Pronator Teres, Humeral Division (484)
Quadratus Plantae, Lateral Division(944)
Rectus Abdominis, Fourth Div., Lateralis(708)
Semispinalis Capitis (332)
Tensor Fascia Lata, Posterior Division (792)
Teres Major, Inferior Division(434)

THE CORRELATIONS OF MUSCLES, ORGANS, TISSUES, CRANIAL BONES, FOOT BONES AND ACUPUNCTURE POINTS (ADDITIONS AND CORRECTIONS)

by René Espy, D.C. and Nancy McBride, D.C.

ABSTRACT

As stated in the The Proceedings of the ICAK-USA, Summer 1992 -1993, muscles are a storehouse of information and are supreme indicators of various interrelated functions in the body. 2. This paper includes the interrelationship of a muscle with its organs, tissues, cranial bone, foot bone and acupuncture point and includes the electromagnetic frequency lock. 1.

The project to update and refine the muscles manuals of Alan G. Beardall, D.C. is now complete. 5. We present these manuals as one paper and include in this volume of collected papers, interrelationships and correlations for the Applied Kinesiologist to be able to have a quick reference to the multiple interrelated correlations that are available. The muscle is such a beautiful holistic phenomenona and allows us to gather data from deep within the body. Hopefully this information will provide useful data to the college.

ARM POSITIONS

In the The Proceedings of the ICAK-USA, Summer 1992 -1993, two arm positions were described. 2. This year we present the third position, namely the electromagnetic frequency position. 3. This position is used to access data related to acupuncture, flower remedies and homeopathy and other subtle body energies.

Yin - 7 Hz.
Electromagnetic - 120 ° arm abduction
Yang - 4 Hz.

We are also including the chemistry frequency lock as in the previous paper the position was incorrectly presented. With new measurement capabilities we are now able to refine our calculations and now present a corrected description. 4.

Determine if the therapy will be structure (refer to The Proceedings of the ICAK-USA, Summer 1992 -1993), chemistry or electromagnetic:

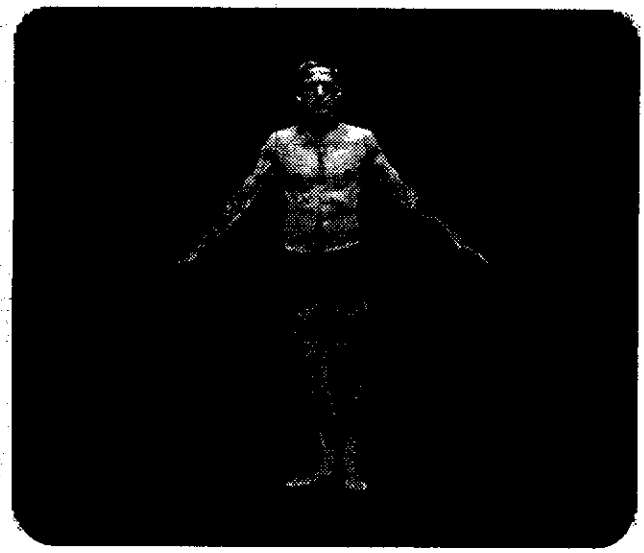
1 - Chemistry:
To determine if specific Chemistry for the muscle is necessary, put the left arm of the patient in the Chemistry Frequency Position. If a strong indicator muscle tests weak, Chemistry is necessary .

Enter the Chemistry Frequency position. The patient will display a YIN CBR. Determine the dosage by whatever means you presently use.

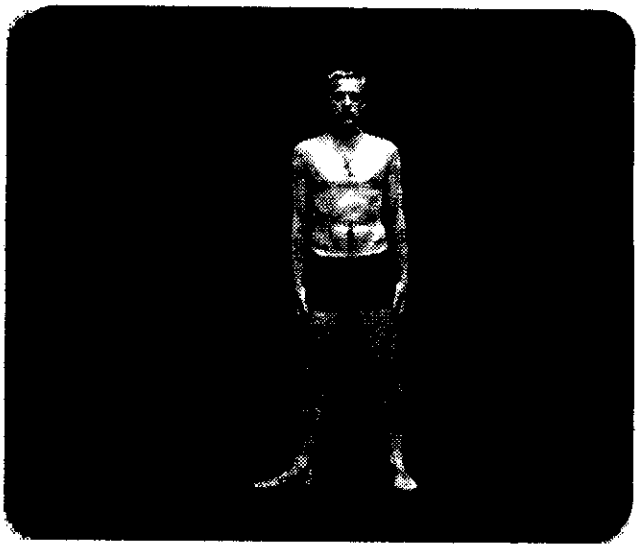
2 - Electromagnetic:
To determine if specific Electromagnetic therapy for the muscle is necessary, put the left arm of the patient in the Electromagnetic Frequency Position. If a strong indicator muscle tests weak, Electromagnetic therapy is necessary.

Enter the Electromagnetic Frequency position. The patient will display a Yin CBR.

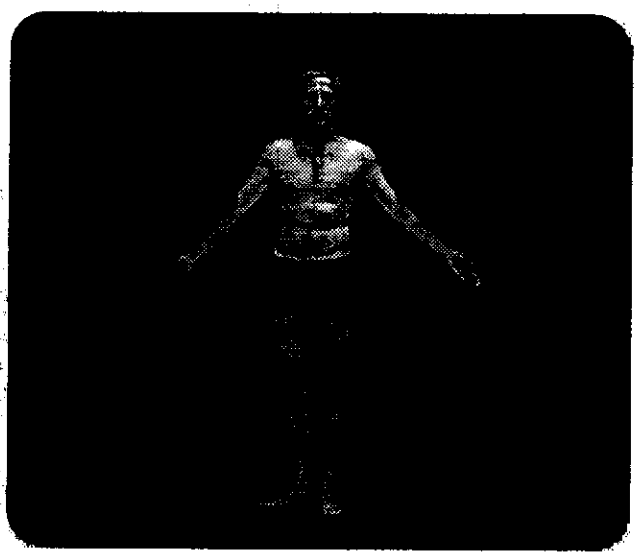
CHEMISTRY FREQUENCY LOCK



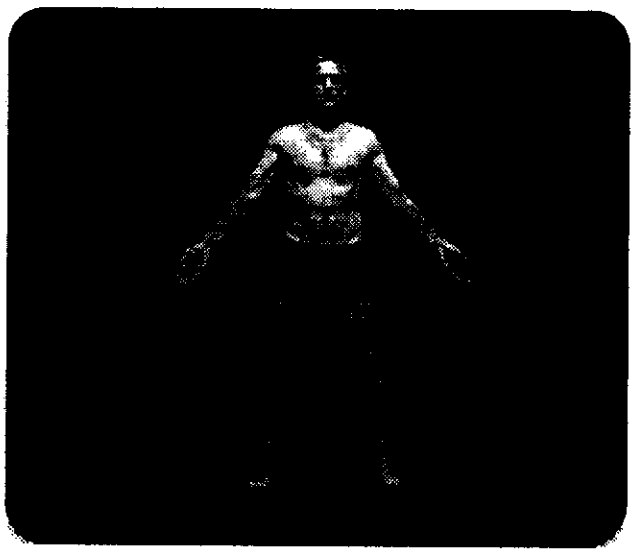
Procedure:
1 – Abduct Arms 45°.
2 – Abduct Legs 20° with Right Foot externally rotated.



3 – Eyes Open:
4 – Eye Motion: Continuous Up-Down, Down-Up
5 – Adduct Legs and Arms

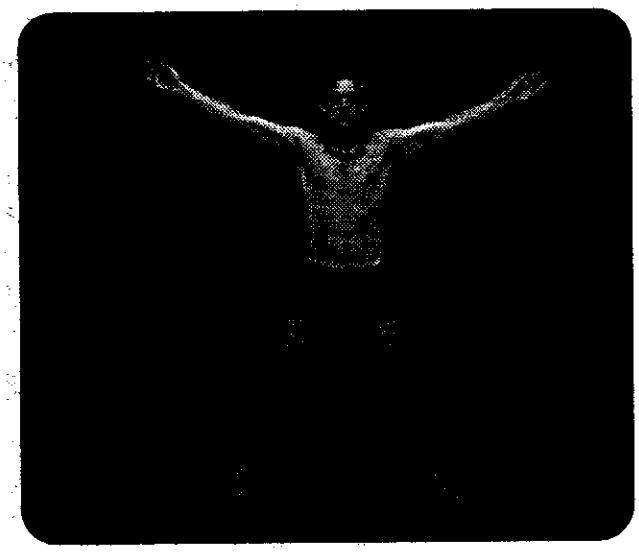


6 – Eyes Closed:
7 – Eye Motion: Continuous Up-Down, Down-Up
8 – Abduct Legs and Arms

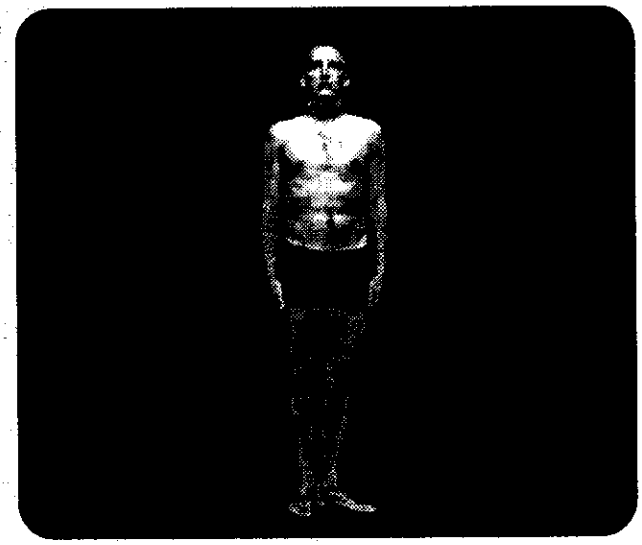


9 – Right Foot Neutral

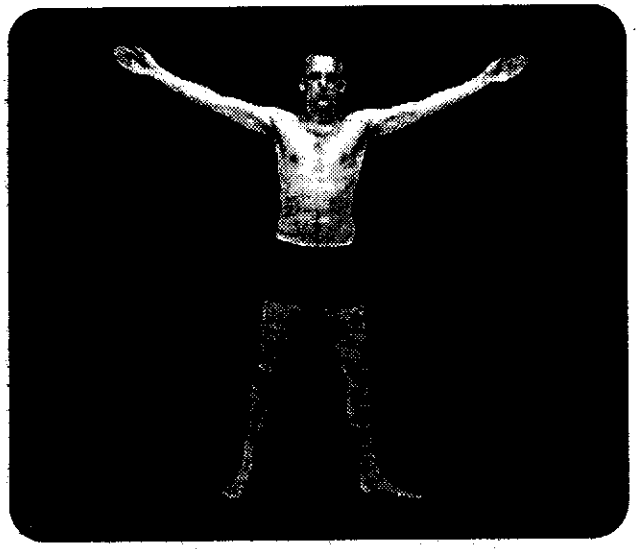
ELECTROMAGNETIC FREQUENCY LOCK



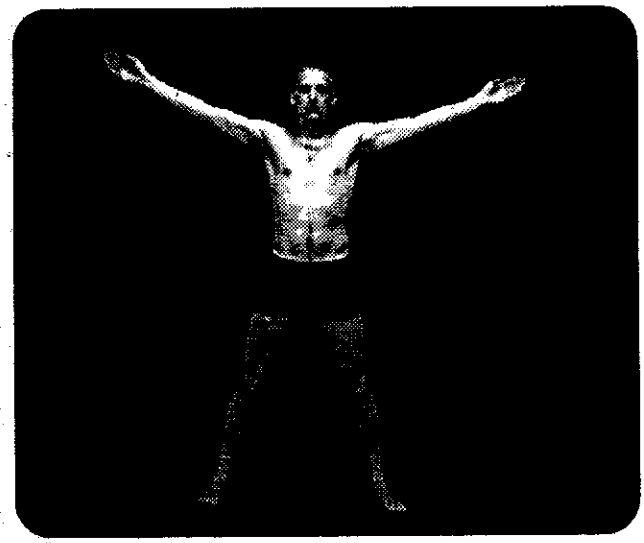
Procedure:
1 – Abduct Arms 120°.
2 – Abduct Legs, externally rotate the left foot.



3 – Eyes Open.
4 – Eye motion: Eyes at a 120° angle.
5 – Adduct Arms. Adduct Legs.



6 – Eyes Closed.
7 – Eye motion: Eyes at a 120° angle.
8 – Abduct Arms
9 – Abduct Legs



10 – Left foot neutral.

Correlations....Espy/McBride

Included in this paper are the correlations of the body sections according to muscle testing procedures included in the six volume muscle manuals. Thes are used for patient education and diagnostic correlations.

PROCEDURE

1 - Test the specific muscle to be treated. If weak, retest and perform a Neutral Lock.

2 - Retest the muscle again. It should now respond in strength. This allows the body to concentrate on the specific area being treated.

3 - Determine if the therapy will be structure, chemistry or electromagnetic:

1 - **Chemistry:**
To determine if specific Chemistry for the muscle is necessary, put the left arm of the patient in the Chemistry Frequency Position. If a strong indicator muscle tests weak, Chemistry is necessary.

Enter the Chemistry Frequency position. The patient will display a YIN CBR. Determine the dosage by whatever means you presently use.

Refer to the following correlations to determine the specific nutrient.

2 - **Structure:** Refer to the The Proceedings of the ICAK-USA, Summer 1992 -1993 for illustration.
To determine if specific Structure for the muscle is necessary, put the left arm of the patient in the Structure Frequency Position. If a strong indicator muscle tests weak, Structure is neces sary.

Enter the Structure Frequency position. The patient will display a Yang CBR.

DL (TL) the specific vertebral level or myomere level for the muscle you have tested. If the patient responds with a Yang CBR then adjust the segment.

Refer to the following correlations to determine the specific vertebral level or myomere level.

3 - **Electromagnetic:**
To determine if specific Electromagnetic for the muscle is necessary, put the left arm of the patient inthe Electromagnetic Frequency Position. If a strong indicator muscle tests weak, Electromagnetic Therapy is necessary.

Enter the Electromagnetic Frequency position. The patient will display a Yin CBR.

DL (TL) the specific acupuncture point for the muscle you have tested. Pulse the positive acupuncture point to the belly of the muscle.

Refer to the following correlations to determine the specific acupuncture point.

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CORRELATIONS

NECK, FACE, HYOID, TMJ, EYE AND THROAT EXAM CORRELATIONS

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group I - Muscles Affecting Neck							
308	Ovary-Testicle/Jejunum	XL 3	T4	C3	Vomer	2nd Metatarsal	Core Folic Acid
282	Lymph. of Submandibular/Gallbladder	Cx 9	L4	C4	Occiput, Universal	Calcaneus	Core Carbo Gest
284	Kidney/Urethra	Tw 12	T12	C5	Ethmoid	1st Cuneiform	Spore-X
278	Ovary-Testicle/Bladder	Sp 10	L1	C4	Parietal, Bulge	Talus	Core Kidney
314	Ant. Pituitary/Uterus-Prostate	Tw 3	T6	C1	Temporal, Internal	Cuboid, Inferior	Core D-Tox
276	Lymphatics of Submandibular/Larynx	Lv 6	T6	C3	Glabella	4th Metatarsal	Core Niacin
274	Pancreas(Sugar)/Gallbladder	Cx 2	T8	C2	Occiput, Lateral	5th Metatarsal	Core Selenium
270	Colon(A/D)/Eye	B 58	L2	C2	Occiput, Lateral	5th Metatarsal	Core Selenium
272	Thymus/Ear(Internal)	St 10	T10	C3	Maxillary, (M-L)	3rd Metatarsal	Core Calcium
052	Gallbladder Duct/Thyroid	Xa 3	T9	Cr.VII	Styloid	Distal Phal. Gr. Toe	Core Thyro

Group II - Muscles Affecting Neck (Prone)							
332	Spleen/Vagina-Penis	K 9	T4	C3	Temporal, External	Cuboid, Lateral	Core Potassium
346	Spleen/Lungs	G 40	T10	C2	Occiput, Lateral	5th Metatarsal	Core Rutin
326	Adrenal/Heart	Lv 7	C7	T1 R	Occiput, Lateral	Fifth Metatarsal	Core Folic Acid
322	Anterior Pituitary/Pancreas(Protein)	Tw 16	C3	C2	Temporal, External	Cuboid	Core Potassium
334	Sublingual Gland/Uterus-Prostate	Tw 16	T12	T2	Mandible	Prox. Phal. Gr. Toe	Core Thyro
350	Colon(A-D)/Stomach(Fundus)	St 37	L5	C6	Parietal, Bulge	Talus	Core Thiamine

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group III - Muscles Affecting Face							
058	Spleen/Vagina-Penis	K 9	T4	C3	Temporal, External	Cuboid, Lateral	Core Potassium
060	Thyroid/Internal Ear	Tw 4	L3	Cr. VIII	Mandible	Prox. Phal. Gr. Toe	Core E
062	Thymus/Pancreatic Duct	H 7	T8	Cr. VII	Occiput, Universal	Calcaneus	Prosta-X/Uter-X
070	Ovary-Testicle/Parathyroid	K 2	T6	Cr. VII	Frontal, External	Navicular	Core Inositol
098	Liver/Gallbladder(Arteries)	B 6	C3	Cr.VII	Parietal, Bulge	Talus	Core Manganese
100	Anterior Pituitary/Ovary-Testicle	G 2	T1	Cr.VII	Sphenoid	3rd Cunei., Lateral	Bl. Currant Seed Oil
286	Pancreas (Sugar)/Duodenum(Desc. Port.)	Cv 17	T1	Cr.VII	Glabella	4th Metatarsal	Core Carbo Gest
288	Lymphatics of Rectum/Pancreatic Duct	St 45	T11	Cr.VII	Nasal	Distal Phal. 2nd Toe	Core Panto. Acid
Group IV - Muscles Affecting Hyoid							
290	Pancreatic Duct/Bladder	K 25	C3	C2	Glabella	4th Metatarsal	Core Vitamin E
292	Gallbladder(Arteries)/Stomach(Veins)	K 17	L4	C2	Ethmoid	1st Cuneiform	Core Bone Matrix
294	Thyroid/Ureter	Cv 15	L5	C1	Styloid	Distal Phal. Gr. Toe	Core Calcium
296	Bladder/Tonsil	Li 7	T12	C2	Frontal, External	Navicular	Core Folic Acid
298	Thyroid/Eye	H 8	T9	C2	Vomer	2nd Metatarsal	Core Bile
300	Gallbladder Duct/Uterus-Prostate	Lv 8	T2	C3	Temporal, Internal	Cuboid, Inferior	Core Panto. Acid
302	Kidney/Adrenal	Li 13	T3	C3	TMJ, A-P	3rd Cunei., Medial	Core Manganese
304	Thymus/Nose	G 33	L3	C3	Sphenoid	3rd Cunei., Lateral	Core Manganese
306	Spleen/Eye	H 7	L3	C3	Maxillary (A-P)	Prox. Phal. 5th Toe	Core Folic Acid
Group V - Muscles Affecting TMJ							
114	Adrenal/Lung(Bronchiole)	G 39	L5	Cr.V	Mandible	Prox. Phal. Gr. Toe	Core Adrenal
118	Posterior Pituitary/Thyroid	G 38	L4	Cr.V	Vomer	2nd Metatarsal	Core Pituitary
120	Thyroid/Posterior Pituitary	Si 5	L5	Cr.V	Lacrimal	Prox. Phal. 2nd Toe	Core Health Reserve
122	Thymus/Tonsil	Lv 4	C3	Cr.V	Occiput, Universal	Calcaneus	Core Magnesium
124	Pineal/Ovary-Testicle	St 38	L4	Cr.V	Occiput, Universal	Calcaneus	Core Ovary/Orchic
126	Ovary-Testicle/Parathyroid	St 41	L1	Cr.V	Palatine	1st Metatarsal	Uter-X/Prosta-X
110	Pancreas(Sugar)/Pharynx	B 56	T5	Cr.V	Parietal, Bulge	Talus	Core Niacin
112	Pineal/Pancreatic Duct	Tw 10	L3	Cr.V	Vomer	Second Metatarsal	Core Niacin

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group VI - Muscles Affecting Eyes							
130	Kidney/Stomach(Special Cells)	G 1	T2	Cr. III	Lacrimal	Prox. Phal. 2nd Toe	Core C-TR
134	Liver/Gallbladder Duct	G 38	T3	Cr. III	TMJ, A-P	3rd Cuneiform	Core Vitamin A
136	Pancreas(Sugar)/Urethra	Sp 5	T7	Cr. VI	Frontal, Internal	Prox. Phal. 3rd Toe	Core Carbo Gest
138	Liver/Lymphatics of Gallbladder Duct	G 28	T12	Cr. IV	Mandible	Prox. Phal. Gr. Toe	Core Dent Matrix
140	Liver/Lymphtaics of Jejunum	Lu 4	T3	Cr. III	Parietal, Bulge	Talus	Core Magnesium
132	Stomach(Spec. Cells)/Lymph. of Gallbl.	Si 14	T3	Cr. III	Temporal, External	Cuboid, Lateral	Core Rutin
Group VII – Muscles Affecting Throat							
370	Uterus-Prost.(Dig. Por.)/Pancreas(Protein)	Sp 4	L3	C3	Temporal, Internal	Cuboid, Inferior	Core Folic Acid
380	Liver/Pharynx	Tw 2	L3	C2	TMJ, M-L	Distal Phal. 4th Toe	Core Thyro
362	Pancreatic Duct/Parotid Gland	St 44	T4	C2	Palatine	1st Metatrasal	Core Potassium

SHOULDER EXAM CORRELATIONS

Muscle #	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group I – Muscles of the Shoulder (Arm Straight)							
410	Ear(External)/Pancreatic Duct	Li 13	T11	C6	Temporal, Internal	Cuboid, Inferior	Core RNA
412	Posterior Pituitary/Liver	H 7	L2	C6	Sphenoid	Third Cuneiform, Lateral	Core Adrenal
402	Lung/Ovary-Testicle	St 33	C5	C5	Parietal, Bulge	Talus	Core Thyro
460	Uterus-Prostate (Digestive Portion)/Heart	G 30	C5	C7	Parietal, Bulge	Talus	Core Folic Acid
462	Mammary/Liver	Sp 4	T8	T1 L	Frontal, Internal	Navicular	Core Carbo Gest
464	Anterior Pituitary/Vagina-Penis	Cx 7	C2	C7	Temporal, External	Cuboid, Lateral	Core Panto. Acid
414	Lymphatics of Ileum/Pancreas(Sugar)	Tw 15	T7	C7	Sphenoid	Third Cuneiform, Lateral	Core Zinc
416	Parotid Gland/Lymphatics of Duodenum	Lu 5	C6	T1 L	Sphenoid	Third Cuneiform, Lateral	Spore-X
418	Urethra(Membranous Port.)/Colon(Trans.)	Tw 3	L5	T1 R	TMJ (A-P)	Third Cuneiform, Medial	Core Potassium
306	Spleen/Eye	H 7	L3	C3	Maxillary (A-P)	Prox. Phal. Fifth Toe	Core Folic Acid
404	Thymus/Lung	Tw 6	T5	C7	Maxillary (M-L)	Third Metatarsal	Thym-X
392	Bladder/Kidney	Tw 10	C7	C4	Parietal, Bulge	Talus	Core Manganese
390	Posterior Pituitary/Internal Ear	Gv 22	C7	C3	Frontal, Internal	Prox. Phal. Third Toe	Core Pituitary
422	Anterior Pituitary/Esophagus	B 60	T8	C5	Ethmoid	First Cuneiform	Thym-X
420	Thymus/Maxillary Sinus	St 43	T7	C5	Mandible	Prox. Phal. Great Toe	Thym-X
444	Kidney/Nose	Lv 10	C5	C7	Styloid	Distal Phal. Great Toe	Core Inositol
446	Bladder/Lung	B 62	T5	C7	Vomer	Second Metatarsal	Pare-X
408	Kidney/Lung	St 28	T5	T1 R	Temporal, External	Cuboid, Lateral	Core Vitamin E
406	Lung/Heart	G 21	L2	T1 L	TMJ (A-P)	Third Cuneiform	Core Lung
Group II – Muscles of the Shoulder (Arm Flexed)							
400	Liver/Mammary	St 36	T8	C5	Vomer	Second Metatarsal	Core Liver
398	Posterior Pituitary/Liver	Lv 8	T7	C5	Frontal, Internal	Prox. Phal. Third Toe	Core Liver
394	Frontal Sinus/Spleen	Li 15	L3	C4	Occiput, Lateral	Fifth Metatarsal	Core Methionine
396	Parathyroid/Parotid Gland	Li 14	L2	C4	Temporal, External	Cuboid, Lateral	Pare-X
430	Thyroid/Ovary-Testicle	Li 4	T3	C6	Nasal	Distal Phal. Second Toe	Core Thyro
428	Thymus/Gallbladder Duct	Lu 6	L1	C6	Frontal, External	Navicular	Thym-X
426	Eye/Gallbladder	Tw 5	T4	C6	TMJ (A-P)	Third Cuneiform	Splen-X
424	Thyroid/Lymphatics of Lung	Cx 1	C5	C6	Nasal	Distal Phal. Second Toe	Splen-X

Muscle #	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group II (continued) – Muscles of the Shoulder (Arm Flexed)							
436	Heart/Posterior Pituitary	Tw 9	T4	C7	Maxillary (M-L)	Third Metatarsal	Core Pancreas
438	Sphenoid Sinus/Thyroid	Tw 3	T3	C7	Temporal, Internal	Cuboid, Inferior	Spore-X
440	Esophagus/Heart	Cx 4	T2	C6	Palatine	First Metatarsal	Core Potassium
442	Pancreatic Duct/Uterus-Prostate	Tw 5	C4	C6	Maxillary (M-L)	Third Metatarsal	Uter-X/Prosta-X
432	Pineal/External/Ear	K 11	L3	C7	Occiput, Lateral	Fifth Metatarsal	Core Health Reserve
434	Vagina - Penis/Lung	St 40	T7	C7	Frontal, Internal	Prox. Phal. Third Toe	Spore-X
456	Lung/Adrenal	G 31	L2	C5	Sphenoid	3rd Cuneiform, Lateral	Core Carbo Gest
458	Stomach (Special Cells)/Lung	G 43	L4	C6	Sphenoid	3rd Cuneiform, Lateral	Core Liver
452	Spleen/Lung	Lv 3	C5	C6	Sphenoid	3rd Cuneiform, Lateral	Core Lung
454	Lung/Gallbladder	Cx 3	T8	C6	Sphenoid	3rd Cuneiform, Lateral	Thym-X
450	Eye/Colon	Lv 6	L2	C6	Maxillary (A-P)	Prox. Phal. Fifth Toe	Core Ileoduodenal
448	Spleen/Tonsil	Lu 8	C6	C6	Nasal	Distal Phal. 2nd Toe	Thym-X

ELBOW, FOREARM, WRIST, THUMB AND FINGERS EXAM CORRELATIONS

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group I – Muscles of the Elbow and Forearm							
470	Duodenum(Super. Port.)/Eustachian Tube	B 50	L4	T1	Lacrimal	Prox. Phal. Second Toe	Core Health Reserve
466	Kidney/Spleen	Sp 9	T12	C5	Styloid	Dist. Phal. Great Toe	Core Folic Acid
476	Bladder/Parotid Gland	Lv 11	L5	T1 R	Palatine	First Metatarsal	Core Niacin
472	Sublingual Gland/Gallbladder	G 39	T10	T1 L	TMJ M-L	Distal Phal. Fourth Toe	Pare-X
474	Thyroid/Uterus-Prostate	Li 2	T3	T1 L	Mandible	Prox. Phal. Great Toe	Core Potassium
482	Kidney/Lung	Tw 14	T8	C6	Temporal, External	Cuboid, Lateral	Core C-TR
480	Ileum(Peyer's Patches)/Ovary-Testicle	St 32	L5	C7	Maxillary(A-P)	Prox. Phal. Fifth Toe	Pare-X
478	Tonsil/Jejunum	Tw 9	L5	C6	Temporal, Internal	Cuboid, Inferior	Core Thyro
468	Ovary-Testicle/Colon(Sigmoid)	Tw 8	L5	C6	TMJ A-P	Third Cuneiform	Core Health Reserve
490	Pancreatic Duct/Lymph. of Gallbladder	St 40	T7	T1 L	Sphenoid	Third Cuneiform	Pare-X
488	Bladder/Heart	Li 9	L4	T1 L	Styloid	Distal Phal. Great Toe	Core Manganese
492	Ovary-Testicle/Ear(Internal)	K 3	T5	C7	Frontal, External	Navicular	Core B6
494	Uterus-Prostate/Liver	Tw 6	T2	C7	TMJ-A-P	Third Cuneiform	Spore-X
484	Adrenal/Vagina-Penis	B 64	T9	C7	Temporal, Internal	Cuboid, Inferior	Core Folic Acid
486	Kidney/Posterior Pituitary	St 14	T2	C7	Maxillary(M-L)	Third Metatarsal	Core Pituitary
496	Stomach(Fundus)/Eustachian Tube	Li 6	T5	C7	Frontal, Internal	Prox. Phal. Third Toe	Spore-X
498	Sinus(Maxillary)/Kidney	St 18	T12	T1 R	Frontal, Internal	Prox. Phal. Third Toe	Core Kidney

WRIST, THUMB AND FINGERS EXAM CORRELATIONS

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group II – Muscles of the Wrist							
500	Kidney/Lymphatics of Throat	Si 7	T9	T1 L	Parietal Bulge	Talus	Core Carbo Gest
502	Gallbladder/Esophagus	B 66	L3	C7	Mandible	Prox. Phal. Great Toe	Core Pant. Acid
504	Thyroid/Adrenal	H 2	L3	T1 L	Maxillary (M-L)	Third Metatarsal	Core Thyro
506	Ovary-Testicle/Ileum	Sp 9	T9	T1 L	Sphenoid	Third Cuneiform, Lat.	Core B6
508	Uterus-Prostate/Nose	Lu 11	L3	T1 R	Temporal, Internal	Cuboid, Inferior	Uter-X/Prosta-X
510	Colon(Appendix)/Stomach(Pyloric Vestib.)	K 24	T6	C6	Mandible	Prox. Phal. Great Toe	Core Ileoduodenal
512	Kidney/Uterus-Prostate	G 30	T2	C7	Temporal, Internal	Cuboid, Inferior	Uter-X/Prosta-X
514	Duodenum(Special Cells)/Nose	St 43	T1	C7	Maxillary(M-L)	Third Metatarsal	Core Thyro
516	Stomach(Pyloric Antrum)/Kidney	B 2	C5	C7	Parietal Descent	Distal Phal. Third Toe	Core Niacin
518	Pancreatic Duct/Liver	K 6	T11	C6	Temporal, External	Cuboid, Lateral	Core Iron
Group III – Muscles of the Thumb (Distal Phalanx)							
528	Pancreatic Duct/Stomach(Pyloric Gland)	K 9	T7	C7	Occiput, Universal	Calcaneus	Spore-X
520	Anterior Pituitary/Gallbladder Duct	Lu 3	T6	C7	Maxillary(A-P)	Prox. Phal. Fifth Toe	Spore-X
522	Lymph. of Thoracic Duct/Ovary-Testicle	Lv 6	L2	C6	Sphenoid	Third Cuneiform, Lat.	Core Carbo-Gest
Group IV – Muscles of the Thumb (Proximal Phalanx)							
536	Bladder/Sinus(Nasal)	Lu 8	L2	T1 L	Parietal Bulge	Talus	Core Folic Acid
544	Heart/Ileum	Sp 5	T11	T1 L	Ethmoid	First Cuneiform	Core RNA
546	Anterior Pituitary/Lymphatics of Colon	Tw 15	C6	T4	Sphenoid	Third Cuneiform, Lat.	Core Niacin
534	Adrenal(Cortex)/Adrenal(Medulla)	B 51	T10	T4	Frontal, External	Navicular	Core Manganese
542	Pancreas(Sugar)/Gallbladder Duct	G 35	C3	C7	Parietal Bulge	Talus	Core Zinc
526	Adenoid/Pancreatic Duct	St 24	C2	C7	Parietal Bulge	Talus	Core Rutin
524	Gallbladder Duct/Liver	K 2	C6	T1 L	Vomer	Second Metatarsal	Core Inositol
Group V – Muscles of the Thumb (First Metacarpal)							
538	Posterior Pituitary/Ovary-Testicle	H 6	T5	C6	Palatine	First Metatarsal	Core Inositol
540	Pharynx/Pineal	B 51	T9	C6	Occiput, Universal	Calcaneus	Core Dent Matrix
532	Pancreas(Sugar)/Ovary-Testicle	Tw 4	T1	C7	Vomer	Second Metatarsal	Core Rutin
530	Lymphatics of Face & Neck/Nose	Lu 9	T7	T2	Maxillary (M-L)	Third Metatarsal	Core B6

Group VI – Muscles of the Fingers (Extensors)							
550	Pancreas(Sugar)/Spleen	Tw 9	T8	C6	Parietal Bulge	Talus	Spore-X
548	Ovary-Testicle/Pharynx	Cx 7	T6	C7	Glabella	Fourth Metatarsal	Core B6
554	Pancreas(Sugar)/Kidney	Li 13	C4	C7	Temporal, External	Cuboid, Lateral	Spore-X
552	Lung/Posterior Pituitary	St 37	L1	T1 L	Lacrimal	Prox. Phal. Second Toe	Core Brain/Spinal
570	Anterior Pituitary/Bladder	Lv 9	T10	T1 R	Sphenoid	Third Cuneiform, Lateral	Core Thiamine
568	Spleen/Sinus(Frontal)	Si 8	T6	T1 L	Maxillary(M-L)	Third Metatarsal	Core Carbo Gest
566	Pancreatic Duct/Esophagus	St 39	T7	T1 L	Maxillary(M-L)	Third Metatarsal	Super EPA
564	Heart/Thymus	K 25	T1	T8	Frontal, External	Navicular	Core Heart
Group VII – Muscles of the Little Finger							
560	Bladder/Spleen	Li 15	L4	T2	Palatine	First Metatarsal	Core Magnesium
556	Pancreas(Sugar)/Lymphatics of Jejunum	G 40	T5	T1 L	Vomer	Second Metatarsal	Core Vitamin A
572	Nose/Lymphatics of Colon	K 8	C6	T4	Ethmoid	First Cuneiform	Core Pancreas
590	Spleen/Larynx	Tw 14	T2	T1 L	Occiput, Lateral	Fifth Metatarsal	Core Rutin
580	Pancreas(Sugar)/Lymph. of Gallbladder Duct	G 22	L1	T1 L	Ethmoid	First Cuneiform	Core Dent Matrix
582	Lymphatics of Colon/Adrenal	H 4	T2	C7	Ethmoid	First Cuneiform	Core Heart
584	Ileum(Special Cells)/Lung(Bronchiole)	Si 7	L3	T1 L	Inferior Conchae	Prox. Phal. Fourth Toe	Core Ileoduodenal
588	Pancreas(Protein)/Uterus-Prostate	Cx 3	T2	T1 R	Parietal Bulge	Talus	Core Bone Matrix
586	Jejunum/Sinus(Maxillary)	St 18	T10	T1 L	Inferior Conchae	Prox. Phal. Fourth Toe	Core Bile
Group VIII – Muscles of the Ring Finger							
560	Bladder/Spleen	Li 15	L4	T2	Palatine	First Metatarsal	Core Magnesium
556	Pancreas(Sugar)/Lymphatics of Jejunum	G 40	T5	T1 L	Vomer	Second Metatarsal	Core Vitamin A
574	Parathyroid/Lymph. of Rectum(Anal Canal)	Lv 3	L3	T2	Frontal, External	Navicular	Core Parathyroid
592	Ovary-Testicle/Pharynx	K 1	T10	T2	Occiput, Lateral	Fifth Metatarsal	Core Bone Matrix
Group IX – Muscles of the Middle Finger							
562	Jejunum/Liver	Si 1	T6	T1 L	Styloid	Distal Phal. Grt. Toe	Black Curr. Seed Oil
558	Tonsil/Anterior Pituitary	Li 8	L2	T1 R	Glabella	Fourth Metatarsal	Core Pituitary
576	Parathyroid/Ovary-Testicle	Tw 17	T7	T2	TMJ A-P	Third Cuneiform, Med.	Core B6

Group X – Muscles of the Index Finger							
562	Jejunum/Liver	Si 1	T6	T1 L	Styloid	Distal Phal. Grt. Toe	Black Curr. Seed Oil
558	Tonsil/Anterior Pituitary	Li 8	L2	T1 R	Glabella	Fourth Metatarsal	Core Pituitary
578	Posterior Pituitary/Liver	Gv 23	C2	T2	TMJ A-P	Third Cuneiform, Med.	Core Liver
594	Ileum(Special Cells)/Sublingual Gland	Sp 13	L5	T1 L	Vomer	Second Metatarsal	Spore-X

Group XI – Muscles of the Hand							
596	Gallbladder/Sublingual Gland	Cv 9	T9	T4	Parietal Bulge	Talus	Core Vitamin E

With the patient prone, doing measurements after postural analysis and manual muscle testing, and then palpating and correcting the spinal fixations followed by re-measurement, I felt that improved lower thoracic function should result in a shortening of the distance between T1 and T12 as well as an improvement in posture. This I found to be supported in most cases by findings on a series of patients I treated.

MATERIAL AND METHODS:

A plastic tape measure was used that had markings in both centimeters and inches. The patient was first evaluated posturally posterior to anterior, and from the side (laterally), to observe for thoracic spine kyphosis as well as abduction and elevation of the scapulae(2). The test for inhibition of the lower trapezius was done as described in "Applied Kinesiology, Volume 1", page 366, and "Applied Kinesiology Synopsis", page 307. The evaluation and correction of fixations was as described in "Applied Kinesiology Synopsis", page 82 - 84. To determine the position of T1 and T12, I passively moved the patient's head into extension a number of times while palpating for the spinous that moves forward with extension. This I took as being C7, and then the first spinous tip below it, which moved very little, I took to be T1. As for T12 I would palpate for the last rib (the 12th. rib) and follow it's course back to the spine at T12. I would use a red marker and mark the tip of the spinous process of T1 and T12. I then measured them in 3 phases of respiration; in normal, quiet respiration which I referred to as neutral, in full inspiration, and full expiration.

BILATERAL LOWER TRAPEZIUS INHIBITION RELATED TO DORSO LUMBAR FIXATIONS AND INCREASED POSTURAL KYPHOSIS CORRECTED BY A FIXATION RELEASE.

HANS W BOEHNKE,D.C.

ABSTRACT:

One of the most common findings in my practice and that of many colleagues I know, is the occurrence of fixations at the junction of the thoracic and lumbar spines. In most cases, according to applied kinesiology literature,(1)(2)(3) the patient will have an increased thoracic kyphosis and a bilateral weakness of the lower fibres of the trapezius muscles on manual muscle testing. As applied kinesiology is frequently challenged on the reproduceability and accuracy of manual muscle testing, I felt that an objective measurement of a postural change related to improved lower trapezius function would be of value. This would then give some additional support to the treating doctor's findings of improved muscle function. This I found could be done in most cases by using a simple measuring tape.

INTRODUCTION:

In a copy of "Joint Motion. Method of Measuring and Recording" published by the American Academy of Orthopaedic Surgeons, in 1965(4), a tape measure method was shown for measuring true motion of the spine in flexion. In that, they measured C7 - S1 in the erect and forward flexion modes. They found a 4 inch increase in forward flexion, stating that 1 inch occurred in the thoracic spine, and 3 inches in the lumbar spine as the spinous tips separated. I felt that this type of measurement could also be applied in a more finite way by measuring just the length of the thoracic spine from T1 - T12.

DIVISION II - CRITICAL REVIEW PAPERS

CASE REPORT (continued)

December 6, 1988 - occiput indicated L.P.
C-6-R.P., sacral fix, CX two pointed
with L - Tapped CX-LUO pt.

By December 13th he reported pain free for two nights. Two days later pain returned.

Times of remission slowly extended, recurrent spells were of short duration though the pain intensity seemed the same.

CONCLUSION: Finally, by mid '91 all signs of Peyronie's Disease were gone. Medical urological exam confirmed that all plaque had disappeared.

Though this was a protracted case, I believe, in view of the poor results of alternative methods, further study and application of AK is warranted.

References:
(1) Dorlands Medical Dictionary
27th edition
(2) The Merck Manual
14th edition
(3) Chiropractic Office Procedural Manual
Fred Stoner, D.C.
(4) Applied Kinesiology Synopsis
David S. Walther

PEYRONIE'S DISEASE

George N. Koffeman, D. C.

ABSTRACT: Peyronie's Disease is defined as an induration of the corpora cavenosa of the penis producing a fibrous chordee called also fibrous cavernitis, penis plasticus and penile induration.(1) This paper illustrates the positive input of standard AK procedures on one patient contrasting it with standard allopathic methods administered over a 6 year period.

INTRODUCTION: November, 1988, Mr. G., age 70 presented with a diagnosis of Peyronie's Disease. His condition had become progessively worse over a six year period. He had consulted three urologists and had the diagnosis confirmed at the U. of M. hospital. He had been through a "pain clinic" and was taking pain medication and Valium. Despite this, he reported 4 to 6 spontaneous erections per night with severe pains. He estimated that he slept no more than two hours per night in aggregate.

This condition rates two paragraphs in the Merck Manual. It is defined as dysplasia of the cavernous sheaths, fibrous thickening and contracture of the investing facia of the corpora. Cause unknown.

Medical Treatment:

(1) Surgical removal of plaque and replacement with patch graft (may improve problem or make it worse!).
(2) Local injections of high potency Corticosteroids.
(3) Ultrasound.

These treatments are usually ineffective and, barring spontaneous resolution over months, the condition is considered incurable. (2)

DISCUSSION: Over a three year period of removing N root interference, it gradually became clear that a pattern was emerging relative to the patient's symptomatology. Whenever he presented with an exacerbation the findings were CX, CX/L (tap CX-6), or CX/B (tap B58 or CX-6), etc., adjust T-4 and sometimes T-7. One of these combinations was always there and was easily corrected.

Finally, by mid 1991, all signs of Peyronie's were gone. (3)(4)

CASE REPORT: Patient had disc surgery approximately ten years previous to onset. General health was good.

Examination: BP $\frac{168}{90}$ Gluteus medius weakness as a 5l percenter therapy localized at CX alarm point, N.L. and N.V. pts. Feet subluxated. Sacrum had both a fixation and a subluxation.

X-rays - nothing remarkable, slight short left leg with pelvic tilt.

First adjustment November 29, 1988. Gluteus medius. CX, N.V., N.L.
T-4 associate vertebra of CX
T-7 lovett
Sacral fixation.
A 3mm heel lift in left shoe. Other adjustments followed along this line of approach.

SUMMARY OF PROCEDURES

1. Right & left piriformis muscle test strong

YES | NO

Check factors associated with a weak piriformis.

2. Tapping sedation points of cirulation-sex weakens each piriformiis muscle.

NO | YES

Normally temporary inhibition of piriformis

3. Tap luo point of cirulation- sex meridan or triple warmer & repeat step 2.

4. One at a time perform the following & repeat step 2.
Have patient taste sugar, alcohol , baking soda, caffeine, decaffinated coffee, and tea.
Have patient perform an anaerobic / aerobic challange .
Have patient recall several images of stressful events.
If piriformis now stays on during screening procedure, repeat step 3 until list is completed.

Treatment:
See if a source of (TPP)thiamine pyrophosphate or magnesium restores normal functioning at step 2.
Supply appropriately, either TPP or magnesium and review those lifestyle factors with patient that caused dysfunction to return. If needed, tap luo point of circulation sex or triple warmer meridian.

Desensitize patient by using Schmitt's Injury Recall Technique - tug forward on back of head while it is fully flexed +
1. Tasting factor(s) that caused a return;
2. Therapy localize emotional neurovascular point while recalling stress;
3. Therapy localize neurolymphatic point that negates anaerobic or aerobic challenge.

Normally the muscle should be initially strong but on tapping the sedation point it temporarily weakens. If it remained strong, tap the luo point for either the circulation sex meridian or the triple warmer meridian and retap the sedation point. Now the muscle should respond by weakening.

If the luo point need to be reset, introduce some of the common reasons for increased need of thiamine. One by one have the patient taste sugar, caffeine, decaffeinated coffee, tea, alcohol and baking soda. Have the patient think of some current stress in his/her life. Have the patient perform some anaerobic or aerobic challenge.(4) After each see if the piriformis still responds to its sedation point by temporally weakening, restimulating the luo points as needed to regain your indicator. See if tasting a source of thiamine pyrophosphate and or magnesium restores workings of the sedation point or if they cause a inhibited muscle to strengthen.

Now restimulate the points as the patient insalivates the substance & advise patient to abstain from the substance or use Dr. Schmitt's injury recall technique to nullify substance, emotional neurovascular or neurolympathic points which negate the effects of the exercise. Supply patient with thiamine pyrophosphate and or magnesium until it is no longer needed.

RESULTS

Of the 20 people tested 17 showed an inappropriate response to tapping of the circulation sex sedation point. All seventeen had a recurrence of overactivity when stimulated with one of the factors. Fourteen had more than one. All seventeen responded to a source of thiamine pyrophosphate and three also responded to magnesium citrate.

DISCUSSION

Although the case size was small it points to the importance of a healthy lifestyle as a cause of overactivity of the circulation sex meridian. In the future I'm sure other factors which either increase need or inhibit the conversion of thiamine to thiamine pyrophosphate will be discovered.

CONCLUSION

Many patient's lifestyles have factors which are contributing to their complaints. Muscle testing along with a good case history are ways in which to help pinpoint the causes of recurring clinical signs.

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Lifestyle Factors to Consider in Overactive Circulation-Sex Meridian and the Need for Thiamine Pyrophosphate

John N. Kane D.C.

ABSTRACT

Muscle testing was used to evaluate some possible causes for the need of thiamine pyrophosphate, the active form of B1 and over activity of the circulation sex meridian.

INTRODUCTION

Goodheart has shown that a large percentage of patients require rebalancing of the circulation sex meridian and that it is often associated with a need for thiamine pyrophosphate. (1)

There are many factors which require more thiamine in the diet. There are some foods which have anti-thiamine activity- Raw fish, blueberries, red cabbage, red chickory, baking soda, black currants, and brussels sprouts.(2) Most people will not consume enough of these foods on a daily basis for it to be a problem.

Coffee and tea however are consumed daily and in some people many times a day. Coffee contains chlorgenic acid and the tannins in tea, both have anti-thiamines activity.(2, 3)

Caffeine inhibits phospodiesterase, an enzyme that breaks down the secondary messenger CAMP. It therefore prolongs the effects of CAMP which were stimulated by hormones like epinephrine or T3, which increases metabolism. (6)

Other factors which increase energy requirements also put demands on thiamine supply. These include hyperthyroidism, pregnancy, lactation, administration of thyroid hormone, stress and exercise. (7) Exercise not only requires the creation of more energy but the metabolism of lactic acid requiring thiamine pyrophosphate (TPP).

Alcohol has two effects on thiamine. It is known to decrease B1 absorption from the small intestine. (3) Alcohol is metabolized to Acetyl-CoA which requires thiamine pyrophosphate, as a cofactor.

In liver only - Alcohol------NAD---------->acetaldehyde----->AcetylCoA
ROH Deydrogenase NAD TPP

Excess sugar supplies the fuel to be burned without the nutrients needed to metabolize it. Sugar is eventually broken down to pyruvic acid which needs thiamine pyrophosphate (TPP) to be converted to Acetyl-CoA.

Pyruvic acid----------*>Acetyl-Coa + CO2*
TPP/Lipolic Acid/Fad/Nad/HS-Coa(B5)
Pyruvate Dehydrogenase Complex

Synthesis of thiamine pyrophosphate requires magnesium. Principle sites of phosphorylation are the liver, the RBC, & the cells of the cerebral cortex. (8) Conversion compromised in liver disease by reducing thiamine availability.(7)

Thiamine---------ATP----Mg---------->Thiamine pyrophosphate (TPP)
Thiamine Pyrophosphokinase

METHODS

By using Dr. Goodheart's over active circulation sex technique we can see which factors are linked to the patients need for thiamine pyrophosphate. (1) Twenty people were examined . Fourteen were women and six were men. Ages ranged from twenty one to sixty four years.

Procedure is simple. Screen for the need for the over activity of the circulation sex meridian by tapping the sedation point and testing an intact associated muscle with that meridian, the piriformis.

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and go on to step nine.
8. Use temporal tapping or "priority" hand mode to determine the priority therapy.
9. Click heels together. Leave together for a second or two, then separate feet.
10. Repeat the procedure beginning with step two, until the south polar challenge weakens the indicator muscle from step one.

Note: A "yes" answer to a question posed by a hand mode is an indicator changing its strength from either strong to weak, or weak to strong. When using hand modes, the examiner must be touching the patient with at least one of his hands.

CONCLUSION

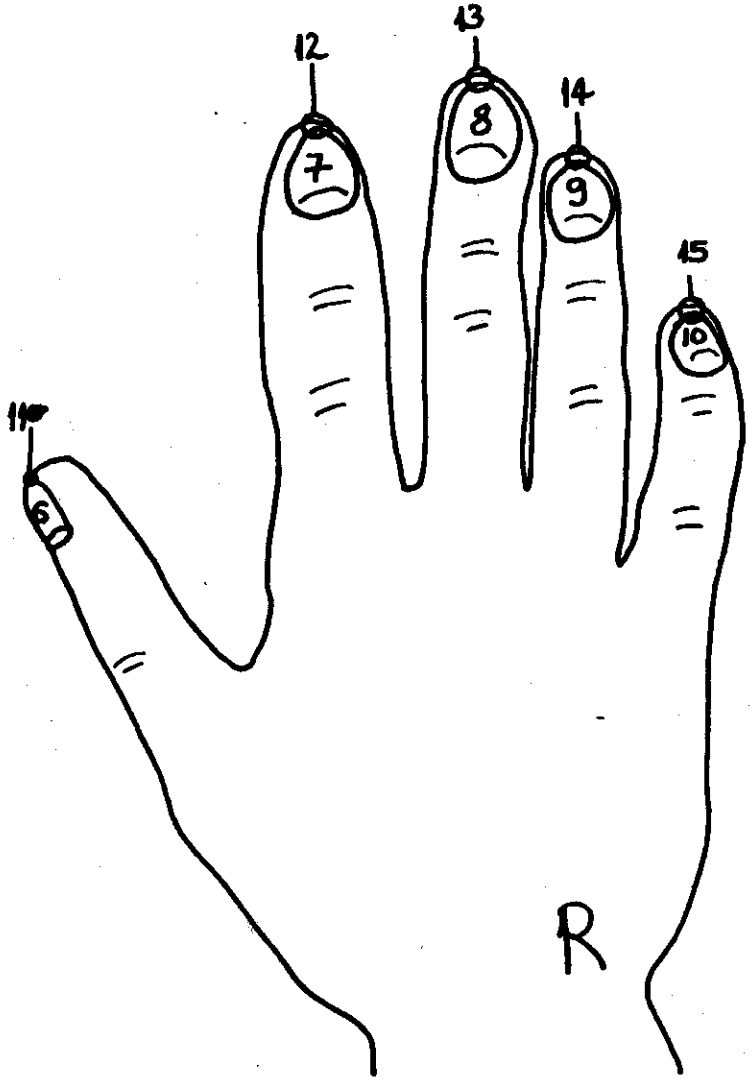
The placing of the north pole of a 3,000 gauss ceramic magnet over the muscle belly of a muscle to be tested should weaken an intact indicator muscle. The mechanism involved is presumably the same as pushing together the spindle cells of the muscle belly. The test makes use of the fact that north polar energy causes muscle fibers to contract. This method can be used periodically during an exam/treatment to test the reliability of an indicator muscle.
The placing of the south pole of a 3,000 gauss ceramic magnet over a muscle belly, and then testing a previously determined intact indicator muscle, should weaken the indicator muscle if the muscle over which the south pole has been placed is normal. The mechanism for this response is not as clear cut as for a north polar challenge. Presumably, the sedating, relaxing properties of the south pole interact with the muscle spindles. But how this causes a remote muscle of the body to weaken is unknown and needs to be investigated.
The south polar challenge is most useful in detecting "51%-er" muscle dysfunctions. The hand moding and lock in procedures can be utilized to determine how to correct that dysfunctioning muscle. The toxicity and the acupressure/emotion hand modes are the modes that this examiner has found to occur most ofter in normalizing south polar challenges.

For example, 1-2 means thumb pad to index finger pad; 1-G means thumb pad to first crease of pinky, radial side.
Example: The examiner starts with his heels touching each other and places the south pole of a 3,000 gauss ceramic magnet over the belly of the left vastus lateralis. Now the examiner tests the right PMS. During the actual muscle test of the right PMS, if the right PMS remains strong, then the examiner spreads his feet. Remove the magnet from the muscle belly. Retest the right PMS. It should now test weak. At this point, revert to hand modes to see which factor will strengthen the indicator muscle (in this case, the right PMS.) Apply the therapy indicated. Re-challenge the belly of the vastus lateralis with the south pole. Retest the right PMS. If the right PMS remains strong, repeat the hand mode procedure for a second layer, etc. When challenging the vastus lateralis belly with the south pole causes weakening of the indicator muscle, we would conclude that we are done. However, to find out if we are really done, we need to ask the body if there is more to do, by placing the fingers of one of the examiner's hands into the "more mode" position (index and middle finger pads on proximal and distal sides of distal joint of posterior thumb), and then retest the right PMS. If the right PMS suddenly becomes strong, click heels together and then quickly spread them a few inches. This locks in the "more mode" finding. Now go through the various hand modes in table B until the test indicator weakens. Fix the muscle using the indicated therapy. If more than one therapy is indicated, temporal tapping or use of a "priority" hand mode can be used to determine the priority therapy. To do this, lock in the first hand mode that strengthens the indicator by quickly clicking your heels together and separating your feet several inches. Release the hand mode, and now temporal tap both tempero-sphenoidal lines at the same time (this must be done while the priority hand mode is strengthening the indicator.) Retest the previously strong indicator. If it weakens, the therapy indicated by the hand mode is a priority. Repeat the south polar challenge of the vastus lateralis using the "more mode" again. If the right PMS remains weak, now there is "no more" to do at this time. Most therapies indicated by the twenty hand modes follow regular applied kinesiological procedures. Two, however, do not: the toxicity mode and the acupuncture/emotion mode. These two, in my experience, also show up the most. For the toxicity mode you need to determine, again using hand modes and pause locking, whether the toxicity is chemical or emotional. You also need to find out which organ/meridian system is involved. For chemical toxicity, tap the gait point associated with that organ/meridian system. For emotional toxicity and acupressure/emotion use the neuro-emotional techniques developed by Scott Walker, D.C.[10]

Summary of Procedure to Correct South Polar Challenge

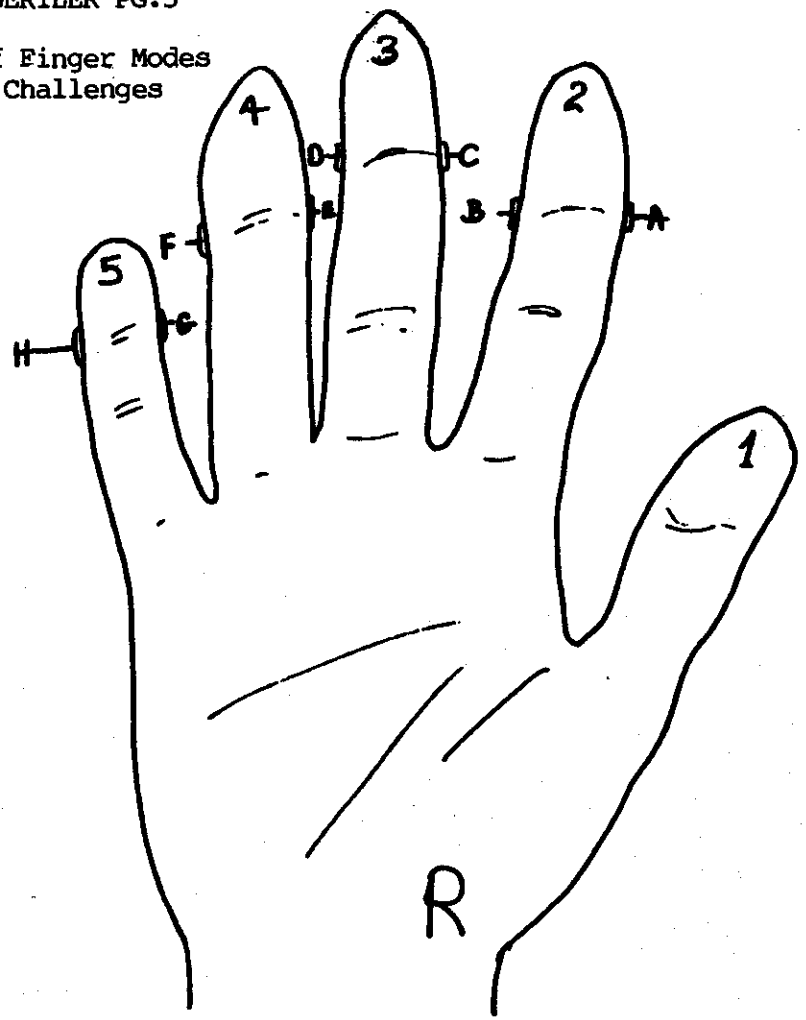
1. Choose an indicator muscle which tests strong in the clear and weakens with a north polar challenge.
2. Examiner keeps heels together and places south pole of magnet over belly of muscle to be tested.
3. Retest indicator. If weak,the muscle that the magnet is on is normal. Go back to step two and choose a different muscle. If strong, the examiner spreads his heels several inches and goes on to step four.
4. Remove the south pole magnet from muscle belly and the body.
5. Retest the indicator muscle. It should test weak.
6. While the indicator is testing weak, the examiner quickly clicks his heels together and then spreads his heels several inches apart. This sustains the weakness of the test indicator muscle.
7. Using hand modes, discover which one(s) strengthens the indicator. Apply the appropriate therapy. If more than one hand mode strengthens the indicator, go on to step eight. If only one hand mode strengthens the indicator, skip steps eight

Chart A: Graphic Representation of Finger Modes
Used in Correcting South Polar Challenges



Numbers 6,7,8,9,10 represent the nail.

Numbers 11, 12,13,14,15 represent the pad plus the nail edge.



Numbers 1,2,3,4,5 represent finger pads.

A,B,C,D,E,F,G,H represent sides of the fingers at the first crease.

BIOMAGNETIC MUSCLE CHALLENGE - GERTLER PG.4

Table B: Positions for Hand Modes in Correcting South Polar Challenge

1 - 2	Structure
1 - 3	Nutrition/Lymphatics/Chemistry
1 - 4	Emotions
1 - 5	Acupressure
1 - 7	Specific Muscle
1 - 8	Toxicity
1 - 9	Bachflower
1 - 10	Acupuncture
1 - 12	Intrinsic Spinal Muscles
1 - 13	Candida
1 - 14	Karma
1 - 15	Reflexology (hand, foot, NV, NL, S/r, gait, ear acupuncture)
1 - A	NL, NV of specific muscle
1 - B	Reactive Muscle
1 - C	Fascia
1 - D	Reactive Fascia
1 - E	Ligaments
1 - F	Tendons
1 - G	Subluxation
1 - H	Fixations

Priority Mode: Tip of 3rd finger to first crease of thumb
Pause Lock: Separate touching ankles during TL or challenge
Examiner Interference: TL emotional points on left and right forehead
"More Mode" (More to do?): Index and middle finger pads/tips on posterior thumb, distal and proximal to joint of thumb
Acupressure/Emotion: Thumb pad to pads of ring fingers and pinky

To correct a step one PMS, place the PMS in its normal test position. Now shorten the fibers as much as possible by putting the PMS in the most contracted position you can by obliquely adducting the arm across the chest. The examiner now pushes the arm into further oblique adduction as the testee resists and tries to move the arm into extension/abduction. Do this several times to fatigue the PMS. Retest with north pole over the muscle belly. It should test weak.

Example number two for correcting step one: The biceps do not weaken with a north polar challenge. Have the patient flex his arm as much as possible, and then try to extend his forearm while the examiner continues to apply pressure in the direction of flexion (contraction). Continue to pump the forearm into flexion to fatigue the biceps.

Once we have established an intact indicator muscle from step one, we can use step two to screen many muscles very quickly without actually manually muscle testing them. Like the tempero-sphenoidal line, this becomes a great time saver and it focuses you on where remaining muscle dysfunction remains. For example, suppose you have a patient who has been complaining of left knee problems, but you cannot find any subluxations or weak muscles in the clear. Regular applied kinesiological methods do not reveal dysfunctioning muscles. At this point, suspect an "all muscles strong" situation in and around the left knee. One by one test each muscle above and below the knee as outlined in step two: Place the south pole of a 3,000 gauss ceramic magnet over the belly of the left vastus lateralis and test the right PMS. If the right PMS does not go weak, the vastus lateralis is dysfunctioning. Do this for each muscle above and below the knee joint. That is, continue to place the south pole over the various muscle bellies and then test the right PMS. Make a list of the muscles that do not pass the south pole screening test.

CORRECTION FOR STEP TWO: Correcting all the muscle that have failed the south polar challenge in step two is a simple, but sometimes intricate, process. I utilize a series of hand modes and a neutral locking-in procedure (which evidently come from early clinical kinesiology notes) to ask the body what specifically needs to be corrected next in each dysfunctioning muscle.[8] This works by having the examiner use his own feet in a certain way to "lock in" a finding. In this way, continuous therapy localization or continuous muscle testing does not have to be done to see if a muscle will respond in a certain manner. To do the lock-in procedure, simply have the examiner spread his own feet when the muscle test or challenge procedure is being done. This will lock in a muscle response. This procedure is equivalent to a "still-frame pause" on a VCR. Once the picture, or muscle readout, is locked in we can study the picture and ask the body questions about it. The examiner's own heels have to be touching before the feet are spread. To get the body out of a "lock-in" simply have the examiner touch his heels together again. This entire locking-in procedure can be done using the patient's feet instead of the examiner's feet.

Hand Modes

Hand modes ask the body questions. One needs to know about twenty hand modes in total to clear most south pole challenges.[9] The accompanying chart and table explain the hand mode positions.

BIOMAGNETIC MUSCLE CHALLENGE - GERTLER PG.2

Table A: Various Effects of North and South Pole Magnetic Energy

Contracts (tonifies) muscles fibers	Expands (sedates), relaxes muscle fibers
Reduces inflammation	Increases inflammation
Slows metabolism	Increases metabolism
Decreases acute pain	Increases acute pain
Dissolves calcium deposits around arthritic joints	Softens hardened soft tissues, liquifies tissues
Sedates, contracts, closes acupuncture points	Tonifies, expands, opens acupuncture points
Cools	Warms
Alkalinizes	Acidifies
Slows growth; slows micro-organism multiplication	Increases protein, sugar and oil content in plants; increases micro-organism multiplication

Knowing that the north pole contracts muscle fibers while the south pole expands and relaxes muscle fibers can be used as the basis for an incredibly simple method to detect dysfunctional ("frozen," "locked," "hypervigilent") non-reliable test-indicator muscles.

PROCEDURE

1. Placing the negative, north pole of a 3,000 gauss magnet over the muscle belly of a muscle to be tested should WEAKEN that muscle if that muscle is normal. If the muscle doesn't weaken it is dysfunctional. (The muscle must test strong in the clear to begin with.)

2. Placing the positive, south pole of a 3,000 gauss magnet over the muscle belly of a muscle to be tested should WEAKEN any other muscle found to be normal in step one above. If it doesn't, the muscle over which the magnet is placed is dysfunctional.

Let's illustrate the procedure for step one by using, for example, the pectoralis major sternal muscle. First, test the right PMS in the clear. It must test strong in the clear to proceed. Second, place the north pole of a 3,000 gauss ceramic magnet over the muscle belly of the right PMS and retest the right PMS. If it weakens this is NORMAL. If it doesn't weaken, this is a dysfuntionally strong PMS and can be normalized by corrective procedures discussed next.

CORRECTION FOR STEP ONE: To quickly, albeit temporarily, correct a step one dysfunction, place the muscle to be corrected in its contracted position. Pump the muscle into further contraction while the testee attempts to bring the muscle into extension. Continue until the muscle reaches temporary fatigue.

THE BIOMAGNETIC MUSCLE CHALLENGE

BY

LARRY GERTLER, M.Ed., D.C.

ABSTRACT

The purpose of this paper is to show that north and south poles of 3,000 or more gauss magnets interact objectively and predictively with muscles and muscle spindles. These interactions can be used as a quick screening procedure to find hidden or overt dysfunctional muscles. Methods will be described and suggested to correct these dysfuntional muscles.

INTRODUCTION

Recently I happened to read an article entitled "Daniel David Palmer: The Frontier Years, 1845-1887."[1] It states that our forefounder was a magnetic healer before he discovered chiropractic. The term "magnetic" is used literally. He used magnets to heal his clients. Accounts of his early years show that he was successful enough as a magnetic healer to support his family. This fact, plus the recent use by Michael Lebowitz, D.C.[2] of the south pole of a magnet to enhance the body's response to nutritional challenges, has prompted me to submit this paper portraying how magnetic energy interacts with muscle testing. Goodheart in his 1977 workshop manual states that, "normally, placing 3,000 gauss magnets on any area of the body should cause no muscle weakness. The body should adapt to this."[3] Further investigation has led me to extend these conclusions.

The phenomenon of muscles "locking" or "freezing" or becoming suddenly hypervigilent -- i.e., becoming unable to be weakened by normal applied kinesiological methods such as spindle cell manipulation, therapy localization to a known active reflex, etc. -- became the subject of my investigation when, for example, I would find a normally strong anterior deltoid which, if the patient was a positive pineal-type patient, would weaken when I turned off the lights. But then, on some patients, before doing a mandibular spread to correct the pineal-related holographic mandibular fixation, the anterior deltoid would suddenly strengthen with the lights still off. I was relieved to find this phenomenon (of a muscle suddenly testing strong immediately after being tested weak) encountered by other practitioners as well. I believe Wally Schmitt, D.C. alludes to this phenomenon in his 1991 flow chart entitled "All Muscles Strong".[4] The situation I am describing, however, does not involve the entire body, but only the muscle in question. If all muscles are in fact found to be strong, the method described in this paper will allow the practitioner to quickly, albeit temporarily, reset any individually strong muscle to its normal functioning so that you can use this muscle as an intact indicator muscle during a single treatment. Correcting an all-muscles-strong situation would be advantageous to do if time permits.

DISCUSSION

Yin-Yang theory classifies the north pole of a magnet as the negative, yin pole and the south pole as the positive, or yang pole. North polar energy, among other things, contracts muscle fibers, cools and slows metabolism, alkalinizes, and reduces inflammation and pain. South polar energy, among other things, expands (sedates, relaxes) muscle fibers, increases metabolism, acidifies, vasodilates, increases fluid flow and pain.[5] Rats bred under north polar energy showed fewer, smaller, less aggressive offspring than normal. Rats bred under south polar energy showed bigger, heavier, more numerous and more aggressive offspring than normal.[6]

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

Patient: Patient 3-fixation 03/09/1993,Tue
asymmetry table, seated neutral in freq. band 100- 200Hz

%DF	PSD	NSD	NORM	uV	SITE	uV	NORM	NSD	PSD	%DF
-->	+	0.9	2.5	3.7	C1	4.1	2.8	1.0	+	11
22		1.0	2.6	3.0	C3	2.5	2.6	0.8		<--
29		0.8	2.6	3.0	C5	2.3	2.4	0.7		<--
-->		1.2	3.0	3.2	C7	3.3	2.7	1.0		2
-->	++	1.5	3.2	6.5	T1	6.9	2.8	1.1	+++	7
8	+++	1.0	2.9	7.5	T2	6.9	2.9	1.2	+++	<--
50	+++	1.1	3.2	6.7	T4	4.5	3.4	1.2		<--
-->	++	1.2	3.7	7.2	T6	7.5	3.8	1.3	++	5
-->	++	1.3	4.3	7.5	T8	8.9	4.5	1.4	+++	19
1	+++	1.2	4.3	8.9	T10	8.8	4.6	1.6	++	<--
21	+++	1.4	4.0	9.3	T12	7.7	4.1	1.3	++	<--
9	+++	1.4	3.9	10.0	L1	9.2	3.9	1.4	+++	<--
-->		1.3	3.3	2.3	L3	2.4	3.5	1.3		7
-->		1.6	3.3	2.6	L5	2.7	3.4	1.7		3
-->		1.4	2.9	2.4	S1	3.3	2.8	1.4		35

key: uV=scan NORM=normal data NSD=normal standard deviation
PSD=patient standard deviations from normal %DF=percent differ.
* = no normal data available for this site / freq.band

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

Patient: Patient 3 - fixation (continued)
comparison of one session, 1st most recent, on 03/09/1993,Tue
to another session, 2nd most recent, on 03/09/1993,Tue
freq. band is 100- 200Hz

LEFT SIDE uV	COMP	%diff	SITE	%diff	RIGHT SIDE COMP	uV
3.7	3.9	-6	C1	-4	4.3	4.1
3.0	4.9	-38	C3	-38	4.0	2.5
3.0	4.4	-32	C5	-36	3.6	2.3
3.2	5.3	-40	C7	-45	5.9	3.3
6.5	4.0	62	T1	18	5.9	6.9
7.5	5.0	48	T2	-33	10.4	6.9
6.7	8.2	-18	T4	-53	9.6	4.5
7.2	7.8	-7	T6	-1	7.6	7.5
7.5	7.9	-5	T8	14	7.8	8.9
8.9	6.8	31	T10	38	6.4	8.8
9.3	9.4	-1	T12	-3	7.9	7.7
10.0	9.3	8	L1	16	7.9	9.2
2.3	5.8	-61	L3	-31	3.5	2.4
2.6	2.7	-3	L5	10	2.5	2.7
2.4	3.7	-35	S1	24	2.6	3.3

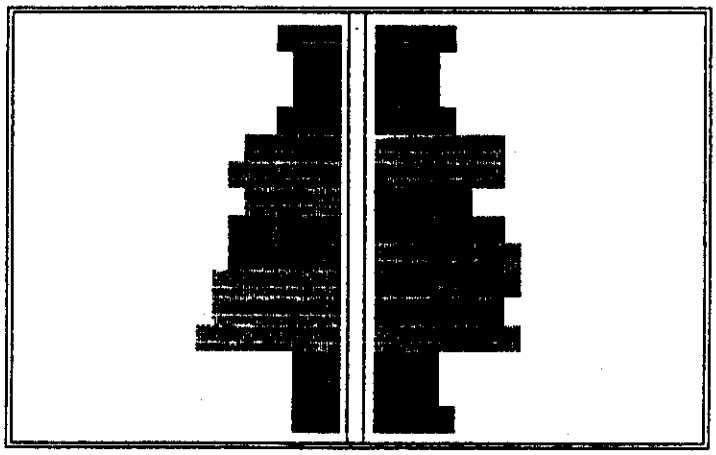
note: positive (+) percent (%) difference means comparison data is lower
values with a "??" were not taken during the comparison session

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

performed by: Post

Patient: Patient 3- fixation
SSN: height: 0.0 weight: 0.0
age: 0 sex: male ref.ID:
Static-Scan, spinal on 03/09/1993,Tue (1st most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 25.0 uV

left sites		right sites	
3.7uV	C1	C1	4.1uV
3.0uV	C3	C3	2.5uV
3.0uV	C5	C5	2.3uV
3.2uV	C7	C7	3.3uV
6.5uV	T1	T1	6.9uV
7.5uV	T2	T2	6.9uV
6.7uV	T4	T4	4.5uV
7.2uV	T6	T6	7.5uV
7.5uV	T8	T8	8.9uV
8.9uV	T10	T10	8.8uV
9.3uV	T12	T12	7.7uV
10.0uV	L1	L1	9.2uV
2.3uV	L3	L3	2.4uV
2.6uV	L5	L5	2.7uV
2.4uV	S1	S1	3.3uV



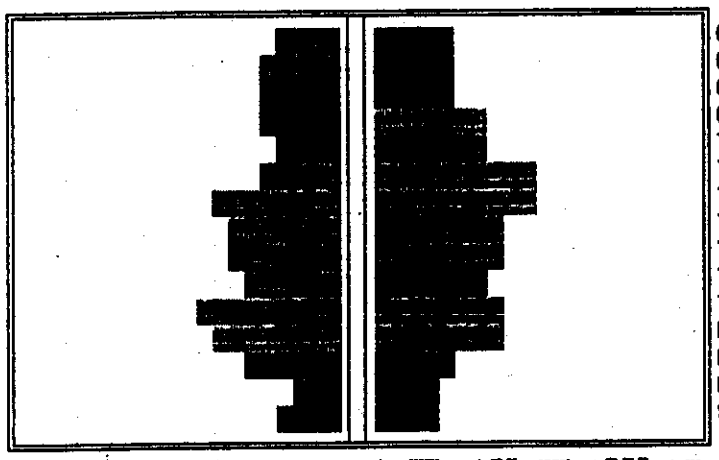
within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

performed by: K. C

Patient: Patient 3-fixation
SSN: height: 0.0 weight: 0.0
age: 0 sex: male ref.ID:
Static-Scan, spinal on 03/09/1993,Tue (2nd most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 25.0 uV

left sites		right sites	
3.9uV	C1	C1	4.3uV
4.9uV	C3	C3	4.0uV
4.4uV	C5	C5	3.6uV
5.3uV	C7	C7	5.9uV
4.0uV	T1	T1	5.9uV
5.0uV	T2	T2	10.4uV
8.2uV	T4	T4	9.6uV
7.8uV	T6	T6	7.6uV
7.9uV	T8	T8	7.8uV
6.8uV	T10	T10	6.4uV
9.4uV	T12	T12	7.9uV
9.3uV	L1	L1	7.9uV
5.8uV	L3	L3	3.5uV
2.7uV	L5	L5	2.5uV
3.7uV	S1	S1	2.6uV



within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

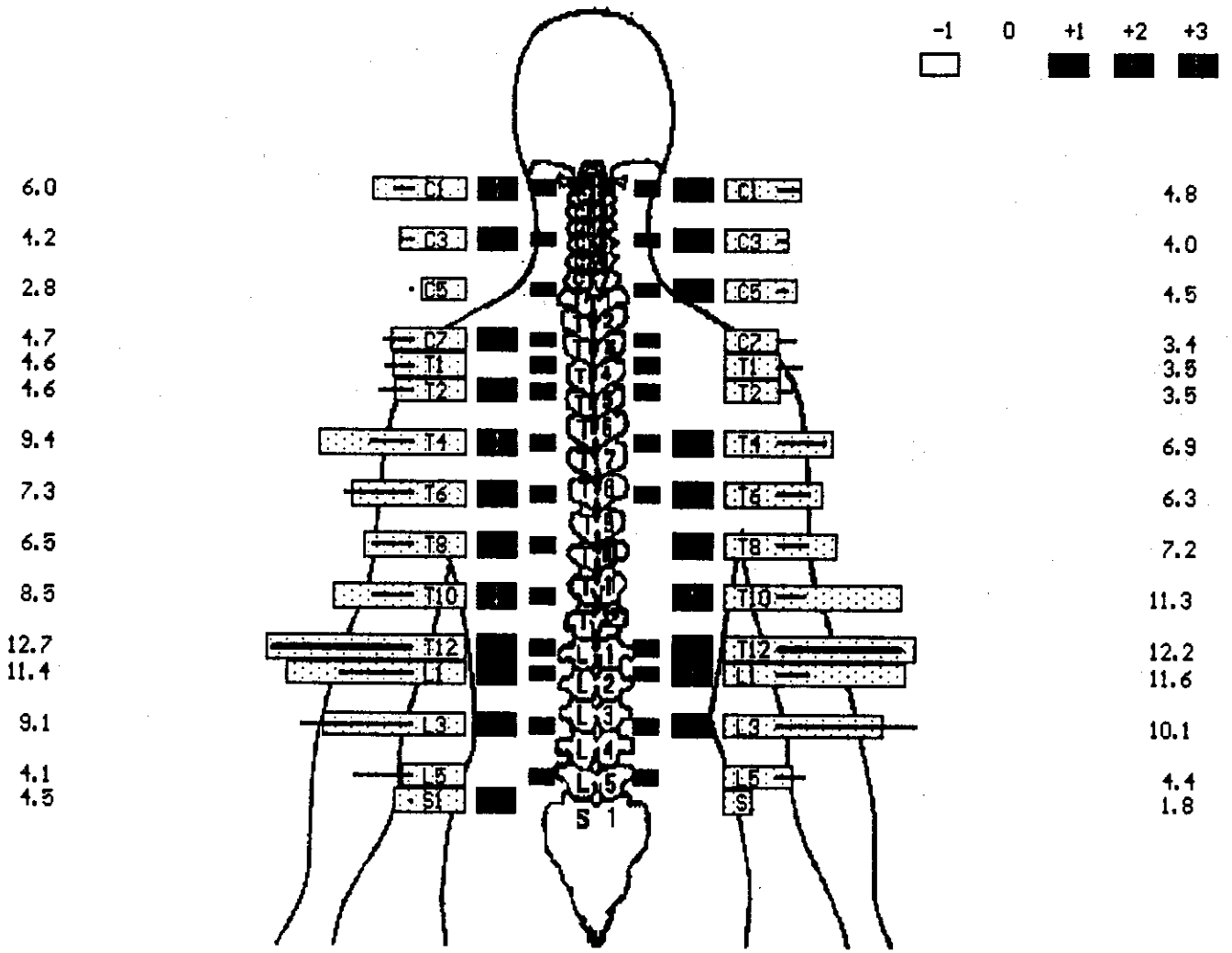
patient:
SSN: Patient 2 - fixation
date: 03/11/1993, Thu

100- 200Hz SEATED NEUTRAL

STATIC Scan

scale displayed = 0.0 - 25.0 uV

-1 0 +1 +2 +3



(thin solid bars and small boxes, if any, are signals from "compared-session")

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

Patient: Patient 2-fixation (continued)
comparison of one session, 1st most recent, on 03/11/1993,Thu
to another session, 2nd most recent, on 03/11/1993,Thu
freq. band is 100- 200Hz

LEFT SIDE uV	COMP	%diff	SITE	%diff	RIGHT SIDE COMP	uV
6.0	4.6	31	C1	2	4.7	4.8
4.2	4.1	1	C3	-1	4.0	4.0
2.8	3.6	-22	C5	13	4.0	4.5
4.7	5.3	-11	C7	-25	4.5	3.4
4.6	5.2	-12	T1	-28	4.9	3.5
4.6	5.6	-19	T2	-18	4.2	3.5
9.4	6.1	54	T4	7	6.4	6.9
7.3	7.8	-7	T6	16	5.4	6.3
6.5	6.0	8	T8	37	5.3	7.2
8.5	5.9	44	T10	119	5.2	11.3
12.7	12.4	2	T12	6	11.5	12.2
11.4	8.0	43	L1	114	5.4	11.6
9.1	10.5	-13	L3	-18	12.3	10.1
4.1	7.1	-43	L5	-14	5.1	4.4
4.5	3.5	30	S1	-22	2.3	1.8

note: positive (+) percent (%) difference means comparison data is lower
values with a "??" were not taken during the comparison session

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Patient: Patient 2 fixation 03/11/1993,Thu
asymmetry table, seated neutral in freq. band 100- 200Hz

%DF	PSD	NSD	NORM	uV	SITE	uV	NORM	NSD	PSD	%DF
25	+++	0.9	2.5	6.0	C1	4.8	2.8	1.0	++	<--
5	+	1.0	2.6	4.2	C3	4.0	2.6	0.8	+	<--
-->		0.8	2.6	2.8	C5	4.5	2.4	0.7	+++	63
41	+	1.2	3.0	4.7	C7	3.4	2.7	1.0		<--
31		1.5	3.2	4.6	T1	3.5	2.8	1.1		<--
32	+	1.0	2.9	4.6	T2	3.5	2.9	1.2		<--
36	+++	1.1	3.2	9.4	T4	6.9	3.4	1.2	++	<--
16	++	1.2	3.7	7.3	T6	6.3	3.8	1.3	+	<--
-->	+	1.3	4.3	6.5	T8	7.2	4.5	1.4	+	11
-->	+++	1.2	4.3	8.5	T10	11.3	4.6	1.6	+++	33
4	+++	1.4	4.0	12.7	T12	12.2	4.1	1.3	+++	<--
-->	+++	1.4	3.9	11.4	L1	11.6	3.9	1.4	+++	1
-->	+++	1.3	3.3	9.1	L3	10.1	3.5	1.3	+++	11
-->		1.6	3.3	4.1	L5	4.4	3.4	1.7		8
156	+	1.4	2.9	4.5	S1	1.8	2.8	1.4		<--

key: uV=scan NORM=normal data NSD=normal standard deviation
PSD=patient standard deviations from normal %DF=percent differ.
* = no normal data available for this site / freq.band

Fixation/Correction EMG Effects . . . Garrett
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Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
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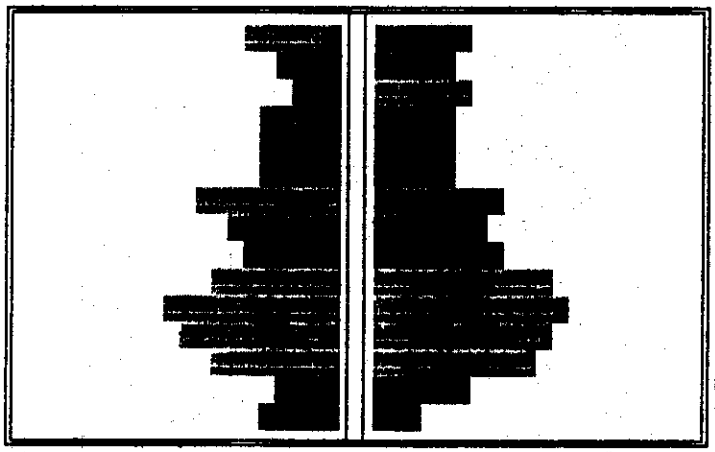
performed by: POST

Patient: Patient 2- fixation
SSN: height: 0.0 weight: 0.0
age: 0 sex: male ref.ID: 787
Static-Scan, spinal on 03/11/1993,Thu (1st most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 25.0 uV

left sites		right sites	
6.0uV	C1	C1	4.8uV
4.2uV	C3	C3	4.0uV
2.8uV	C5	C5	4.5uV
4.7uV	C7	C7	3.4uV
4.6uV	T1	T1	3.5uV
4.6uV	T2	T2	3.5uV
9.4uV	T4	T4	6.9uV
7.3uV	T6	T6	6.3uV
6.5uV	T8	T8	7.2uV
8.5uV	T10	T10	11.3uV
12.7uV	T12	T12	12.2uV
11.4uV	L1	L1	11.6uV
9.1uV	L3	L3	10.1uV
4.1uV	L5	L5	4.4uV
4.5uV	S1	S1	1.8uV



within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

Fixation/Correction EMG Effects . . . Garrett
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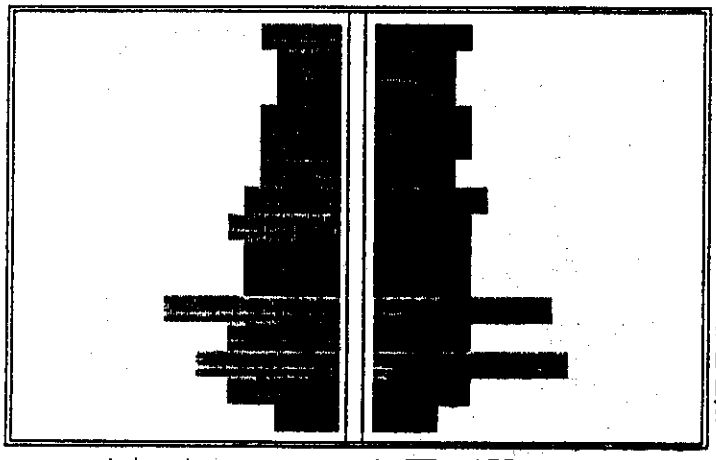
Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

performed by: Pre -

Patient: Patient 2 - fixation
SSN: height: 0.0 weight: 0.0
age: 0 sex: male ref.ID: 787
Static-Scan, spinal on-03/11/1993.Thu (2nd most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 25.0 uV

left sites		right sites	
4.6uV	C1	C1	4.7uV
4.1uV	C3	C3	4.0uV
3.6uV	C5	C5	4.0uV
5.3uV	C7	C7	4.5uV
5.2uV	T1	T1	4.9uV
5.6uV	T2	T2	4.2uV
6.1uV	T4	T4	6.4uV
7.8uV	T6	T6	5.4uV
6.0uV	T8	T8	5.3uV
5.9uV	T10	T10	5.2uV
12.4uV	T12	T12	11.5uV
8.0uV	L1	L1	5.4uV
10.5uV	L3	L3	12.3uV
7.1uV	L5	L5	5.1uV
3.5uV	S1	S1	2.3uV



within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

Fixation/Correction EMG Effects . . . Garrett
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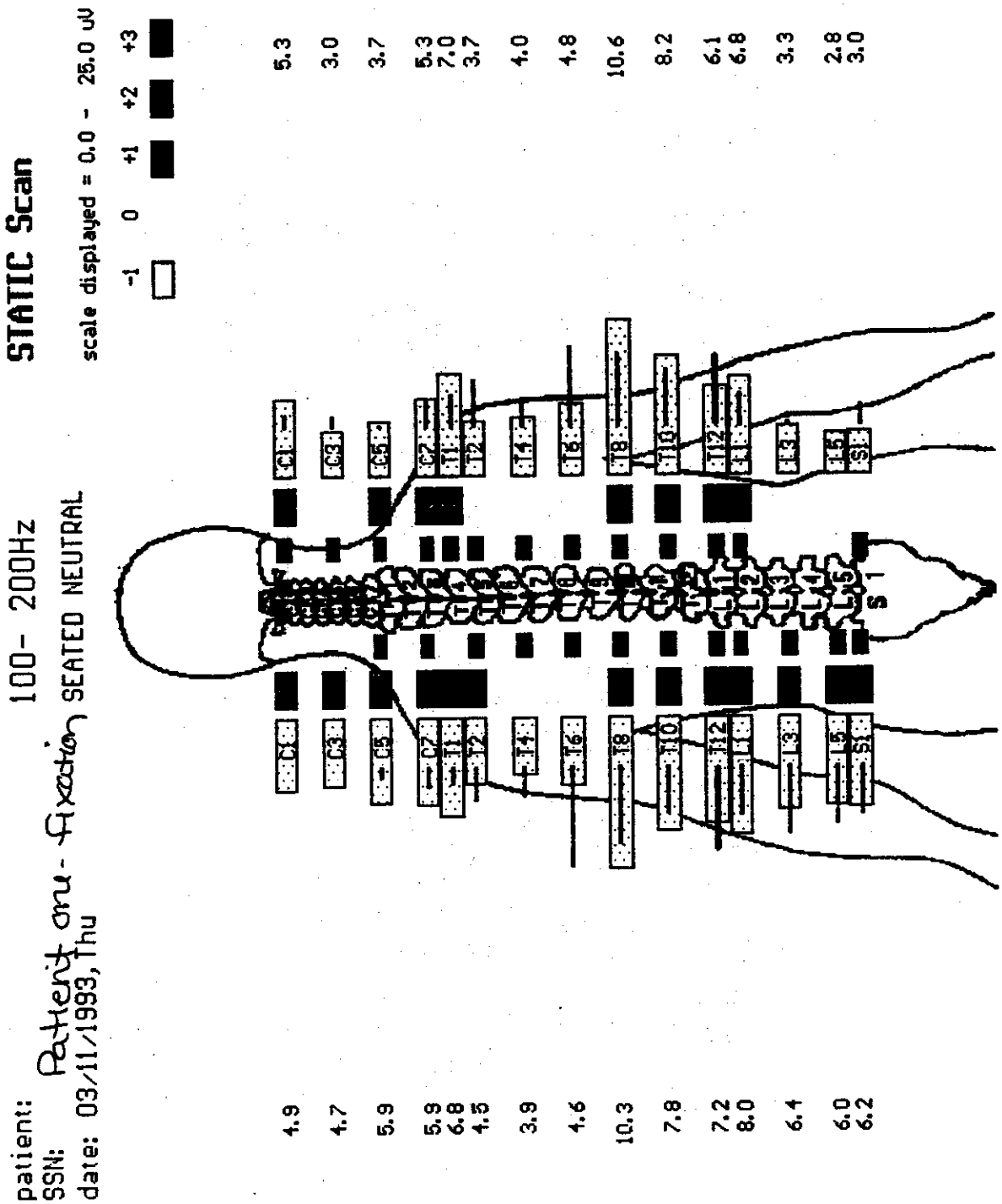

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

Patient: Patient one-fixation (continued)
comparison of one session, 1st most recent, on 03/11/1993,Thu
to another session, 2nd most recent, on 03/11/1993,Thu
freq. band is 100- 200Hz

LEFT SIDE uV	COMP	%diff	SITE	%diff	RIGHT SIDE COMP	uV
4.9	2.9	67	C1	23	4.3	5.3
4.7	3.1	52	C3	-26	4.1	3.0
5.9	4.2	41	C5	6	3.5	3.7
5.9	4.8	22	C7	0	5.3	5.3
6.8	4.6	48	T1	25	5.6	7.0
4.5	5.7	-21	T2	-44	6.6	3.7
3.9	5.4	-27	T4	-24	5.3	4.0
4.6	10.2	-55	T6	-45	8.8	4.8
10.3	8.7	19	T8	29	8.2	10.6
7.8	6.5	19	T10	13	7.2	8.2
7.2	9.2	-21	T12	-26	8.3	6.1
8.0	6.7	19	L1	22	5.6	6.8
6.4	8.1	-21	L3	-7	3.5	3.3
6.0	7.4	-18	L5	-8	3.1	2.8
6.2	6.8	-8	S1	-36	4.7	3.0

note: positive (+) percent (%) difference means comparison data is lower
values with a "??" were not taken during the comparison session

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

Patient: Patient one- fixation 03/11/1993,Thu
asymmetry table, seated neutral in freq. band 100- 200Hz

%DF	PSD	NSD	NORM	uV	SITE	uV	NORM	NSD	PSD	%DF
-->	++	0.9	2.5	4.9	C1	5.3	2.8	1.0	++	8
54	++	1.0	2.6	4.7	C3	3.0	2.6	0.8		<--
57	+++	0.8	2.6	5.9	C5	3.7	2.4	0.7	+	<--
11	++	1.2	3.0	5.9	C7	5.3	2.7	1.0	++	<--
-->	++	1.5	3.2	6.8	T1	7.0	2.8	1.1	+++	4
21	+	1.0	2.9	4.5	T2	3.7	2.9	1.2		<--
-->		1.1	3.2	3.9	T4	4.0	3.4	1.2		2
-->		1.2	3.7	4.6	T6	4.8	3.8	1.3		5
-->	+++	1.3	4.3	10.3	T8	10.6	4.5	1.4	+++	3
-->	++	1.2	4.3	7.8	T10	8.2	4.6	1.6	++	5
18	++	1.4	4.0	7.2	T12	6.1	4.1	1.3	+	<--
18	++	1.4	3.9	8.0	L1	6.8	3.9	1.4	++	<--
93	++	1.3	3.3	6.4	L3	3.3	3.5	1.3		<--
115	+	1.6	3.3	6.0	L5	2.8	3.4	1.7		<--
107	++	1.4	2.9	6.2	S1	3.0	2.8	1.4		<--

key: uV=scan NORM=normal data NSD=normal standard deviation
PSD=patient standard deviations from normal %DF=percent differ.
* = no normal data available for this site / freq.band

Fixation/Correction EMG Effects . . . Garrett
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Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
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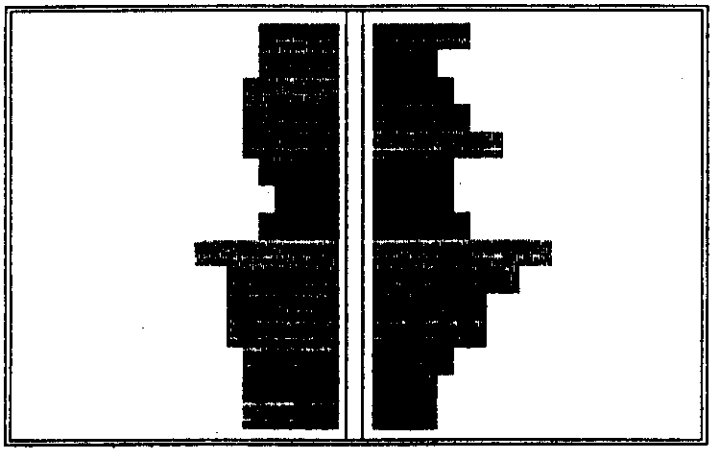
performed by: post

Patient: - Patient one - fixation
SSN: height: 0.0 weight: 0.0
age: 27 sex: female ref.ID: 002
Static-Scan, spinal on 03/11/1993,Thu (1st most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 25.0 uV



left sites		right sites	
4.9uV	C1	C1	5.3uV
4.7uV	C3	C3	3.0uV
5.9uV	C5	C5	3.7uV
5.9uV	C7	C7	5.3uV
6.8uV	T1	T1	7.0uV
4.5uV	T2	T2	3.7uV
3.9uV	T4	T4	4.0uV
4.6uV	T6	T6	4.8uV
10.3uV	T8	T8	10.6uV
7.8uV	T10	T10	8.2uV
7.2uV	T12	T12	6.1uV
8.0uV	L1	L1	6.8uV
6.4uV	L3	L3	3.3uV
6.0uV	L5	L5	2.8uV
6.2uV	S1	S1	3.0uV

within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

Fixation/Correction EMG Effects . . . Garrett
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Advanced Chiropractic Care
Dr. Brian F. Garrett
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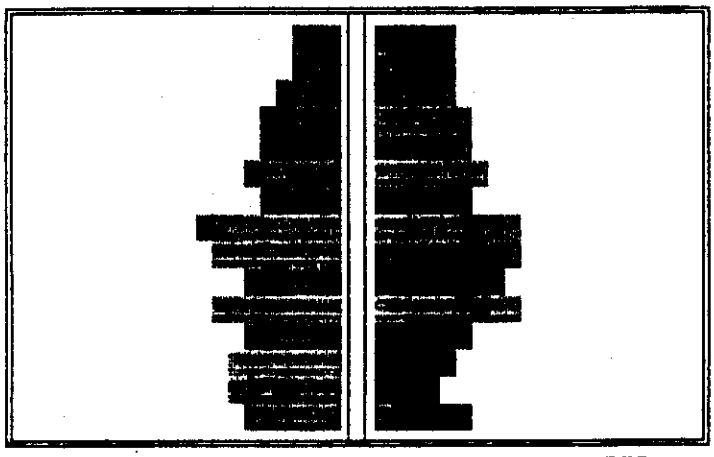
performed by: Pre

Patient: Patient one - fixation
SSN: height: 0.0 weight: 0.0
age: 27 sex: female ref.ID: 002
Static-Scan, spinal on 03/11/1993,Thu (2nd most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 25.0 uV



left sites		right sites	
2.9uV	C1	C1	4.3uV
3.1uV	C3	C3	4.1uV
4.2uV	C5	C5	3.5uV
4.8uV	C7	C7	5.3uV
4.6uV	T1	T1	5.6uV
5.7uV	T2	T2	6.6uV
5.4uV	T4	T4	5.3uV
10.2uV	T6	T6	8.8uV
8.7uV	T8	T8	8.2uV
6.5uV	T10	T10	7.2uV
9.2uV	T12	T12	8.3uV
6.7uV	L1	L1	5.6uV
8.1uV	L3	L3	3.5uV
7.4uV	L5	L5	3.1uV
6.8uV	S1	S1	4.7uV

within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

REFERENCES

1. Kent, C. and Gentempo, P., Protocols and Normative Data for Paraspinal EMG Scanning in Chiropractic Practice; Journal of Chiropractic Research and Clinical Investigation (1990); 6(3): 64.

2. Walther, D., Applied Kinesiology Synopsis, Systems, D.C., (1988); pages 80-87.

3. Kent, C., [Personal Communication between myself and doctor on 3/16/93].

Post: 9 at 3 standard deviations; as compared to normal
6 at 2 standard deviations; as compared to normal
2 at 1 standard deviations; as compared to normal
13 were found to be within normal limits.

Patient #3: Little change in symmetry of muscular activity, although regions within normal more than doubled.

Pre: 7 at 3 standard deviations; as compared to normal
10 at 2 standard deviations; as compared to normal
7 at 1 standard deviations; as compared to normal
6 were found to be within normal limits.

Post: 9 at 3 standard deviations; as compared to normal
6 at 2 standard deviations; as compared to normal
2 at 1 standard deviations; as compared to normal
13 were found to be within normal limits.

CONCLUSION

Based on the analysis of muscular activity, it appears that correction of fixation patterns as described by standard applied kinesiology protocol's significantly affects normal paraspinal muscle tone and activity, although not symmetry at least in the short term. Further investigation is indicated as to the long-term effect and underlying causation of recurrent fixation patterns. [3]

Surprising to me, is the effect of this treatment on two of these patients in raising the number of segmental areas over three standard deviations from the norm. One possible explanation is increase in paraspinal muscle activity in the body's overall corrective compensation mechanism. Further evaluation is necessary to come to a valid conclusion.

It appears static paraspinal surface electromyography may be helpful in the objective documentation of the effectiveness of applied kinesiological structural correction and its correlation to standard chiropractic care.

RESULTS

Pre-test findings were as follows: $_2$

Patient #1: Bilateral weakness:
Psoas: associated with right occiput fixation
Popliteus: associated with C3/4/5 fixation.
Lower trapezius: associated with thoraco-lumbar fixation.
Unilateral neck extensors: associated with sacral fix.

Patient #2: Bilateral weakness:
Psoas: associated with right occiput fixation
Popliteus: associated with C4/5/6 fixation.
Lower trapezius: associated with thoraco-lumbar fixation.
Unilateral neck extensors: associated with sacroiliac fixation.
Teres major: associated with T4/5/6 fixation.

Patient #3: Bilateral weakness:
Lower trapezius: associated with thoraco-lumbar fixation.
Unilateral neck extensors: associated with left sacro-iliac fixation.

SPECIFIC EMG CHANGES

Patient #1: Greater symmetry in overall scan of muscular activity present post-treatment.

Pre: 7 at 3 standard deviations; as compared to normal
6 at 2 standard deviations; as compared to normal
12 at 1 standard deviations; as compared to normal
5 were found to be within normal limits

Post: 4 at 3 standard deviations; as compared to normal
13 at 2 standard deviations; as compared to normal
4 at 1 standard deviations; as compared to normal
9 were found to be within normal limits

Patient #2: Greater asymmetry was found in overall post-treatment scan, although more regions returned to within normal limits.

Pre: 7 at 3 standard deviations; as compared to normal
10 at 2 standard deviations; as compared to normal
7 at 1 standard deviations; as compared to normal
6 were found to be within normal limits

THE EFFECTS OF CORRECTION OF APPLIED KINESIOLOGY FIXATION PROTOCOL ON STATIC PARASPINAL ELECTROMYOGRAPHY

By

Brian T. Garrett, DC, CCSP

ABSTRACT

Bilateral muscle weakness patterns as determined by applied kinesiology manual muscle testing, associated with spinal fixation patterns, are correlated with pre- and post-treatment static surface paraspinal electromyography.

INTRODUCTION

Objective substantiation of treatment outcome is essential in the health care field in this day and age. In A.K. practice, as well as standard chiropractic care, it is crucial to show pre-treatment and post-treatment differences that correlate with the symptomatic picture.

It is well accepted in chiropractic that muscular abnormalities are present and important in the evaluation of the vertebral subluxation complex. Electromyographic analysis can objectively document the efficacy of our treatment procedures.[1]

In this paper, I will take one procedure, bilateral muscle weaknesses associated with fixation patterns in the spine, out of context in the overall treatment protocol of applied kinesiology and observe its effect on paraspinal muscular electrical activity, and it's deviation from a statistical normative data base.

MATERIALS/METHODS

Static scans using the Insight 5000 EMG™ were performed on these patients in the seated position, feet on the floor flat and palms up, resting on their lap.

Insight EMG's have statistical normals built into the computer analysis program.

Bilateral muscle weakness patterns associated with spinal fixations were found and the fixations corrected according to standard applied kinesiology protocol.

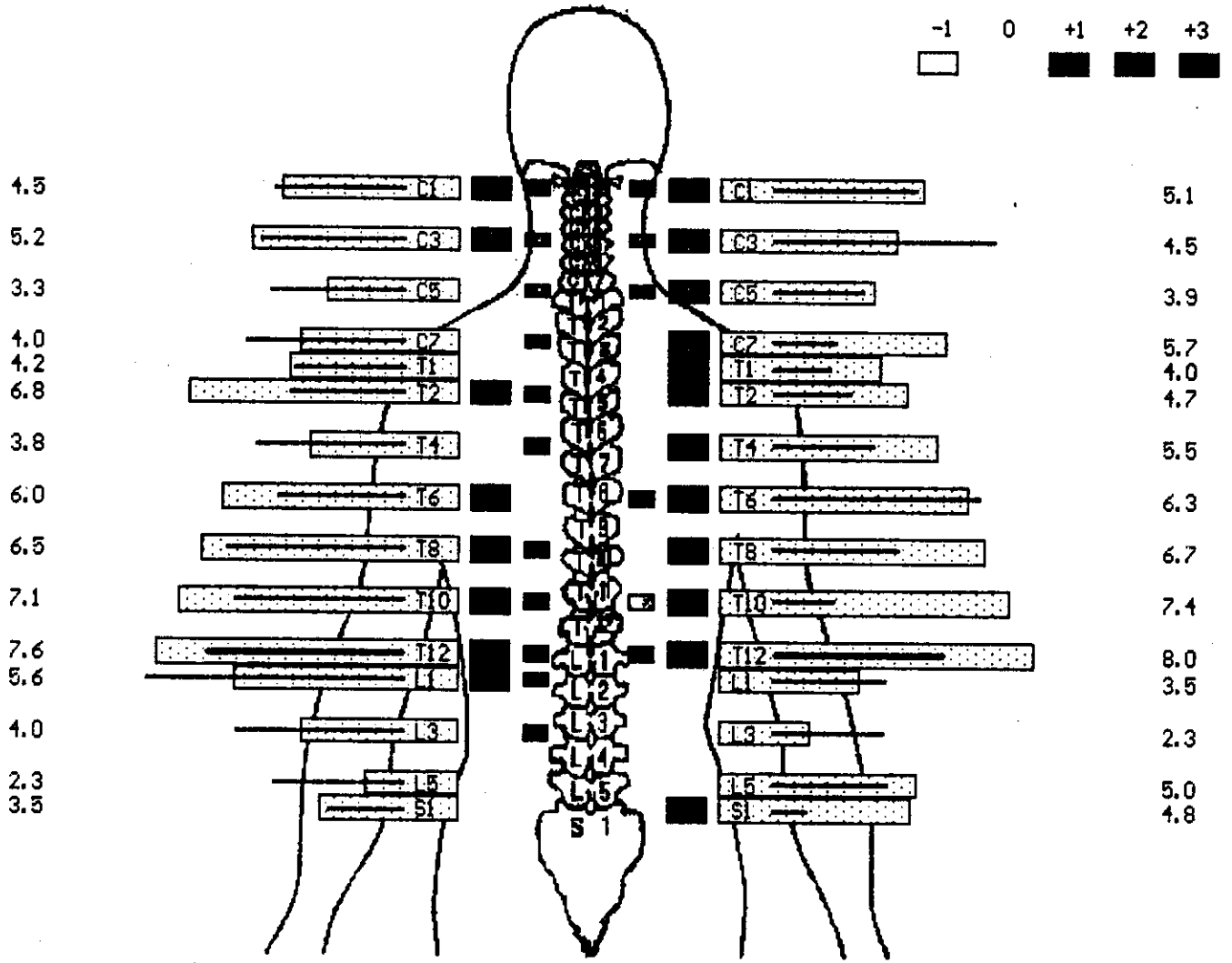
patient:
SSN: Patient Three 's
date: 03/09/1993, Tue

100- 200Hz
SEATED NEUTRAL

STATIC Scan

scale displayed = 0.0 - 10.0 uV

-1 0 +1 +2 +3



(thin solid bars and small boxes, if any, are signals from "compared-session")

Cranial/Sacral EMG Effects . . . Garrett
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Advanced Chiropractic Care
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Patient: -- Patient three c/s 03/09/1993,Tue
asymmetry table, seated neutral in freq. band 100- 200Hz

%DF	PSD	NSD	NORM	uV	SITE	uV	NORM	NSD	PSD	%DF
-->	++	0.9	2.5	4.5	C1	5.1	2.8	1.0	++	14
16	++	1.0	2.6	5.2	C3	4.5	2.6	0.8	++	<--
-->		0.8	2.6	3.3	C5	3.9	2.4	0.7	++	18
-->		1.2	3.0	4.0	C7	5.7	2.7	1.0	+++	44
5		1.5	3.2	4.2	T1	4.0	2.8	1.1	+	<--
44	+++	1.0	2.9	6.8	T2	4.7	2.9	1.2	+	<--
-->		1.1	3.2	3.8	T4	5.5	3.4	1.2	+	47
-->	+	1.2	3.7	6.0	T6	6.3	3.8	1.3	+	4
-->	+	1.3	4.3	6.5	T8	6.7	4.5	1.4	+	3
-->	++	1.2	4.3	7.1	T10	7.4	4.6	1.6	+	4
-->	++	1.4	4.0	7.6	T12	8.0	4.1	1.3	++	4
59	+	1.4	3.9	5.6	L1	3.5	3.9	1.4		<--
72		1.3	3.3	4.0	L3	2.3	3.5	1.3		<--
-->		1.6	3.3	2.3	L5	5.0	3.4	1.7		117
-->		1.4	2.9	3.5	S1	4.8	2.8	1.4	+	37

key: uV=scan NORM=normal data NSD=normal standard deviation
PSD=patient standard deviations from normal %DF=percent differ.
* = no normal data available for this site / freq.band

Advanced Chiropractic Care
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Patient: Patient three 95 (continued)
comparison of one session, 1st most recent, on 03/09/1993,Tue
to another session, 2nd most recent, on 03/09/1993,Tue
freq. band is 100- 200Hz

LEFT SIDE uV	COMP	%diff	SITE	%diff	RIGHT SIDE COMP	uV
4.5	4.7	-4	C1	3	5.0	5.1
5.2	5.0	4	C3	-35	7.0	4.5
3.3	4.8	-30	C5	7	3.6	3.9
4.0	5.4	-27	C7	94	3.0	5.7
4.2	4.1	2	T1	44	2.8	4.0
6.8	4.3	61	T2	42	3.3	4.7
3.8	5.2	-27	T4	42	3.9	5.5
6.0	4.6	31	T6	-5	6.6	6.3
6.5	5.9	11	T8	48	4.5	6.7
7.1	5.7	25	T10	155	2.9	7.4
7.6	6.4	19	T12	39	5.7	8.0
5.6	7.9	-29	L1	-16	4.2	3.5
4.0	5.6	-30	L3	-45	4.2	2.3
2.3	4.7	-51	L5	18	4.3	5.0
3.5	3.3	7	S1	117	2.2	4.8

note: positive (+) percent (%) difference means comparison data is lower
values with a "??" were not taken during the comparison session

Cranial/Sacral EMG Effects . . . Garrett
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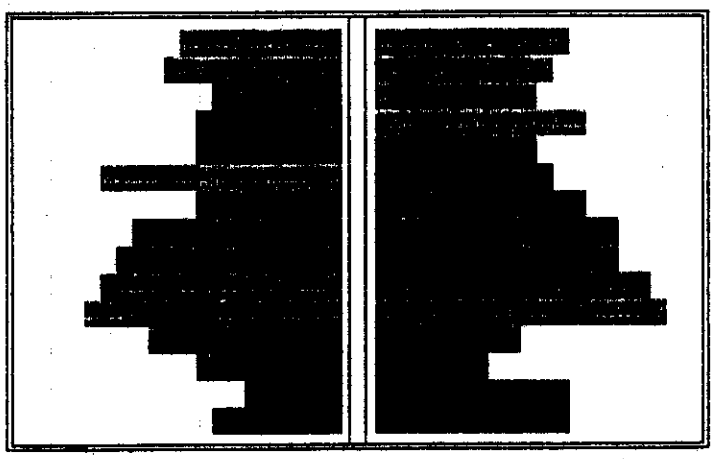
Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

performed by: Post

Patient: Patient three cls
SSN: height: 61.0 weight: 113.0
age: 27 sex: female ref.ID:
Static-Scan, spinal on 03/09/1993,Tue (1st most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 10.0 uV



left sites		right sites	
4.5uV	C1	C1	5.1uV
5.2uV	C3	C3	4.5uV
3.3uV	C5	C5	3.9uV
4.0uV	C7	C7	5.7uV
4.2uV	T1	T1	4.0uV
6.8uV	T2	T2	4.7uV
3.8uV	T4	T4	5.5uV
6.0uV	T6	T6	6.3uV
6.5uV	T8	T8	6.7uV
7.1uV	T10	T10	7.4uV
7.6uV	T12	T12	8.0uV
5.6uV	L1	L1	3.5uV
4.0uV	L3	L3	2.3uV
2.3uV	L5	L5	5.0uV
3.5uV	S1	S1	4.8uV

within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

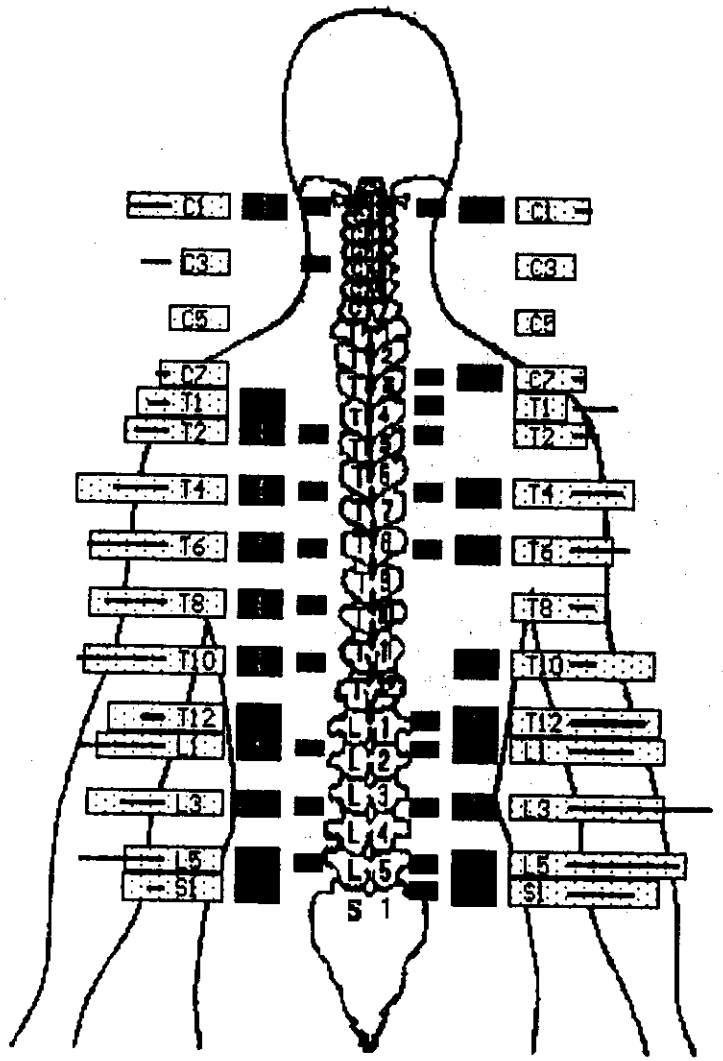
patient:
SSN: Patient two c/s
date: 03/09/1993, Tue

100- 200Hz
SEATED NEUTRAL

STATIC Scan

scale displayed = 0.0 - 25.0 uV

-1 0 +1 +2 +3



5.8	C1	C1	4.2
2.7	C3	C3	3.4
3.3	C5	C5	2.1
3.9	C7	C7	4.0
5.2	T1	T1	2.9
5.8	T2	T2	4.1
8.5	T4	T4	6.8
7.8	T6	T6	5.7
7.7	T8	T8	5.2
8.1	T10	T10	8.2
6.7	T12	T12	8.6
7.3	L1	L1	8.8
7.7	L3	L3	8.9
5.5	L5	L5	10.1
5.7	S1	S1	8.6

(thin solid bars and small boxes, if any, are signals from "compared-session")

Cranial/Sacral EMG Effects . . . Garrett
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Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

Patient: Patient two c/s 03/09/1993,Tue
asymmetry table, seated neutral in freq. band 100- 200Hz

%DF	PSD	NSD	NORM	uV	SITE	uV	NORM	NSD	PSD	%DF
39	+++	0.9	2.5	5.8	C1	4.2	2.8	1.0	+	<--
-->		1.0	2.6	2.7	C3	3.4	2.6	0.8		28
55		0.8	2.6	3.3	C5	2.1	2.4	0.7		<--
-->		1.2	3.0	3.9	C7	4.0	2.7	1.0	+	3
76	+	1.5	3.2	5.2	T1	2.9	2.8	1.1		<--
41	++	1.0	2.9	5.8	T2	4.1	2.9	1.2		<--
25	+++	1.1	3.2	8.5	T4	6.8	3.4	1.2	++	<--
37	+++	1.2	3.7	7.8	T6	5.7	3.8	1.3	+	<--
47	++	1.3	4.3	7.7	T8	5.2	4.5	1.4		<--
-->	+++	1.2	4.3	8.1	T10	8.2	4.6	1.6	++	1
-->	+	1.4	4.0	6.7	T12	8.6	4.1	1.3	+++	29
-->	++	1.4	3.9	7.3	L1	8.8	3.9	1.4	+++	20
-->	+++	1.3	3.3	7.7	L3	8.9	3.5	1.3	+++	15
-->	+	1.6	3.3	5.5	L5	10.1	3.4	1.7	+++	82
-->	++	1.4	2.9	5.7	S1	8.6	2.8	1.4	+++	51

key: uV=scan NORM=normal data NSD=normal standard deviation
PSD=patient standard deviations from normal %DF=percent differ.
* = no normal data available for this site / freq.band

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

Patient: patient two cls (continued)
comparison of one session, 1st most recent, on 03/09/1993,Tue
to another session, 2nd most recent, on 03/09/1993,Tue
freq. band is 100- 200Hz

LEFT SIDE uV	COMP	%diff	SITE	%diff	RIGHT SIDE COMP	uV
5.8	5.8	0	C1	-1	4.2	4.2
2.7	5.1	-48	C3	7	3.2	3.4
3.3	2.4	38	C5	-24	2.8	2.1
3.9	4.1	-5	C7	3	3.9	4.0
5.2	4.5	14	T1	-49	5.8	2.9
5.8	5.3	8	T2	-15	4.8	4.1
8.5	6.5	31	T4	11	6.2	6.8
7.8	7.8	-1	T6	-15	6.6	5.7
7.7	6.8	13	T8	14	4.6	5.2
8.1	8.5	-5	T10	73	4.7	8.2
6.7	4.8	38	T12	10	7.8	8.6
7.3	8.4	-14	L1	26	7.0	8.8
7.7	5.8	33	L3	-23	11.5	8.9
5.5	8.1	-32	L5	3	9.8	10.1
5.7	4.2	35	S1	21	7.1	8.6

note: positive (+) percent (%) difference means comparison data is lower
values with a "??" were not taken during the comparison session

Cranial/Sacral EMG Effects . . . Garrett
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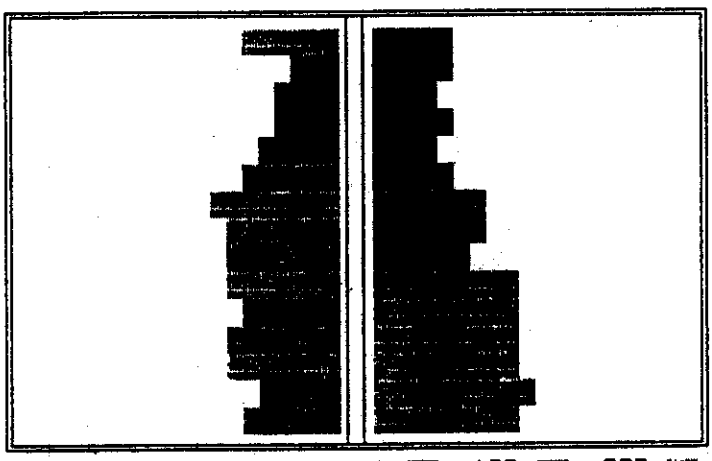
Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

performed by: Post

Patient: Patient two cls
SSN: height: 71.0 weight: 152.0
age: 34 sex: male ref.ID:
Static-Scan. spinal on 03/09/1993,Tue (1st most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 25.0 uV

left sites		right sites	
5.8uV	C1	C1	4.2uV
2.7uV	C3	C3	3.4uV
3.3uV	C5	C5	2.1uV
3.9uV	C7	C7	4.0uV
5.2uV	T1	T1	2.9uV
5.8uV	T2	T2	4.1uV
8.5uV	T4	T4	6.8uV
7.8uV	T6	T6	5.7uV
7.7uV	T8	T8	5.2uV
8.1uV	T10	T10	8.2uV
6.7uV	T12	T12	8.6uV
7.3uV	L1	L1	8.8uV
7.7uV	L3	L3	8.9uV
5.5uV	L5	L5	10.1uV
5.7uV	S1	S1	8.6uV



within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

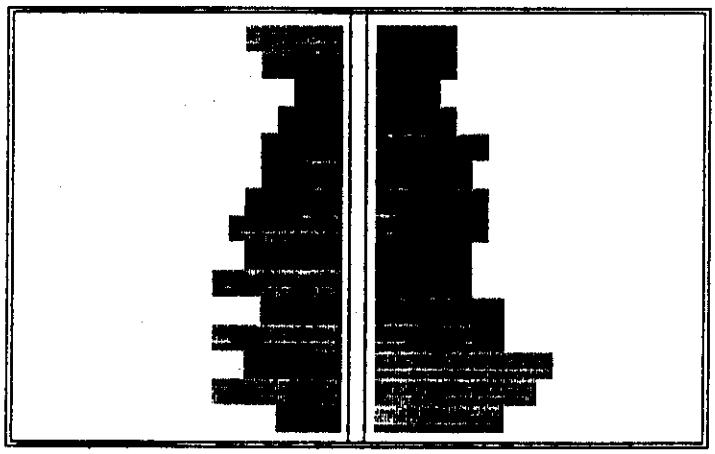
Cranial/Sacral EMG Effects . . . Garrett
Page 10

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

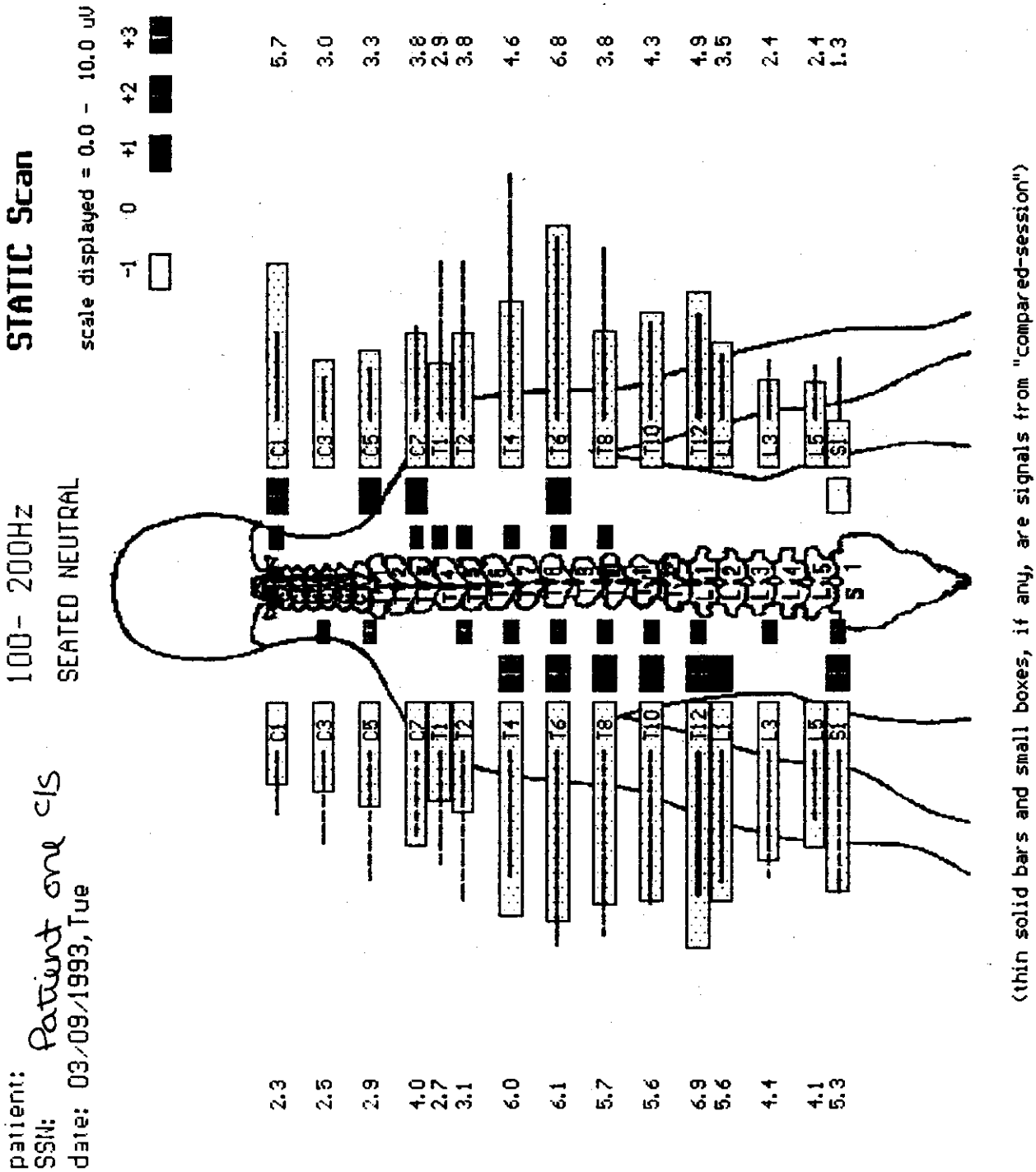
performed by: Pre

Patient: Patient two cls
SSN: height: 71.0 weight: 152.0
age: 34 sex: male ref.ID:
Static-Scan, spinal on 03/09/1993,Tue (2nd most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 25.0 uV

left sites		right sites	
5.8uV	C1	C1	4.2uV
5.1uV	C3	C3	3.2uV
2.4uV	C5	C5	2.8uV
4.1uV	C7	C7	3.9uV
4.5uV	T1	T1	5.8uV
5.3uV	T2	T2	4.8uV
6.5uV	T4	T4	6.2uV
7.8uV	T6	T6	6.6uV
6.8uV	T8	T8	4.6uV
8.5uV	T10	T10	4.7uV
4.8uV	T12	T12	7.8uV
8.4uV	L1	L1	7.0uV
5.8uV	L3	L3	11.5uV
8.1uV	L5	L5	9.8uV
4.2uV	S1	S1	7.1uV



within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=


STATIC Scan
100- 200Hz
SEATED NEUTRAL
scale displayed = 0.0 - 10.0 uV
-1 0 +1 +2 +3
patient: Patient one c/s
SSN:
date: 03/09/1993,Tue
C1 C3 C5 C2 T1 T2 T4 T6 T8 T10 T12 L1 L3 L5 S1
5.7 3.0 3.3 3.8 2.9 3.8 4.6 6.8 3.8 4.3 4.9 3.5 2.4 2.4 1.3
2.3 2.5 2.9 4.0 2.7 3.1 6.0 6.1 5.7 5.6 6.9 5.6 4.4 4.1 5.3
(thin solid bars and small boxes, if any, are signals from "compared-session")

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

Patient: Patient one cls (continued)
comparison of one session, 1st most recent, on 03/09/1993,Tue
to another session, 2nd most recent, on 03/09/1993,Tue
freq. band is 100- 200Hz

LEFT SIDE uV	COMP	%diff	SITE	%diff	RIGHT SIDE COMP	uV
2.3	3.1	-27	C1	50	3.8	5.7
2.5	4.0	-37	C3	18	2.5	3.0
2.9	5.0	-42	C5	16	2.8	3.3
4.0	3.8	6	C7	-5	3.9	3.8
2.7	4.5	-40	T1	-50	5.8	2.9
3.1	5.6	-45	T2	-35	5.8	3.8
6.0	4.9	23	T4	-44	8.2	4.6
6.1	6.8	-10	T6	4	6.5	6.8
5.7	6.5	-13	T8	-38	6.2	3.8
5.6	5.6	-1	T10	6	4.0	4.3
6.9	5.4	26	T12	14	4.3	4.9
5.6	5.1	10	L1	9	3.2	3.5
4.4	4.9	-11	L3	-18	3.0	2.4
4.1	3.3	25	L5	-17	2.9	2.4
5.3	5.4	-1	S1	-58	3.1	1.3

note: positive (+) percent (%) difference means comparison data is lower
values with a "??" were not taken during the comparison session

Advanced Chiropractic Care
Dr. Brian T. Garrett
702 C Street
San Diego, CA 92101

Patient: ... Patient one cks 03/09/1993.Tue
asymmetry table, seated neutral in freq. band 100- 200Hz

%DF	PSD	NSD	NORM	uV	SITE	uV	NORM	NSD	PSD	%DF
-->		0.9	2.5	2.3	C1	5.7	2.8	1.0	++	149
-->		1.0	2.6	2.5	C3	3.0	2.6	0.8		20
-->		0.8	2.6	2.9	C5	3.3	2.4	0.7	+	12
6		1.2	3.0	4.0	C7	3.8	2.7	1.0	+	<--
-->		1.5	3.2	2.7	T1	2.9	2.8	1.1		6
-->		1.0	2.9	3.1	T2	3.8	2.9	1.2		23
30	++	1.1	3.2	6.0	T4	4.6	3.4	1.2		<--
-->	++	1.2	3.7	6.1	T6	6.8	3.8	1.3	++	10
49	+	1.3	4.3	5.7	T8	3.8	4.5	1.4		<--
30	+	1.2	4.3	5.6	T10	4.3	4.6	1.6		<--
41	++	1.4	4.0	6.9	T12	4.9	4.1	1.3		<--
60	+	1.4	3.9	5.6	L1	3.5	3.9	1.4		<--
80		1.3	3.3	4.4	L3	2.4	3.5	1.3		<--
71		1.6	3.3	4.1	L5	2.4	3.4	1.7		<--
312	+	1.4	2.9	5.3	S1	1.3	2.8	1.4	-	<--

key: uV=scan NORM=normal data NSD=normal standard deviation
PSD=patient standard deviations from normal %DF=percent differ.
* = no normal data available for this site / freq.band

Cranial/Sacral EMG Effects . . . Garrett
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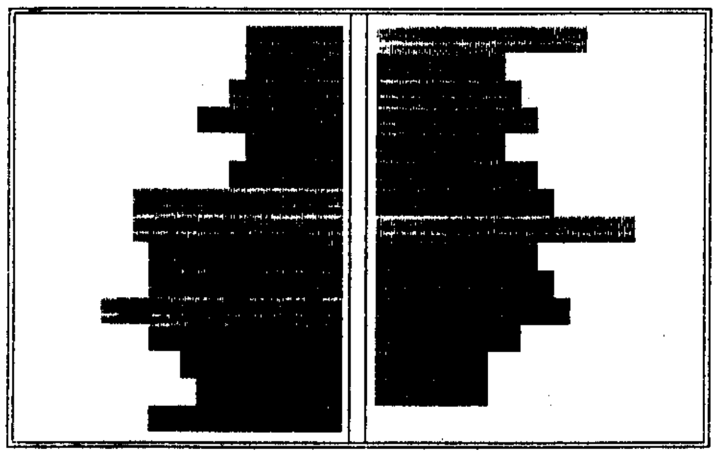
[illegible] Chiropractic Care
[illegible] Garrett
[illegible]
San Diego, CA [illegible]

performed by: Post

Patient: patient one c/s
SSN: height: 0.0 weight: 0.0
age: 0 sex: female ref.ID:
Static-Scan, spinal on 03/09/1993,Tue (1st most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 10.0 uV

left sites			right sites	
2.3uV	C1		C1	5.7uV
2.5uV	C3		C3	3.0uV
2.9uV	C5		C5	3.3uV
4.0uV	C7		C7	3.8uV
2.7uV	T1		T1	2.9uV
3.1uV	T2		T2	3.8uV
6.0uV	T4		T4	4.6uV
6.1uV	T6		T6	6.8uV
5.7uV	T8		T8	3.8uV
5.6uV	T10		T10	4.3uV
6.9uV	T12		T12	4.9uV
5.6uV	L1		L1	3.5uV
4.4uV	L3		L3	2.4uV
4.1uV	L5		L5	2.4uV
5.3uV	S1		S1	1.3uV



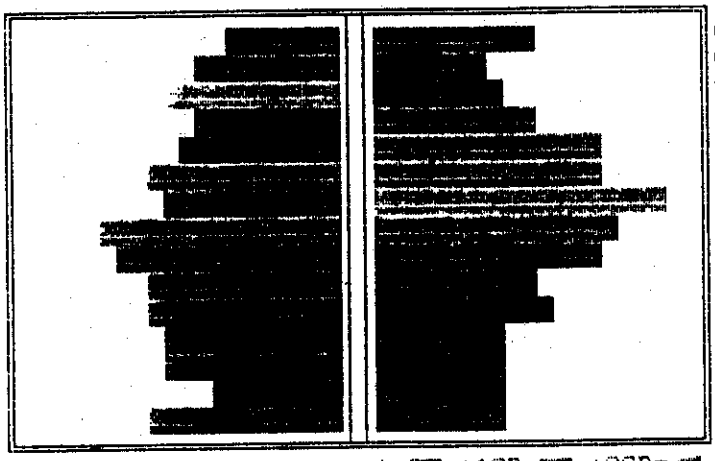
within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

Cranial/Sacral EMG Effects . . . Garrett
Page 5

performed by: Pre

Patient: Patient one cls
SSN: height: 0.0 weight: 0.0
age: 0 sex: female ref.ID:
static-scan, spinal on 03/09/1993,Tue (2nd most recent)
seated neutral in freq. band 100-200Hz scale shown = 0.0 - 10.0 uv

left sites		right sites
3.1uV	C1	3.8uV
4.0uV	C3	2.5uV
5.0uV	C5	2.8uV
3.8uV	C7	3.9uV
4.5uV	T1	5.8uV
5.6uV	T2	5.8uV
4.9uV	T4	8.2uV
6.8uV	T6	6.5uV
6.5uV	T8	6.2uV
5.6uV	T10	4.0uV
5.4uV	T12	4.3uV
5.1uV	L1	3.2uV
4.9uV	L3	3.0uV
3.3uV	L5	2.9uV
5.4uV	S1	3.1uV



within one SD of norm. data (or no norm.)= +1SD= +2SD= +3SD= -1SD=

REFERENCES

1. Kent, C. and Gentempo, P., Protocols and Normative Data for Paraspinal EMG Scanning in Chiropractic Practice; Journal of Chiropractic Research and Clinical Investigation (1990); 6(3): 64.

2. Walther, D., Applied Kinesiology Synopsis, Systems, D.C., (1988); pages 351-367; 371-374.

3. Kent, C., [Personal Communication between myself and doctor on 3/16/93].

Post: 2 at 3 standard deviations; as compared to normal
8 at 2 standard deviations; as compared to normal
10 at 1 standard deviations; as compared to normal
8 were found to be within normal limits

Patient #3 Was not improved post treatment.

Pre: 8 at 3 standard deviations; as compared to normal
8 at 2 standard deviations; as compared to normal
5 at 1 standard deviations; as compared to normal
13 were found to be within normal limits

Post: 10 at 3 standard deviations; as compared to normal
6 at 2 standard deviations; as compared to normal
6 at 1 standard deviations; as compared to normal
10 were found to be within normal limits

CONCLUSION

I am unable to come to any conclusions on the effect of cranial-sacral balancing on static paraspinal muscular activity and symmetry in the short term (15 minutes post treatment). In conversation with Dr. Christopher Kent of EMG Consultants, his opinion was that further investigation was necessary regarding greater time for the paraspinal muscles to re-equilibrate, and further correlation with permanent correction to the cranial/sacral primary respiratory pumping mechanism.[3]

RESULTS

Pre-test findings were as follows:[2]

Patient #1 Sphenobasilar inspiration assist -- left
Universal fault

Patient #2 Sacral wobble inspiration assist -- left
Occipital/atlantal counter-torque -- left

Patient #3 Sphenobasilar inspiration assist -- left
Temporal bulge -- left
Parietal descent -- right
External frontal fault -- left
Lambdoidal sutural fault -- left
Sacral inspiration assist -- left

All faults were corrected, and remained corrected with patient ambulation.

SPECIFIC EMG CHANGES

Patient #1 Better symmetry of paraspinal muscle activity was observed post-correction. Deviations from normative data increased in number, but lowered in amplitude.

Pre: 1 at 3 standard deviations; as compared to normal
7 at 2 standard deviations; as compared to normal
8 at 1 standard deviations; as compared to normal
13 were found to be within normal limits

Post: 2 at 3 standard deviations; as compared to normal
5 at 2 standard deviations; as compared to normal
10 at 1 standard deviations; as compared to normal
18 were found to be within normal limits

Patient #2 Symmetry was not improved post-treatment, number of deviations from normative data increased post-treatment.

Pre: 1 at 3 standard deviations; as compared to normal
7 at 2 standard deviations; as compared to normal
8 at 1 standard deviations; as compared to normal
9 were found to be within normal limits

EFFECTS OF APPLIED KINESIOLOGICAL CRANIAL/SACRAL CORRECTION ON STATIC PARASPINAL EMG

By

Brian T. Garrett, DC, CCSP

ABSTRACT

Applied Kinesiology cranial/sacral analysis and correction was performed upon three patients with pre-treatment and post-treatment surface static paraspinal electromyography at 15 minutes post-treatment, evaluating balance of paraspinal muscular electrical activity and evaluating the effect of this treatment.

INTRODUCTION

Objective substantiation of treatment outcome is essential in the health care field in this day and age. In A.K. practice, as well as standard chiropractic care, it is crucial to show pre-treatment and post-treatment differences that correlate with the symptomatic picture.

It is well accepted in chiropractic that muscular abnormalities are present and important in the evaluation of the vertebral subluxation complex. Electromyographic analysis can objectively document the efficacy of our treatment procedures.[1]

In this paper, I will take one procedure, cranial-sacral respiratory motion, out of context in the overall treatment protocol of applied kinesiology and observe its effect on paraspinal muscular electrical activity, and it's deviation from a statistical normative data base.

MATERIALS/METHODS

Static scans using the Insight 5000 EMG™ were performed on these patients in the seated position, feet on the floor flat and palms up, resting on their lap.

Insight EMG's have statistical normals built into the computer analysis program.

Cranial/sacral analysis was performed according to standard applied kinesiological methods and corrected as indicated.

References:

1. Deal, Sheldon,"Seven Conditions of a Muscle", International College of Applied Kinesiology, Collected Papers, Summer 1986
2. Lebowitz, Michael,"Universal Muscle Strength and Copper Deficiencies", International College of Applied Kinesiology, Collected Papers, Summer 1987.
3. Gerz, Wolfgang, "Superchallenge", International College of Applied Kinesiology - Europe, Collected Papers, Spring 1992.
4. Farkas, Jeff, "More on Superchallenge",International College of Applied Kinesiology - Europe, Collected Papers, Spring 1992.
5. Gerz, Wolfgang,"Working with Homoeopathic Preparations in AK",International College of Applied Kinesiology - Europe, Collected Papers, March 1990
6. Pischinger, A., Matrix and Matrix Regulation; Heine ed.,(Brussels; Haug International,1991)
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9. Farkas, Jeff, "More on Superchallenge",International College of Applied Kinesiology - Europe, Collected Papers, Spring 1992.
10. Rauch, Erich, Diagnostik nach F.X. Mayer,(Heidelberg, Germany; Haug Verlag,1977)
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5. A female patient, approx 45 yr. old, Lycopodium type. Diagnosis: liver/gall bladder associated headaches, diverticulosis, repeated candidiasis, food allergies, etc. One visit, while being examined during a typical headache, all muscles tested hypertonic. Normotonic Challenge: Lycopodium D200 made all lower extremity muscles normotonic; however, not those of the upper extremity. Lycopodium CM normalized both upper and lower extremity muscles. Five globules were given and 2 hours later the patient was totally symptom free.

6. Patient with chronic headache which would arise suddenly, usually after forms of stress. Upon examination, all muscles tested hypertonic. First exam revealed a Superchallenge: knots in the upper bilateral trapezius muscles. These were treated with bloody cupping, after which a normotonic state was noted. This was followed by mercury amalgam replacement and cleansing, as well as lymphatic therapy, which produced noticeable improvement. After 6 months, the patient suffered from only occasional headaches.Naturally, he longed for complete relief. He was advised to return immediately in the event of an acute episode. Just that occured only 2 weeks later. All muscles, at this time, tested hypertonic. Normotonic Challenge: Orally given magnesium citrate in pure form (Pure Encapsulations). 1 ampule of Magnesium-Diasporal* was intraveniously administered. Within 5 minutes, the headache had disappeared. Since then, the patient has been virtually symptom-free, taking two magnesium capsules as well as an alkalizing powder at the slightest hint of an oncoming headache.

Conclusion:

We wish to correlate the three "Muscle Tone" possibilities with the "Stress Concept" according to Selye :[6]

1. Normotonus

This is equivalent to a relatively satisfactory reaction condition. The body can react to both negative and positive "challenges" with a clear muscle response.

2. Hypertonus

This state suggests, particularly when all muscles are in this phase, a long, massive "Adaptation State" to gross stress - of any type. According to Selye, these patients require rest. In our Natural Therapy practice this means a multidimensional approach aimed at lightening all sides of the patients stress load.The Superchallenge probably indicates what the initial treatment focus must be. In most cases, the various forms of toxity must be addressed: colon cleansed, allergens avoided (physical and spiritual), psychological stress factors managed, nutritional deficiencies supplemented and so forth.

3. General Weakness

The next level, according to Selye, is the "exhaustion stage". This is probably what we in the AK community are observing in patients with all, or nearly all, muscles weak; especially when it is difficult or impossible to find anything which will create a strengthening. Although there are no strict rules to follow, this patient always requires maximal drainage (pure water is most important for the lymphatic system) as well as massive supplementation of amino acids, multiple vitamins, mineral preparations etc.(based, of course, upon AK testing!). Also: Rest, warmth and positive energy - from wherever it may come.

c) a hypertonic muscle: a muscle which is "too strong"; strong and unsedateable by one or more sedation techniques

Case Studies

1. A patient with repeated infections, chronic sinusitis and shoulder-arm syndrome on the right side presented on his first examination with a general hypertonicity, specifically on the side of the involved arm.Superchallenge: TL to the lymphatic area at the right angle of the jaw (not the left). In addition, the history included a tonsillectomy during childhood, with post-operative problems, on the right side as well (a revision was necessary). Proceeding from the TL, the nosode "Angina Compositum" (Pascoe), the immuno-stimulant "Cefasept" (Cefak) and a lymph drainage remedy ("Lymphdiaral", Pascoe) tested well. After the examination, Neural Therapy (according to Hunecke) was performed on the right tonsil scar. Immediately, all muscles tested normotonic. At the next appointment, the patient reported that the shoulder problem was only occasionally and mildly experienced. Further diagnostic and immune-relevant therapies were added, and after 6 months, the patient was completely problem-free.

2.Initial examination of a patient complaining of left knee pain with a history which included a seminoma operation, a cholecystectomy and chronic candidiasis, revealed a weak left popliteus, right PMS, and bilateral TFLs. All were diagnosed and treated using standard AK procedures. Two weeks later, with the exception of an occasional light "pressure" in the right hypochondriac region, the patient was problem free. However, with AK testing, all muscles tested hypertonic. Normotonic Challenge: Graphites D12 (this single remedy was chosen for testing because the entire constitutional remedy picture was evaluated).

3. One of the most common cases: all muscles were hypertonic in the presence of various immune system and intestinal symptoms. The examination (according to Dr. F.X. Mayr) revealed an inflamed, gassy abdomen with massive Radix Mesentery edema. Superchallenge: candida antigen, which was eliminated using various anti-mycotics. At the next visit, after a week of anti-fungal therapy and diet, the patient tested normotonic, had lost 2-3 kilograms, and the intestines demonstrated general improvement. Note: the "mirror image" finding is also common; i.e. bilateral weak quadriceps and multiple weaknesses which strengthen while the patient is tested to Histaminum D12, as well as an anti-mycotic such as Ampho-Moronal* (amphotericin) suspension. This suggests to us the following: histamine oversensitivity due to candidiasis. As a rule, this patient is easy to help, as long as the patient complies with an anti-fungus diet, always followed by a nutritional change as per Dr. Mayr.

4. A patient with an allergic sinusitis and evident candida overgrowth in the sinus area was appropriately treated. During the stringent phase of the anti-fungal diet, clear improvement was noted. After 5 months, a control examination revealed entirely different muscle findings compared to the initial examination - a general hypertonus. Normotonic Challenge: Histaminum D12. A complete food screeening was performed, with Histaminum D12 held in the patients' hand . Hazelnuts, Liverwurst and all milk products produced hypertonicity. Interestingly, the patient noted that, in the evening , when she was quite tired, she often ate hazelnuts, as they "worked like coffee". Diagnosis: addictive allergy against the above mentioned substances. Note: the described procedure - a food screening test with a therapeutic remedy in the hand or with a orthomolecular substance in the mouth, is often the only possible way to adequately test a patient who is completely hypertonic.

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In this case the tester adds additional velocity in order to overpower the muscle and this is precisely what needs to be avoided.

The Sedation challenge

This challenge, performed by the methods described below, can be used to determine whether a muscle is normotonic or hypertonic. Specifically, they are as follows:

- Spindle Cell Manipulation:
A contact is taken on a muscle belly with two fingers approximately 5 to 10 cm apart along the length of the muscle fibers. Pressure can then be applied with both fingers toward each other (approximation of the muscle belly fibers). This should inhibit a muscle for up to 10 sec.

- Stimulation of the acupuncture sedation point of the meridian known to be correlated with the muscle to be tested. This action should inhibit all meridian related muscles for approximately 10 sec.

- "Running the Meridian":
With a flat, soft hand the meridian can be contacted andstroked lightly in the direction from the highest point to the lowest point on the meridian. This also should inhibit related muscles for approximately 10 sec. For example: The Deltoid muscle is associated with the Lung Meridian. Running this meridian by taking the forementioned contact at Lung 11, which is on the thumb, and running the meridian in the direction of Lung 1, which is found underneath the lateral clavicle, should be done 4 or 5 times in order to produce the desired effect.

- Placing the sedating pole of a strong magnet on a muscle belly
Note: Be aware of the various definitions of magnetic pole dependent upon the origin of the magnet. in reality, there should be only one side of a magnet with the capacitiy to inhibit a muscle. Occasionally, there are cases where the reverse side of the magnet can weaken.We consider this to be "magnetic switching" and it needs to be corrected. As a rule, as long as the magnet remains on the muscle belly, the muscle should remain inhibited.

Procedure:
Test strong muscle and then perform one or more of the above mentioned methods to determine if that muscle can be weakened/sedated.

Muscle test >>>>>> tests strong >>>>>> sedateable? Yes = normotonic
No = hypertonic

There are essentially 3 possible muscle test results:

a) a weak muscle: - a muscle, absent any contraindication to testing, which reacts to an AK-test with a clear weakness

b) a normotonic muscle: a muscle which reacts strong to testing without any evidence of hypertonicity

neurolymphatic, eyes up, neurovascular stress points, emotional challenge, foci such as teeth, scars , tonsils, etc., or any challenge which indicates a functional neurological disorganization, formerly refered to as "switching".

Definitions of Superchallenge and Normotonic Challenge

1. Superchallenge refers to any challenge which weakens 1 or more hypertonic muscles.

2. Normotoner challenge refers to any challenge which returns 1 or more hypertonic muscles to a normotonic condition.

What if the muscle goes from weakness to either normotonic or hypertonic? Unfortunately, there is an opposite case phenomena which is also observable. Specific challenges can produce a hypertonic reaction in both a normotonic or a weak muscle. This condition is probably the equivalent of the addictive allergy phenomena observed in clinical ecology. Perhaps the most drastic example of this state is that of the heroin addict. At the onset of testing, all muscles will test weak and strengthen when heroin is given. However, while he is "high", the muscles will be in a "high-pertonic" state. Clearly, it is inappropriate for both tester and patient to continue an AK Examination at such a time.We have seen exactly that reaction in a number of patients with allergies and sensitivities.

On occasion, we have also observed an interesting form of psychological "Challenge". It may be seen not only when the patient thinks about a strong stress (we have all seen this phenomenon creating weakness), but also when the patients preferred examiner interrupts the testing to address another patient or problem - thereby making the original patient feel ignored and less important.

Discussion:

All the statements made in this article refer to a specific method of muscle testing, namely, that which originally was taught by Goodheart, the "patient-induced" test. With this type of test, at various times also described by several authors as a Gamma-2-Test, the patient, after specific instructions from the examiner, begins a muscle contraction. The examiner responds with a parallel increase in strength against the patients force. As a result, the patients muscle should remain in an isometric contraction. After the patient has arrived at a maximum contraction, the examiner adds a light force of no more than 3 - 4 % and observes if the patient has the muscular integrity to respond with what we call a "locking in".

A crucial prerequisite for performing this type of test, in addition to sufficient knowledge of muscle physiology, is that the examiner in no way sees this test as a form of competition (for example: does the examiner say such things as "you will now see that I can force your muscle down.")

Much more important is for the examiner to recognize his body strength relative to that of the patient, as well as the test position; always being sure to choose the correct length of lever so as to allow the patient to achieve maximum strength level. Only then can the examiner say that he has followed the advice of Goodheart who suggested that "all the advantages must be given to the patient". Unfortunately the opposite is too often true- the strength level of the patient is greater than that of the examiner, the lever chosen is too short or the examiner doesn't realize that the patient or a particular muscle may be hypertonic!

Like Goodheart, I had found a disturbance in the Triple Heater, but it led me to an entirely different conclusion: is it possible that even in the most severe hypertonicity, there is at least one challenge available which will produce weakness ? This could then be considered a "Superchallenge"!

From that day forward I approached each hypertonic patient with that in mind and it worked! Suddenly, a large group of patients who, previously, could not be examined using Applied Kinesiology, were testable - and it was quite effective. Specifically, whenever such a Superchallenge was found, (sometimes two were found and occasionly even three) we were able to direct therapy quickly and intensively toward the discovered priority - with excellent results!

During the winter of 1991/92, with the arrival of Jeff Farkas, DC, further development was accelerated. It became evident that the Superchallenge was not unique to my experience but rather, as long as the AK-testing assumptions made in this paper were maintained, the Superchallenge could be witnessed and used by other examiners as well. In addition, we discovered in rapid sequence that with specific patients, particularly in relation to certain areas of the Triad of Health, the following reactions are possible:

<u>A weak muscle can</u>:

stay weak - become normotonic - become hypertonic

<u>a normotonic muscle can:</u>

become weak - remain normotonic - become hypertonic

<u>a hypertonic muscle can:</u>

remain hypertonic - return to normotonic - become weak

How can this be explained physiologically?

What we noted of great interest is that patients who exhibited a hypertonic muscle condition were all extremely "stressed". This term refers to immune system correlated disturbances such as: allergies, generalized weak immune defense, rheumatic and pseudo-rheumatic problems, clinical-ecological disease such as poisoning and intoxication, enteropathic syndromes including dysbiosis, (i.e. Candidiasis), psychosomatic problems, adrenal and/or thymus weakness, vegetative stress-syndrome etc. Often, these patients were under antirheumatic, cortisone or psychoactive medication.

We found the following medicaments and procedures to be commonly helpful in returning these hypertonic patients to a normotonic condition:
Zinc, Magnesium, Copper, Histaminum D12, homoeopathic remedies (usually a single remedy in either middle or higher potencies), neural therapy to a focal problem (as long as that problem had been found to be a Superchallenge), Bach flowers remedies, ICV protocol as per AK, etc. Until a better suggestion is made, I would refer to the above mentioned findings and protocol as the "Normotonic Challenge" (NC).

Once again to review: the "Superchallenge" refers to each challenge which has the capacity to break a hypertonic state; in other words- to make a hypertonic muscle weak. Commonly, this occurs using Histidine, PCCK, candida antigen, TL to thymus or adrenals, pectoralis minor

From Superchallenge to Modern Allergy, Focus and Medicine Testing or weak - normotonic - hypertonic: 3 ways in which a muscle may test.

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Abstract:

In the last two years we have developed in our practice - with the help of colleague Natalia Budsa - what we believe to be an extremely important addition to current AK-testing procedures. Essentially, it is as follows: we need to determine whether each patient belongs to that group of patients which we have, until now, almost always seen- those that respond to a muscle test with either weakness or strength -or does the patient belong to what is, unfortunately, an increasingly larger group of patients, those that respond to a muscle test or a particular challenge with a hypertonic reaction.

Introduction:

In the mid-1980s, the topic of muscles which do not weaken was introduced into AK as the "frozen muscle" concept by Sheldon Deal, DC.[1] In May of 1992, during a discussion with George Goodheart, he indicated that he had noted the phenomenon of hypertonic muscles earlier. He thought it always to be indicative of a meridian dysbalance, most commonly the Triple Heater meridian. An author who discussed this concept further was Michael Lebowitz DC who, in 1987, discussed the "All-Muscle-Strong-Patient"[2], who often responded to orally given copper (pure orthomolecular substance) by becoming testable. I was forced in 1989/90 to utilize Lebowitz´ observations because I had, at that time, an increasing number of patients, with various types of problems, exhibiting this "all muscles strong" condition and remaining so regardless of which challenge, or what therapy localization I attempted. Copper did sometimes work, but only sometimes. I also tried, when the homoeopathic remedy picture fit the case, a high potency of Cuprum Metallicum homoeopathic. This also proved helpful in only a few cases. Further attempts were made to use Elpimed-injections and fasting, as I had heard from Gerlinde Kania that, at the Buchinger Clinic, fasting patients always seemed particularly testable. These two methods proved effective in some cases. However, is it realistic in a normal practice to expect patients to be on a fasting regiment before they have even been examined?

All in all, an unsatisfying situation. The breakthrough came at last when, "by coincidence", I found a patient with a general muscle hypertonicity, who exhibited a total body muscle weakness with therapy localization to Triple Heater 3 on the left. The patient had, coincidentally, placed one hand over another during the testing procedure and therefore, had therapy localized Triple Heater 3.

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group V – Muscles of the Third Toe							
912	Uterus-Prostate/Heart	H 6	T10	L5	Vomer	2nd Metatarsal	Core Health Reserve
976	Thymus/Tonsil	Cx 8	L2	S2	Lacrimal	Prox. Phal. 2nd Toe	Core Thymus
908	Anterior Pituitary/Stomach	Si 11	L4	S1	Maxillary, M-L	3rd Metatarsal	Core Zinc
946	Ovary-Testicle/Larynx	Cx 6	T6	S1	Ethmoid	1st Cuneiform	Core Selenium
942	Stomach (Spec. Cells)/Sublingual Gland	G 33	C4	S3	Styloid	Dist. Phal. Great Toe	Pare-X
952	Ear (External)/Kidney	Li 4	T11	S2	Nasal	Dist. Phal. 2nd Toe	Core Kidney
960	Bladder/Spleen	Si 7	T6	S3	TMJ, M-L	Dist. Phal. 4th Toe	Core Thyro
968	Adrenal/Ureter	Tw 11	L2	S3	Mandible	Prox. Phal. Great Toe	Core Panto Acid
Group VI – Muscles of the Fourth Toe							
914	Thyroid/Posterior Pituitary	B 27	T10	S1	Occiput, Universal	Calcaneus	Core Magnesium
976	Thymus/Tonsil	Cx 8	L2	S2	Lacrimal	Prox. Phal. 2nd Toe	Core Thymus
910	Anterior Pituitary/Kidney	G 27	T4	S2	Parietal, Bulge	Talus	Core Rutin
948	Colon (A-D)/Sublingual Gland	Li 5	T5	L5	Sphenoid	3rd Cuneiform, Lateral	Core Carbo Gest
944	Vagina-Penis/Larynx	G 42	T11	S2	Mandible	Prox. Phal. Great Toe	Core Niacin
954	Thymus/Vagina-Penis	Lv 9	T1	S3	Ethmoid	1st Cuneiform	Core Vitamin E
962	Submandibular Gland/Eye	Li 20	T9	S3	Styloid	Dist. Phal. Great Toe	Core Potassium
970	Uterus-Prostate (Dig. Port)/Thyroid	G 43	C4	S2	Lacrimal	Prox. Phal. 2nd Toe	Core Manganese
Group VII – Muscles of the Fifth Toe							
914	Thyroid/Posterior Pituitary	B 27	T10	S1	Occiput, Universal	Calcaneus	Core Magnesium
910	Anterior Pituitary/Kidney	G 27	T4	S2	Parietal, Bulge	Talus	Core Rutin
948	Colon (A-D)/Sublingual Gland	Li 5	T5	L5	Sphenoid	3rd Cuneiform, Lateral	Core Carbo Gest
944	Vagina-Penis/Larynx	G 42	T11	S2	Mandible	Prox. Phal. Great Toe	Core Niacin
956	Liver/Sinus (Maxillary)	Lv 6	T12	S3	Mandible	Prox. Phal. Great Toe	Super EPA
964	Sinus (Nasal)/Tonsil	H 8	T8	S3	Temporal, External	Cuboid, Lateral	Core Vitamin A
972	Nose/Lung	Lv 2	T1	S2	Maxillary, A-P	Prox. Phal. 5th Toe	Core C-TR

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group III – Muscles of the Great Toe							
906	Urethra/Heart (Left Ventricle)	Si 8	L2	S1	Palatine	1st Metatarsal	Core Magnesium
904	Thymus/Anterior Pituitary	Lv 1	L3	S1	Mandible	Prox. Phal. Great Toe	Thym-X
940	Pancreatic Duct/Jejunum	B 43	T9	S2	Temporal, External	Cuboid, Lateral	Core D-Tox
900	Liver/Anterior Pituitary	H 9	L2	S2	Vomer	2nd Metatarsal	Core Iron
902	Esophagus (Abdominal Port.)/Kidney	B 58	C5	S1	Inferior Conchae	Prox. Phal. 4th Toe	Core Selenium
924	Pineal/Lymphatics of Stomach	Cv 7	T1	S2	Lacrimal	Prox. Phal. 2nd Toe	Core Folic Acid
926	Tonsil/Pancreatic Duct	H 8	T8	S2	Frontal, External	Navicular	Core Thyro
928	Submandibular Gland/Lung	Sp 3	L3	S2	TMJ, A-P	3rd Cunei., Medial	Core Oxidate
930	Jejunum/Lung	Tw 10	L5	S2	Palatine	1st Metatarsal	Core Potassium
932	Stomach/Adenoid (Palatine)	B 51	T10	S3	Nasal	Dist. Phal. 2nd Toe	Core Thyro
934	Nose/Lung	Cv 5	C4	S3	Occiput, Lateral	5th Metatarsal	Core Carbo Gest
936	Ileum/Ovary-Testicle	Tw 12	T9	S2	Mandible	Prox. Phal. Great Toe	Core Rutin
938	Lymphatics of Jejunum/Colon (Rectum)	Si 4	T12	S3	Styloid	Dist. Phal. Great Toe	Core Carbo Gest
Group IV – Muscles of the Second Toe							
912	Uterus-Prostate/Heart	H 6	T10	L5	Vomer	2nd Metatarsal	Core Health Reserve
976	Thymus/Tonsil	Cx 8	L2	S2	Lacrimal	Prox. Phal. 2nd Toe	Core Thymus
908	Anterior Pituitary/Stomach	Si 11	L4	S1	Maxillary, M-L	3rd Metatarsal	Core Zinc
946	Ovary-Testicle/Larynx	Cx 6	T6	S1	Ethmoid	1st Cuneiform	Core Selenium
942	Stomach (Spec. Cells)/Sublingual Gland	G 33	C4	S3	Styloid	Dist. Phal. Great Toe	Pare-X
950	Lymphatics of Ileum/Sinus (Maxillary)	B 63	T10	S2	Inferior Conchae	Prox. Phal. 4th Toe	Pare-X
958	Pancreatic Duct/Eye	B 65	L4	S2	Occiput, Universal	Calcaneus	Core Zinc
966	Spleen/Thymus	B 2	T7	S3	Vomer	2nd Metatarsal	Core Iron

CALF, ANKLE & FOOT EXAM CORRELATIONS

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group I – Muscles of the Calf and Ankle							
878	Adrenal/Anterior Pituitary	Cx 8	T3	S2	Frontal, External	Navicular	Core Rutin
880	Colon (Rectum)/Liver	Si 12	T9	S2	Frontal, External	Navicular	Core Liver
874	Colon (A-D)/Liver	Li 6	T9	S2	Occiput, Lateral	5th Metatarsal	Core Thyro
872	Duodenum/Kidney	Tw 11	T11	S2	Occiput, Universal	Calcaneus	Core Ileoduodenal
876	Jejunum/Lymphatics of Tonsil	Tw 13	L4	S1	Zygoma	2nd Cuneiform	Core Magnesium
870	Vagina-Penis/Gallbladder	G 32	L1	L5	Temporal, Internal	Cuboid, Inferior	Core Inositol
Group II – Muscles of the Arch							
882	Ileum/Pancreas (Protein)	Li 8	C3	L5	Parietal, Bulge	Talus	Core Zinc
884	Heart/Ovary-Testicle	H 3	T7	L5	Sphenoid	3rd Cuneiform, Lat	Core Potassium
922	Heart/Liver	Lu 2	L3	S1	Frontal, Internal	Prox. Phal. 3rd Toe	Core Potassium
920	Ant Pituitary/Uterus-Prostate (Dig. Port.)	K 10	L3	S1	Ethmoid	1st Cuneiform	Core Potassium
896	Jejunum/Urethra (Prostate Portion)	Sp 6	L2	L4	Frontal, External	Navicular	Core Calcium
898	Spleen/Ovary-Testicle	Lu 4	C5	L5	Frontal, External	Navicular	Spore-X
888	Parathyroid/Gallbladder	Li 7	T8	S1	Parietal, Bulge	Talus	Core Manganese
886	Bladder/Duodenum	H 2	T11	L5	Ethmoid	1st Cuneiform	Core Ileoduodenal
890	Lung/Urethra (Membranous Portion)	G 32	T11	L5	Parietal, Bulge	Talus	Core Iron
892	Bladder/Nose	B 67	T5	S1	Temporal, Internal	Cuboid, Inferior	Core Health Reserve
974	Nose/Posterior Pituitary	St 33	C5	S2	Occiput, Lateral	5th Metatarsal	Core Vitamin A
894	Lung/Bladder	Tw 9	C4	L5	Temporal, Internal	Cuboid, Inferior	Core Manganese

KNEE EXAM CORRELATIONS

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group V – Muscles of the Knee (Knee Flexed)							
810	Adrenal/Lymphatics of Ileum	Si 9	T10	L3	Frontal, External	Navicular	Core Adrenal
816	Lung/Duodenum (Special Cells)	K 5	L1	L4	Glabella	4th Metatarsal	Core Vitamin A
818	Adrenal/Gallbladder	Li 7	T9	L3	Glabella	4th Metatarsal	Core Pancreas
820	Posterior Pituitary/Ileum	Sp 12	T8	L3	Styloid	Dist. Phal. Great Toe	Core Magnesium
830	Thyroid/Thymus	Li 8	T6	L3	Parietal, Bulge	Talus	Core Thyro
832	Pancreatic Duct/Submandibular Gland	K 4	C3	L3	TMJ, A-P	3rd Cunei., Medial	Core Methionine
834	Ileum/Pancreatic Duct	Tw 4	T7	L2	Parietal, Bulge	Talus	Core Parathyroid
836	Ileum/Parotid Gland	Tw 14	L5	L4	Lacrimal	Prox. Phal. 2nd Toe	Pare-X
838	Tonsil/Sublingual Gland	Sp 1	T3	L4	Parietal Descent	Dist. Phal. 3rd Toe	Spore-X
840	Ovary-Testicle/Adrenal	B 63	C4	L3	Occiput, Lateral	5th Metatarsal	Core Carbo Gest
Group VI – Muscles of the Knee (Leg Straight)							
808	Adrenal/Uterus-Prostate	St 32	T2	L4	Frontal, External	Navicular	Core Uterus/Prost.
Group VII – Muscles of the Knee (Internal and Extrernal Rotators)							
824	Colon (A-D)/Eustachian Tube	Lu 10	L5	S1	Zygoma	2nd Cuneiform	Core Rutin
826	Colon (A-D)/Spleen	Lu 5	L5	S2	Occiput, Universal	Calcaneus	Core Health Res.
828	Uterus-Prostate (Dig. Port)/Subl. Gland	Li 11	L4	S2	Temporal, External	Cuboid, Lateral	Core Niacin
854	Posterior Pituitary/Adrenal	G 30	T9	L5	Frontal, External	Navicular	Core Oxidate
856	Lymphatics of Submandibular/Adrenal	Sp 8	L5	S1	Sphenoid	3rd Cunei., Lateral	Core Magnesium
858	Ileum (Peyer's Patches)/Pancreatic Duct	B 25	C2	S1	TMJ, M-L	Dist. Phal. 4th Toe	Core Ileoduodenal

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group III – Muscles of the Thigh (Prone)							
846	Nose/Lymphatics of Thyroid	G 44	T10	L2	Occiput, Universal	Calcaneus	Core Iron
852	Bladder/Colon (Rectum)	B 13	C6	S1	Palatine	1st Metatarsal	Core Potassium
848	Pancreas (Sugar)/Pancreatic Duct	St 41	C4	S2	Inferior Conchae	Prox. Phal. 4th Toe	Core Pancreas
850	Post. Pituitary/Uterus-Prostate (Dig. Port.)	H 1	L4	S3	Nasal	Dist. Phal. 2nd Toe	Core Vitamin E
860	Uterus-Prostate (Dig. Port)/Ovary-Testicle	Sp 8	C2	L5	Occiput Lateral	5th Metatarsal	Core Vitamin E
862	Lung/Uterus-Prostate	Li 17	L1	L5	Occiput, Lateral	5th Metatarsal	Core Magnesium
864	Heart/Ovary-Testicle	Si 2	T2	L5	Zygoma	2nd Cuneiform	Core Thiamine
Group IV – Muscles of the Pelvis							
740	Ileum/Pancreatic Duct	Lv 5	T1	S3	Palatine	1st Metatarsal	Core C-TR
742	Stomach (Pyloric Canal)/Thymus	Li 10	C4	S2	Maxillary, M-L	3rd Metatarsal	Core Thymus
746	Thyroid/Uterus-Prostate	G 41	L2	S2	Lacrimal	Prox. Phal. 2nd Toe	Core C-TR
744	Lung/Heart	Gv 26	L5	S2	Occiput, Universal	Calcaneus	Core Pepsin

PELVIS & THIGH EXAM CORRELATIONS

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group I – Muscles of the Thigh (Leg Straight)							
780	Gallbladder Duct/Vagina-Penis	St 39	T4	L5	Maxillary, M-L	3rd Metatarsal	Core Bile
782	Uterus-Prostate/Mammary	Cx 2	L4	L5	Parietal Descent	Dist. Phal. 3rd Toe	Core Iron
784	Lung/Ovary-Testicle	Lu 11	L2	L5	Occiput, Lateral	5th Metatarsal	Core Zinc
786	Pancreas (Sugar)/Lung	Sp 3	L5	S1	Temporal, Internal	Cuboid, Inferior	Core Manganese
788	Pancreatic Duct/Vagina-Penis	Li 1	C6	L5	Maxillary, M-L	3rd Metatarsal	Pare-X
790	Colon (A-D)/Eustachian Tube	G 34	L2	L5	Parietal, Bulge	Talus	Pare-X
792	Thyroid/Vagina-Penis	Lu 1	T12	S1	Parietal, Bulge	Talus	Core D-Tox
794	Thyroid/Liver	Tw 7	T4	L3	Occiput, Lateral	5th Metatarsal	Core Calcium
796	Jejunum/Lymphatics of Ileum	Lu 6	T3	L4	TMJ, M-L	Dist. Phal. 4th Toe	Core Niacin
800R	Gallbladder Duct (Amp. of Vater)/Rt. Eye	St 41	T10	L4	Ethmoid	1st Cuneiform	Core Health Res.
800L	Duodenum/Left Eye	St 41	T4	L4	Ethmoid	1st Cuneiform	Core Health Res.
804	Ileum/Vagina-Penis	Si 6	T7	L3	Parietal Descent	Dist. Phal. 3rd Toe	Core Selenium
806	Liver/Ovary-Testicle	B 65	T1	L4	Maxillary, A-P	Prox. Phal. 5th Toe	Core Carbo Gest
842	Uterus-Prostate (Broad Ligament)/Bladder	Xl 2	T2	L4	Vomer	2nd Metatarsal	Core Magnesium
844	Ant. Pituitary/Uterus-Prostate (Broad Lig.)	Li 3	C2	L3	TMJ, A-P	3rd Cunei., Medial	Core Panto. Acid
Group II – Muscles of the Thigh (Knee Flexed)							
812	Heart/Spleen	Tw 13	L4	L4	Temporal, Internal	Cuboid, Inferior	Core Zinc
814	Ovary-Testicle/Nose	Lu 11	T5	L5	Styloid	Dist. Phal. Great Toe	Core Parathyroid
822	Lung/Larynx	Li 5	T9	S2	Vomer	2nd Metatarsal	Core Lung

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group VI – Muscles Affecting Diaphragm							
656	Lymphatics of Jejunum/Lymphatics of Legs & Ing. Nodes/Heart	R G 30 L Sp 21	T12	C4	Maxillary (M-L)	3rd Metatarsal	Thym-X
662	Spleen/Lymph. of Legs & Ing Nodes/ Lymphatics of Pancreas	G 30 Sp 21	T12	C5	Maxillary (M-L)	3rd Metatarsal	Spore-X
612	Pineal/Heart	B 62	T3	T3	TMJ, A-P	3rd Cuneiform, Medial	Core Magnesium
610	Heart/Bladder	G 31	T6	T6	Mandible	Prox. Phal. Great Toe	Core Selenium
614	Stomach/Pancreatic Duct	H 5	C6	T7	Temporal, Internal	Cuboid, Inferior	Health Reserves
618	Adrenal/Mammary	St 32	T8	T10	Ethmoid	1st Cuneiform	Uter-X/Prosta-X

ABDOMEN, LOW BACK AND DIAPHRAGM EXAM CORRELATIONS

#	Organ	MAP	VL	MM	Cranial	Foot	Nutrient
Group I - Muscles Affecting Abdomen							
690	Bladder/Nose	Sp 11	L3	T12	Vomer	2nd Metatarsal	Core Heart
700	Adrenal/Colon (A/D)	Cx 4	T9	T5	Temporal, Internal	Cuboid, Inferior	Core D-Tox
702	Duodenum (Hor. Por.)/Sublingual Gland	Li 14	L4	T7	Sphenoid	3rd Cuneiform, Lat.	Core Thyro
704	Ileum/Eye	Tw 12	T4	T9	Sphenoid	3rd Cuneiform, Lat.	Splen-X
728	Lymphatics of Stomach/Post. Pituitary	St 29	L5	L2	Maxillary (M-L)	3rd Metatarsal	Core Thyro
706	Duodenum (Asc. Port.)/Esophagus	G 31	L4	T8	Frontal, Internal	Prox. Phal. 3rd Toe	Core Folic Acid
708	Vagina-Penis/Bladder	Li 9	T6	T7	Sphenoid	3rd Cuneiform, Lat.	Core Kidney
Group II - Muscles Affecting Abdomen							
714	Sublingual Gland/Post. Pituitary	Si 5	T7	T10	Parietal, Desc.	Distal Phal. 3rd Toe	Core Oxidate
692	Colon (A/D)/Sinus (Frontal)	Li 14	T11	T9	Occiput, Universal	Calcaneus	Core Selenium
Group III - Muscles Affecting Abdomen							
698	Tonsil/Uterus-Prostate	Cx 8	C3	T11	Frontal, Internal	Prox. Phal. 3rd Toe	Core Folic Acid
696	Colon (Hepatic/Splenic Flexure)/Eye	Si 8	T3	T10	Ethmoid	1st Cuneiform	Core Folic Acid
718	Thymus/Duodenum (Desc. Portion)	H 4	L1	T9	Parietal, Bulge	Talus	Thym-X
694	Lymphatics of Colon/Sinus (Sphenoid)	Lv 4	L1	T9	Parietal, Bulge	Talus	Thym-X
Group IV - Muscles Affecting Low Back							
730	Liver/Colon (A/D)	St 38	L4	L3	Vomer	2nd Metatarsal	Core Potassium
732	Gallbladder/Tonsil	Sp 8	T4	L2	Palatine	1st Metatarsal	Spore-X
734	Ovary-Testicle/Colon (Sigmoid)	Li 15	L1	L3	Styloid	Distal Phal. Great Toe	Core Magnesium
736	Lung/Stomach (Pyloric Valve)	Si 9	T5	L4	Palatine	1st Metatarsal	Core Iron
738	Jejunum/Eye	Tw 1	L2	L2	Maxillary (M-L)	3rd Metatarsal	Core Iron
Group V - Muscles Affecting Low Back							
798	Colon (Transverse)/Pancreatic Duct	St 38	L4	L3	Maxillary, M-L	3rd Metatarsal	Core Magnesium
710	Colon (A-D)/Lymphatics of Jejunum	Lu 10	L4	L3	Occiput, Universal	Calcaneus	Core Manganese
712	Pancreas (Sugar)/Kidney	G 32	T9	L3	Frontal, Internal	Prox. Phal. 3rd Toe	Core Methionine
722	Kidney/Colon (Rectum)	G 37	L1	L4	Occiput, Lateral	5th Metatarsal	Core Kidney
724	Kidney/Mammary	Lv 4	T12	L3	Occiput, Universal	Calcaneus	Core Kidney
726	Adrenal/Bladder	Gv 2	T12	L2	Maxillary (M-L)	3rd Metatarsal	Core Adrenal
752	Spleen/Pancreatic Duct	K 7	T8	L3	Occiput, Universal	Calcaneus	Core Iron

RESULTS:

In a series of 23 patients I found an average change of 1.48 mm. That is the measurement got shorter by 1.48 mm. in the average case. It got longer in 2 cases and showed no change in 7 cases.

When I compared the overall change from full inspiration to full expiration I got an average increase of 1.25 mm. with 5 showing no change, 13 showing an increase in overall change, and 4 showing a decrease in overall change.

DISCUSSION:

It appears that in most cases of bilateral lower trapezius inhibition due to fixations in the dorsal lumbar area of the spine, the correction of fixations appears to result in a shortening of the distance between T1 and T12 in 60.87% of the cases studied. This is not as high a correlation as I felt I would find. I had to do the experiment twice. At first I only took one measurement prior to, and one measurement after treatment, that was in the neutral, quiet respiration phase. My first 17 patients showed confirmation of my original hypothesis, but then I started to get exceptions. I then felt that I may have measured those exceptions on inspiration on the second measurement, and tried to reduce that possibility by measuring the next group in all 3 phases of respiration. I do not have a full explanation of the exceptions. There must be other factors involved. I know one of the cases has a case of mild apophysitis in the thoracic spine which may make him react differently. One case even showed a dramatic improvement of the kyphosis which seemed contradictory. It is possible that a height measurement should have been done or a whole spine measurement, as Goodheart does, from coccyx to the external occipital protuberance.

DORSO LUMBAR FIXATIONS.....BOEHNKE
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	Comp. ref #	Date	Expiration	Neutral	Inspiration
1	1198	Feb 9/93	38.7	39.0	39.5
			38.4	38.9	39.2
2	1326	Feb 9/93	31.7	31.9	32.5
			31.4	31.7	32.2
3	1428	Feb 9/93	32.5	33.0	33.2
			32.0	32.5	33.0
4	45	Feb 9/93	33.6	33.7	33.9
			33.2	33.3	33.5
5	282	Feb 9/93	33.9	34.0	34.3
			33.8	33.8	34.1
6	1427	Feb 10/93	35.3	36.0	36.2
			35.4	36.0	36.2
7	1288	Feb 11/93	29.5	29.7	30.2
			29.2	29.7	30.2
8	1387	Feb 11/93	28.3	28.7	29.0
			27.9	28.3	29.1
9	7	Feb 11/93	31.3	31.3	31.5
			31.0	31.3	31.5
10	136	Feb 11/93	37.5	37.6	38.0
			37.3	37.4	38.0
11	1344	Feb 11/93	33.7	33.8	34.0
			34.4	34.5	35.0
12	51	Feb 12/93	33.4	33.5	34.0
			33.4	33.5	34.0
13	450	Feb 12/93	32.4	32.5	33.0
			32.1	32.4	33.0
14	11	Feb 12/93	32.2	33.5	34.7
			33.5	34.0	35.0
15	209	Feb 12/93	36.2	36.4	36.5
			35.7	36.0	36.5
16	246	Feb 23/93	36.3	36.5	37.0
			36.5	36.5	37.2
17	838	Feb 23/93	33.2	33.5	34.1
			33.2	33.5	34.2
18	214	Feb 23/93	32.9	33.0	33.1
			32.4	32.5	32.7
19	87	Feb 12/93	33.4	33.5	34.0
			34.4	34.5	34.8
20	1347	Feb 12/93	33.8	34.4	34.8
			33.5	33.8	34.2
21	5	Feb 13/93	37.3	37.5	38.2
			36.9	37.0	38.5
22	1326	Feb 9/93	31.7	31.9	32.5
			31.4	31.7	32.2
23	772	Feb 17/93	28.3	28.8	29.0
			28.0	28.5	29.0

Measurement of T1-T12 in cm.
Upper readings prior to treatment. Lower readings post treatment.

CONCLUSIONS:

In 60.87% of cases of dorso lumbar fixations resulting in a bilateral inhibition of the lower trapezius that were studied, a decrease in the measurement of T1 to T12 was obtained by the correction of the fixations. This change averaged 1.48 mm. and in my opinion was associated with the facilitation of the lower trapezius muscles.

SUMMARY OF PROCEDURES:

1) Do a postural evaluation looking for dorsal spine kyphosis.
2) With the patient prone test the lower trapezius muscles for an inhibition or weakening pattern.
3) If present palpate the cervical thoracic junction to determine T1. On passive extension of the cervical spine C7 will move anterior much more than T1. Mark T1 with a marker.
4) Palpate for the 12th. rib, and follow it along to the spine, that will be T12. Then mark the spinous of T12.
5) Using a measuring tape measure the distance from T1 to T12 in centimeters. Take this measurement in neutral quiet respiration, full inspiration, and full expiration.
6) Correct the fixations present.
7) Retest the lower trapezius muscles for facilitation.
8) Remeasure the T1 to T12 measurement as in number 5 above.

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THE USE OF MANUAL MUSCLE TESTING AND PATIENT POSITION TO ASSIST IN THE DIAGNOSIS AND TREATMENT OF DIFFICULT CASES OF FORAMINAL AND/OR LATERAL DISC LESION

HANS W BOEHNKE, D.C.

ABSTRACT:

In a series of difficult cases of spinal pain associated with radicular symptoms I have found that a little creative use of body position combined with muscle testing can reveal valuable information about the injury and its treatment. It appears to be successful in the small sampling of cases that I have been able to try it on. The procedure involved having the patient identify the segmental sensory innervation on a chart, testing muscles related to that segmental innervation by means of a doctor started test and then use patient positions i.e. left lateral bending, right lateral etc. to either facilitate an inhibited muscle or to inhibit an apparently intact muscle. The treatment is based on usual treatment modes but with the addition of a patient position opposite that of the aggravating position. I have had very good result to date using this protocol.

INTRODUCTION:

In the past year I have had a number of very difficult cases of radicular pain or numbness associated with low back pain. I had tried our standard Chiropractic and Applied Kinesiology approaches with moderate improvement. I then remembered that back in college, I had heard of a Chiropractor who would treat some cases in a side lying position with the body in a lateral bending position to open the foramina and disc spaces on the elevated side (1). His technique intrigued me for a while but as the years went on and other techniques caught my interest I let it fade from my conscious memory. Then while having difficulty with a case taking longer than I felt it should, I remembered the basic concept of the technique and applied it.

In a sense it is similar to BID (Body Into Distortion) (2). I have a copy of the CIBA Clinical Symposium Volume 32 Number 6, 1980 on low back pain in each treatment room (3). I turned it to page 7 and had the patient identify the radicular symptoms on the diagram thereby giving me the segmental sensory innervation or dermatome involvement. I then simply went to the lower diagram which gave the segmental innervation of lower limb movements and looked for the corresponding muscle to test. For instance the L5, S1 dermatome symptoms should relate to the hamstrings which get their innervation from L5, S1. I then tested the muscle in the prone position. If the muscle was inhibited (demonstrated weakness) I would place him in a position of right lateral bending, in the hope that it might reduce pressure or irritation to the innervating nerve. If that position caused facilitation of the previously inhibited muscle I would challenge test him in the neutral position to find what structural corrections were necessary and add the component of a lateral bend to the right at the time of treatment. This was quite easy to do with my treatment tables (Zenith Vertilifts) which can be wedged to cause distraction when the patient is in the side lying position. This patient responded very well to this approach. In his case I frequently found that I only needed to place him in a side lying lumbar roll position with slight torque with a left convex bend of his spine, and hold him in that position for 30 - 60 seconds and a series of audible releases would occur.

There are also cases much like the well known 51% phenomenon where we have the patient therapy localize a factor relating to a suspected muscle inhibition to have it reveal itself. These cases in my protocol show the muscle related to the segmental innervation as being intact until the patient is put into a position that tends to accentuate the lesion, then the inhibition pattern reveals itself.

The treatment mode would be to challenge the patient in the position that shows the lesion, to see what treatment would decrease the effect of the lesion and treat them with adjustment of a lumbar segment, a sacro iliac, a sacral lesion or disc succession, or respiratory treatments etc., but in a position opposite to that which accentuated the lesion.

DISCUSSION:

In these cases I have found that a progression of improvement can be noted that can be seen by manual muscle testing. For instance, let us say a patient has a left sciatic pain causing radicular symptoms in the S1 dermatome as indicated in the segmental sensory innervation (after Keegan). We then test the left hamstring and find it inhibited. However, placing the patient in the right lateral bend position, i.e. spine convex to the left, facilitates the muscle. This would indicate that there is some space occupying lesion which has its effect lessened by the opening of the disc spaces and/or foramina at L5, S1 on the left. This would in my experience correlate with the standard tests for discs such as a positive straight leg raise on the ipsilateral side of the lesion and sometimes in more severe cases a positive straight leg raise on the contralateral side.

I would do most of the standard tests, valsalva manoeuvre, Bechterew's test, etc., which we are all familiar with. If at that point we suspect a diagnosis of disc injury (protrusion) on the left (lateral to the nerve root), we can proceed with challenge testing to determine treatment which may include the respiratory treatment of the intravertebral disc as proposed by Goodheart (4), light adjustment of L5, and/or sacro iliac treatment, or just disc succession. The only difference is that the treatment is done in a right lateral bending position, i.e. side lying on the right side with the table wedged, or cushions under the right side causing a convexity to the left. If the treatment is successful the left hamstring will appear intact. On the next visit the left hamstring may still appear intact with likely some improvement in symptoms, however we can now put the patient in the left lateral bending position to accentuate the lesion's effect on the hamstring. If it shows inhibition in this position we know that although improvement was noted, more treatment is indicated. To this protocol can be added the Applied Kinesiology factors such as dural tension, and torque to help in determining the progress of the patient. Thus I have found that treatment progress can be assessed with some degree of objectivity and can be demonstrated to the patient even though they may not have had a noticeable change in symptomatology on that particular treatment visit.

CONCLUSIONS:

Applied Kinesiology can be used with some creativity to augment standard diagnostic tests, and to determine treatment modes and the progress of treatment. The determination of improvement by the treatment applied may be seen in advance of the changing symptoms, thus we can assure the patient that the treatment done was correct, and that the symptom improvement should come as other factors such as inflammation etc. are brought under control.

PATIENT POSITION DIAGNOSIS.....BOEHNKE
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PROCEDURE OVERVIEW:

1) The patient presents with spinal pain and radicular symptoms in the lower back and leg.
2) Show the patient the diagram by the medical artist Frank Netter on segmental sensory innervation (dermatomes) of the lower limb (after Keegan). Have the patient identify the dermatome involved as closely as he or she can.
3) Go to the lower diagram of Frank Netter, which shows segmental innervation of lower limb movements (after Last), and test the muscles indicated for that segmental innervation related to the same dermatome as indicated in 2) above.
4) If the muscle shows inhibition place the patient in various positions, i.e. first in a position which causes convexity on the side of the symptoms and suspected lesion. Try to find a position that facilitates the muscle. The position in the case of the lesion medial to the nerve root may even be in a position which causes concavity on the lesioned side.
5) If the muscle related to the dermatome involved appears intact find a body position that inhibits the muscle. A case such as this would obviously be less severe than in 4) above.
6) Challenge for various Applied Kinesiology treatment modes in the position that demonstrates inhibition of the related muscle.
7) Treat the patient with the normal treatment modes but place him or her in the body position that facilitates rather than inhibits the muscle response.
8) Retest and determine the effect of the treatment.

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ADDENDUM:

I would like to point out that body position for diagnosis may not be a new concept. Dr. Goodheart first proposed body into distortion in 1986, and Bandy expanded on the idea with his proposals of weakness of specific muscles not associated with the five factors of the IVF indicating disc involvement. He further used specific muscles (after Beardall) to determine the level and their strengthening when the body was placed in lateral bending. I acknowledge and applaud their work. The main thrust of my article is to point out that it appears to be helpful to not only be able to determine the level of the lesion but to also use what appears to be a more ideal body position for treatment. I have not studied Beardall's methods, and am not sure how much overlap of procedure may be present. I have not done an exhaustive review of the past literature on this. If these proposals are in some way a differnet slant on a previous author's idea, let my paper be a confirmation of the effectiveness of the method and lead others to expand further on it.

Case report of Applied Kinesiology Treatment and Allergic Cutaneous Vasculitis

David B. Dauphiné, D.C.

Abstract:

Allergic cutaneous vasculitis identified and eliminated through applied kinesiological examination and treatment.

Introduction:

Allergic cutaneous vasculitis or nodular angiitis is an inflammation of blood or lymph vessels of the dermis or epidermis. This condition is marked by papules, macules, vessicles, urticarial wheals or purpura. It is often accompanied by pruritis and erythemia.(1).

Until a cause is identified, dermatitis remains a visibly frustrating mystery to physician and patient. The patient is often inflicted emotionally as well as physically. Continuing pain, itching, and/or discolorations, progression of lesions, subsequent scarring, and a future, more systemic diagnosis, are discomforting possibilities.

A non-invasive, inexpensive identification and treatment of this condition through Applied Kinesiology examination, diagnosis, and treatment, would be valuable.

Case Presentation:

Suzanne is a 57 year-old, white female, weighing 230 pounds, at 5 feet 5 inches in height. She is a self-employed bookkeeper, married with two children. Her soft weight gain was dispersed between the elbows and knees and would be classified as 'hypo-thyroid'.(2). Her presenting complaint was a medically diagnosed 'allergic vasculitis', without known cause.

What I saw was a very obvious skin rash of bright red blotches upon extremely white skin. The erythematous, segmental lesions were on this patient's feet, stomach, chest, neck, and forearms. Some were raised and palpable nodules of varying sizes, while others were flat macules and

patches. There was an interconnecting system of superficial blood vessels, telangiectasias, that usually did not itch, appeared to be deeper in the dermis, and had a red, 'glassy' or hyaline reflective quality. The vasculitis was first noticed in March, 1991. Many medical visits and referrals later to doctors, allergists, and dermatologists, the condition was diagnosed as "allergic vasculitis", without the identity of any causal allergen.

Several possible associations were looked at by the treating physicians prior to my examination of the patient. In May, 1989, a dermatologist had prescribed a sulfa-medicated topical cream for a long-standing 'sensitive skin' condition. Suzanne has skin sensitivity to dyed toilet paper, perfumes, and artificial, rough, or tight-fitting clothing and fibers. She experienced a contact dermatitis from using the cream, and an exacerbation of the vasculitis. She was allergic to sulfa drugs in the past, and they thought this reaction, or perhaps another drug side-effect, or interaction, responsible for the vasculitis.
I was more interested in her past need for sulfa medication.

Suzanne had terrible acne as a teenager. For this condition, she was also given xray 'treatments' to her face, chest, upper arm and back and fascial plastic surgery. She has had a history of basal-cell carcinoma extractions, and is taking Synthroid for "hypothyroidism". Other current medications were: Premarin since a hysterectomy in 1975; Capotin for hypertension; Bayer aspirin; and Xanax for anxiety and arthritis. Medications were substituted and eliminated without affecting the vasculitis. While on Prozac, Suzanne had several serious nose bleeds, and painful sinuses.

Another possible association that had been examined was the patient's exposure to the chickenpox virus in 1990, which she had had in her childhood. Also, Suzanne had been briefly hospitalized for acute anxiety in February, 1991. She was then diagnosed as diabetic, type II(non-insulin dependent). Perhaps there was an association between the diabetes and the vasculitis? Water contamination in Suzanne's town or a reaction to artificial sweeteners were concerns of hers upon our first meeting. She was mentally anguished and embarrassed to be in public.

I examined Suzanne on 7/26/91. Unusual initial examination findings included an acid urine of 4.5ph, a Koenisburg's test result of 40(3), positive pulpilary dilation of 5 seconds to bright light in an extremely large pupil. She was on smaller, more frequent meals, and a complex carbohydrate, diabetic diet. She did not exercise. Her periods before the hysterectomy were irregular with heavy bleeding and cramps. She had a progressive history of

ovarian cysts. Her family medical history included heart attacks, high-blood pressure, and strokes on both sides of her family, with no identified allergies.

Through gamma II(eccentric) muscle testing(4), there were bilateral weaknesses of the psoas, piriformis, and subscapularis muscles. Therapy localization to C5, and oral challenge with niacinamide negated these weaknesses. Oral challenge with milk(both casein and lactate proteins) and cocoa reweakened these same three muscle pairs. I adjusted C5 right posterior and recommended a strict elimination diet, free of cocoa and milk proteins. Also, I recommended increasing alkaline foods, an adrenal body-type diet (2), and four 500mg of niacinamide daily. I scheduled a return visit in four weeks.

At the next visit, on 8/23/91, Suzanne reported that she had adhered to my recommended diet. It wa disappointing not to see a change in the vasculitis. It had occasionally receded and she thought that being around freshly cut grass affected it. I localized and adjusted C5 right posterior, T5 anterior, and treated the neurolymphatic reflex(5) of the right semispinalis capitis muscle through muscle testing, therapy localization, and challenge. Gamma II testing revealed strong bilateral psoas, piriformis, and subscapularis muscles and a bilateral weakness of subclavius, teres minor, and pectoralis major sternal. These strengthened to therapy localization and challenge to L4 right which I adjusted, right posterior. I asked the patient to remain on her elimination diet as oral challenge with milk/cocoa brought back the original bilateral piriformis and subscapularis gamma II weaknesses.

On 9/6/91, She disclosed that one week after the last visit, all of the dermatitis healed and disappeared. She then ate milk proteins in a lasagne dish, and the pruritis and vasculitis returned. This meal also brought on a headache, sinusitis, and a sore low back. I adjusted a L4 right and C5 right as described above. this patient has been strictly on a milk protein and cocoa free diet. Her vasculitis has healed, disappeared, and not recurred. She reports that her skin is not abnormally sensitive.

Discussion:

The body's immune response to consumed foods is not well understood. This case is unusual in the type of lesion provoked by a food antigen. It is also remarkable in the length of elimination period necessary to detect a clearing

of symptoms. The total elimination period required was five weeks. This is in contrast to the routine one week elimination period, if this patient was successful in identifying all milk proteins in the foods consumed. Experience has taught me that a very small lapse in as allergy-free diet can stimulate a reaction. I am indebted to John V.N. Bandy, D.C. of Austin, Tx., for his knowledge and work in this area, including a three to four week elimination period for a suspected food allergen.

Of greater interest, perhaps, is that the allergy in this case was uncovered by applied kinesiology techniques through muscle testing. Walther writes, " The applied kinesiology approach to allergy and hypersensitivity is to identify items to which the patient is allergic or sensitive by how the nervous system reacts, as observed by its control of muscles evaluated by manual muscle testing.".(6). This same author quotes Scopp, "'Correlation between foods identified as provocative by muscle testing and by the fast[ed.Philpott-type fast] was .81.'" and, "'Observation of clinical results obtained with muscle testing suggests the method has substantial clinical utility.'".(6). I refer you to Dr. Walther's text for a documented critical discussion of other means of allergy testing.

A large number of diseases are characterized by vasculits. These include the collagen vascular diseases, such as: systemic lupis erythematosis(SLE); scleroderma; rheumatoid arthritis.(7)(8). This case demonstrates that vasculitis can be associated with a food allergy. This has also been demonstrated through the work of Reading, M.D.(8), and Philpott, M.D.(9).

Conclusion:

This case demonstrates the value of applied kinesiology muscle testing in determining the cause of a medically diagnosed allergic cutaneous vasculitis. It is time that such a potentially beneficial discipline be researched and examined for wider, more intense use in our health care delivery system.

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Case Report of Applied Kinesiology Treatment and Infant Hydrocephalus

David B. Dauphiné, D.C.

Abstract:

Skull circumference reduction, personality, and behavior change in diagnosed case of infant hydrocephalus following Applied Kinesiologic examination and treatment.

Introduction:

"Hydrocephalus, characterized by enlarged ventricles of the brain, can result either from increased or decreased absorption of cerebrospinal fluid or from blockage of one of the normal outflow pathways of the ventricular system. The most common forms of hydrocephalus occur in infants. Because cranial sutures are not yet fused, head size increases progressively; thus, periodic measurement of the skull's circumference to detect such enlargement is important in neonatal and infant care."(1)

Communicating, rather than obstructive hydrocephalus, is the most common reason for enlarged skulls in neonates and can result from meningeal inflammation from infection or blood within the subarachnoid space.(2) Medical treatment depends on etiology. Lumbar punctures are used for temporary reduction in cerebrospinal fluid(CSF) in many instances. Progressive hydrocephalus often requires a shunt from the ventricles into the right atrium of the heart or to the peritoneal cavity.

Periodic surgery for shunt revisions, necessary extensions, and antibiotic therapy are required during the child's growth. The course of congenital, versus acquired, hydrocephalus is usually progressively downhill, with death in the first or second year of life, resulting from recurrent infections.(3) Some hydrocephalics arrest spontaneously. However, careful monitoring for skull enlargement, along with behavioral and other signs and symptoms, is necessary to detect the progression of this condition.

Any reversal of the progression by means of manual medicine, facilitated by Applied Kinesiologic diagnosis and treatment, would be valuable.

Case Presentation:

Jake's mother is an educated, long-time chiropractic patient in an applied kinesiology practice. Jake was born 10/8/90, 5 weeks premature, by C-section, after concern over placenta prevara. At 6 pounds and 5 ounces, he was slightly jaundiced, but not needing artificial support. He stayed in the hospital for 3 days of monitoring. He was listed as a 36 week term baby. Skull size was within 25% below average when born.

He endured recurrent colds and ear infections during his first 10 months as a "normal and calm" baby. On 8/5/91, Jake began a constant scream, and was brought to the local hospital ER with a temperature of 104 degrees. Tests were done to rule out pneumonia and meningitis, including 2 chest xrays, blood profile, urine, and a spinal tap. Tylenol and emoxocilin were prescribed and he was released. Diagnosis was a low grade viral or bacterial infection.

I first saw Jake on 8/6/91, the day after his ER visit. His temperature was normal. He was sedated. I adjusted his mid-cervicals and right sacrum, using his mother in surrogate testing, after examining and adjusting his mother. During August and September, Jake's parents noticed more aggressive behavior than they thought normal and had experienced with their other two children. Jake learned the behavior of repeatedly slapping his hand to his forehead. He repeatedly woke up agitated and fearful, as if from a bad dream. These were changes from his previous behavior.

On 9/23/91, his pediatrician was concerned with Jake's head size during a routine exam. It measured 50 cm which was above 98% of infants his age, and was radically different from within the 25% below normal girth when born. Hydrocephalus was suggested with resulting brain damage, the possible need for surgical shunts, and the need to pursue diagnosis. CT scans were ordered at the county hospital. On 9/25/91, the radiologist diagnosed hydrocephalus and possible brain damage and referred to a pediatric neurologist. On 9/26/91, the chief of pediatric neurology at the regional hospital measured the head at 51cm and down-played this increase in size after a neurologic exam. He placed Jake under close observation. On 10/15/91, Jake measured 51 cm on a repeat visit. On 11/18/91, Jake saw a local pediatrician for persistent ear infections. Head circumference measured 52cm and had grown 2cms in less than two months. This placed Jake significantly above the 95th percentile of recorded head size for his age. This prompted new concerns and a new referral to a pediatric neurosurgeon at an even larger regional, teaching hospital. Upon examination on 11/25/91, and after a second CT scan, the diagnosis was progressive hydrocephalus. The treatment

course of periodic surgical shunt operations was outlined for the parents. Jake returned home to await surgery at a more critical moment in signs and symptoms. The parents were asked to monitor behavioral changes such as: sleeping or being awake too much; more aggressive personality or sudden lethargy; increase in moodiness; vision changes caused by pressure increase in the eyes; other neurologic dysfunction or dementia.

On 11/30/91, I examined Jake and treated him via surrogate testing, after examining and treating his mother. He appeared agitated, fearful, and sensitive to sound. I employed therapy localization and challenge to locate a right posterior occiput, T5 anterior, and a right frontal bone subluxation. I adjusted these in the sequence in which they were found. I then located and adjusted a right, fixated sacrum with some force in side posture with a loud audible and palpable release. My first two attempts at the sacral adjustment did not eliminate the surrogate's upper trapezius muscle indicator, nor the challenge to the patient's right sacrum. I tapped triple-warmer 23 to the right, posterior sacrum(beginning and end technique), and adjusted the right, posterior fibula.

The mother and I noticed an instant calming effect of these adjustments, and particularly of the sacral adjustment upon Jake. Following this treatment, the hand-head banging and Jake's anxious state of fear and tension ceased. Interest in the world around him has blossomed.

On 12/3/91, upon a repeat examination with the overseeing pediatric neurosurgeon, Jake's skull measured 51.25cm. This was down from 52cm. The diagnosis was changed to a mild hydrocepphalus in remission. Continued monitoring was advised by the parents and the local pediatrician.

I have seen this patient three times in 1992, and once in 1993. At 28 months, the skull measures 52cm, which is in the 95th percentile of head sizes.(3). Jake excels in his physical and mental realms. My treatment during these visits was noteworthy on one visit, when the Mother brought my attention to a lump over the patient's asterion. This appears to be subsiding after correcting occipital and frontal inspiration faults and a parietal descent.

Discussion:

In hydrocephalus, the increased intrecranial pressure from lack of cerebrospinal fluid circulation causes calvarium enlargement before the sutures are closed. In contrast, the facial bones are normal in size. Other diagnostic signs include: thinning of the scalp, distented scalp veins,

papilledema, eyes displaced downward("sunset eyes"), bulging fontanels, thinning of cranial bones on imaging, cerebrospinal fluid pressure measurements and protein measurements, trans-illumination(ala candling an egg), and Macewen's sign(tympany upon percussion over the lateral ventricles). Diagnostic symptoms include: headache, earache, vomiting, agitation, mood swings, weakness of limbs, incoordination, emaciation.(1)(3)(4)(5).

Head circumference is measured in cm by placing a tape over the external occipital protruberance to just above the supraorbital ridges. The average for the normal child at birth is 35cm. It is recognized that premature infants need to be measured according to a separate scale than term infants. At 10 months, the average head circumference for term-delivered boys is 46cms. Plus two standard deviations, which includes 98% of this population, is a skull measurement under 48.5cm.(see growth charts accompanying this paper). At birth, Jake's skull circumference was in the lower 25% of term deliveries. However, at eleven and twelve months Jake's circumference was 50 and 51cms, respectively. 95% of the full-term size at this age is below 49cms. At 13 months, mean head size is 47.5cm for full-term infants, compared to 52cm for jake. 52cm is the mean circumference skull of a seven year old boy.(1)(5). There were no neurological deficits that were detected beyond certain behaviors mentioned, such as the aggressiveness, forehead-slapping, agitation, and startled awakenings.

The reduction in skull circumference and immediate behavioral changes were remarkable and directly followed chiropractic manipulation assisted by applied kinisiological diagnosis and treatment.

Could the spinal tap, performed during Jake's visit to the emergency room on 8/5/91, have induced an inflammation of the meninges which commonly results in communicating hydrocephalus. Hydrocephalus commonly results from intraventricular hemorrhage in premature infants. In this case, blood breakdown products or infection interfere with cerebrospinal fluid absorption, which leads to a form of communicating hydrocephalus.(2). Perhaps this case of hydrocephalus was iatrogenically induced or affected by such an invasive procedure. Aware that bacterial meningitis is a very serious possibility in Jake's emergency room presentation, I question whether monitoring other vital signs over time would have been wiser care.

Lastly, I would like to mention the occiput-sacrum Lovett Reactor association in this case, and the primary cranioscral respiratory mechanism, which are well documented in chiropractic literature.(6).(7).

Conclusion:

The implication that any non-invasive, non-life threatening, inexpensive therapy could help the body correct such a condition, as the one described above, is worthwhile of further investigation. To understand the significance of this applied kinesiological treatment on the resolution of diagnosed progressive hydrocephalus, I would like to know the incidence of hydrocephlus in pre-maturely delivered infants, the incidence of spontaneously resolved cases of progressive hydrocephalus, and other cases of resolved progressive hydrocephus through manual therapy.

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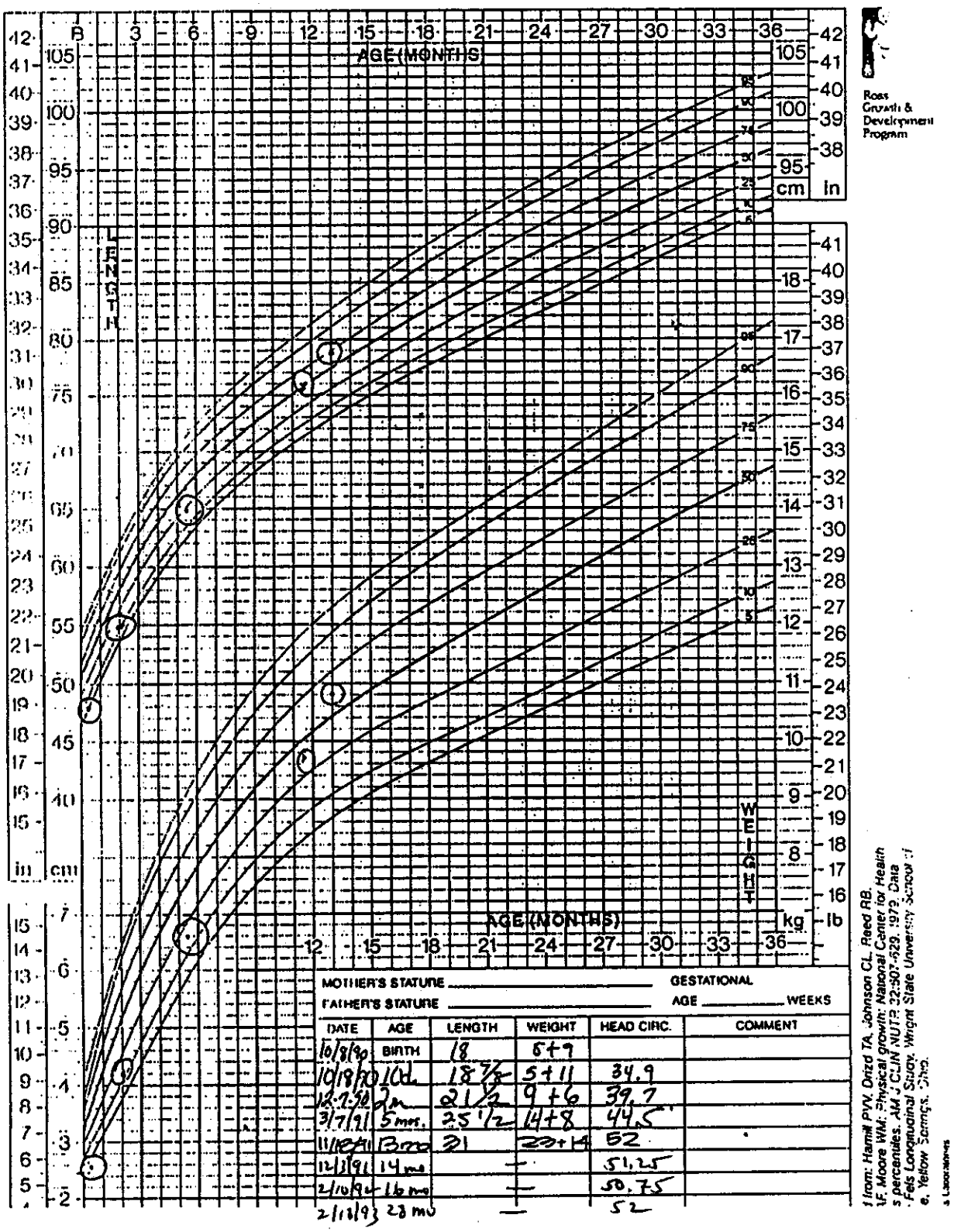
Infant Hydrocephalus---Dauphiné---page 6

clinic growth chart 1/2

PHYSICAL GROWTH
NCHS PERCENTILES*

NAME Jacob RECORD # 10-8-90



MOTHER'S STATURE _____ GESTATIONAL AGE _____ WEEKS

FATHER'S STATURE _____

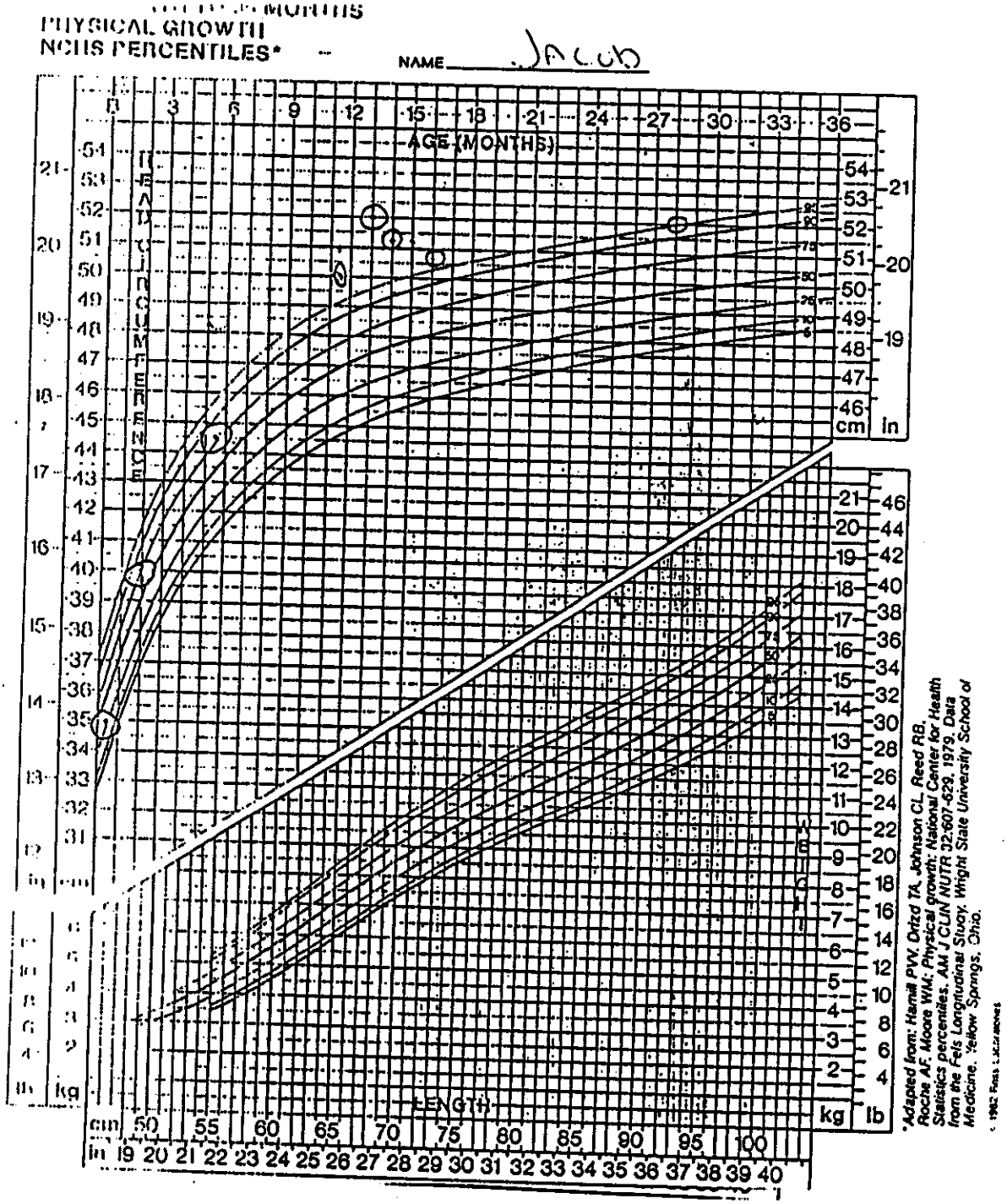
DATE	AGE	LENGTH	WEIGHT	HEAD CIRC.	COMMENT
10/8/90	BIRTH	18	5+7		
10/18/90	10d.	18 1/2	5+11	34.9	
12-7-90	2m	21 1/2	9+6	39.7	
3/7/91	5mos.	25 1/2	14+8	44.5	
11/18/91	13mo	31	23+14	52	
12/3/91	14 mo			51.25	
2/10/92	16 mo			50.75	
2/18/93	28 mo			52	

1 from: Hamill PVV, Drizd TA, Johnson CL, Reed RB, Roche AF, Moore WM: Physical growth: National Center for Health Statistics percentiles. Am J Clin Nutr 32:607-629, 1979. Data from Fels Longitudinal Study, Wright State University School of Medicine, Yellow Springs, Ohio.

* Laboratories

Infant Hydrocephalus---Dauphiné---page 7

clinic growth chart 2/2



MUSCLE STRETCH RESPONSE IN WEAK MUSCLES

David P. Engel, D.C.

Abstract: This paper presents a simple method to differentiate muscle stretch response from other factors which cause a muscle to test weak.

Introduction: The handling of a strong muscle which weakens upon stretching is well described by Walther[1,2]. He attributes weakness of a muscle which also has muscle stretch response to five factor involvement. No mention is made of the weakness being caused by the muscle stretch response itself. In my practice, I have found that muscle weakness can be caused by a positive muscle stretch response. The problem is that the standard test for muscle stretch response is invalid when the muscle is weak "in the clear" i.e., without any stimulus introduced in the test to cause it to test weak.

Methods: When posture, patient complaints or other factors cause you to suspect muscle stretch response involvement, but the suspected muscle tests weak in the clear, you can rule out muscle stretch response simply by performing a mild stretch to the fascia of the suspected muscle and retest. If the weakness is caused exclusively by muscle stretch response, the previously weak muscle will strengthen. Of course, the treatment is the standard treatment for the muscle stretch phenomenon.

Discussion: By simply performing a mild, "trial treatment" version of the myofascial stretching procedures we use to address the classic muscle stretch response involvement, I feel the body reacts temporarily to the procedure by strengthening the muscle. This confirms the involvement of the muscle stretch response and the need for complete treatment. As an additional note, I would like to encourage a deeper, more vigorous approach to this problem than I have generally seen among applied kinesiologists. I have found that using a deeply penetrating, deliberate pass along the involved fascia, without lubrication (the presence of which makes effective stretching of deep fibers more difficult) absolutely necessary to reduce recidivism.

Conclusions: In my experience the addition of this diagnostic procedure helps find hidden causes of certain muscle weaknesses which either do not respond to other procedures or return repeatedly after treatment.

Summary of Procedures:

1: Upon finding a weak muscle for which you suspect a muscle stretch response involvement, perform a mild stretch to the fascia.
2: Retest the muscle. If it strengthens, treat with complete stretching of the fascia.

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MAKING CRANIAL SETPOINT THERAPY WORK

Gary N. Klepper, D.C.

ABSTRACT: Cranial setpoint therapy can be very effective at enhancing metabolic corections if performed with an understanding of the mechanisms involved. These mechanisms are: 1- the cranial mechanism, 2- information transfer activated by the setpoint itself, 3- the divergent channel, 4- integration of the target organ with the primary respiratory mechanism, and 5- status of the metabolic nutritional reserves. The main reason that cranial setpoints often fail to give a good clinical response is due to stagnation in the meridians. This aspect will be addressed in this paper.

INTRODUCTION

Cranial setpoint therapy has been used within the body of applied kinesiology technique as a method of regulating metabolic processes. First described in 1980 by George Goodheart, the technique consisted of identifying a meridian beginning or endpoint on the head which then could be treated by tapping with the intention of removing vascular congestion from the pituitary and hypothalamus area[1]. It was believed that by doing so, the hypothalamus would be enhanced in its ability to "smell" body chemistry aberrations and to pass on appropriate instructions to the pituitary to correct the problem. Goodheart recommended the use of a biofeedback thermistor taped to the glabella in order to detect a drop in surface temperature caused by tapping of the setpoints, feeling that this would measure the achievement of an effect of cooling off of the hypothamamic-pitutary area. He also felt that the cranial setpoints were involved with proper energizing of the intracranial dura[2].

Wally Schmitt popularized this technique further and made it more accessible. His simplified method of diagnosing the need for the setpoint therapy involved establishing a therapy localization in the clear or after inducing a biochemical stress to an organ, and finding a setpoint that would neutralize this therapy localization. He theorized that the setpoint therapy would be useful in cases where there was a history of biochemical stress which has since been corrected, but which continues to be actively symptomatic[3]. An example would be a person who depleted their system of essential nutrients in their prodigal youth through overconsumption of refined sugar, has since improved their diet and restored depleted nutrients through supplementation, but still reacts badly when eating a small amount of refined sugar. In a case like this, the setpoint therapy would enable the body to reinventory its biochemical reserves and begin to behave appropriately.

Michael Liebowitz expanded the use of cranial setpoints greatly and introduced their treatment using laser. He found that setpoint therapy can be used to neutralize a majority of abnormal reflexes including Chapman's reflexes and pelvic categories. He also introduced the use of additional points on the head which he called the master setpoints, which he feels are more powerful than the normal setpoints[4]. Among his concepts on cranial setpoints is that their treatment helps restore the organizing effect on the body that would normally be provided by adequate exposure to natural magnetic fields such as the Earth's magnetic field.

Yet another layer of depth to the understanding of cranial setpoints was developed by Richard Holding in conjunction with myself. Richard Holding

developed a method of using a cranial setpoint diagnostically to relate a visceral area back to a deep cranial adaptive pattern[5]. By establishing a connection between a dysfunctional viscera, the cranial setpoint, and a cranial lesion, the doctor can then operate a cranial therapy which takes into account the pattern of adaptation involving the divergent channel connection to the viscera and its lymphatic drainage. Performing a correction in this manner feels very much like removing a magnetic lock that has been holding in a deep cranial lesion for some time.

DISCUSSION

A method of cranial setpoint correction involves clearing stagnation from the meridian system on multiple levels. Clearing of stagnation is necessary because the communication between the cranial setpoint and the involved viscera is not a direct one.

The major vital internal organs are classified in traditional Chinese medicine as yin. The associated channels are lung, heart, liver, spleen, and kidney. All of the classical cranial setpoints are on yang channels, these being bladder, stomach, large intestine, gall bladder, triple warmer, and small intestine. The connection between the yin and the yang is via the divergent channel. This channel has the function of protecting the organs from pathogenic influences entering the channels[6].

The divergent channel splits off from the primary channel at about the level of the knee or elbow at the point known as the he or uniting point[7]. It passes to the appropriate visceral organs, then again rejoins the associated yang primary channel on the head or neck. Each yin/yang meridian pair has a point of meeting near the beginning of the divergent pathway, another on head or neck, and all of them meet at GV20. Thses meeting points become the basis for treatment of the divergent channel, along with the classic cranial setpoint.

Unfortunately, just treating the setpoint and the divergent channel will usually not give an adequate response. It is necessary to remove stagnation from the more superficial levels of the meridian system as well as the deeper levels. Otherwise the result will be similar to what happens when a deep acting homoeopathic remedy is given without establishing proper drainage. It either doesn't work or it creates reactions. The levels of clearing necessary are: 1- surface clearing, 2- extraordinary vessel balancing, 3- hara therapy, and finally treatment of the setpoint and divergent channel will work.

SURFACE CLEARING

The most superficial level of meridian flow is that of the wei qi or protective qi in the musculotendinous channels. Stagnation at this level is relatively easy to improve. If fact, anyone who does bodywork is routinely clearing qi stagnation at this level. The most superficial level should be cleared first.

One way to do this is to perform routine corrections removing major areas of structural lesions, especially those of the pelvis, spine, and extremities. This tends to disperse major areas of qi stagnation at the wei qi level.

Another way is to identify the major channels displaying stagnation by palpation, and to release the surface via superficial needling techniques such as sesshoku shin. The sesshoku shin needling involves taking a light guage

needle and inserting in and out no deeper than 3 fen (.3 cun) into the painful areas of the musculotendinous channels. The following chart shows areas that are most useful to check[8].

Taiyang Complex (BL, SI)
- BL 10 occipital, splenius capitus area
- SI 9-12 latissimus dorsi, scapular area
- BL 31-34 medial gluteus maximus area
- BL 40 posterior knee
- BL 57-58 gastrocnemius area
- BL 60 lateral malleolus area

Shaoyang Complex (GB, SJ)
- GB 20-21 splenius cervicis and trapezius area
- SJ 15 superior scapular area
- SJ 5 extensor indicis area
- GB 28-30 mid gluteus maximus area
- GB 31 tensor fascia lata area
- GB 34 peroneus longus
- GB 41 lateral extensor digitorum longus

Yangming Complex (ST, LI)
- ST 12 superior clavicular area
- ST 30 lower iliopsoas area
- LI 4 first dorsal interosseous area
- LI 10 superior bracheoradialis area
- ST 36-40 anterior tibial area

Taiyin Complex (SP, LU)
- LU 10 adductor pollicis
- LU 4-6 extensor carpi radialis area
- LU 1-2 pectoralis minor area
- SP 3-4 abductor hallucis area
- SP 6-9 medial tibial area
- SP 21 mid serratus anterior

Shaoyin Complex (KI, HT)
- HT 1 subaxillary area
- HT 3 superior palmaris longus area
- KI 2-6 medial plantar to inferior malleolus
- KI 10 inferior popliteal transeverse crease
- KI 16 lateral umbilical area
- KI 27 subclavian area

Jueyin Complex (LV, PC)
- LV 3 flexor digitorum longus area
- LV 5 anterior tibial area
- LV 13 11th rib area
- PC 6 palmaris longus area
- PC 3 inferior biceps bracheii area

Master points of muscle meridians
- ST 3 all leg yang muscle meridians
- GB 13 all arm yang muscle meridians
- Ren 3 all leg yin muscle meridians
- GB 22 all arm yin muscle meridians

It is best to choose one or two of the above complexes and treat them rather than needling all of the painful areas.

EXTRAORDINARY MERIDIAN BALANCING

The 8 extraordinary channels represent an intermediate depth in that they run deeper than the surface channels but do not work on the deepest or zang-fu level. These vessels are one of the most fascinating topics of study in all of Chinese medical theory. While much could be shared about them, for the purpose of this paper I will just say that they are a major regulator of function in the meridian system.

Of the 8 extra meridians, only 2 have their own points (conception and governing vessel). The rest utilize points on the other primary meridians. For our purposes, the only points that need to be considered in evaluating and treating the 8 extraordinary meridians are their master points. These are generally treated unilaterally and in pairs. The master couples of the 8 extra meridians are as follows:

SP4	penetrating (chong mai)	PC6	yin linking (yin wei mai)
SI3	governing (du mai)	BL62	yang heel (yang qiao mai)
GB41	girdle (dai mai)	TW5	yang linking (yang wei mai)
LU7	conception (ren mai)	KI6	yin heel (yin qiao mai)

These points will often not therapy localize. The easiest way to identify those needing balance is by challenging all of the above points using a magnet. In general, those showing a kinetic response to the south pole of the magnet need tonification. Those showing the kinetic response to the north pole of the magnet need sedation. These channels are typically treated by tonifying one member of the pair and sedating the other[9]. One pair and up to all four pairs can be treated. My favored method of treatment is needling with ion pumping cords. Many other treatment options are possible.

HARA THERAPY

The deepest level of treatment in the meridian system is hara therapy. This system of therapy consists of identifying areas of stagnation or deficiency in the abdomen and correcting them. Areas of stagnation will feel hard and be painful to palpation. Areas of deficiency will feel soft and devitalized. Usually areas of stagnation are treated first, unless deficiency is the predominant presentation.

To treat an area of stagnation, find a predominant painful abdominal area, establish a therapy localization (often with back of hand) to it, and find a meridian point that abolishes the therapy localization. A general hint about where to find the meridian point is based on this model[10]:

middle abdomen	spleen channel
upper abdomen	heart channel
lower abdomen	kidney channel
right abdomen	lung channel
left abdomen	liver channel

The procedure is to identify the point on the meridian that abolishes the abdominal therapy localization. Then, while palpating the site of abdominal

pain, find the exact location and direction of the point which softens the abdominal area and diminished the pain. Insert a needle in this vector and retain for 10 minutes. Multiple treatment points may be found.

If an area of deficiency is to be treated, this is best done by direct moxabustion.

DIVERGENT CHANNEL TREATMENT

Perhaps the most complex part of the treatment involves that of the divergent channel itself. This involves a deep treatment of a yin-yang polarization problem. The treatment involves selecting the appropriate upper point and the appropriate lower point. The upper point will be the classical cranial setpoint, its alternate, or the upper meeting point of the divergent channel. The lower point will be the uniting point of the involved channel or the lower meeting point of the divergent channel. The upper point is treated by sedation and the lower point by tonification. Points can be selected from this chart[11,12]:

Cranial setpoint	BL1	ST1	LI20	GB1	TW23	SI19
Upper meeting	BL1	ST1	ST12	GB1	GB12	BL1
or	BL10	BL1	LI18		TW16	
Alternate	BL10	ST8		GB20		
Uniting (yin)	KI10	SP9	LU5	LV8	PC3	HT3
Uniting (yang)	BL40	ST36	LI11	GB34	TW10	SI8
Lower meeting	BL40	ST30	ST12	CV2	CV12?	GB22

PROCEDURAL SUMMARY

The steps in the procedure of clearing channel stagnation in order to make the cranial setpoint therapy work are as follows:

1- Release the surface using structural corrections or superficial needling to relieve stagnation in the musculotendinous channels.
2- Balance the 8 extraordinary channels.
3- Release stagnation and/or tonify the hara.
4- Treat the cranial setpoint and/or divergent channel points.

CONCLUSIONS

Cranial setpoint therapy is an effective treatment for metabolic regulation. However, it is the very factors that prevent the setpoint therapy from giving good clinical response that make the therapy necessary in the first place. In other words, if there were no stagnation in the channels, the setpoint mechanism would be self regulating and require no treatment. Therefore, it is best to correct the channel stagnation in order to be getting at the root level of treatment.

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Muscle Testing and Upper Extremity Peripheral Nerve Entrapments

David W. Leaf

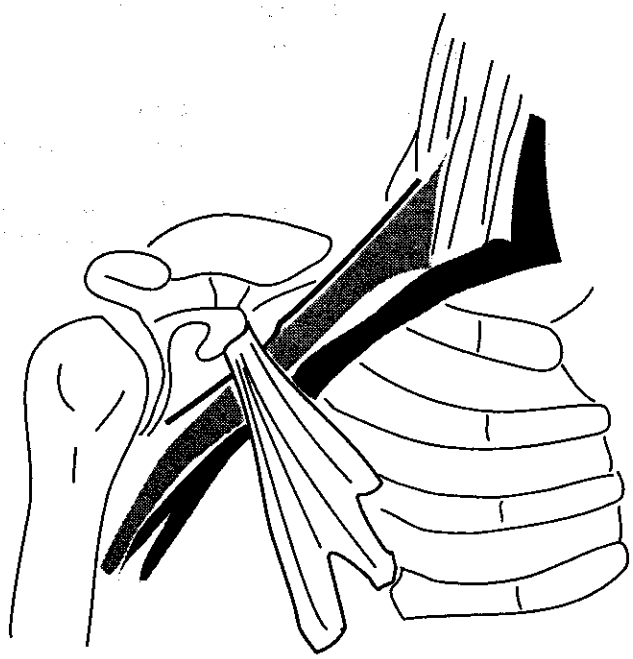
Abstract: Muscle testing can be used as a diagnostic key to determine the existence of peripheral nerve entrapments. This paper discusses the basic entrapment syndromes of the upper extremity and the related findings using muscle testing.

Introduction

In examining a patient, symptoms in the upper extremity are often confused and improperly related to a spinal causative factor. Especially following any fall or automobile accident the upper extremity must be examined for peripheral entrapment syndromes. The major entrapment syndromes are presented here with their symptomatic picture and muscle testing findings.

Discussion

Anterior Scalene Syndrome



Compression of the brachial plexus, the subclavian artery and the subclavian vein between the anterior and medial scalene muscles and the first rib can cause symptoms throughout the arm. The muscle fibers, if continually contracted or hypertrophied, first effect the lower sections of the brachial plexus. When this becomes chronic, the entire plexus is composed of nerve roots from C - 5 to T - 1 can be effected. Vascular symptoms occur due to the compression of the artery and/or vein.

Symptoms reported by the patient will usually begin with numbness in the hand and fingers radiating up into the forearm, pain from the shoulder to the hand and cold hands with symptoms similar to Raynaud's phenomenon. The entrapment of the brachial plexus causes sensory symptoms on the ulnar side of the hand. Travell reports that trigger points in the scalene muscles will cause referred pain on the radial aspect of the hand. On inspection, the small muscles of the hand may appear to have atrophied.

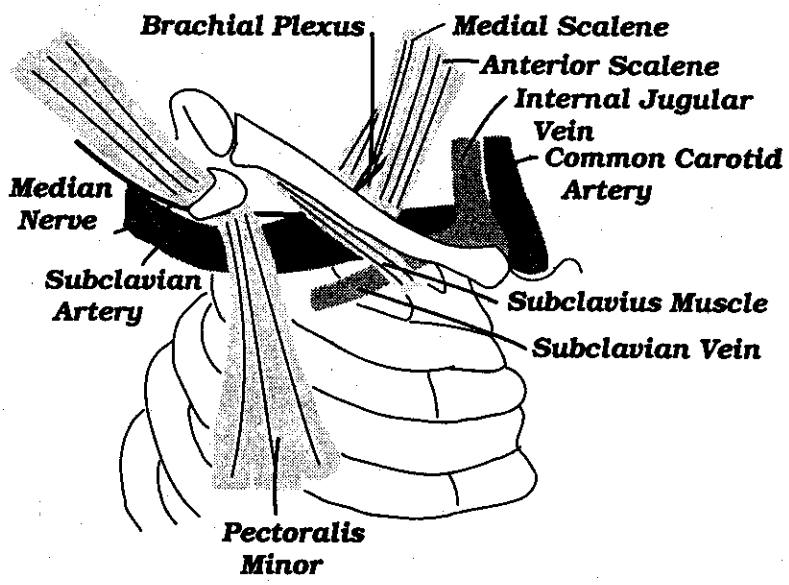
Muscle testing will usually reveal no overt signs of weakness unless the anterior scalenes are stressed. The stress is applied by varying the position of the patient, standing, leaning over, fully inspiring or having the patient elevate the head while lying supine. Examination of the anterior scalene will uncover an intact muscle that has trigger points. These are usually the result of another weak muscle. Frequently the latissimus dorsi is found involved.

If weakness is found in the arm with the patient sitting or standing, raising the arm above the shoulder with anterior rotation of the shoulder elevates the clavicle reducing any neurovascular entrapment. This same position can be used in the following three syndromes to decompress the neurovascular bundles.

Costoclavicular Syndrome

This syndrome refers to entrapments of the brachial plexus, the subclavian artery and/or the subclavian vein as they traverse beneath the clavicle and over the first rib.

Symptoms of entrapment are usually transient and brought on by motions of the clavicle or the first rib. The symptomatic pattern is the same as in the anterior scalene syndrome.



Muscle testing will reveal no overt weakness patterns until the clavicle or the first rib is stressed. Hand muscles can be tested for weakening. There are two different positions that can elicit a weakness pattern. First, the arm is flexed to 140 degrees and arm or hand muscles are tested. This motion rotates the clavicle involving the subclavius muscle. The shoulder can also be rotated posteriorly with the arm extended to 30 degrees. This shoulder position is similar to the position used in the military. Finally, the patient is asked to fully inspire. This activates the scalene muscles elevating the first rib. If the clavicle has been displaced inferiorly or the first rib is superior a weakness pattern will be created in the arm.

Pectoralis minor syndrome

In this syndrome, neurovascular entrapment of the brachial plexus, the axillary artery and the subclavian vein can occur between the fibers and tendon of the pectoralis minor muscle, the head of the humerus and the coracoid process of the scapula.

Symptoms of entrapment are usually transient. The symptomatic pattern is the same as in the anterior scalene syndrome. However, these patients have more vascular symptoms as well as signs of lymphatic blockage. This differs from the scalene involvement where venous blockage is paramount. Commonly, this is found in people who work over their heads or who have excessively developed the pectoral muscles. Symptoms are aggravated by sleeping with the

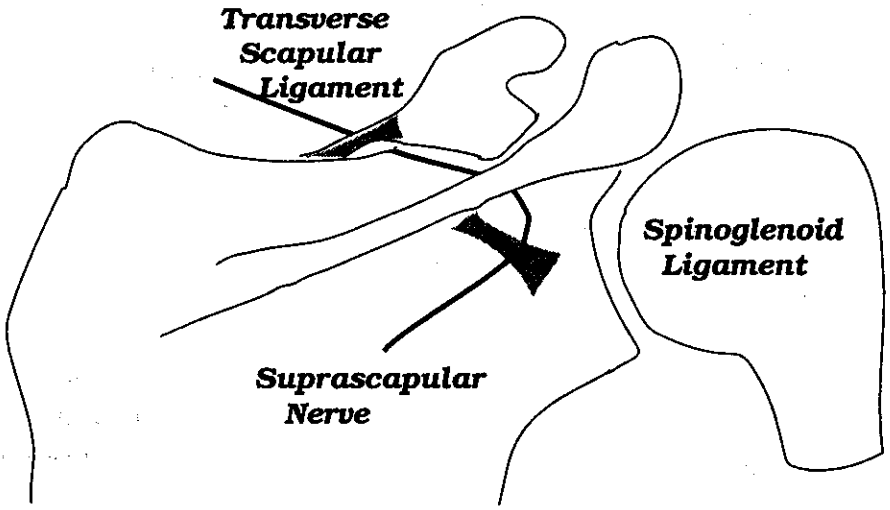
arm elevated or by carrying heavy objects.

The classical test for entrapment of the axillary artery is the Wright maneuver where the arm is placed in external rotation and the shoulder is abducted. This same position will elicit weakness if the costoclavicular syndrome is present.

Muscle testing will reveal no overt weakness patterns in some positions. Creation of the weakness depends upon the contraction or relaxation of the pectoralis minor muscle. If the latissimus dorsi is weak allowing elevation and anterior rotation of the shoulder, the pectoralis minor will be shortened with trigger points in the belly of the muscle. In this case, testing in the standing or sitting posture will reveal weakness of the hand muscles that will immediately strengthen if the arm is elevated above the horizontal with slight flexion of the arm. Care should be taken not to maximally elevate the arm as that will cause the weakness pattern to return.

Suprascapular nerve syndrome

This is a very commonly overlooked syndrome that can lead to atrophy of the infraspinatus and the supraspinatus muscles. The suprascapular nerve is composed of fibers arising from either the C-5 or C-6 nerve roots. It traverses through the suprascapular fossa and the scapular notch to arrive at the supraspinatus fossa. After supplying fibers to the supraspinatus muscle, the acromio-clavicular joint and the subacromial bursa, it twists around the base of the spine of the scapula and enters the infraspinatus fossa. Due to this tortuous path, the nerve is stretched as the scapula moves if there is any scapula instability.

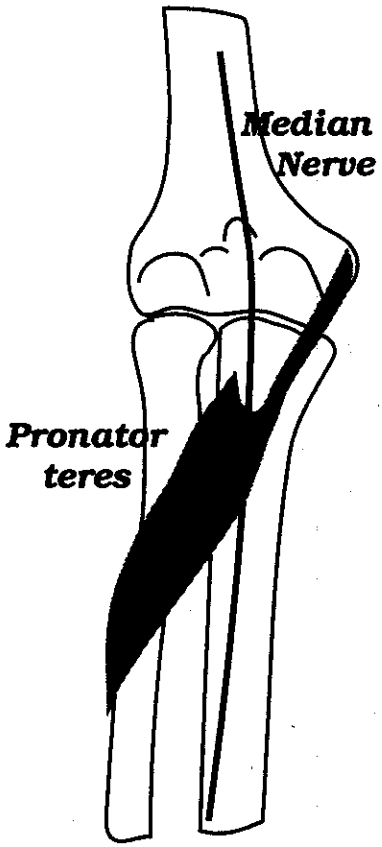


Symptoms, reported by patients, will run the gamut from diffuse shoulder pain that is hard to localize to complete atrophy of the infraspinatus first and then the supraspinatus muscles. Symptoms are usually aggravated by any activity that requires extensive motion of the scapula.

In most cases, there is no overt weakness pattern. However, if the scapula stabilizers are weak, especially the serratus anterior or rhomboids, the infraspinatus will test weak if tested with the arm flexed to 90 degrees with anterior rotation of the shoulder. This position creates additional torsion on the suprascapular nerve and if the scapula has inadequate support, the infraspinatus will weaken.

Pronator teres syndrome

This entrapment syndrome is of the median nerve as it passes between the ulnar and radial heads of the pronator teres muscle. After the nerve passes the pronator teres, it divides and supplies the flexor muscles of the wrist and hand except for the flexor carpi ulnaris and the ulnar portion of the flexor digitorum profundus.



Symptoms reported by the patient will include loss of strength throughout the hand, difficulty writing, paresthesia throughout the hand and especially the palm.

Muscle testing will reveal weakness of the finger flexors that resolves when the radius and the ulnar are approximated just distal to the elbow.

Supinator syndrome

In this entrapment syndrome the radial nerve becomes compromised as it passes beneath the supinator muscle.

Symptoms occur during repeated motions of the forearm. The throwing motion uses all of these. These can include pronation, wrist flexion and forearm extension. The pain pattern is described as deep on the posterior aspect of the forearm. Hand weakness is reported.

The easiest muscle to test for this syndrome is the extensor carpi ulnaris. If it is found weak, the head of the radius is approximated to the ulna and the muscle is retested. The extensor carpi ulnaris can also be tested when the supinator is placed in a strain counterstrain position. This position fully relaxes the muscle.

Ulnar sulcus syndrome

The ulnar nerve passes down the posterior surface of the humerus and passes through a sulcus on the medial epicondyle of the humerus. The epicondylo-olecranon ligament stabilizes the ulna and the humerus. It also stabilizes the ulnar nerve at the sulcus and prevents it from moving during forearm motions.

When the ligament is hypertrophied or stretched, entrapment of the ulnar nerve occurs. The nerve supplies the flexor carpi ulnaris, the ulnar portion of the flexor digitorum profundus, the interossei and hypothenar muscles, the adductor pollicis and the deep head of the flexor pollicis brevis.

Symptoms include paresthesia and pain over the ulnar nerve distribution and weakness of the above muscles.

Testing of the flexor carpi ulnaris with the elbow first in extension and then in flexion may uncover entrapment of the ulnar nerve. If found, the relationship between the humerus and the ulna needs further inspection. Any chronic subluxation, dislocation or avulsion can create ulnar nerve entrapment. After correcting any imbalances, direct attention should be applied to the integrity of the ligament.

Flexor Carpi Ulnaris Syndrome

As the ulnar nerve leaves the sulcus, it descends between the two heads of the flexor carpi ulnaris muscle. The muscle has a tendinous arch which is formed by the arcuate ligament. This runs from the medial epicondyle to the olecranon.

The symptomatic pattern is exactly the same as the syndrome of the ulnar sulcus. This makes differential diagnosis difficult without muscle testing.

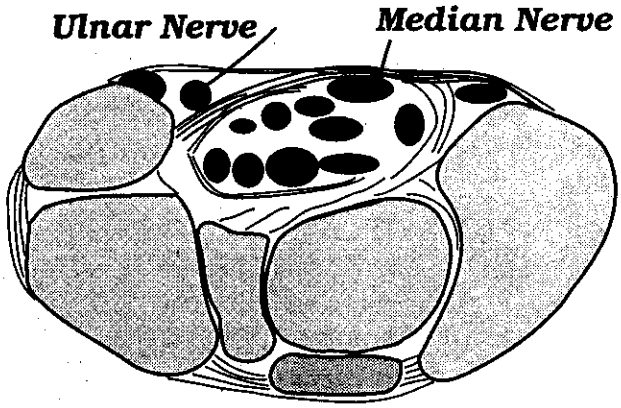
Accurate testing of the adductor pollicis with the forearm first in a neutral state, then with full contraction of the flexor carpi ulnaris and then with a relaxed approximated (strain counterstrain) position of the flexor carpi ulnaris allows insight into the status of the ulnar nerve as it passes between the heads of the flexor carpi ulnaris muscle.

Carpal Tunnel Syndrome

This syndrome consists of compression of the median nerve at the fibro-osseous canal at the wrist. The tunnel is formed by four major bony prominences, the pisiform, the navicular, the hamate and the trapezium. Between these bony prominences runs the transverse carpal ligament. After this tunnel, the median nerve gives sensory branches to supply the palmar surfaces of the first and second fingers and motor branches to the opponens pollicis, abductor pollicis brevis and the superficial head of the flexor pollicis brevis. Compression of the contents of the tunnel can occur due to edema, local subluxation, fracture, etc..

The patient reports symptoms ranging from paresthesia, thenar atrophy and weakness. These symptoms worsen as the condition becomes chronic.

As noted above, the median nerve innervates the opponens pollicis muscle. Weakness of this muscle becomes the diagnostic key to isolating the problem. The muscle should be tested with the forearm and wrist in a neutral position, full pronation, full supination, wrist extension and wrist flexion.



Carpal Tunnel

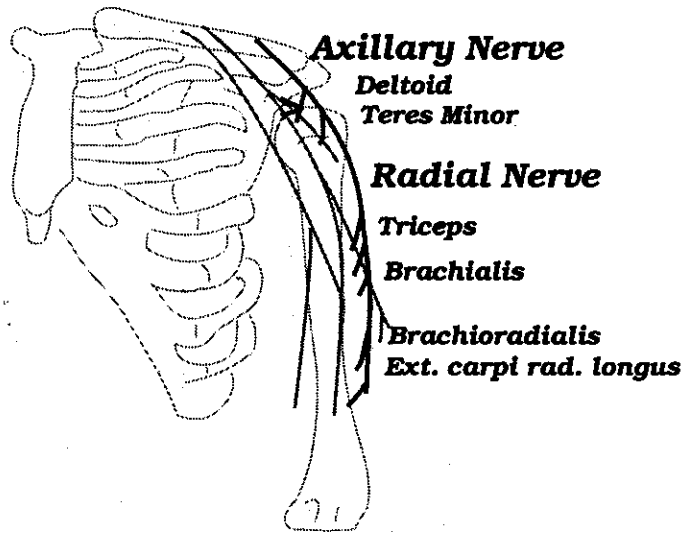
Ulnar tunnel syndromes

The ulnar nerve can become compressed at the level of the proximal carpal bones. This tunnel is bordered by the pisiform and the hamate as well as the transverse carpal ligament and the flexor carpi ulnaris muscle. There are two entrapment syndromes in this area. These correspond to the superficial and deep branches of the ulnar nerves which run through the ulnar tunnel. These nerves supply the sensory distribution to the palmar aspect of the fifth finger and the ulnar side of the fourth finger. The deep branch of the ulnar nerve supplies the interossei muscles the small muscles of the fourth finger and the adductor pollicis muscle.

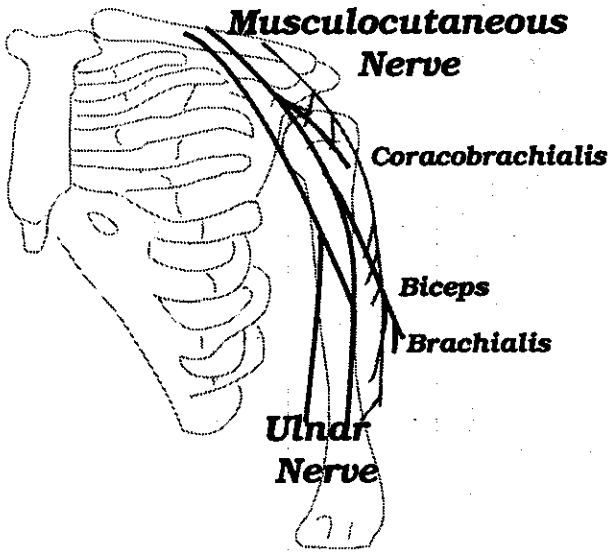
Weakness of either the flexor digiti minimi or the opponens digiti minimi muscles is the key that indicates entrapment of the ulnar nerve at the wrist. Once the weakness is found, directional pressure is applied against the pisiform and the hamate until a vector is found that strengthens the weak muscle.

Neurology Review

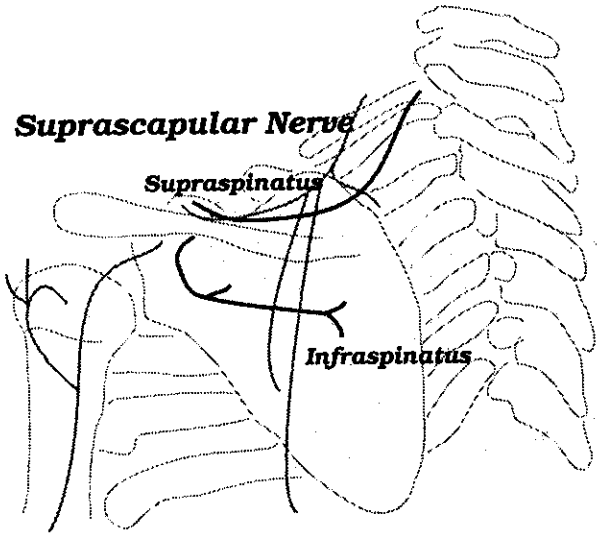
A review of the muscular innervation of the upper extremity will aid in reviewing the muscle testing sequence for determining entrapment syndromes.



The diagram to the right demonstrates muscles that are innervated by the axillary and radial nerves in the upper arm. Radial palsy has been reported after repeditive forceful contractions of the upper arm muscles. Weakness of the deltoid, especially the medial and posterior sections, is commonly found in acromioclavicular strains. Severe thoracic outlet syndromes can entrap the superior sections of the brachial plexus creating weaknesses in the deltoids and the teres minor.



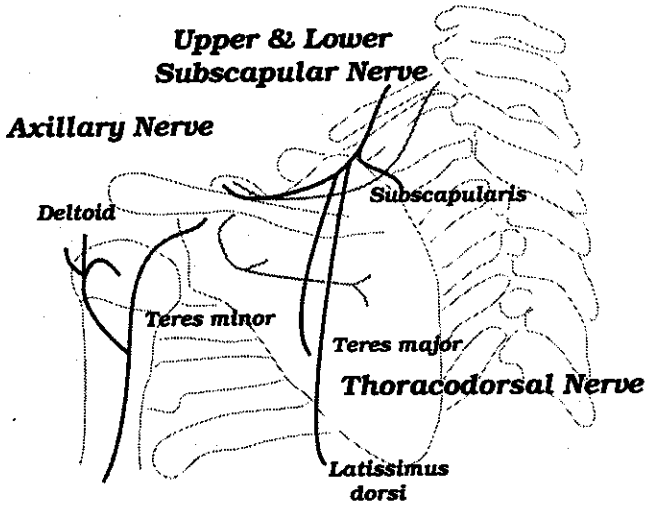
Depicted here are the three muscles innervated by the musculocutaneous nerve in the upper arm. Weakness of all three of these muscles, the coracobrachialis, biceps and the brachialis may indicate a



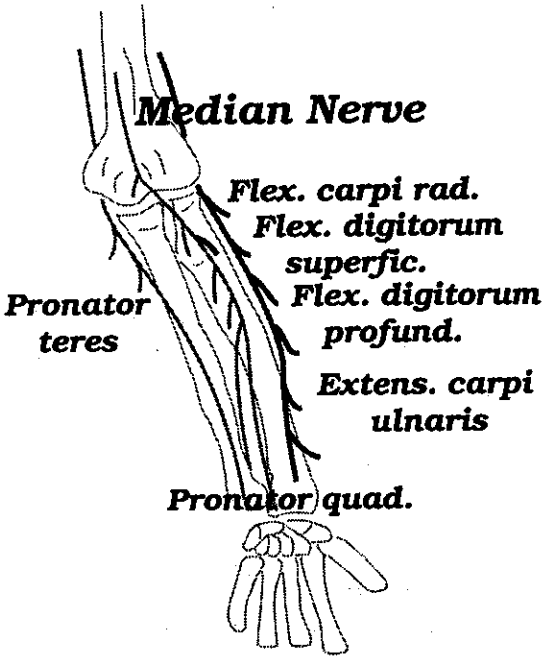
thoracic outlet syndrome.

Notice that the ulnar nerve does not supply any muscles until it is well down the humerus.

On the posterior aspect of the shoulder, the major missed entrapment syndromes involve inadequate stabilization of the scapula. As you can see, the suprascapular nerve supplied the supraspinatus before it bends and winds itself around the spine of the scapula. After this tortuous path, the nerve supplies the infraspinatus muscle.



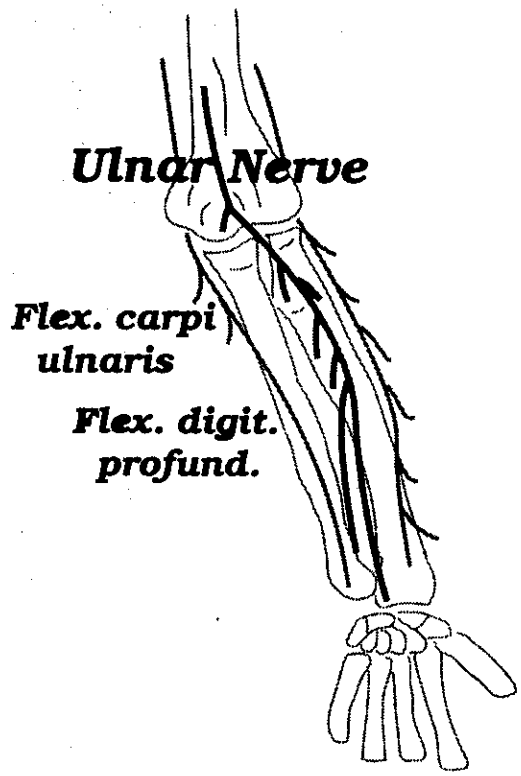
The axillary nerve is shown again as it supplied the deltoid and the teres minor muscles. Notice that before the nerve bifurcates to supply these muscles, branches are given off to the subscapularis, latissimus dorsi and the teres major muscles. Again, these are primary muscles to be used in ascertaining the existence of a peripheral nerve entrapment in the are of the shoulder girdle.

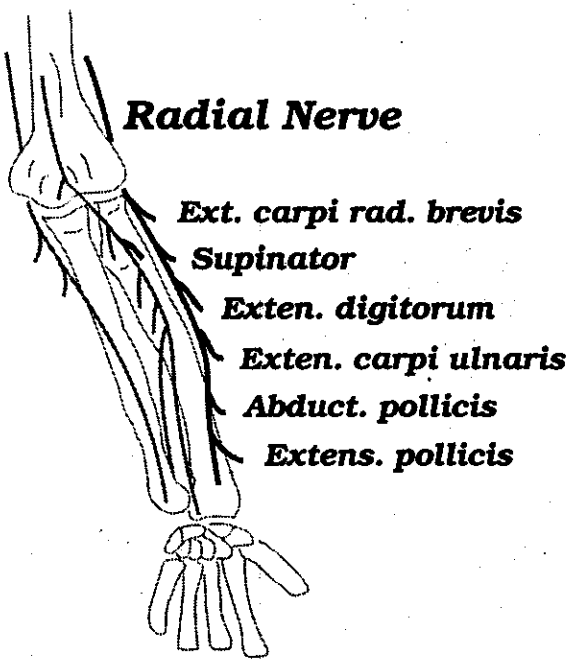


As you progress farther down the arm, the pronator teres is the last muscle supplied superior to the elbow by the median nerve. Knowing the status of the pronator teres aids in diagnosing problems arising from entrapment syndromes inferior to the el-

bow. For example, if the flexor carpi radialis is weak and the pronator teres is strong, then the median nerve is involved at the elbow.

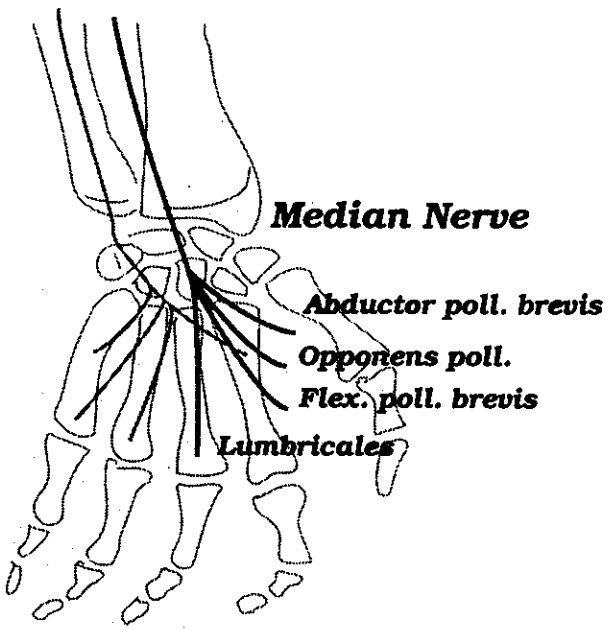
When the ulnar nerve is entrapped at the elbow, the flexor carpi ulnaris is the first muscle supplied inferior to the elbow. The integrity of this muscle is important if the flexor digiti minimi or opponens digiti minimi is found weak. Weakness of muscles above and below the carpal bones indicates an entrapment syndrome at the elbow and not just at the wrist.



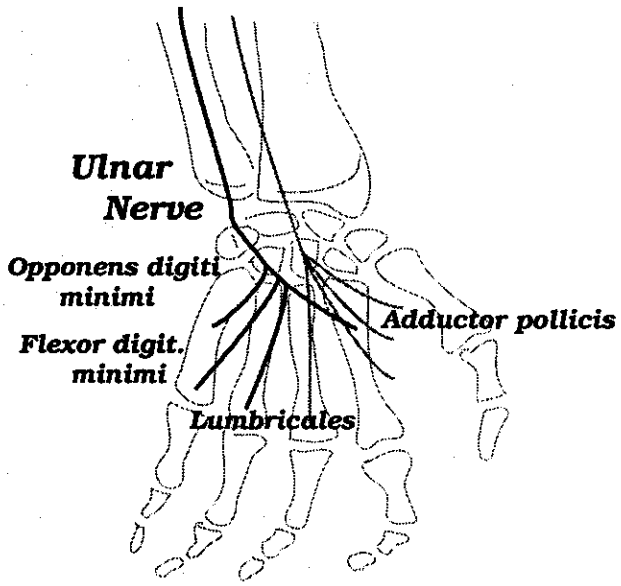


When examining the radial nerve, important muscles to test include the supinator, the extensor digitorum muscles and the abductor and/or extensor pollicis muscles. Entrapment syndromes as the radial nerve passes down the forearm will create weakness patterns relative to the level of the first muscle found weak. Generally, all of these muscles will be weak if the radial nerve is entrapped.

In the hand, weakness patterns of the opponens pollicis, with a finding of strength in the muscles innervated superior to the wrist by the median nerve, indicates the probable presence of a



carpal tunnel syndrome. A common testing procedure will find the flexor digitorum superficialis and profundus strong and the opponens pollicis weak. If all three muscles are found weak, the entrapment syndrome is at the elbow or mid-forearm.



Testing of the ulnar nerve at the wrist consists of testing for the relative strength of the opponens digiti minimi or flexor digiti minimi in comparison with the strength of the flexor carpi ulnaris. If the flexor carpi ulnaris is weak along with the muscles of the fourth finger, then the ulnar nerve is entrapped at the level of the elbow. Weakness of the finger muscles in the presence of strength of the flexor carpi ulnaris indicates a probable entrapment at the level of the carpal bones. Note the innervation of the adductor pollicis. Improper testing of the opponens pollicis will result in recruitment of the adductor pollicis and erroneous findings.

Conclusions

Muscle testing is an art as well as a science. One of the best uses of muscle testing is in aiding your examination procedures. Reproducible results depend on consistent testing. This testing must not only be reproducible from one examination to another, but must also be done accurately. A common error is improper testing of the opponens pollicis muscle. If this test is properly performed, you are testing median nerve function. If the test is improperly performed, you could be testing the ulnar nerve or a combination test that would challenge the integrity of the median as well as the ulnar nerve.

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AN ALTERNATIVE TO STIMULATING OLFACTORY RECEPTORS IN CHEMICAL EVALUATION

MICHAEL LEBOWITZ D.C.

ABSTRACT: Stimulating the olfactory receptors in chemical evaluation can prove problematic when testing the sensitive patient. Bio-magnetic testing over GV-19 yields similar muscle testing results while avoiding adverse reactions.

INTRODUCTION: On her first visit to our clinic, on chemically testing a patient on breathing in a chemical passed out and remained so for hours. A lawyer who was a patient as well as myself inhaled geranium oil to test it as an appropriate therapy. Both of us felt "stoned" for about four hours (it didn't work a second time). I referred by phone an environmentally ill patient to an ICAK member in southern California who performed a clorox sniff test on her. She had a severe sensitivity reaction and took two weeks to recover. Isolated cases like these plus the accumulative effects on the practitioner utilizing the sniffing of clorox, aldehydes, etc. over a busy day or week has led me to the obvious conclusion that stimulating the olfactory receptors to do chemical evaluation is not an ideal method.

DISCUSSION: Three or four years back we substituted bio-magnetic testing (1) for oral testing with excellent results that have been confirmed by other practitioners (2,3). I did find there were a small per centage of cases where olfactory testing on a chemical, air sample, mold, essential oil, etc. was positive while bio-magnetic testing was negative. This perplexed me and I decided to investigate. My investigation led me to the finding that placing a substance under the south pole of a magnet over GV-19 yielded the same muscle testing results as olfactory stimualtion. We have done it hundreds of times and find it a quicker, easier, and safer screening method for the practitioner and patient. GV-19 also appears to be some type of olfactory reflex that is therapeutic when treated manually or with a laser.

CONCLUSION: Bio-magnetic testing over GV-19 appears to be an effective, safer alternative to stimulating olfactory receptors in chemical evaluation.

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A TECHNIQUE TO EVALUATE AND TREAT DENTAL FOCI AND INFECTIONS (INCLUDING SUBCLINICAL)

MICHAEL LEBOWITZ D.C.

ABSTRACT: Dental infections can perpetuate many problems in the chronic patient. A simple screening technique along with treatment of positive areas can be quite useful in helping these patients.

INTRODUCTION: The role of dental foci, acute and subclinical infections of the teeth and underlying structures as a cause of chronic disease has been debated throughout the twentieth century and is beyond the scope of this paper. Let it suffice to say that clinically we have observed that subclinical dental infections of either bacterial or fungal origin can contribute to chronic sinus infections, chronic intestinal dysbiosis, fatigue, etc.

DISCUSSION:I must state at the onset that although symptomatic change has been significant in a number of cases, I have not worked in conjunction with a dentist to either confirm or assist in these findings.
On each patient with chronic symptoms I find it useful to look at their teeth. I observe the number of fillings, caps, crowns, bridges, etc.; the materials they are composed of, corrosion of metals, etc. A few patients of ours have had chronic dysbiosis or sinus infection that did not respond as quickly as usual to my usual dysbiosis treatment(1).
I began to investigate and found the following technique useful. I would take the south pole of a stick or diagnostic magnet and place it one by one at the root of each tooth and check for an indicator muscle to weaken. Wherever it did we would check to see which of the following antibacterial and/or antifungal supplements would negate the weakness: Berbercap (2), Enterocap(2), Goldenseal, SF722(2), Undecyn(2). In all cases to date at least one of the supplements would negate the weakness. After screening all teeth we would treat the neurolymphatics for the positive teeth. We found that if you were to lay the jaw out directly inferior to the clacicle, that the particular tooths neurolymphatic would be as lateral as the tooth is in the mouth and directly inferior to the clavicle. Stimulation of the neurolymphatic is often quite painful. We follow it with laser stimulation to the tooth root, and master set point treatment(3). We then supplement appropriately. On return visits the teeth usually test negative. If not retreat and if the finding is not resolved in a reasonable time period, referral to a dentist should be considered.
I feel this can be an important procedure at times though fixing intestinal and sinus dysbiosis can in many cases correct the teeth findings without treating them specifically (though not always). Many dentists feel that their techniques to uncover infections leave something to be desired and this technique might complement their procedures.

CONCLUSION: Treating subclinical dental infections or foci can be another piece of the puzzle in helping the chronic patient. Overlooking the state of the oral cavity can lead to less than optimal results in these patients.

SUMMARY OF PROCEDURES

1. Take the south pole of a stick or diagnostic magnet (4) and place it one at a time at each tooth root and see if a strong indicator muscle weakens.
2. Wherever muscle weakness occurs see which of the following placed between the south pole and tooth root negates the weakness: Berbercap(2), Enterocap(2), Golden Seal, SF722(2), Undecyn(2).
3. Rub the appropriate tooth neurolymphatic (inferior to the clavicle as lateral as the tooth is) and laser(4) the tooth root for three minutes. Supplement with appropriate products from step 2.
4. If the tooth root keeps testing positive for many weeks, consider referral to a dentist for evaluation.

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SPECTRA VIEW TECHNIQUE

MICHAEL LEBOWITZ D.C.

ABSTRACT: The different frequencies of the visible light spectrum are like nutrients. Selective malabsorption of certain frequencies can lead to organ dysfunction, chronic illness, repression of traumatic memories, etc. A procedure to discern which frequencies need to be supplied as well as how to supply them along with a case study is presented. Supplying these frequencies can lead to profound changes both physically and emotionally.

INTRODUCTION: Color therapy, full spectrum lighting, etc. have been studied for a number of decades at least. Pioneers such as Spitler, Ott, Babbitt, Dinshah, etc. have contributed vast amounts of information on the effect of visible light on human beings. The work we developed that is discussed in this paper in an outgrowth of the work of Jacob Liberman O.D.,PhD. whose work was as outgrowth of the work of the above mentioned pioneers. Liberman (1) feels there are three factors that block biological receptivity or absorption of selective frequencies of visible light. The first is excessive time spent under artificial light. Artificial light being imbalanced compared to sunlight causes certain body sensors to lose part of their function according to Liberman. The second is excessive use of sunglasses or tinted glasses. The third, physical or emotional trauma, may cause certain sensors to close down such that even if people are exposed to certain frequencies of light, they may not "receive" them.
Liberman hypothesized that stimulating the body (through the eyes) with the portion of the spectrum that is blocked will cause the "unstimulated sensor to awaken". In his clinical experience he found that having people look at certain colors would evoke different emotional responses varying from depression to elation. He decided to treat (having the patient look at certain colors through an apparatus) with the colors that made people uncomfortable or exacerbated their symptoms. By doing this peoples addictive behavior would change- often intensifying at first but then resolving. I for instance went on one of the biggest food binges of my life after my first treatment. He also found that old unresolved emotional issues and memories would resurface and with further treatment resolve along with their physical symptoms. Being intrigued by his work an inventor I knew developed a machine called the Spectra View (2) that would expose people to 106 different colors and we started experimenting.

DISCUSSION: We felt that we should treat with colors that caused universal muscle weakness, that that would be similar to treating with colors that caused discomfort. We also supplemented the patient during this time with either L-Tyrosine (3), Phosphatidyl Choline(3), and/or Pineal Plus(3) according to muscle testing results. We found that at least three sessions per week were necessary for optimal results. We also found that patients that felt they were "O.K." and beyond having emotional problems did not respond to treatment. Patients also had to be willing to stay with treatment until finished (20-70 sessions) or else they could develop nightmares, color allergies, etc. We found quite a number of cases recalling early childhood sexual abuse they were totally unaware of before but were able

to verify in a number of cases when pressing relatives for details.
One of the most interesting findings we had was that if we tested patients for dysbiosis, food and chemical sensitivities, etc. prior to treatment and after all the sessions were completed (assuming no other therapies were performed) the findings remained virtually unchanged but the symptomotology greatly lessened or ceased. We had always wondered why two patients could have very similar finds yet the severity of symptoms would differ greatly. Perhaps the frequency imbalance or emotional undercurrent was the answer.
To give an idea of the consequences of treatment I will give a case history in which I found the results particularly rewarding. Alice is a personal friend, about 45 years old. She suffered from continual shoulder pain, chest pain, irregular heart beat, debilitating left rhomboid pain aggrevated by stress. She had lost vision in one eye seven years ago as the result of a car accident. The top eye hospital on the east coast felt the loss was permanent. She suffered from depression and unknown to me had unsuccessfully tried to commit suicide just weeks before starting treatment. Previous treatment by me yielded very temporary relief only and we discontinued all other therapy during Spectra View so we could evaluate its effectiveness better. Alice also had a doormat personality- wanting people to step on her. Her self worth was nonexistant. After session one the left rhomboid pain as as well as the shoulder and chest pain left, never to return except for a brief period during another session that was especially traumatic. After session four her energy which had been very low since her accident returned to normal. The psychological abuse her husband gave her no longer affected her- even her children saw a remarkable change. Session six caused extreme pain behind her blind eye and session seven brought vision back to the eye. In session ten she saw her mother being beaten by her father when she was an infant and her father trying to strangle her (she was later able to confirm many of the details of this memory). Subsequent sessions brought more memories, headaches, nightmares at times. By the time we were finished (31 sessions) Alice was asymptomatic, no longer a doormat, and had a zest for life, eager to see what the future would bring. Over a year has passed and she remains very well.
We have also seen others turn their lives around. Another interesting observation is that after treatment people crave sunlight and healthy food while before treatment it was usually the opposite. One D.C. after a session gave up a 25 year habit of chewing tobacco which he could never previously quit despite his efforts to do so.
CONCLUSION: Treating receptive people with properly selected frequencies of the visible light spectrum can cause profound physical and emotional changes.

SUMMARY OF PROCEDURES
1.Take a strong indicator muscle. Going in order, insert one slide at a time into the instrument and see which frequencies or colors cause universal muscle weakness. Keep testing until two slides are found.
2. See which of the following negates the weakness: a)Pineal Plus(3), b)L-Tyrosine(3), c)Phosphatidyl Choline(3) and supplement with these (usually two of the three are needed). Recheck these weekly and change if needed.

3. Treat with each of the two slides, ten minutes each in a quiet, darkened room. After the first ten minutes unless the patient is in the middle of a significant memory or emotion or feels very uncomfortable, switch to the next slide for ten minutes. If they are in the middle, let them continue until they are done with the memory, etc. After ten minutes of the second slide (again if not in the middle) turn the machine off and record the patients comments on the session. Some sessions are non-eventful, maybe 20% are very significant though the others prepare the way for these. Some patients experience emotions or memories between sessions (either awake or asleep).
4. Three to five sessions weekly are optimal. On each subsequent visit recheck the slides used the last visit. Treat again with those as long as they cause muscle weakness. Often a particular slide or frequency takes at least a few treatments to complete its work. During the process the patient may experience headaches, bad dreams, irritability, etc. Usually they will peak with a significant memory or release and then the frequency will no longer evoke symptoms. It is important never to have a long break between sessions if you are in the middle of treating with a certain frequency or the patient may experience recurrent nightmares or even "color allergies".
5. Once all the slides have been screened and all appropriate frequencies treated, start the sequence over again, but have the patient therapy localize the emotional neurovasculars with one hand during the testing. This will make new frequencies show up. After all are tested and appropriated treated, you are finished.
6. It can take 20-70 visits to take someone to completion. This can often be a rollercoaster like time for the patient as they remember, experience, and purge unresolved issues. We do not recommend doing this type treatment unless the patient has a support patient and unless you or a therapist you work with is willing to be there for the patient as needed.

RESOURCES

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2. Spectra View available from Sun Designs P.O.Box 18944, Asheville. N.C. 28814, 704-253-5336
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Jugular Compression:
A Diagnostic Technique for Craniosacral Dysfunction

by Joseph Shafer

Abstract

Manual compression of the jugular veins (Queckenstedt's phenomenon) is regularly used during routine lumbar puncture procedures. In healthy persons, a rise in CSF pressure is expected at the site of puncture during venous compression. The author has observed consistent clinical significance in the use of the maneuver for craniosacral and dural membrane evaluation. In patients with a normal metabolic rate and exhibiting normal craniosacral, stomatognathic and dural membrane mechanics, compression of the jugular veins causes no change in pre-compression muscular strength. When dysfunction is present, immediate and significant changes in pre-compression muscle strength is observed.[1] It is hypothesized that the use of the technique during routine applied kinesiology evaluation is an invaluable adjunct to therapy and readily uncovers missed or hidden problems within the the primary respiratory system.

Key Words

Queckenstedt's maneuver, jugular compression, cerebrospinal fluid (CSF), craniosacral mechanics, primary respiratory system

Introduction

Hans Heinrich Georg Queckenstedt, a German physician (1876-1918), made the astute discovery that compression of the jugular veins made immediate increases in cerebrospinal fluid (CSF) pressure.[2] Bilateral compression of the veins produces the greatest increase in CSF pressure while unilateral compression produces a moderate rise in pressure.[3] Today, the maneuver/test is used during lumbar puncture procedures to evaluate for the presence of blockage of CSF flow between the jugular veins and site of the puncture. [3]

The internal and external jugular veins are responsible for 90 to 95% of the venous drainage from the brain. [4,5] In the field of craniopathy, it is believed that disturbances in the jugular foramina often produce circulatory back pressure and congestion into the brain which precipitates a vast array of symptoms.[4] "Through the jugular foramina passes the jugular venous drainage of blood from the cranial vault. ...deformation or dysfunction of these foramina often results in symptoms relating to intracranial fluid congestion." [6] Mechanical block of cranial venous drainage, due to osseous lesions and dural membrane tension, is an insidious perpetrator of abnormal intracranial pressure.

Manually induced jugular compression temporarily restricts venous drainage and results in a significant rise in CSF pressure. Increase in pressure is maintained until compression is released. This appears to be consistent with the mechanisms of production and absorption of CSF within the brain. Major production of the CSF is believed to occur in the choroid plexuses; ninety-five percent of this in the lateral ventricles. Greatest absorption is believed to be by the arachnoid villi which project and drain into the dural venous sinuses.[3]

The ability of jugular compression to maintain an increase in CSF pressure indicates that the regulatory mechanism for CSF is independent from changes in pressure gradients. Apparently, production of fluid by the choroid plexus follows a cellular metabolic rhythm governed by intracellular metabolism. This production continues uninhibited, despite increases in pressure, causing reflexive skull expansion. A pathologic state reflective of this same phenomenon is seen in hydrocephaly which causes skull expansion due to a physical block of CSF flow within the brain.

Jugular compression, while not effecting production of CSF, does seem to effect the ability of the arachnoid villi to pump against a high pressure gradient into the venous sinuses. Thus, the skull will expand and assume a flexion-type shape. The expansion, while felt generally throughout the skull, is primarily lateral, due to the major increase being reflected in the lateral ventricles of the skull. The phenomenon follows the "ram horn" folding and unfolding of the ventricles in response to CSF fluctuation and is well described by Magoun.[4] If allowed to continue, the skull tends to assume a 'mini-version' of the hydrocephalic skull with the frontal, parietal and temporal bones in external rotation (expanded skull) and disengaged from normal sutural articulation.

Discussion

Spontaneous changes can be noted in cranial bone movement if manual compression is performed while an operator skilled in the palpation of the cranial rhythm index (CRI) monitors the skull. The operator will feel a sudden general expansion of the lateral dimensions of the skull and the paired bones will externally rotate as the skull moves into an extreme flexion, forced by increases in CSF pressure.

The induced change is so strong that if compression is performed during the early extension phase, the cranium will reverse direction and expand as if filling water into a balloon. The skull, arriving at full expansion, remains there, the continued increase in CSF pressure keeping it swollen. To the operator the skull will seem to undulate slightly at the extremes of flexion. This is probably due to the fact that incremental amounts of cerebrospinal fluid do manage to escape into the venous sinuses causing small fluctuations in skull dimension. Normal cranial rhythm will not return until jugular compression is discontinued. This appears to support the theory that CSF production and absorption are primarily responsible for the cranial rhythmic cycle.

The internal forces cause a pull on and swelling of the dura, tensing the entire dural membrane structure from the skull to the coccyx. To allow for this the cranial bones must also balloon outward and eventually disengage at the extremes of cranial expansion and the sacral-coccygeal attachments must be unrestricted. A normally functioning craniosacral mechanism permits this temporary change and no negative reaction will be noted, the effects remaining within the physiologic adaptive range of the body. Nevertheless it initiates a powerful unfolding of the skull and can even be used adjunctively in home therapy, along with respiratory phasing for patients with craniosacral dysfunction.

Clinically, when a disturbance inhibiting normal craniosacral function is present, notable changes in pre and post-compression muscle strength can be observed. This offers a significant and simple diagnostic input for the physician to evaluate the entire craniosacral and stomatognathic system dynamics. Several combinations of positive reactions have been observed by the author in response to jugular compression, along with its use as a therapeutic modality.

1. A previously strong muscle will weaken to bilateral and or unilateral jugular compression.
2. A previously weak muscle will strengthen to bilateral jugular compression.
3. A previously strong muscle will weaken to bilateral jugular compression combined with jaw motion, head motion or some gait position.
4. Jugular compression as a therapeutic adjunct.

Group one

By far the most common positive finding and represents the inability of the craniosacral system to adapt to pressure changes resulting from jugular compression. The system is further evaluated by compressing first one side then the other. If only unilateral compression causes a strong muscle to weaken, one can assume that some cranial/dural lesion exists on the side of positive jugular challenge. Should compression of both sides, individually, cause weakness the entire craniosacral system should be re-evaluated as the problem is more global in character, involving the entire cranial bowl; thus, more severely lesioned.

Maintaining compression, the patient is asked to respire - first inspiration, then expiration. If the cause of cranial dysfunction is respiratory in nature, one or the other phase of respiration will reverse the positive CSF challenge. Certain cranial lesions, non-respiratory in nature, may be the underlying cause of the problem and should be evaluated in the absence of changes from phases of respiration. These can be somewhat difficult to locate as the physician has little real clue as to the location of the lesion. Unilateral jugular compression will indicate which side the lesion is present, but the physician must use both knowledge and experience to locate non-respiratory lesions.

Cranial lesions not responding well to respiratory challenge are as follows:

a. Sphenobasilar compression
b. Frontal bone fixation complexes
c. Zygomatic bone sutural fixation involving all three sutures at once (An observation by the author, but is a common finding which can only be therapy localized using one finger on each of the three articulations.)
d. Severe bilateral temporal bone compressions - rare, but can be present (auricular proprioceptive technique - Goodheart)
e. Certain lesions involving only the dural membranes; most especially the tentorium cerebelli lateral pole torsioning.

When none of the above problems are present in the cranium, non-respiratory faults can also be traced to the temporo-mandibular (TMJ). The patient is asked to clench, open, lateralize, protrude and retrude the jaw. Often, one, or a combination of these movements will negate the positive challenge. The physician must conclude that craniosacral mechanics are seriously impaired by temporo-mandibular dysfunction. Vertical dimension evaluation and other applied kinesiology - TMJ evaluations can be made simultaneously.

Group two

Offers the physician another dimension for the evaluation of the patient. In patients whose CRI is below 6 full cycles per minute, jugular compression will cause NO weakening of pre-

compression muscle strength, despite the presence of cranial lesions and dural membrane tension. Rather, CSF pressure increases will STENGTHEN pre-compression muscle weaknesses. This is the patient suffering from low metabolic rate; the production of CSF within the choroid plexus not of sufficient volume to stimulate great increases in CSF pressure during jugular compression. It appears that the central nervous system, perceiving the pressure rise, temporarily assumes that the metabolic rate has also risen and muscle weaknesses associated with the lowered metabolism will return to strength.

The physician should be alerted to an underlying metabolic problem which must be treated prior to or simultaneously with mechanical corrective procedures. Because of the extreme significance of the low metabolic rate, the author recommends that patients be evaluated for muscle weaknesses and strengths prior to initiating the jugular compression so that the physician is not ignorant of its existence. Failure to be aware of this reaction may lead the doctor into concluding that the body is functioning within normal craniosacral limits when, in fact, it is not.

It is the experience of the author that manual therapy should await evaluation and correction of the metabolic disturbance. These patients have a characteristic tendency to either react negatively to manual therapy or not at all, reflecting the inability of innate to successfully utilize the treatment given. Low metabolism can be found in many problems ranging from the simple to the more complex. Thyroid conditions, drug therapy, long-term environmental toxicities and allergic responses coupled with fungal infections are only a few of the many combinations which can cause the post-compression strengthening response. They often challenge the ability of the physician to correct, but the positive jugular compression test will send the doctor in the right direction already on the first visit. (It is interesting to note that not only will the muscle strength return when the appropriate metabolic therapy is given, but the CRI will respond immediately upon ingesting the correct substance showing the marvelous integration of the nervous system.)

Group three

Constitutes the finding when craniosacral and dural membrane mechanics are essentially functioning within normal limits, but upon introducing another factor influencing those mechanics into the test, post-compression weakness occurs. This indicates that the introduced factor is negatively influencing the craniosacral dynamics and should be corrected; otherwise it will cause an eventual return of dysfunction. When the patient can perform all jaw movements and obtain all gait positions without inducing post-compression changes in muscle strength, the physician can assume that adequate correction has been made.

Group four

Jugular compression is an extremely powerful internal mechanism for aiding in correction of difficult craniosacral lesions. When used in combination with respiratory phasing and specific manual correction, the lesion remisses much more rapidly and effectively. A secondary effect of using compression therapeutically is that it will automatically correct almost all minor lesions, immediately. Inspiration and expiration faults, when not coupled to more severe lesions, can be removed at home by the patient using compression together with deep respiratory phasing. The cranial bones tend to disengage and physiologic tension is placed on the entire dural membrane structure, removing adverse tension and torque.

Procedure

1. Test the patient for general muscle strength, making sure that the muscle tested is not over-facilitated using any of the several applied kinesiology techniques. (North pole of a magnet, backwards running of a meridian.) Failure to locate an over-facilitated muscle will cover any possible positve finding. [The author has previously reported an alternate method of determining the over-facilitated muscle through the use of the acupuncture system.[7] The patient is asked to therapy localize the point of K-27 on the same side as the muscle being tested for over-facilitation. If the previously strong muscle weakens, it is indicative of hypertonicity and cannot be used as a successful strong indicator muscle. A full discussion of this technique is not possible in this paper. A more complete and updated discussion of this finding can be found in the 1992 collected papers of ICAK-E.[8]]
2. Apply bilateral jugular compression. Compressive force is applied at a point just below the mastoid processes along the line of the sternocleidomastoid muscle. Here the internal and external jugular veins cross each other as they progress down the neck.[9]
3. Test a previously strong indicator muscle for weakening. If found, introduce phases of respiration, jaw movements, etc. One or more may be found negating the weakness to challenge. These may be corrected as found or the physician may use temporal tap to find the primary problem.
4. If no weakness occurs, include other factors influencing the craniosacral system into the test - jaw motion, gait position, head & neck movement. If some other factor provokes weakness, it can be assumed that the craniosacral system has been negatively influenced. It should be evaluated and corrected. If no weakness occurs, assume that the system is functioning normally.
5. If the doctor suspects a low metabolic rate, muscles found weak prior to jugular compression should be tested for post-compression strengthening. If this occurs, the problem is metabolic in nature and should be corrected prior to continuing craniosacral and manual therapy.
6. Treat the most severe cranial lesions using respiratory phasing along with jugular compression. After each correction, rechallenge for minor lesion patterns. Most often, because of the powerful unfolding effect caused by the jugular compression, many lesions will spontaneously correct themselves.

Conclusion

The author has not previously read nor heard of any other applications of jugular compression as described above. Thus, it is believed that the above observation constitutes a new application of the discovery made by Queckenstedt many years ago. Although all the applications for the technique have not yet been thoroughly developed, it is thought that the technique will be a significant contribution to the clinical application of precise and lasting craniosacral therapy.

The technique appears to have some application during therapy. Especially during treatment for lesions requiring inspiration assist, simultaneous jugular compression aids in the correction. During therapy, compression seems to aid in "disengaging the sutures" and in "unfolding" the dura, making the total correction less time-consuming and more lasting.

Since the inception of the procedure, the author has observed that the methods available to the physician using applied kinesiology often fall short of fully correcting the entire craniosacral and dural membrane mechanics. This was made evident by the work of Christopher Smith, D.O., DICAK, who has brought to applied kinesiology tests for sphenobasilar compression, sphenobasiclar lateral and vertical shifts and frontal bone fixations, among others.[10] These have often proven invaluable in making lasting corrections of the system. This is most evident when a patient has a sphenobasilar compression. No significant movement of the sphenobasilar is possible and respiratory challenging is negative, falsely leading the physician into believing the craniosacral mechanics are normal.

Because of an inability of the author to make sufficient corrections in the craniosacral mechanics, it was found that only by incorporating the concepts of Magoun and Upledger into the work-up, final corrections could be made. To do so, it became necessary to develop applied kinesiology techniques for the evaluation of the facial bones and dural tension based upon Upledger.[6] Sphenomaxillary compression, sphenomaxillary and vomer shear and torsion and three point zygomatic sutural faults, have all resisted correction of the primary respiratory mechanics. Finally, adverse dural tension, evaluated and treated according to Upledger, along with the therapeutic effects of jugular compression, have allowed for much more profound and immediate cranial vault changes. The procedures found by the author have been reported in seminar notes.[11]

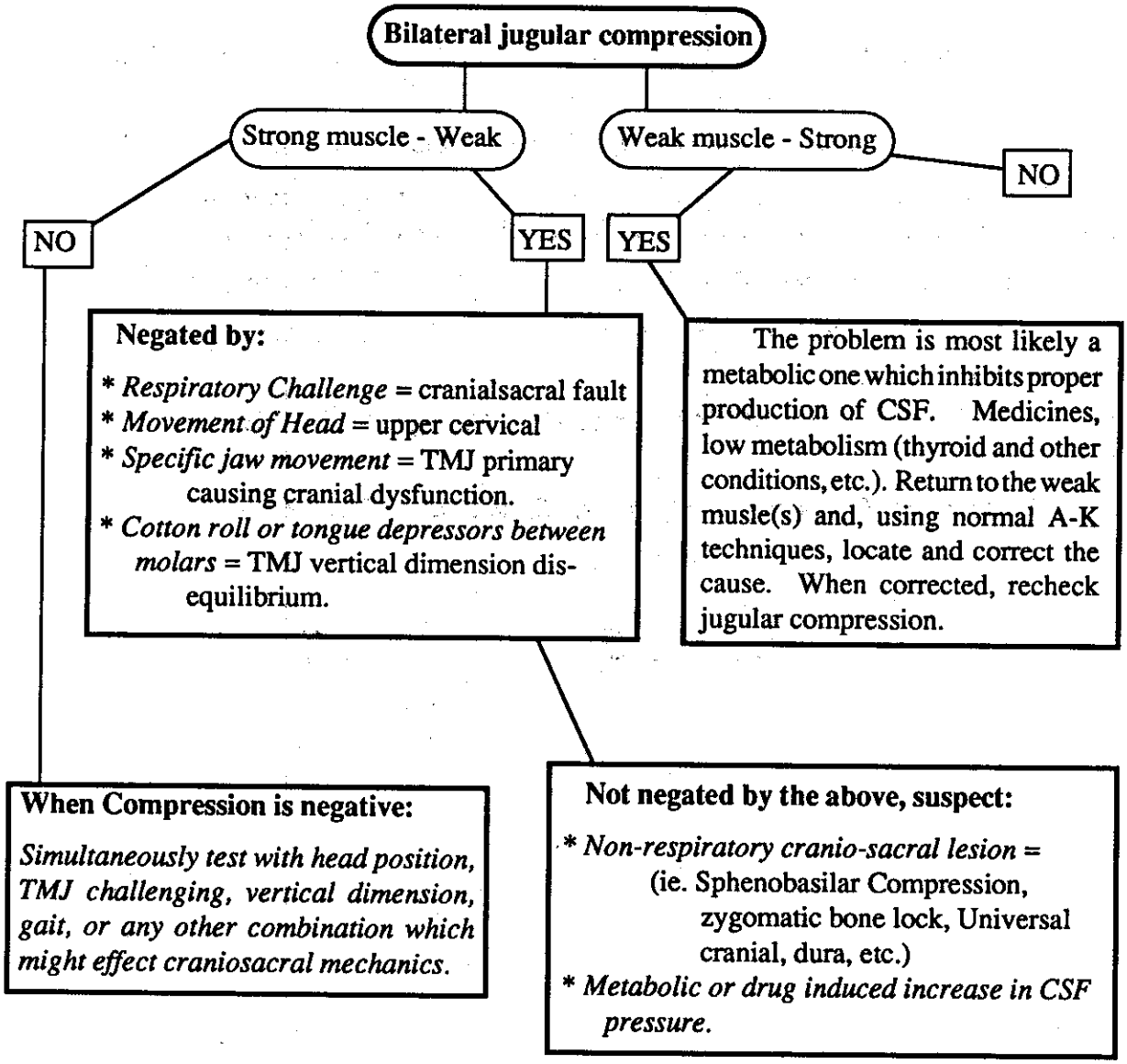
To date, the author has not found a single patient experiencing low metabolism who doesn't strengthen during jugular compression. It appears as if strengthening of a weak indicator during jugular compression is almost pathagnomonic for low metabolic states. The beauty of the reaction is that it alerts the doctor immediately to the problem of low metabolism. It becomes obvious that mechanical correction of the primary respiratory mechanics in the presence of lower metabolism will tend to have short-term gain, but long-term futility, as the patient cannot maintain adequate cranial motion.

The physician must not be discouraged should a positive jugular compression test return on a follow-up visit. It is the experience of this author that the depth of correction made when following the indications of the jugular compression are much more profound. These deeper corrections cause much more rapid changes in facial bone and cranial bowl structure to which the body must compensate. Especially in adults who evidence osseous asymmetry, follow-up visits need to be made in order to eliminate minor problems which tend to develop during physiologic skull remolding.

Finally, in the absence of a positive jugular compression test (assuming no low metabolic rate), the physician may use the test creatively in order to locate any hidden abnormalities which, although not present in a neutral position, may present themselves under various conditions. Positive reactions can be elicited from TMJ dysfunction, gait disturbances and shoulder dysfunction which effects proper sternocleidomastoid and upper trapezius coordination and facilitation. Any manner of singular or combined problems may covertly effect the primary respiratory system and can be located with this test procedure.

Under normal circumstances, jugular compression is a painless, safe procedure. However, caution should be used when attempting jugular compression on patients with recent head injuries and suspected of having intracranial tumors. The increase in CSF pressure may abruptly precipitate further bleeding or cause hernitation of the cerebellar tonsils with resulting medullary compression. This possibility should not be cause for alarm, as it is rare and the compression procedure is maintained only a short period of time. However, it is indicative of the powerful mechanism of action offered the physician who uses jugular compression for diagnostic and treatment procedures.

Further research into this phenomenon is needed and may lead to other discoveries or an alteration of the procedure presented above as further clinical evaluation is made.



Nb. Make any cranial corrections with jugular compression for greater effect.

(Figure 1)

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DIVISION III - COMMENTS ON PUBLISHED PAPERS

DR. GEORGE J. GOODHEART
RESEARCH REPORT

DR. GOODHEART'S RESEARCH TAPES
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-Arginine cycle ultimately gives off nitric oxide which is important in blood pressure regulation, immunity, platelet adherence, and levels of cholesterol.

-Place arginine (from any supplier, GJG uses Biotics) in the mouth of a patient and if this weakens a previously strong muscle, i.e. pectoralis major sternal, place Arginex (Standard Process) in the mouth with the arginine to check for neutralization of weakness. Arginex is a source of arginase. Provide arginex.

-Nitric oxide sythase controls metabolism of arginine to ornithine to citrulline. Nitric oxide synthase is both calcium and magnesium dependent. If patient weakens with arginine and is not helped with arginex, then leave the arginex on the tongue and add a source of calcium and magnesium (Calsol from Standard Process). If this negates the weakness, need to provide both arginex and calcium-magnesium product.

-If the calsol and arginex does not neutralize the muscle weakness to arginine, then place folic acid-B12 (Standard Process) on the tongue with the others and check for neutralization of the weakness. Tetrahydropteridine delivers folic acid for the nitric oxide pathway.

-If the arginex, calsol, and folic acid-B12 does not neutralize the weakness to arginine, then add SOD (superoxide dismutase) to the rest of the supplements in the patient's mouth. So far, GJG has not had one patient who did not respond at this point (these were all patients who had an indication for this approach, i.e. hypertension, immunity problems, impotency).

-The last two issues of the New England Journal of Medicine have dealt with the effect of nitric oxide on impotent and potent males and affecting circulation in the corpus cavernosum.

-Nitric oxide maintains the endothelial relaxing factor (ERF). ERF is responsible for maintaining a normal blood pressure.

-As people age, there is often an increase in the systolic pressure, and a lesser degree of rise in the diastolic. There are some people who are still normal at 120/80, this is due to the ERF.

-Nitric oxide is a new neurotransmitter. It is a simple gas that is produced by the intima of blood vessels. University of Michigan has done the work to show that the intima produces nitric oxide. When the intima is stripped from the blood vessel, there is no longer production of the relaxing nitric oxide.

-May 1992, Scientific American, The Biological Roles of Nitric Oxide. "This previously elusive and obscure chemical is proving to be of vital physiological significance. Nitric oxide may be the first of a novel class of neurotransmitters." By Solomon Snyder, Director of the Department of Neuroscience, professor of neuroscience, psychiatry, and pharmacology at Johns Hopkins. Received an award for basic biomedical research. He has pioneered the identification of receptors for neurotransmitters and responsible along with Candace Pert for identifying the location of the receptors for both morphine and other

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narcotic agents, endorphins and enkephalins.

-Phagocytes produce nitric oxide and aid in immunity against parasites, viruses, and bacteria, and along with interferon and interleukin 1 and 2, will interfere with the production of new malignant cells.

-Patients who weaken with clorox sniff (Schmitt), manipulation of LI4 would negate this weakness to clorox, and the free radical quenchers would not be needed. Apparently, manipulation of LI4 helps the enterohepatic circulation of bilirubin. Bilirubin is the best free radical quencher, even exceeding vitamin E, glutathione, etc.

-Superoxide dismutase is also useful in continuing the function of nitric oxide.

-A large intestine visceral pattern could be influenced by treating LI4.

-The thrust philosophically in chiropractic is to reduce the art of chiropractic practice to that of a very narrow musculoskeletal vision, when in reality the more appropriate should be neuromusculoskeletovisceral.

-Visceral Manipulation, by Barral and Mercier, 1988, Eastland Press, P.O. Box 12689, Seatle, Washington 98111. Discusses basic concepts of visceral movement and the thoracic cavity, abdominal/pelvic cavity, liver/biliary system, esophagus, stomach, small intestine, colon, kidneys, perineum, bladder, female and male reproductive systems, and coccyx.

-Original concept of muscle/organ-gland relationship came from some observations: patients with kidney stones evident on an x-ray would have a lumbar scoliosis rotating away from the kidney stone side, or looking in urological texts, would see the same thing. Supraspinatus associated with drainage of the head and neck, even though textbooks say there is no lymphatics in the brain, there is evidence that the glial cells contain lymphatics. Observation that around April 15 (tax time) there was an increase in shoulder problems, i.e. bursitis, associated with using adding machines; the handedness of the patient did not always correlate with the side of the shoulder problem. This is a mental strain which would be associated with the supraspinatus. Bilateral weakness of the supraspinatus in the child is associated with having difficulty with one subject in school and doing well in the rest of the subjects, and doing the neurolymphatic associated with the supraspinatus improves this.

-Victor Portelli, D.C. has found that when an organ is out of position, if you push it further out of position and test the muscle associated with the organ, the muscle that was originally strong will weaken.

-Have noted this in the past, i.e. in females, if they void urine when they cough, this is associated with a weak levator ani and uterine drop. A bearing down that weakens the piriformis or gluteus medius is the diagnosis for the need to correct a uterine lift. Correction is a contact above the pube with the thumb and index fingers spread out, as the patient exhales, she raises her arms and legs and the doctor applies a pressure to lift the uterus (cephalad).

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-Can perform this in the male, have them bear down and if there is weakening of the piriformis, go rectally and lift the prostate.

-Portelli and Marcellino have done a lot of research into visceral manipulation, including finding older textbooks on the subject. They have developed a system of visceral technique.

-Moving the pancreas by a squeezing/pinching motion will weaken only one or the other latissimus (if the pancreas is out of position), and it will not weaken any other muscle.

-Exception is if there are numerous adhesions and movement of one organ may influence another organ and possibly result in muscle weakness not related to the original organ tested, i.e small intestine.

-The treatment method is to lift the organ in the abdomen while the patient coughs, this breaks tension in the mesentary and peritineal folds that hold the viscera in position.

-Recommend study of Portelli and Marcellino's work. Portelli wrote a paper for the Proceedings of the Members of ICAK called Ptosis of the Transverse Colon, in the Winter 1987 edition, contains a description of adjusting the colon as well as some home excercises.

-Pressure on the transverse colon posteriorly and caudally will weaken only the tensor fascia lata.

-Visceral technique is a difficult thing to teach because you depend upon feel. Portelli has simplified it with the ability to make a definite diagnosis to perform the visceral manipulation.

-When you have a cold with a runny nose, the mucous eventually will turn into strings of mucous. Normally there are enzymes that will digest the mucous strings. The same exudation occurs on the outside of the bowel, and you get spider web adhesions that literally fix the viscera into position.

-Evidence on magnetic resonance imaging of organ motion like the early osteopaths and others noted in the early years of bloodless surgery.

-Portelli's paper, Ptosis of the Transverse Colon: "This paper is presented to help in the evaluation and management of a condition that has not been addressed before in AK literature, that is ptosis of the transverse colon. Recurrent ICV, premenstrual tension, weakness of the abdominals could all be due to ptosis of the transverse colon. Effects of gravity on the colon producing weakness of the abdominals. It doesn't allow a return to normal function. Constant nagging pain in the lower abdominals, high blood pressure, occasional radiculitis due to traction of the lumbar and sacral plexi,..., ptosis of the gall bladder into the lower abdominal quadrant. No consistent information on how to therapy localize this condition is available. Using a strong indicator muscle, use a holding challenge in a caudad direction on the most cephalic point of the bloated bowel (this point represents the upper edge of the transverse colon). If a strong indicator muscle weakens then it is indicative of ptosed transverse colon. Correction is achieved by applying a cephalad scooping posteriorward pressure on the lower portion of the transverse colon and gradually lifting the bowel towards it normal position. Asking the patient to cough several times during this procedure helps for a more speedier correction. The patient needs to learn how to correct this condition at home before

retiring...The home instruction is that the patient performs this procedure every night for approximately 3 months."

-GJG finds a consistent need to perform jugular decompression with visceral adjustment corrections. One of the signs of interference with the glossopharyngeal nerve is deviation of the uvula (with the patient in a seated position). Disturbance with the vagus is indicated when you ask the patient to say "ah", and one side of the levator palatini will rise higher than the other side. Occulocardiac reflex: pressure on the eyeball slows the heart in vagal disturbance, i.e from 72 beats per minute to 57, a drop of 15 or more.

-These are indications to perform jugular decompression, hold the fingers in a cantelever position between the occiput and atlas, prior to doing any visceral techniques.

-GJG's father used to say: "A patient complains of their lungs, digestion, voice, constipation, diarrhea, etc. I don't know what you call this, but it's all the same nerve."

-Pilot's on transcontinental flights get weak voices at the end of the flight due to this.

-There is a difference in facial temperature as measured on liquid crystal thermography, differences in pupil contraction, and a difference in toe turn in (relative hypertonicity and hypotonicity of the psoas muscles) are also indicators to perform jugular decompression. Perform the jugular decompression long enough to eliminate these signs, takes about 3-4 minutes. Then perform the visceral adjustment, and balance the pyramidalis muscle. Then the home excercises are not so necessary.

-Psuedo-Category 2, positive therapy localization of the symphysis pubis right, left, or both against the sartorius/gracilis. Maintain therapy localization to the symphysis pubis and now the evidence of a UOMS short leg posterior ilium or LLL long leg posterior ischium will be present, but when they take their hand away, the signs are gone.

-Dvorak and Dvorak spondylogenic reflex for the sacrotuberous and sacrospinous ligaments are at fault. Get a palpatory pain from C1-T8, then contact into the belly of the ligaments with your thumb and then repalpate the painful areas and note a diminished palpatory pain. This is commonly found in people that have arm or shoulder complaints.

-GJG finds (like Sheldon Deal described before) that you can't turn off muscles of the circulation sex meridian. Sometimes the sartorius/gracilis will test weak in the clear and you can't turn off the piriformis in the same patient (with sedation points). In classical acupuncture, if the CX meridian is over, the TW should be under. When you test the TW via teres minor, you don't find the teres minor to be weak (under). If you stimulate CX9 on the middle finger with 3 or 4 vigorous taps, the TW will now show weak (teres minor). Tapping the sedation point at the wrist should turn off the piriformis, but it does not; so go to the connecting point for either the CX or TW and tap this first, then tap the sedation point at the wrist, and then the piriformis should turn off. Try the connecting point of CX first, if that doesn't work, then try TW. In a rare

instance, you have to use SP21/KI27.

-The gonads are always on and the thyroid is off, and the patient complains of fatigue, trouble with weather. It's like leaving your car running, eventually you will run out of gas even though the engine is still running.

-Don't forget to check the associated point on the spine for CX and TW for a subluxation, T5 and L2.

-This often results in an increase in axillary temperature, improvement in constipation.

-Dr. Richard Murray discussed myelin health at the ICAK-Europe meeting in Belgium. Myelin is composed of 70-80% fat, and myelin shealth is composed of galactosphingolipoids, galactose, specialized lipids, cerebrosides, sulfatides, galactosides. Long chain fatty acid metabolism and catabolism requires many bioavailable nutrients. The only source of bioavailable lactose which the body can synthesize into delta galactose which is in turn incorporated into the galactosides is raw milk. Raw milk is practically impossible to find. There is a substitute product called BioLac by NutriWest, 500 milligrams per tablet, 190 milligrams of lactose.

-Muscle weakness in patients with neuromuscular disorders like multiple sclerosis show strengthening with BioLac on the tongue. Using Arginex, Calsol, folic acid-B12, SOD, for the nitric oxide production is also helpful. Also check them for repeated muscle testing, if weakness occurs, check them against fats like Linum-B6, vitamin F perles, Black Currant Seed Oil, primrose oil, etc.

-All of these factors are important in the rebuilding of damaged myelin.

-Wulzen factor is found in raw cream and is useful in arthritic pains. The BioLac is useful in place of the raw cream.

-Murray, "Cholesterol represents the major lipid, the fat of the myelin sheath. Interference with cholesterol synthesis production affects myelin synthesis as a whole. This is from Gray's Anatomy 36th Edition. One of the basic phospholipids in myelin is sphingomyelins which resembles lecithin, not the purified fractional type sold as a supplement, but a fatty acid complex that is present in some of the fatty acids. Cerebrosides are another essential lipid group in myelin from the glycolipid category, and its chemical composition includes a linkage with galactose, which is a sugar available in unheated milk and made unavailable in heated milk. Galangosides, a type of glycolipid, specifically helps to form the myelin sheath and the white matter of nerves. The sulfatides, also a glycolipid, are found in myelinated nerve fibers, and are composed in part of sulfates, a form of sulfur which is extremely important in the repair and maintenance. Many of these are from raw milk. Multiple sclerosis lesions are placques, scar-like fibrous patches along the nerve fibers. These patches allow a "leak". An effective placque will stop the leak, thus the patient enjoys freedom of symptoms as long as the patch of the myelin sheath is able to stop the leak. Swank has treated MS for over 30 years. He feels that MS is tiny heart attacks in the spinal cord and not the heart. Diet should consist of the best raw fruits and vegetables, natural fats and oils, raw cream or milk. Alpha lactose

predominates in milk. When it is heated, alpha lactose is converted to beta lactose. This conversion interferes with the normal biochemical conversion of lactose to delta galactose. Delta galactose is essential to the building of galactocerebrosides, the chemical constituents of nerve tissue. So the development of the entire myelin sheath is dependent upon the availability of galactocerebroside, from raw milk. This was virtually unavailable until the advent of this product."

-The combination of the nitric oxide precursors, fats, and BioLac has helped many patients with neurological degenerative diseases.

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-Medial meniscus syndrome: on the injured knee there is a spot on the medial knee about the circumference of a dime at the level of the medial meniscus. Method of correction (after balancing the muscles) is: patient supine, point toe towards center of midline, a sudden sharp traction is exerted in order to relieve the trapped meniscus. Weak sartorius/gracilis often involved which produces a wedge pattern with the wide side of the wedge on the underside of the knee which allows the meniscus to become entrapped. Discussed in previous manuals.

-GJG patient with knee pain. Previously treated one year prior successfully, with return of the knee pain, especially upon climbing stairs. When GJG supported the knee with his hands and walked up and down stairs with the patient, the patient could then perform climbing the stairs. There was a disturbance in the vastus lateralis as it relates to the vastus medialis obliquus (like the reins of a horse). When atrophy occurs, it is very specific. If the lateral muscle is stronger than the medial, the patella will be thrown medially. The patella moves laterally when the knee goes from extension to 20 degrees flexion.

-Patellofemoral pain syndrome: pain is a diffuse ache in the anterior compartment of the knee. Exacerbation of pain upon climbing stairs or following changes of position, especially of flexion. There is a palpable crepitus. Patient describes that the knee feels as though it is going to give out. There is a mild, diffuse swelling, lateral and/or medial. The knee giving way is a reflex inhibition of the quadriceps as a group, especially with ascending or descending stairs.

-The patellofemoral joint absorbs 3-4 times the individual's weight in stair climbing, deep knee bends, squatting, etc. When GJG's patient returned one year later, this is what he complained of. Treatment to the quadriceps did not produce the same results as the initial time. GJG felt atrophy on the medial side of the knee than on the lateral side. The lateral side muscle was throwing the patella in a medial direction. Holding the patella in a lateral direction with his hands allowed the patient to climb stairs with less pain. Knee supports or taping at that time did not reproduce this effect, and balancing the muscles did not give the pain relief as it had done the first time. This particular patient had knee arthroscopy and a laminectomy performed eventually.

-Australian physiotherapist discovered a taping procedure that allowed the patella to be held in the lateral direction during the first 20-35 degrees of flexion. She developed the McConnell taping treatment.

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-The normal patellofemoral joint under normal conditions, the patella sits lateral to the trochlea when the joint is fully extended. This is due to a vector force that pulls the patella laterally. At 20 degrees of flexion, the patella sinks itself into the trochlea, but at 80-90 degrees flexion, the patella moves laterally again.

-McConnell says that you fall behind on the lateral side largely because there is a strong lateral connection of the vastus lateralis in the iliotibial band, but on the medial side, there is a very thin medial retinaculum that is only one layer thick, and there is only one muscle that would pull the patella into the right position, the vastus medialis obliquus.

-Place a red mark on the center of the patella with the knee in normal extension, measure with the Metrecom probe. Then measure right of center and left of center. In GJG's observation, there should not be more than 5 millimeters of difference (given error of placement on the medial and lateral marks). Disturbance in VMO (vastus medialis obliquus), there is not enough lateral pull and there will be a difference in the position of the patella. Pressure should be exerted from medial to lateral to hold it in position. That positioning is such that there is more than 5 millimeters difference on the lateral side (from the center to the lateral side than to the medial). May be able to see it with just placing the marks on the patella.

-Balancing the muscles or using tape failed at holding the patella in position until this new tape was discovered that was made in Germany.

-Travell and Simons, Myofascial Pain and Dysfunction, Trigger Point Manual, The Lower Extremities, Volume 2, Williams and Wilkins. In the discussion of the vastus medialis muscle, she states that the vastus medialis attaches distally not only to the medial border of the patella and through the patellar ligament to the tibial tubersity, but also by a strip of muscle to the medial patellar retinaculum. The distal fibers of the vastus medialis are markedly angulated as they attach in the region of the patella. They can clearly be separated from the rest of the vastus medialis by fiber direction and by fascial plane. These distally angulated fibers often attach proximalto the femur, but chiefly to the adductor magnus, partially to the adductor longus. and to the medial intermuscular septum. The lateral obliquely oriented fibers have been designated the vastus medialis obliquus or VMO.

-The quadriceps themselves may show signs of any of the 5 IVF factors, if found, correct.

-The VMO (lower division) test: patient supine, flex knee and hip to place the heel at the level of the contralateral ankle. 20 degrees of thigh flexion, internally rotate the tibia. The doctor braces on the lateral side of the knee involved, cup the calcaneous, and pulls laterally to internally rotate the femur along the plane of the table. Rib pump area is the 7th costal cartilage and

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costovertebral junction. The VMO may have a positive TL to any of the 5 factors, origin-insertion, or the rib areas (strain/counterstrain).

-The vastus medialis (middle division) test: patient supine, flex knee and hip to place heel on opposite tibia (about half way up), 35 degrees of thigh flexion, then internally rotate the femur. Rib pump area is the 8th costal cartilage and costovertebral junction.

-In this type of knee problem, the atrophy that occurs in the vastus medialis, oblique portion is different than that which occurs in the lateral aspect. The taping procedure is such that there is a pressure from medial to lateral on the patella. Measurements will reveal that the patella is too medial.

-Genu articularis test: patient supine, flex knee and hip to 60 degrees with no rotation of tibia, have the patient push up on their ankle while the doctor resists. Rib pump is the 2nd costovertebral and costosternal.

-The vastus medialis is innervated by a separate branch of the femoral nerve. This was made note of by Travell: Part of the femoral nerve branches to the vastus intermedius, penetrates that muscle to supply the articularis genu and the vastus medialis oblique.

-Tape: Medco Supply Co. 705 South Nichols Ave, Munsee, Indiana 47303.

-Excercise the vastus medialis (8-10 pounds and extend the knee) and tape for 2 weeks (patient must tape on their own).

-Why does the stomach and duodenal mucosa resist ulceration? This is due to the combination effects of the urea-urease enzyme system and carbon dioxide-carbonic acid anhydrase system (which is zinc dependent). They are biochemically associated in the gastric and duodenal mucosa. Their products neutralize acid and alkali respectively, and the two systems interact to maintain the acid-alkaline balance in the mucosa. Carbonic acid anhydrase, in addition, supports normal gastric and duodenal secretion.

-If there is a crack in the gastric mucosa, the urease enzyme system secretes urea (which is ammonia and carbon dioxide) and any erosion of the mucosa by hydrochloric acid is stopped.

-American Journal of Gastroenterology, Jan. 1960, Goodfriend and Goodfriend (a dentist and gastroenterologist), Vol. 33, No. 1, pages 80-89.

-Patients with gastritis, ulcers, or hiatal hernia can be given carbamide, which is basically urea, a half to one teaspoon 2-3 times per day is good prevention. Standard Process produces AC Carbamide which is urea and vitamins A and C.

- Conway: Urea's action neutralizes gastric acidity at the surface of the mucosa. They found increased urease action at the margins of excised human ulcers and decreased urease action in the mucosa most susceptible to ulceration. The level of urease is increased in animals by adding low concentration of urea to the

drinking water, by the hormone enterogastrone and by a high protein diet, and reduced urease action after diets low in protein, in other words, if you lower the protein intake, it is ulcerogenic.

-Conway: "The high acidity of the parietal secretions is associated with an equivalent alkalization within the secreting cells. The restoration of the hydrogen ions in the cell becomes essential. Likewise, the non-parietal cells, chiefly, as would be seen secreting mucous, go to secreting mucous to protect from the acidity of the gastric juice. Two intracellular enzymes have been shown to catalyze neutralizing reactions, they are carbonic anhydrase and urease. Carbonic anhydrase catalyzes the hydrogen ions of the oxyntic cell, and urease the formation of ammonia which protects the non-parietal cells."

-Clinical studies with two placebo tests in the six years prior to the publication of the article in 1960 showed that carbamide relieved symptoms in 90% of 115 cases of gastric or duodenal ulcer and 93% of 41 cases of upper gastrointestinal disorders, i.e. hiatal hernia, esophagitis. Therapeutic dose was a rounded teaspoon of carbamide in one third glass of water between meals and before retiring. A single dose before retiring lengthened intervals between and diminished the severity of reoccurance. The safety and effectiveness gave it wide use in gastrointestinal disorders.

-Helps in burping, heartburn.

-Also check for small intestine.

-These patients may require zinc. Test for through the Zinc Tally test. Use small amounts of chelated zinc three times a day. Carbonic acid anhydrase is zinc dependent.

-Irritable Bowel Syndrome, reoccurring cystitis; these patients often have very alkaline urine.

-1983, Cohen, article concerning pathogenesis of recurrent urinary tract infections, the bowel, bladder, and hypokalemia connection. "A conceptual approach to the understanding of the pathogenesis of recurrent, non-obstructive urinary tract infections is presented. Abnormal colonic function is associated with potassium wastage which alters smooth muscle function of both the bowel and bladder. Hypokalemia additionally results in the abberation of an alkaline urine and bladder dysfunction which leads to bladder stasis, vaginal-perineal contamination from the bowel flora are etiologically important in such infections, thus a linkage exists between bowel dysfunction, potassium wastage and recurrent urinary tract infections."

-If sniffing clorox weakens, treatment of LI4 will negate the weakness. This is also present upon sniffing ammonia. Clorox and ammonia are found separate or occurring together. If the patient has a relative hyperammonemia, they will weaken to sniffing ammonia.

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-GJG tests saliva potassium (obtained from Ann Arbor Scientific Organization). This parallels the blood potassium. In general, the patients will have low or low normal potassium and alkaline urine and require potassium supplementation, i.e. Organic Minerals from Standard Process.

-This is also been useful in migraine headaches.

-Cohen: "It is clear that abnormal colonic function plays a central role in the genesis of recurrent urinary tract infections. Colonic dysfunction is often associated with potassium wastage either on an intermittent or continuous basis. The biological consequences of such potassium loss are four fold: first, potassium loss might result in further alteration of colonic smooth muscle function with further potassium loss occurring, second, the alterations of colonic dysfunction are often accompanied by abdominal distension and fluid retention, third, the same problem that affects the smooth muscle of the colon also results in altered urinary bladder tone which is associated with varying degrees of urine retention and stasis, the final consequence of potassium depletion is the effect on urinary pH, the relative and resultant alkaline urine along with various amounts of standing bladder urine provide the setting for bacterial growth."

-Cohen has also published an article in Medical Hypothesis, The Hypokalemic Bowel, Bladder, Headache Relationship and New Syndrome, The Role of the Potassium-Ammonia Axis. "A conceptual approach that relates vascular headaches, bowel, and bladder dysfunction to abnormalities of the ammonia potassium axis is presented. Hypokalemia alters smooth muscle function of both bladder and bowel and results in the elaboration of an alkaline urine. The occurance of an alkaline urine along with bladder dysfunction and urinary stasis predisposes to recuurent urinary tract infections. Hypokalemia and/or alkalosis increases the renal return of ammonia exposing the brain to chronically higher concentrations of ammonia, and it facilitates its passage into the central nervous system. Increased blood levels of ammonia predispose to hyperventilation which results in superimposed respiratory alkalosis on a pre-existing hypokalemia and/or alkalosis, therefore causing intense cerbral vasoconstriction, varying degrees of cerebral ischemia and hypoxia occur giving rise to higher brain concentration of ammonia, vasodilation occurs during the headache phase and maybe a consequence of sudden increases in brain ammonia and/or due to the release of other vasoactive mediators as a consequence of increased blood ammonia. A reduction of protein intake may result in alterations in amino acid precursors for brain uptake, therefore, further interference with the modulation of cerebral blood flow and brain function."

-Ammonia is detoxified to glutamine in the brain and results in a depletion of brain glutamic acid. The neurotoxic effects are magnified in the presence of hypokalemia and/or alkalosis. Glutamic acid is a precursor of GABA (a mediator of central inhibition). In an exaggerated condition it can lead to convulsion or seizure activity.

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The precise biomechanical considerations for the genesis of seizures remains unknown, it is likely that chronic elevations of blood ammonia play a role. The depletion of glutamic acid may only be one facet of the effects of the myoclonic effects of ammonia. The long term elevations of ammonia may result in other nutritional consequences.

-If patient weakens to ammonia, consider that they may have a hypokalemic pattern. Place a potassium supplement on the tongue and retest the ammonia sniff, if it negates the sniff, give 4-8 Organic Minerals, or other potassium supplements. They may have symptoms of irritable bowel syndrome, recurrent urinary infections, migraine, or convulsions/seizures. Vigorous manipulation of LI4 also negates the ammonia sniff test (both thumb webs).

-Refer to Schmnitt's material on ammonia for patient's who do not clear the ammonia sniff with the above treatments.

-Journal of the American Medical Association, July 19, 1985, Vol. 254, No.3. Refers to the above listed Medical Hypothesis article on bowel, bladder, and vascular headache relationship. Since this initial study, we have performed echocardiograms on 23 consecutive patients with the bowel-bladder-headache syndrome. In each instance varying degrees of mitral valve prolapse was observed. Furthermore, this constellation of findings is often associated with esophageal dysfunction. Hypomagnesia is also frequent concommitant. Many of the symptoms including anxiety, panic attacks, fatigue, and fibrocytic-like symptoms respond to the correction of the hypokalemia and alkalosis."

-Can use Organic Minerals for the potassium or can use a quarter teaspoon of potassium bicarbonate in a small amount of water for testing in the patient's mouth against the ammonia sniff test. Can use the potassium bicarb, a quarter teaspoon three times a day, or the organic minerals up to 6 a day.

-Therapy localization is positive whether the patient TL's with the fingers spread apart or with the fingers touching, in other words, the patient is not TLing himself, for those of you that are concerned with the relationship of other hand applications.

-A spread finger double hand TL is much different than the original work of Perlman and the later work of Beardall. None of these observations apply here. The interdigitated TL does not, of itself, offer any muscle test change, until the interdigitated fingers are placed palm down on the area that is being TLed. Earlier palm and finger opposition was used to TL sacral subluxations, with just the ulnar surface of the hands placed on the sacrum. The traditional TL with palm opposition to the back of the hand when applied with both hands does not give the same effect as the dual interdigitation form. Traditional TL utilizing both hands can be altered with both right and left brain activity. The interdigitation adds a possible "mass action" effect because of the right hand-left

brain, left hand-right brain sequential pathways are now effectively multiplied. This doubling effect is then applied to the area that is to be TLed, whether midline, left, right, anterior, or posterior. This temporarily neutralizes switching (which is basically a right and left brain mix-up). No meridian relationships have been noted, although it was an earlier conjecture for the basis of the mass action. The mass effect is probably hologrammic. Random approximations of the digits in flexed, extended, or crossed over patterns yielded no observable results.

-GJG is familiar with hand mode applications and this has nothing to do with interdigitation TL.

-1988-1989 Dorland's Illustrated Medical Dictionary, published by WB Saunders, definition of chiropractic: "Chiropractic is the science of applied neurophysiological diagnosis based on the theory that health and disease are life processes related to the function of the nervous system. Irritation of the nervous system by mechanical, chemical, or psychic factos is the cause of disease. Restoration and maintenance of health depends upon normal function of the nervous system. Diagnosis is the verfication of these noxious irritants and treatment is the removal by the most conservative method."

-The ICA has just changed their definition: "The philosophy of chiropractic holds that the body is a self healing organism and a major determining factor is development of states of disease or dysfunction is the body's inability to comprehend its environment either internally or externally. Directly or indirectly all body functions are controlled by the nervous system, consequently, the central theme of chiropractic theories on health is the premise that abnormal body function may be caused by interference with nerve transmission and expression due to pressure, strain, or tension upon the spinal cord, spinal nerves, or peripheral nerves as a result of displacement of the spinal segments or other skeletal structures (subluxation)."

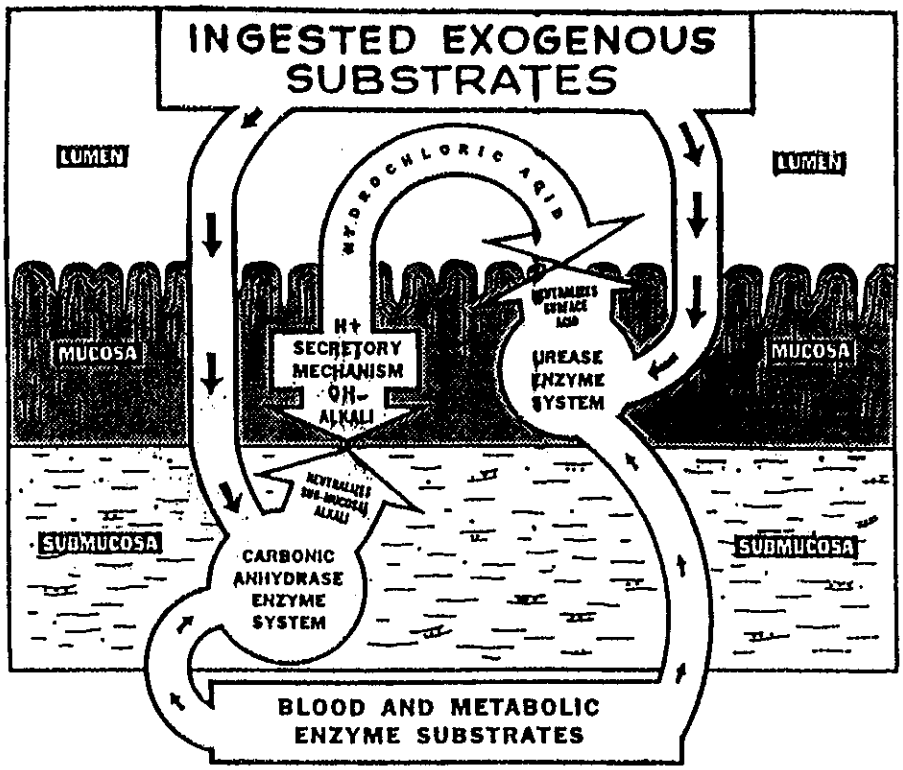
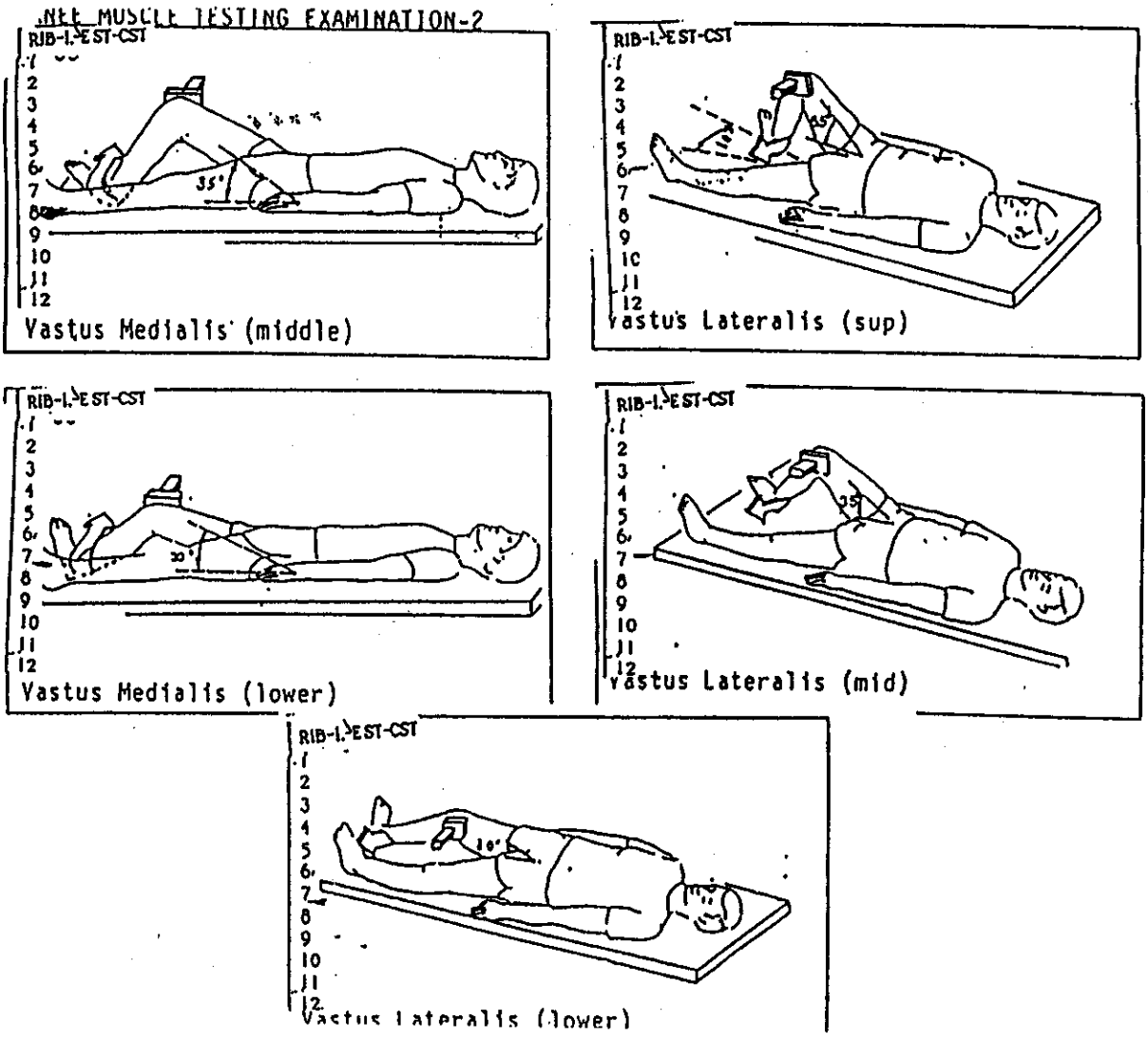


Fig. 5—Action of orally administered enzyme substrates in maintaining acid-base equilibrium and enhancing resistance of mucosa.



DR. GOODHEART'S RESEARCH TAPES
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-Discussion at the 1991 Summer meeting of the ICAK was clinical nutrition. GJG discussed the science of neuroendocrinology (brain to pituitary link) that was discovered by Hinsey and others. This brain-pituitary link is dependent upon hormones flowing within nerve axons (axonal flow). Ernst suggested that hormone messengers were being sent from the brain to peripheral organs through nerve fibers. It was assumed for many decades that axonal flow was always down, away from the brain. Within the last 10 years, it has become clear that hormones also move up nerve fibers from the body to the brain. This was noted utilizing different experimental techniques, i.e. hormones injected into the eye are carried back to the brain, tracers injected into the tongue are carried back to the brainstem, and substances injected into the thigh muscle can be carried into the spinal cord. The best studied molecule for this is called the nerve growth factor. This was in Science, #204, 4-20-88, pages 18-24. The central nervous system operates in the synaptic relationship that we are already aware of, but also in a parasynaptic relationship. Neurotransmitters that affect synaptic relationships are acetylcholine, serotonin, GABA, etc. There are other substances that act in parasynaptic fashion.

-Limbic system is closely connected to the sensations of pain. Pain pathways follow rostral ascending pathways which synapse in the limbic system. The response, repair, or alleviation of pain (what the body does automatically) consists of caudal descending fibers that shut off the same circuits that were turned on by the original pain.

-Mind Body Therapy, Rossi and Cheek, Norton and Co. Professional Book. Quotes will be taken from this book.

-There are localized neuronal networks of the brain that are activated by informational substances (substances that flow within axons). State-dependent memory, learning, and behavior are then encoded by these informational substances (IS) in these neuronal networks. The molecular genetic basis of memory, behavior, and learning is modulated by these IS. The informational receptor communication systems are the psychological or psychobiological basis of state dependent mind-body healing i.e. hypnosis. The neuronal network can be defined in terms of the activation of specifically localized area of neurons that are stimulated by the IS that reach them by the diffusion of the extracellular fluid (ECF). ECF makes approximately 20% of brain volume.

-Frances Schmitt of MIT, in a recent paper on molecular regulations of brain function. "The discovery that more than 50 years ago that contiguous neurons react with each other at a synapse, not by bioelectric modalities, but by the action of chemical mediators called neurotransmitters, which was for many years received with

considerable skepticism by the neurophysiological community. However, this now classical chemical concept with virtually no deviation remains a basic tenant of neurobiology. The working hypothesis here is suggested that neuronal communication may be mediated not only by the dozen-odd classical neurotransmitters, but by many, perhaps hundreds of other neuroactive substances called IS. In some instances they may be delivered in a non-conventional parasynaptic mode (non-synaptic). High specificity of action is achieved from specific structures (synaptic linkages and neuronal networks) but by equally selective bindings of various IS to the receptors not only at the synaptic regions, but over the entire neuronal surface. The IS are contained in the ambient ECF. Neurons may chemically intercommunicate by the mediation not only from the dozen-odd classical neurotransmitters, but also by peptides, hormonal factors, other specific proteins and many other types of IS, a term that is more generally applicable than neuroactive substances that was previously used."

-If you teach a human a memory task, you can demonstrate learning, but if you teach a human a memory task while under the influence of a psychoactive drug (i.e. thorazine, elavil), the person can learn the task. When the drug has been excreted, there is a temporary amnesia. In other words, it is as if the drug was a part of encoding the memory, and excretion of the drug produced a temporary amnesia.

-1. Neuronal networks are defined as activation of specifically localized areas of neurons by IS that reach them via diffusion through ECF. ECF makes up approximately 20% of brain volume. A 15 square millimeter neuronal network could be turned on or off by the presence of a specific IS. That is, the activity of the neuronal network would be state depedent on the presence or absence of the IS.

2. IS are contained in and trasmitted through the ambient ECF to surrounding brain cells where they can encode state dependent memory, learning, and behavior. 3. The molecular genetic basis of memory, learning, behavior (which is now called activity dependent neuromodulation) is then regulated by the IS.

-The traditional concepts of chemical transmission in the nervous system developed largely from detailed studies of the action of acetylcholine, etc. is the fast chemical signal used at the neuromuscular junction. Fast chemical signalling in which the neurotransmitter released at specialized synaptic junctions stimulates the opening of receptor control, ion channels in the post-synaptic cell within a millisecond time frame, and it does occur in the mammalian central nervous system. The amino acids glutamate and GABA (gamma amino butyric acid) may represent the principles of the fast signals used by most of the mainline fast conducting circuits. However, many chemical transmitters in the CNS do not operate in this classical manner. The action of monoamines and neuropeptides are slow acting, acting over a period of seconds or minutes, and rather than

direct, excitatory initiators of inhibition, they are more rapid in character. The slow modulators may not always be released at the morphologically specialized synapses, but sometimes continue to act further away from the site of release. This gave the concept of addressed chemical transmission where information is transmitted by the use of a wide variety of chemical signals acting diffusely, but selectively on the uneven distribution of receptors on the target cells to recognize these signals. Slow mediators act largely by triggering persistent metabolic responses in target areas rather than controlling ion channels. Chemically adressing the differences between IS and their receptors is a relatively slow pattern compared with the classical neurotransmission. There is now good evidence that the chemical addressing of the parasynaptic system is evolutionarily much older than the anatomical addressing of the central and peripheral nervous system in terms of synapses. What is lost in speed is compensated by the much wider bond of information that is mediated in this manner.

-Memory, learning, and behavior patterns are encoded by the IS in the neuronal networks. Example, can't remember something, but later in the day the thought appears. This is dependent upon the relative balance of neuromodulators that are present in the ECF.

-GJG states that thoughts are things. The brain contains steroid hormones that are the IS and perform a duplex action. They initiate a relatively fast (minutes) direct action on synaptic properties that regulate impulse traffic in particular neuronal circuits, and then a slow pattern (hours), like serotonin affecting sleep, a slow, indirect pattern, involving specific gene activation leading to the synthesis of essential proteins, specific receptors. Steroidal hormones regulate behavioral patterns involving reproduction, mood, territory defense, and other affective states. The steroid hormones illustrate the integrative control of both fast bioelectrical events involving the passage of impulses through the neuronal networks, through the classical appreciation of the nervous system, and the neurophysiological processes that underlie specific behavior patterns, and then the slow gene activated processes that lead to the synthesis of protein material which like specific receptors form the molecular substratum of behavioral pattern. The molecular genetic basis of memory, learning, and behavior are now called activity dependent neuromodulators which are regulated by IS.

-Rossi: "There is pretty good eveidence over the last forty years that psychopharmacologists have used the classical state dependent memory and learning experimental paradigms to ask us the psychological and behavioral effect of psychoactive drugs which we now know are mediated apparently by the informational substance receptor systems. The central significance of our working hypothesis is the fact that animal or human subjects are given memory learning tasks while under the influence of psychoactive drugs that either mimic or modulate IS receptor systems. There is a varying degree of amnesia (loss of learning) when the drug has been metabolized out of the system. That is, when memory learning is encoded under drug

conditions, it tends to become state dependent or state bound to that psychophysiological condition such as memory or learning behaviors become disassociated after the drug is metabolized. Readministering the drug reestablishes the original encoding condition and typically results in some gain in memory learning."

-This reversable amnesia is also typical of post-trauma stress syndromes and psychosomatic syndromes. These patterns are encoded in a state dependent manner by the stress released IS (ACTH, beta-endorphin, epinephrine) that is typical in what Selye describes as the General Adaptation Syndrome (GAS). Selye believed that just as a shock evokes such psychosomatic problems and another shock or heightened arousal level could sometimes heal them. The more recent approaches to the mind-body healing and self-hypnosis, such as the relaxation response that are in use, are proported to work by the reduction of the same stress related IS that encode psychosomatic problems.

-Validation of this approach in healing involves understanding the molecular, genetic, and IS receptor system dynamics for the mind-body illness. Survey of the information reveals extensive documentation of how illness can become manifest through the molecular, genetic, and IS receptor pathways. Reliable biochemical assay methods are available for rapidly assessing the molecular pathway that is disturbed in the illness. There are charts that relate the IS and psychoactive drugs that trigger ACTH, beta-endorphin, vasoactive intestinal peptides, etc., which are capable of encoding memory patterns. Presence or absence of illness is readily seen with patient symptoms, testing, etc., but how do we make the mind-body connection/healing?

-GJG relates a personal experience from the 60's where he slipped and almost fell. He experienced pain that made it difficult for him to walk. He was treated by his father, but still had the pain. While he was walking down the hall, he remembered how he almost fell, the pain and distress went away immediately.

-GJG relates the case of a patient of Dan Duffy's that had difficulty with gait where he would take a step with the right leg and fall backwards, then take a normal left step. With each step on the right leg, his body would go backwards. This is when GJG discovered reactive muscles. The right quadriceps and rectus abdominis were strong in the clear, but when the quadriceps were tested and then the rectus tested immediately after, there was weakening of the rectus. Treatment was to turn down the muscle spindle cell of the quadricep and this corrected the gait problem. Triano and Davis documented the concept of muscle reactivity.

-Common to see an old humeral fracture producing problems years after healing due to a reactive muscle pattern. Why did the body heal the fracture, but not take care of the reactive muscle problem? The body cannot heal what it is not aware of. Mental recall of the physical or emotional trauma has been used in the past (asking the patient to recall trauma weakens a muscle, requires treatment to the emotional NV).

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-If the emotional or physical trauma is out of the awareness of the body, the body cannot repair it.

-Correct all structural faults, especially make sure that they pass the Walking Gait Configuration and PLUS pattern, and make proper nutritional recommendations. See if walking makes any patterns return, if so, then check the spinal length lying, sitting, and standing. There are instances where the patient continues to have their symptom even after all corrections have been made.

-Concept that there is an encoding and retention of memory. Drug or nutritional measures to encourage memory, then wait for substance to be eliminated from the body, results in a temporary amnesia. The memory can be reinstituted by giving the substance again. The amnesia is reversible depending upon how you treat the encoding. In GJG's instance, suppose he took a step just at the time he remembered the injury and the encoding principles were at hand. (He had been earlier treated, getting the structure straightened out). The step taken was a corrective step as opposed to the opposite leg that may have kept the lesion active.

-Patient with seizures since 1.5 years of age, onset one month following a fall onto his head. AK treatment reduced seizures from 10 a day to 2 a day, occurring mainly in the morning (associated with blood sugar values and Then and Now Technique). In this patient, Then and Now was lung against small intestine, combined TL of the lung and small intestine alarm points was positive, but individually was negative. Associated points were checked for subluxation and was negative. An upper cervical fixation was found. GJG then asked the patient if he could remember the first seizure, but the patient was too young at the time of the first seizure, so GJG asked him if he could remember the last seizure. The patient spoke very slowly and moved about very slowly due to medication. The patient then remembered an earlier seizure and was describing it aloud (in the slow manner) while turning from supine to prone. GJG then corrected the upper cervical fixation, and he now assumes that the patient was still thinking of the seizure event. The patient was asked to then turn supine again following the correction and he turned very quickly and proceeded to speak describing the seizure event in very clear and normal rate diction. The patient had a restless night that night and only one convulsion. Afterwards there were no convulsions.

-Mental recall of the physical or emotional trauma during the appropriate structural correction literally helps the body to remember the event in past tense and therefore link the correction to the incident and the body clears the circuit effectively so that there is no further revival.

-Correct all spinal and dural problems. After correction, retest your indicator, should now be negative. I.e. upper cervical fixation, bilateral gluteus maximus. After correction of upper cervical fixation, the bilateral gluteus maximus will be strong. Now, have the patient think of the emotional or physical trauma that started all their troubles and the indicator will return, i.e. gluteus maximus

will now weaken again. Treatment is to correct the subluxation while the patient recalls the emotional or physical trauma.

-Diagnose and adjust the dural subluxations and fixations while the patient accurately remembers the primary emotional or physical trauma. Actively adjust the dural areas especially while the patient actively recalls the original or first remembered emotional or physical trauma.

-Holographically the adjustment of dural areas is a time date, object beam-reference beam technique, to effectively file emotional and physical trauma in the past tense. Past tense meaning tight, and where there is a tight one there is a weak one, past weak muscle patterns.

-What if the patient cannot remember the emotional or physical trauma from the past? Have the patient hold the emotional NV on the frontal bone during the adjustment (similar to Dr. Scott Walker's NeuroEmotional Technique, the use of the emotional NV for emotional recall).

-Remember that structure determines function. Don't place the triad of health on its point, structure is the base. Combine the emotional recall with the structural correction.

-Check the patient for emotional NV technique by having them think of a trauma and see if it weakens the bilateral pectoralis major clavicular. If positive, correct with emotional NV. Testing again should now be negative. The emotional recall may not be positive at all. Then check for structural problems. Occipital side-slip can be diagnosed with the lateral thrust of the tongue. Sometimes you need to add chin up or chin down to the tongue thrust in order to make it show due to the anteriority of the lateral side-slip. Challenge and correct, but use a low velocity technique for correction (cervical compaction technique) because you will have to make the correction several times, you don't want to traumatize the patient. Check active vs. passive range of motion with the head in flexion and extension. Laterally flex the patient's head with the head in extension or flexion (opposite that which was found positive) and press on the occiput from right to left or left to right, whatever way challenged, and then slight anterior or posterior directions. Retest for the tongue thrust with chin up and down, should be negative with correction. Have the patient recall the trauma mentally and then retest the tongue thrust, will now weaken when the emotional/physical trauma is remembered that is causing trouble in the patient. Correction must be made again while the patient mentally recalls the trauma. Then retest the indicator while the patient recalls the trauma, and it will now be negative.

-This has probably happened by accident in many patients, but is very random. Now you can diagnose the need for it.

-You still have to diagnose the need, supply the need, and observe the results, give the proper nutrition, provide all the proper structural corrections, but adding this emotional recall has very good results.

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** Material in this paper does <u>not</u> conform with the ICAK Status Statement