COLLECTED
PAPERS OF THE MEMBERS
OF THE
INTERNATIONAL COLLEGE OF APPLIED KINESIOLOGY

PRESENTED MAY 15th THROUGH MAY 17th, 1985

Dale Schusterman D.C.

SHELDON C. DEAL, N.D., D.C.
PAST CHAIRMAN I.C.A.K.

INTRODUCTION

by

Sheldon C. Deal, D.C.

Past Chairman

This nineteenth collection of papers by the members of the International College of Applied Kinesiology represents 50 papers written by 38 authors.

These papers will be presented by their authors to the general membership at the Summer meeting to be held in Santa Monica on May 15, 16, 17, 1985. The authors welcome comments and further ideas on their findings either in Santa Monica or you may write them directly as their addresses are included in the Table of Contents.

These papers do not represent the official educational material of the International College of Applied Kinesiology, but rather areas of special interest to the individual members which have been under research. The papers are presented in an unedited form.

The papers are being mailed out to the members well in advance of the Santa Monica meeting. This will allow the membership at large to read the papers in advance which will save time at the Summer meeting and hopefully stimulate more questions from the members and more demonstrations from the individual author.

We the members of I.C.A.K. can be proud of the amount of research being conducted and feel fortunate to have it at our fingertips in the form of these Collected Papers. It cannot help but be an asset to our health and also to the health of our patients.

*Diplomate

****TABLE OF CONTENTS****

Arm Length Discrepancies in Hyperabducted Position

G. E. Achilly, D.C.

Abstract:

As we normally measure leg length to help diagnose certain structural problems, an effort was made to determine why variations in hyperabducted arm length existed.

Leg length variations are due to several major reasons: posterior ilium, lateral atlas, lateral occiput, congenital short leg, fracture induced short leg, arthritic changes in the acetabular cavity, or anterior ilium on the opposite side.

I observed a certain percentage of patients who exhibited a structurally short arm. This was determined by having the patient hyperabduct both arms above the head with both palms facing each other. The extended finger length was determined to see if one arm was longer than it's opposite. The head, body, and legs were centered. The horizontal and vertical axes bisect the palms, bridge of the nose, and symphysis pubis.

It was reasoned that possibly there was a psoas major muscle involvement, weakness on long arm side, or hypertenacity on the short arm side.

The results observed, which are not that conclusive, reveals a small percentage had a hypertence psoas on one side. By using spindle cell to the belly of the psoas muscle. I was able to normalize arm length. A large percentage on the long arm side responded to neurolymphatic, even though T.S. line indication, muscle tests, and other mechanisms fail to reveal a weak psoas on that side.

Conclusion:

There is none. The incidences mentioned in the tests observed, results were obtained to balance arm length, but I do not know the reason why.

GLUTEUS MEDIUS

POSTERIOR DIVISION

Herbert C. Anderson, D.C.

It has been my experience of testing many athletes (500 or more) for pelvic problems, knee pain, etc., the correcting of the gluteus medius with-the 5 intervertebral foramen factors, the patient would test strong.

When the athlete participated in tennis, racquet ball, jogging, etc., the gluteus medius would again be weak.

In reading articles by Dr. Alan Beardall, where he divided the gluteus medius into middle, anterior and posterior divisions, we found, when testing the posterior division, this would be weak. Correction in the usual testing, with the foot turned lateral, weakness occurred. We used neurolymphatics with the foot lateral, pubes and L4, 5 area and also Beardall's 4th intercostal space right side and posterior, the results were longer lasting.

We had the patient walk in the office, toe-in toe-out, to stress the gluteus medius posterior division and test the patient standing. If any weaknesses were elicited, neurolymphatics were stimulated standing. If he was a tennis, squash or racquet ball player, we would have him in position of serving the ball with knees flexed following through a stroke of the ball and correct in the various structural distortions when playing.

GLUTEUS MEDIUS

POSTERIOR DIVISION page 2

If a baseball or football player, have him throw the ball and stimulate the neurolymphatic in that position.

We have experienced a number of violinists who stand and have complained of nagging low back problems. The gluteus medius was corrected while standing holding the violin under the chin or whenever they complained of pain.

We have found this to be of value when correcting any of the categories which persisted for a period of time.

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Dr. Alan Beardall -- Clinical Kinesiology
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Gray's Anatomy -- Englis Edition
Applied Kinesiology -- David Walther, D.C.
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Goodheart Book II -- George Goodheart, D.C.

PUBO-COCCYGEUS MUSCLE

THE DIAPHRAGM OF THE PELVIS

Herbert C. Anderson, D.C.

It has been our observation that many women, after bearing children, have weak gluteals, pyriformis, intra-abdominals, and, hence, have the problem of prolapse of the internal organs.

Many females who have weak bladder muscles and poor sphynctes control have been helped immensely.

The origin of the pubo-coccygeus in on the posterior surface of the pubes and the anterior portion of the arcus tendinous ileo-pectineal line on the inner aspect of the ishium. The insertion is in the central tendoness points of the perineus around the rectal sphincter. Correction of the pubo-coceygeus has greatly helped support the prolapse but also allieviated the dragged-down feeling they experienced after childbirth.

Enclosed is a photo of the tesing procedure with the permission of Dr. Alan Beardall.

South pole of a magnet to G.V. 20 has also helped a great deal.

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Dr. Alan Beardall -- personal conversation

TEST FOR PUBO COCCYGEUS MUSCLE

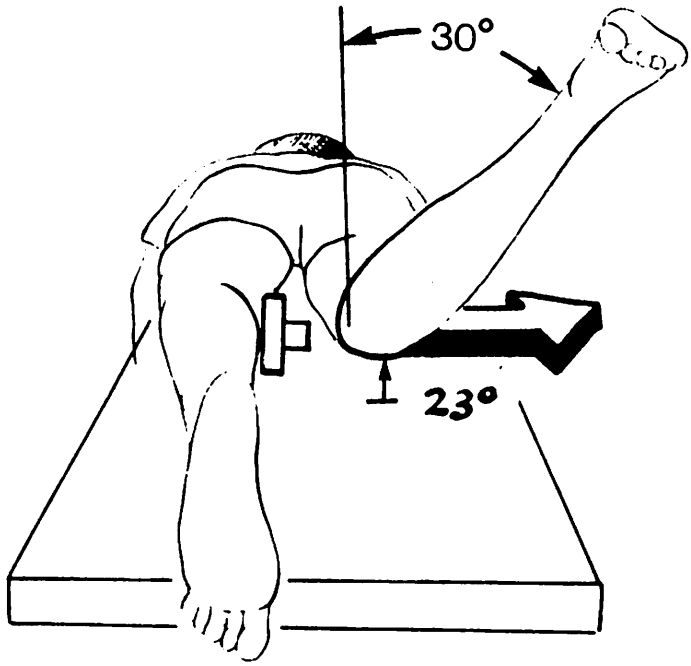

Photo - permission of Dr. Allan Beardall

DRUG WITHDRAWAL AND HYPOADRENIA

Kory Branham

ABSTRACT: A correlation between drug withdrawal and hypoadrenia is observed with suggestions for management.

The observations that led to the writing of this paper began when I was treating a husband and wife on the same day. The husband had recently suffered a spontaneously collapsed lung which was thought to have been provoked by cocaine abuse. The recent stresses caused by her husband's difficulties caused the wife to suffer acute hypoadrenic symptoms including fatigue, insomnia, and emotional instability along with the classical kinesiologic indicators of hypoadrenia. She responded well to basic A.K. therapy and nutritional support with vitamin C, B vitamins and whole adrenal substance. When I saw the husband he was experiencing exactly the same symptoms. He said that his symptoms began, not after his brief hospitalization, but shortly after when he tried to withdraw from his cocaine habit. He had been unsuccessful in his attempts to quit and wondered if I could help.

Having just seen the symptoms in his wife it was clear to me that many of his symptoms were due to hypoadrenia and not some other drug withdrawal related phenomenon. Subsequent nutritional treatment was very helpful in not only making him feel better but decreasing his dependance on cocaine. Shortly thereafter similar observations were made on several patients who were suffering similar symptoms from caffeine and tobacco withdrawal. Appropriate A.K. and nutritional therapy directed at the adrenal glands was helpful in resolving both symptomatic

patterns and cravings for the addictive substances.

The therapies involved and possible theories to explain these observations come from many sources. First, I refer to the observations by Hoffer and Osmond that certain byproducts of adrenal metabolism are hallucinogenic and depressive.[1] These findings have been corroborated and expanded on by many, including Dr. Goodheart.[2] The effects of these adrenal byproducts can be diminished by niacin and other nutrients to support normal adrenal function. Drs. Humphrey and Osmond found that schizophrenics frequently exhibited high blood and urinary levels of these adrenal byproducts (adrenolutin and adrenochromes). These patients benefitted from the same nutritional therapy. Many hallucinogenic drugs affect the same biochemical pathways that are disturbed in schizophrenia.[3] These hallucinogenic drugs along with other non-hallucinogenic drugs (caffeine, nicotine, etc.) are also known to stimulate adrenal secretions. Cheraskin and Ringsdorf have reported success in treating patients with caffeine and nicotine addictions by prescribing nutrients and diets to regulate the hypoglycemia caused by excess adrenal stimulation.[4] Dr. Alfred Libby, a California Chiropractor, has recently popularized a treatment for drug withdrawal that has as its major component megadoses of vitamin C. Dr. Libby started his work with heroin addicts and he quotes research showing that vitamin C blocks opiate receptors in the brainstem as the probable mechanism for alleviation of withdrawal symptoms.[5] I suggest that a further explanation is that vitamin C supports the adrenal gland.

My belief is that use of these substances not only depletes

adrenal reserves but inhibits the normal endocrine and neurologic feedback mechanisms for adrenal regulation. Withdrawal can then result in acute hypoadrenia.

My therapeutic approach has been to fix what I find in regards to adrenal function on a structural basis. In addition I find a need for one or all of the following nutrients which are usually given initially in meganutrient doses for a short period of time followed by much lower maintenance doses.

Vitamin C- One to five grams daily

Niacin- 500 to 2,000 mg. daily followed by Niacinamide-B6

Pantothenic Acid- 500 to 1,500 mg. daily followed by broad spectrum B vitamin supplementation

Adrenal glandular support- Initially whole adrenal extract in frequent doses to be followed by a lower potency glandular support (drenamin)

Tyrosine or phenylalanine- To support adrenergic pathways 500 to 2,000 mg. daily followed by a maintenance dose of 100 to 500 mg. daily

Choline- 175 to 1,000 mg. daily to support cholinergic pathways (Deaner, a choline analog has long been used for schizophrenics and autistic children)

Of course, none of the above supplements are given without appropriate testing and finding a need for them. It is interesting to note that many times vitamins B and C and adrenal glandulars along with the appropriate 5 factor treatment to the sternocleidomastoid and adrenal related muscles will totally abolish any weaknesses. These weaknesses will then be seen to recur with right or left brain activity. This weakness is then usually abolished with the appropriate precursor therapy, either

tyrosine or choline or both.

There are many other factors involved in drug abuse and withdrawal including allergies and psychological factors. Certainly all these factors should be investigated. I feel that the therapies and theories I have discussed can be helpful in understanding and treating this ubiquitous problem and I welcome any further ideas on the subject.

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LOWER THORACIC PAIN AND HYPOADRENIA

Kory Branham

ABSTRACT: A specific type of lower thoracic pain is characterized by rib head instability and quadratus lumborum weakness. The relationship of these symptoms to hypoadrenia is discussed along with suggestions for treatment.

A common type of problem I see in patients is a recurring pain in the middle or lower thoracic area. The onset is usually insidious and non-traumatic, but, occasionally may begin suddenly following a seemingly insignificant lifting stress. The pain is usually on only one side of the spine and ranges from a sharp, constant pain to a vague deep ache that is less localized. The source of the pain can usually be traced to a thoracic-rib subluxation complex. The side of pain usually has an anterior thoracic vertebra with a corresponding posterior rib head. The level of subluxation is most commonly from T-9 to T-12, but areas of pain and subluxation as high as T-4 have been seen to fit this category.

Credit must be given to Dr. Goodheart for helping me understand these problems. It was his observation that some lower thoracic pains were due to unstable ribs caused by a quadratus lumborum weakness that began my investigations. This observation led to the successful treatment of a few patients with these symptoms by appropriate adjustments and treatment of the quadratus lumborum reflexes. A series of subsequent failures led me to further investigation. These failures were characterized by an inability to achieve a lasting correction of the quadratus lum-

borum.

Walther describes the quadratus lumborum as having its "origin on the iliolumbar ligament and posterior part of the iliac crest". The insertion is into the "inferior border of the last rib and transverse processes of the upper four lumbar vertebrae". Its action is described as, "lateral flexion of the lumbar vertebral column. Depresses last rib. Helps action of diaphragm in inspiration." Nutrition is "vitamins A,C,E,", meridian relationship is "large intestine", and specific organ relationship is "appendix".[1]

My experience has been that the quadratus lumborum is also related to ileo-cecal valve function. This observation does not seem contradictory to the above quoted relationships as the appendix is certainly closely allied to the ileo-cecal valve.

I began to test my patients with these thoracic problems and found most of them to have an open ileo-cecal valve. When I added traditional open valve therapy to my treatment of these patients my level of success improved, but not greatly so. My failures were still characterized by recurrence of the quadratus lumborum weakness in addition to recurrence of the open ileo-cecal valve.

One last piece in the puzzle fell into place when I observed several patients with either severe depression or symptoms of schizophrenia. Most of these patients had marked hypoadrenia and open ileo-cecal valves. The interesting fact was that nutritional treatment for the adrenals usually resulted in marked improvement of the ileo-cecal valve function. I then applied this observation to the previous set of patients with the

thoracic pains and recurrent quadratus lumborum weaknesses. Most of these patients were found to have weak adrenal related muscles when challenged on a right or left brain basis. The factor that abolished the weakness was most often nutritional and the nutrients that most commonly were needed were whole dessicated adrenal extracts or the amino acid tyrosine. The addition of appropriate adrenal therapy was helpful in resolving not only the thoracic pain, but also the recurring open valves and quadratus lumborum weaknesses.

The type of nutrients needed suggested that these patients were in the exhaustion stage of adrenal stress. This may explain the connection between the adrenals and the ileo-cecal valve. The ileo-cecal valve, being an intestinal sphincter, is under control of the splanchnic nerves of the sympathetic nervous system.[2] The deficiency of epinephrine and nor-epinephrine in hypoadrenia results in a corresponding decrease in sympathetic tone. The sphincters will likewise lack tone and an open ileo-cecal valve can result. It is interesting to note that Rogoff's sign, one of the physical signs of hypoadrenia, is very similar to the presenting complaint that I describe at the beginning of this paper. Rogoff's sign is described as "definite tenderness at the lower rib junction with the erector spinae muscles".[3]

One other point worth noting may explain why painful sites as high as T-4 seem to respond to this treatment. Patients with symptoms here frequently have a neck extensor weakness that I have been able to abolish with appropriate adrenal therapy. I believe this occurs because the neck extensor weakness is not a true weakness, but an apparent one due to lack of stabilization

of the posterior rib cage by the quadratus lumborum. This is a corollary to apparent neck flexor weakness that responds to treatment of abdominal muscles to stabilize the anterior rib cage.

SUMMARY

I would like to conclude by summarizing the steps I take in correcting these difficult cases of thoracic pain.

1. Check for and correct anterior dorsals and posterior rib heads
2. Check for and correct quadratus lumborum weakness. If it recurs or doesn't strengthen with any of the 5 basic factors:
   a. Check for open ileo-cecal valve
   b. Check for adrenal involvement. This may only show with a right or left brain challenge and is usually abolished by tyrosine or whole adrenal extracts.

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[2]J. J. R. Macleod, Physiology of Modern Medicine (7th ed., C.V. Mosby Co., p. 491)

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THE AK CHALLENGE FOR CATEGORIES I, II, III AND TORK PATTERNS

JOHN W. BRIMHALL, B.A., D.C.

FEBRUARY 1985

ABSTRACT

If you ask a hundred doctors in AK how to therapy localize and or challenge for each of the categories or tork, I think you would get at least eighty different answers. In fact, I never heard anyone say how to challenge category III. So we developed a way where we can quickly screen by challenge or TL each one of the catergories and tork patterns as well as quick structural correction.

Therapy localization has been, I think, heaven sent. The challenge has been an equal blessing and I wonder how people practice without either of them. We have felt a great need for quick challenges and TL's for many structural categories and patterns that have been identified and treated. We have especially felt weak knowing when category III should be used other than symptoms and this was a missing link.

With the marriage of AK and SOT principles mixed in with a little Arizona medicine man insight, the following was discussed and utilized by Doctor Don Peterson, one of my associates and myself. We will not discuss that which is already known other than a brief review of some of the categories to build the new ideas.

As you know, category I is a sacroiliac boot plate subluxation. This is the synovial and moveable part of the sacroiliac articulation. The pelvis is actually fixed in the central position but the vertebral column can make compensations. It is TL'd by the right hand on the right sacroiliac posterior articulation and left hand on the left SI joint, same place. If a previously strong muscle goes weak we then put both hands on the left and then both hands on the right to determine which side is involved and then adjust the opposite side when the blocks are placed. But the way we test this now is by challenge rather than the time consuming TL.

We contact the PSS on one side and the ischium on the opposite or contralateral side and push in opposite directions meaning the posterior superior spine superiorward and the ischium anterior inferiorward and if it blows weak we know its at a category I.

Refer to figure IA and IIA.

An overview of our idea and procedure was to look how the blocks were placed in category I, II and III. The challenge comes in applying pressure in the plain line that the blocks are placed. If the sacroiliac joint is in lesion, it will blow weak and a TL has been accomplished. If the pelvis is properly aligned for that category, it will not blow weak when you challenge in plain line of the blocks. This is the same philosophy as any rebound challenge with a new twist. This idea, is really no different than if you press a vertebrae in the direction it needs corrected, it will rebound and show weakness. If you press contralateral hips in the direction that they need correction, they will show the rebound phenomenon and blow weak. If they are intact, no weakness is shown. Our challenge for category II was not as consistant as we wanted to be, so we rely on a two hand therapy localization while the patient is supine. We place the right hand on the right PSS and the left hand on ischium or vice versa. If it would blow weak in that configuration, we would then block the right PSS and the left ischium as a category II. Please refer to the illustrations Figure IB and IIB. We found some category II's were hidden if the usual one handed therapy we've all used was the only TL.

In accordance to my last years article, where we talked about two hand therapy localization to both the upper and lower hip boot (one at a time) as compared to the opposite shoulder would show us a tork pattern. This still holds true. If we rule out by TL or challenge category I, II and III, we automatically then check with two hand therapy localization to the shoulder and both the upper and lower boot of the hip on both sides. What can happen, is you have category II and still have a torked pattern. After we place the blocks for the category II in the supine position, we have them TL first one shoulder and then the other. If a strong muscle goes weak, we put a block under that shoulder as well. This clears the torking pattern at the same time you clear the category. We find ninety percent of the time, there is an upper cervical subluxation around C-2. This is cleared while they are on the blocks. Any extremity, cranial or cervical subluxation is adjusted while they are on the blocks. We find longer lasting results and much easier adjusting. In both categories I and III, we always check for fixations while they are on the blocks.

Category III has been the hardest to test from an AK procedures as far as I am concerned. Our challenge is simply to press in the plain line of the blocks as afore mentioned. You press for example the right posterior superior spine in an superior direction at the same time you push the left ischium in a lateral to medial direction. This exaggerates the tork pattern the pelvis is into; remembering the lumbar disc, nucleus or subluxation category in one or more of the lumbar foraminal is causing occulsion. Multiple compensatory spinal subluxation and fixations can result with sciatia, etc. As mentioned, we always check for upper cervical fixation while on the blocks. If positive orthopedic findings for a disc also occurred, we may block while the patient is on the Leander or Cox traction table. We do not strap the feet in but do have the table in traction motion.

If you are inclined to use the activators in a typical nine point adjustment you can do this while on the blocks for either category I or III. A drop pelvic piece may also be used while the patient is on the blocks if so inclined.

In the category III, we challenge both sides by pressing the PSS one side and the ischium on the other and then reversing that in the direction we mentioned. Regardless of which side was weak, we block according to the short leg side like SOT teaches. Again refer to the illustrations IC and IIC.

It is good that we have to write articles occasionally, since it reminds us not to complain about others ability to reduce things to writing.

Another observation is that there can be more than one category that has to be cleared on the same visit. We have become much more aware, since employing this technique, how many category III's we have been missing. It helped us with some of those difficult low back cases. A category II, a torque or a category I may surface for correction and must be challenged after correcting a category III. I hope it doesn't have to be said that we are doing all other standard AK procedures from imbrication, five finger concept, TMJ, etc., as we find it. Nothing has changed there, but only a systematic method for screening and treating these structural categories and torking patterns.

SUMMARY

I would hope that this paper will not be forgotten but we would literally put this to the test in the ICAK and see if it doesn't stand the test of time. We have used these techniques for one year and hundreds of cases. We find them to be valid and very helpful. Literally in one to two minutes you can test for all the categories and tork patterns that exist. Then correction can quickly follow and be confirmed by retesting. An interesting thing that we found, was that sometimes a category II would not show by just testing for it by unilateral TL. But by doing two handed TL, it would show a hidden category. Plus it shows you where to place the blocks by where it TL's. Remember the category II is not any fixed position but has suffered a loss of the weight bearing support of the SI joint. Dr. DeJarnette has reminded us for years this causes myological imbalance and vestibular disturbance.

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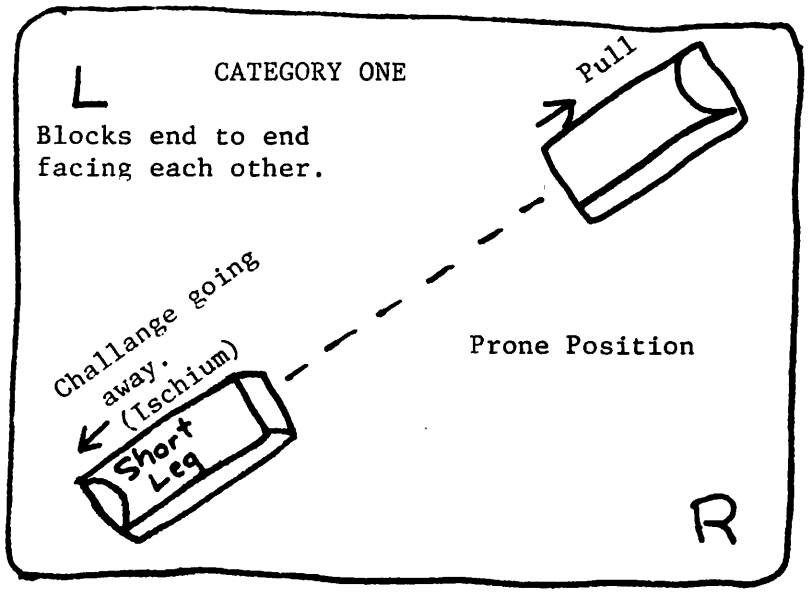
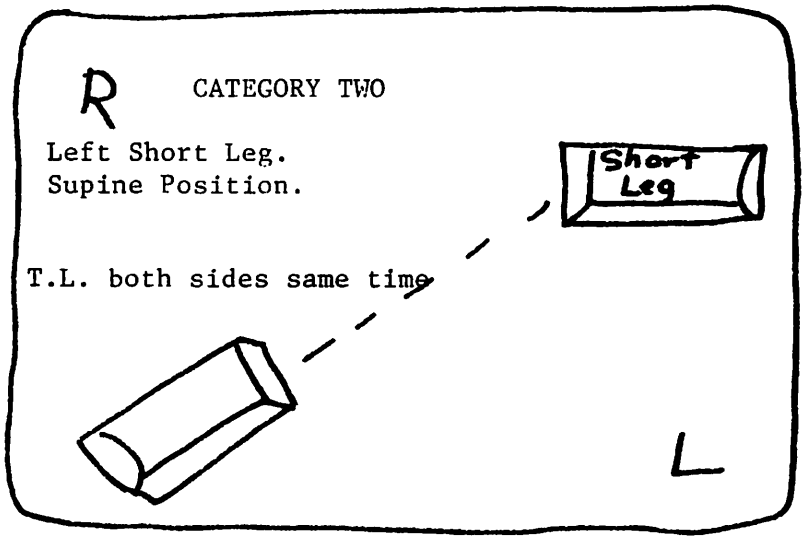
DeJarnette, Bernard Major, D.C. Sacro Occipital Technic, 1977, Nebraska City, Nebraska, U.S.A.

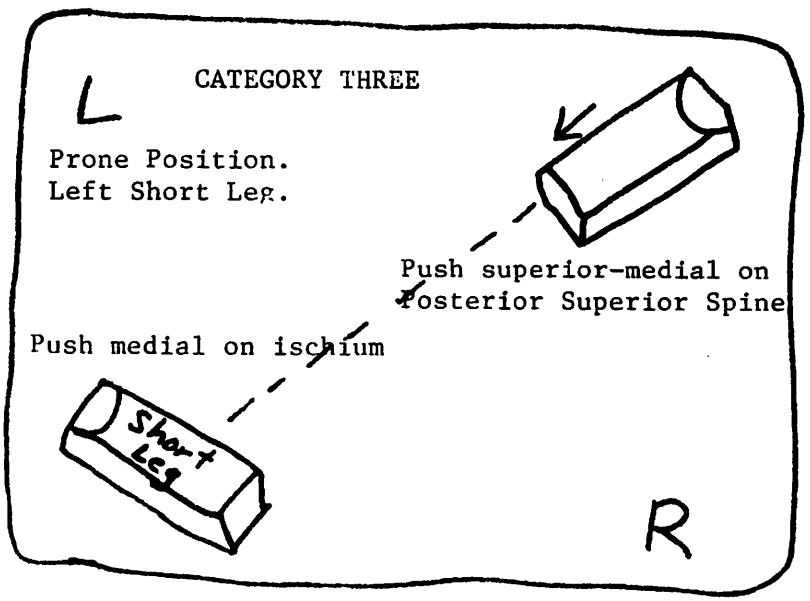
Figure I



Blocks face each other. They are placed on the area that two hand T.L.



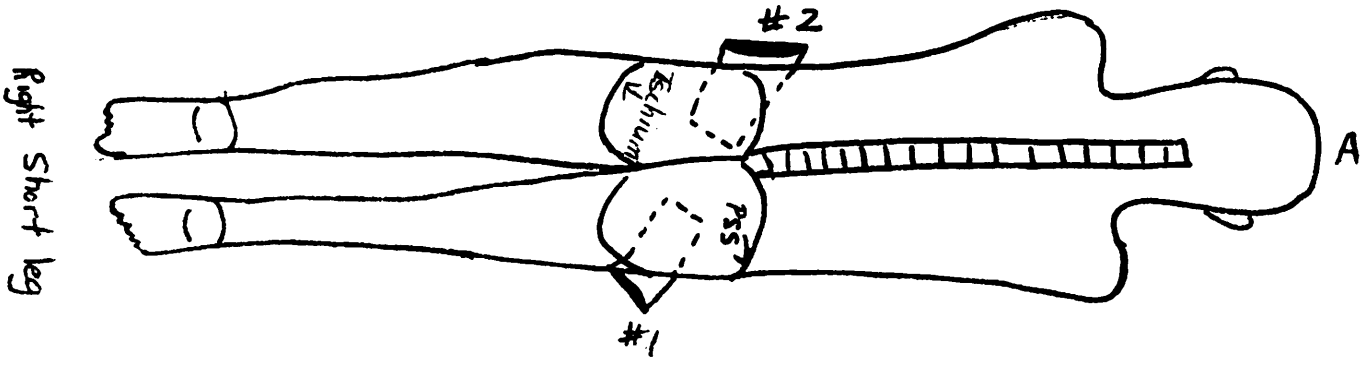
Block on short leg is horizontal with the lower half of the block under crest of iliums. Balance of block supports the back muscles. The block on the long leg side faces horizontal block on short leg side.



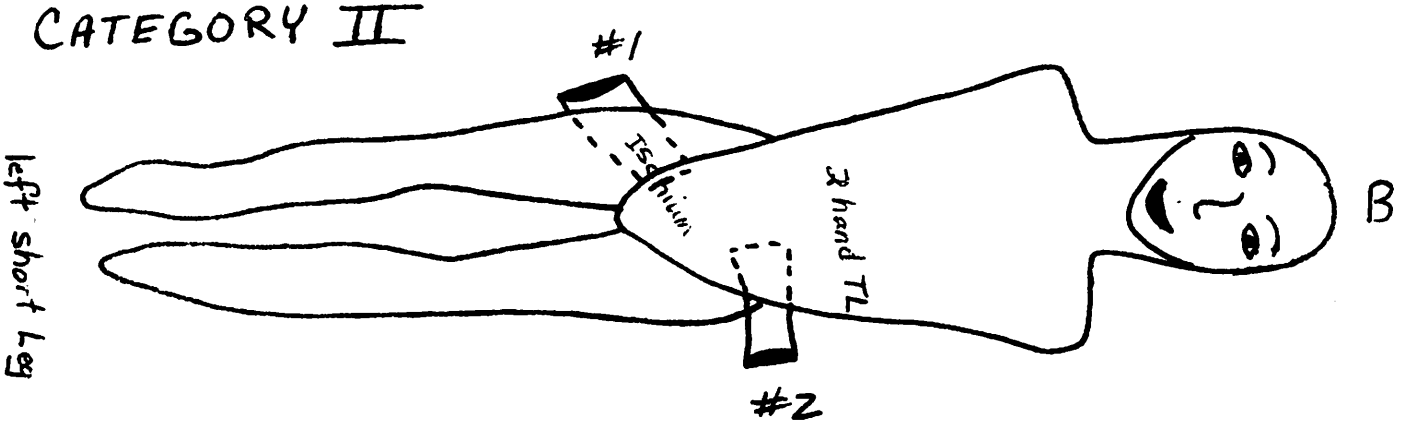
Block 1 is under the pelvis at the acetabulum. Block 2 is under the anterior iliac spine and faces the center of block 1.

Figure II

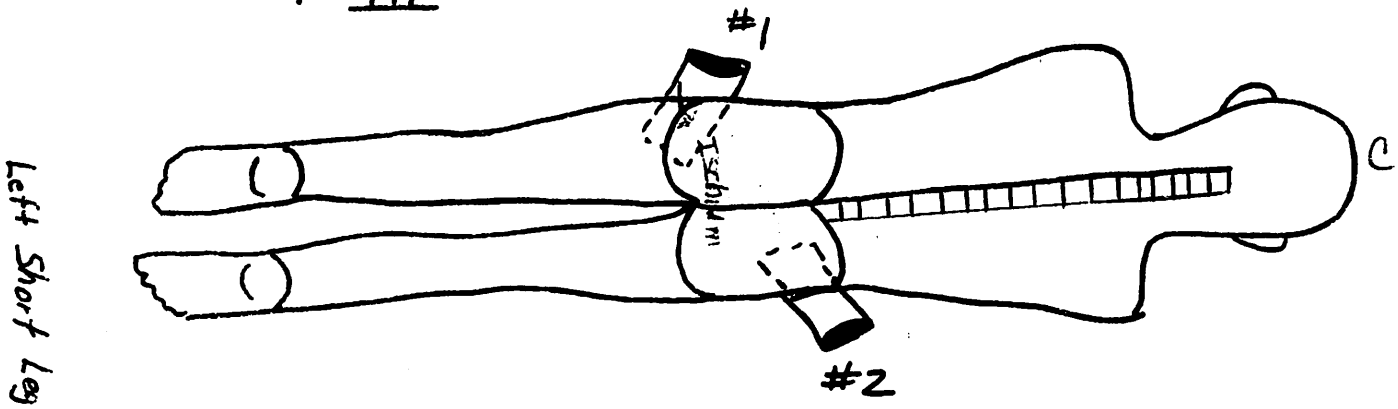
CATEGORY I



CATEGORY II



CATEGORY III



STRAIN COUNTERSTRAIN: A MORE EFFECTIVE APPROACH?

Patrick M. Casey, B.S., D.C.

Abstract: An updated more effective procedure for the treatment of strain counterstrain injuries.

Dr. Goodheart has talked extensively about the strain counterstrain technique developed by Jones and updated by himself. I first saw it demonstrated at the spring convention of the Colorado Chiropractic Association in May of 1984. At that time I thought it was a great addition to my arsenal. Upon returning to my practice I found it worked "alright" but never to the potential Dr. Goodheart had spoken of or to the potential I felt it should have. I was never satisfied with the results and so let the procedure slide and almost quit using it.

About two months ago I had a patient come in who showed a need for the technique and try as I might doing what Dr. Goodheart had demonstrated I ould not get the muscle to release. This patient was a very stocky male who I could not both hold his head and spread the muscle belly as I had been shown. So I thought since this man was stocky I should use an assistant to hold his head while I used both hands to work the muscle. I was totally amazed at the results. The muscle responded and relaxed in about 20 seconds. With the next couple of patients that needed strain counterstrain I returned to the original technique with "airight" results again. Then I had another patient who was resistant to the original technique who also responded very well to the "new" technique.

I have found that using two people gives me greater control over both the body position and over the position or line of drive of my hands in the belly of the muscle. Just a fraction of an inch difference in the belly of the muscle or a degree or two change in the direction the head is turned does make a big difference. A great deal of attention needs to be paid to these particular points for the best results.

During a two month period I used the strain counterstrain technique sixty-seven (67) times. Thirty-one (31) times I have used the original technique where I did both the flexion or extension of the body and the muscle work. The time it took to release varied from twenty-five (25) to fifty (50) seconds with an average of thirty-nine (39) seconds.

The other thirty-six (36) times I used an assistant to move and hold the body while I used two hand or fingers to work on the muscle. The time varied from fifteen (15) seconds to forty-five (45) seconds with an average of twenty-three (23) seconds. In three cases of using the new procedure, one upper Trapezius, one Levator Scapulae, one Gluteus Maximus, pain was cleared from the area that had been there for at least one year. In the case of the upper Trap the patient had had pain in the area for over eight years, ever since he had dislocated his shoulder.

The most significant finding of all this was, of those who had the old technique seven (7) had to have it done twice for it to completely clear but only two (2) had to have the new technique done over to stay clear.

I have stopped doing the procedure with out an assistant. The new procedure is quicker to do and I feel is at least twice as effective. I would very much like to hear from anyone else as to their finding if they try the new procedure.

Patrick M. Casey, D.C.

Colorado Springs, CO

January 1985

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AN ALTERNATE ORDER OF MUSCLE TESTING USING THE MUSCLE TESTS OF CLINICAL KINESIOLOGY

Katharine M. Conable, D.C.

ABSTRACT: An alternative to the order of muscle testing given in Clinical Kinesiology is described, grouped by the major muscles tests of Applied Kinesiology.

The detailed muscle testing presented by Alan Beardall in his series of Clinical Kinesiology books is very useful, even if employed alone and without any of his later refinements of clearing and hand modes. Anyone dealing with musculo-skeletal problems and athletic injuries will find these detailed tests and the accompanying reflexes invaluable in isolating the exact section of a muscle which is dysfunctional and in achieving a lasting correction.

The Clinical Kinesiology texts are set up in an order grouping similar tests together. This is a logical sequence, but not always the most efficient for the doctor. Most of us come from an Applied Kinesiology background, and are familiar with the more general muscle tests of A.K. Our thinking about patients is organized in patterns of these muscles. We often do postural examinations, muscle testing scans or T.S. Line evaluations which lead to a single major muscle or group of muscles (for instance the Hamstrings or the Quadriceps) which may be compromised. Often it is useful to check it with the Kendall and Kendall test, and then evaluate this muscle in greater detail.

At one visit we may want to test only the 11 sections of the Quadriceps listed in Clinical Kinesiology, Vol. II. These tests are found between pages 39 and 85 of the book, with other muscles having similar tests interspersed. This makes it a bit hard to find each section of the Quadriceps without considerable flipping back and forth of pages. Since there are separate NLs, NVs, innervation levels, etc. for each section of

each muscle, it is impractical to commit all the reflexes to memory. Hence, the doctor will want to refer to the charts routinely while working with these muscles, even when the tests themselves are memorized.

To facilitate testing in my own practice I have developed groupings of the detailed Clinical Kinesiology tests which are organized in my mind by the major muscle or muscles being tested, rather than by the similarity of tests. I learned the tests by these groupings and found it helpful to approach the detailed tests as variants on the basic A.K. tests, rather than as one long, overwhelming list of new tests. This is relatively easy, as it builds on an already-established organization in my thinking about muscles.

<u>Volume I</u> comprises the following groups:

Volume III is basically arranged in good testing order - first muscles of the mouth and TMJ, then the neck, then the hyoid. Only the last two muscles - Longus Capitus and Semispinalis Capitus belong in a different order, with the muscles of the neck:

TMJ Group:	Orbicularis Oris	p. 17
	through	
	Pterygoid Externus, lower div.	p. 37
Neck Group:	Upper Trapezius, scapular	p. 39
	through	
	Platysma, post. div.	p. 55
	Longus Capitus	p. 75
	Semispinalis Capitus	p. 77
Hyoid group:	Digastric, Anterior Belly	p. 57
	through	
	Omohyoid	p. 73

I note with pleasure that the recent Volume IV , muscles of the upper extremity, is organized more on the lines I describe. To test and treat an entire group thoroughly usually involves simply paging through the book at the appropriate place, in order.

Obviously, if one plans to test all of the muscle divisions in a book in one session, this is not the most efficient order. However, if your treatment plan involves less than total testing at one time, this pattern works well. In the case of acute injury, the obviously injured muscle group can be tested and treated separately and quickly. For chronic problems, the doctor can systematically work through a single group of muscles each session, correct those found non-intact, and still be within the time allotted for a normal office visit.

I hope this organization of will help Applied Kinesiologists learn and use these excellent muscle tests easily and efficiently.

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page 1.

THE MULTIPLE AETIOLOGIES OF NEUROLOGICAL DISORGANISATION.

by

Richard L. Cook, D.C.

ABSTRACT: This paper is an attempt to show whether the subjective factors commonly present in the problem of neurological disorganisation (switching) are reliable indicators of its actual demonstrable presence, by the usual methodology of applied kinesiology (A.K.).

INTRODUCTION:

Everyone involved with A.K. is familiar with the problem of neurological disorganisation (switching)[1]. This occurs in approximately 10% of the population at large, and probably in a higher proportion in the average clinic of the chiropractor.[2] There are now a number of different ways to detect the presence of switching and it would be interesting to discover which methods are the most useful and reliable.[3]

Switching, in my opinion, covers a broad spectrum of disorders - from the severely disabled to those who are quite unaware of its manifestations. There are, furthermore, differing types of switching.... left - right, anterior - posterior,[4] etc. Thus it would be of value to have some sequence of monitoring switching in the clinical setting.

page 4.

METHODOLOGY:

For the purpose of simplicity a questionnaire was devised that could be easily filled in by the patient and be used to correlate the findings into an ordered sequence. Statistical data could be subsequently derived, concerning the incidence of the various types of switching and the reliability of the tests. This may well be of future value as a way to save time for the busy practitioner.

The use of a questionnaire format for collecting information is by no means new but, I have organised this particular form utilising the commonest subjective findings the switched individual appears to exhibit. As it is straightforward to extract the relevant information, this form has an advantageous set up.

The questionnaire is to be presented to all the new patients arriving at our clinic (which may or may not be a typical example) regardless of their symptomatology. The patient filling in the first part of the form, then it would be possible to test for switching using part two of the form not having seen the first part, to eliminate bias. (This is by way of being a retrospective study rather than a true double blind trial.)

page 3.

THE QUESTIONNAIRE DESIGN:

The pilot questionnaire was carefully thought out so it would be straightforward for patients to answer and easy to select data at a glance. The form was divided into 2 parts, the former for the patient to complete and the latter for the Doctor to fill in. Part 1 was further subdivided into 3 sections for convenience. There are always more questions that could be asked but, a line had to be drawn so as to keep a balance between being unwieldy, and deriving sufficient information. The questions were a mixture of standard medical questions, some from personal observations and others from a variety of ICAK sources.[5,6,7,8,9,10.]

The first part of the questionnaire was primarily a graded response format. With the answers subdivided into categories of positive to negative replies, this was deliberate to avoid the patient's natural embarrassment clouding the issue, and answering negatively to some questions. Numbers from 5 to 0 were assigned in a decreasing factor of positivity:-

5 - Always

4 - Often

3 - Sometimes

2 - Seldom

1 - Never

0 - Don't know

The question layout in Part I was subdivided into 3 major categories:- (see Appendix C.)

1. Speech, reading and numeracy (SRN).
2. Specific learning difficulties (SLD).
3. General physical and childhood problems (GPC).

These subdivisions are mainly for convenience, and to try and elucidate any differences in the type of switching present with the symptoms exhibited.

In the first instance 500 questionnaires will be filled in and the results collated. This will inevitably take some time and the full results will be presented at a later date when all the relevant data is amassed. However, in the meantime,it would be of interest to review some of the facts derived from the latest research.....

GENERAL DISCUSSION:

It is current believed that the process of memory is molecular.[11] In the synaptic clefts between the neurones of the brain, as well as the chemical transmitter substances, there are the so called 'memory molecules'. These chemicals apparently allow the fascilitation of the nervous impulse thereby setting up a link in the chain of events we term memory.

How memory is laid down is still an unravelled mystery but, learning can only occur by either making new

connections or by shutting existing ones down within the brain.[12] These new molecules which have been located may be how the connections are made or broken. It can further be postulated that those individuals who are switched are lacking in some of the memory molecules - which is rather suggestive of a possible biochemical disorder. This too would explain the heredited frequence of the problem, as well as showing how cross-patterning exercises may stimulate the brain to open the correct channels for more ordered responses. It furthermore sets the ball rolling for discovering other ways of determining the presence of switching and for making corrections.

One can readily appreciate that nothing is ever as simple as it would first appear! The phenomenon of 'molecular memory' may give an insight into the multi-factorial aetiology behind switching. There are, as I see it, many factors which either cause the nervous system to run into trouble or promote the continuation of switching. This list is by no means exhaustive but, serves to demonstrate what a wide range of causes can bring about the effect of neurological disorganisation:-

SWITCHING- A LIST OF PREDISPOSING FACTORS

A. Inter-uterine/neo-natal -

page 6.

1. birth trauma → forceps delivery, breech birth, anoxia, brain damage, cranial faults, metabolic/ hormonal problems, schizophrenia.
2. left-handed tendancy.
3. poor brain stimulation - unilaterally deaf/blind.
4. injury to dominant limb or eye -
5. congenital factors - gait/ foot disorders.

B. Childhood -

1. thumb sucking -
2. impaired motor skills -
3. failure to crawl sufficiently -
4. change in hand dominance -
5. restriction of movement - tight clothing, playpens.
6. forced development - early walking, skipping developmental stages, competitive athletics.

C. Later causes -

1. injury -
2. psychological factors -
3. emotional tension -
4. illness - acute or chronic.
5. blood chemistry imbalance - blood sugar.
6. developmental disease - metabolic/hormonal.

page 7.

7. body imbalance - gait, pelvic categories, TMJ, hyoid.
8. stroke - CVA.
9. bacterial/viral invasions -
10. toxicity -

This table admirably illustrates the old chiropractic maxim of "Anything can cause anything" and may provoke the addendum of 'and most of them result in switching!'

There are many eminent individuals around the globe who have become acquainted with switching and call it anything from 'minimal brain disfunction' to 'just plain clumsy'. However, I believe, we as applied kinesiologists can be at the forefront of this expanding body of knowledge, and through our expertise provide much in the way of research and a readily available solution to the misery that switching undoubtedly causes, often without recognition.

DISCUSSION ON PART II OF THE QUESTIONNAIRE:

There are least 5 subdivisions of switching:-

(1) Electromagnetic - polarity differences, ionisation, etc.
(2) Motor - coordination, clumsiness, etc.
(3) Eye/hand - ocular lock, perceptual problems, etc.
(4) Centering - hyoid, cloacal, gait, erroneous feedback.
(5) Schizophrenia - extreme personality disturbances.*

page 8.

As with any test no one in isolation is pathognomonic, only suggestive. We must endeavour to be methodical and avoid preconceptions. This is why I have tried to correlate all the known procedures for the detection of switching and place them in a logical sequence... (left-brain activity?).

Cross-reference of the Doctor's findings to the positive patient responses will then give an evaluation of the presence of switching, and perhaps some insight into its severity. (See Appendix A).

* New evidence is emerging to suggest a biochemical disorder may be involved in schizophrenia.[13] This also relates well to the concept of the molecular memory module and gives some credence to the phenomenon of chiropractic techniques being able to alter the blood chemistry.[14]

THE CONCEPT OF FACEDNESS:

Left or right handedness are usually fairly obviously dominant in most individuals but, 'facedness' (which according to studies is genetically based) is a more reliable guide to brain hemisphere dominance.[15]

Right-faced individuals are more common than left-faced, between 66% and 90% depending on race. The left-faced people tend to be musically talented and artistic, which is right brain activity; those who

page 9.

are right-faced are more linguistic. The idea came about after studying computorised jaw, lip and tongue movements whilst talking. It was noted by Professor K.V. Smith of the University of Wisconsin that most people have one side of the face more active than the other. Observable signs include: - in right-faced persons the right side of the face is less compressed between the jaw and brow, the right eyebrow is higher and any dimples or wrinkles are less marked.
(Hence the old notion of the left half of the face being the more expressive - according to legend.)

The idea of facedness suggests new approaches to the study of all aspects of cerebral dominance and its relation to handedness, speech disabilities, dyslexia and perceptual disorders. To this end I have devised a grid for easy measurement of the facial characteristics, also one could use composite L/L or R/R halves of a face photogragh or observation.

The grid comprises a perspex sheet which one can place in front of the face, on this is superimposed a diamond shape corresponding to the outer borders of the skull then lines are drawn through the eyebrows, lowest portion of the orbit and through the centre of the mouth. Eight measurements are taken in all and analysed to find which side of the person's face is the dominant. (See Appendix B.)

page 10.

CAN CONCLUSIONS BE DRAWN ?

One initial impression is that there is far more to switching than is at first apparent. The subject is most complex and there is still a great deal more to be unravelled. The problem may arise from any aberrant imput from the receptors that relay information from the outside world to the brain - be they mechanical, visual, electromagnetic or even 'negative thought patterns'.[16]

Occasionally, the problem may be masked and TL to K27 does not show positive when testing a strong indicator muscle, and often this will be apparent using dynamic evaluation of K27, ie. in various positions or walking mode rather than the standard supine test position.

Also Professor Peter Behan of Glasgow University, Scotland has drawn attention to the close association of left-handedness (or having a left-handed mother!) with epilepsy, congenital heart disease, severe migraine, allergic disorders, dyslexia, childhood stuttering, the hyperkinetic syndrome and autism.[17] These statistics, together with anatomical differences noted in the brain of dyslexic patients, it would seem to suggest that at least some, if not all, of the children who have been diagnosed as having dyslexia, autism and hyperkinetic syndrome are suffering from a neurological disorder and not psychological damage.

Dr. Behan has demonstrated that these diseases which

page 11.

predominantly affect the male, are related to testosterone levels in intra-uterine life, possibly through this hormone's effect on the thymus gland.

There are some advantages gained in being left-handed - namely being athletic, artistic and mathematical. Good mathematicians it seems are made in the womb. Research suggests that the foetal brain is affected by testosterone[18] which, according to the late Dr. Norman Geschwind of the Harvard Medical School as a counter-balance to mathematical ability, produces a tendancy to left-handedness, dyslexia and allergic diseases such as asthma.

All boys are exposed to quite high concentrations of testosterone in the womb, and most develop normally, ie. not much better or worse than average. But, according to Geschwind, a few are so affected by the hormone - either because there is more of it, or because they are unusually sensitive that the right half of their brain becomes dominant.

It has already been observed that very few girls were highly gifted in mathematics, and the researchers re-checked their talented children for other signs of 'right brain dominance'. These gifted children are found in a frequency of 1:10,000 - but, 20% are left-handed, twice the national average; and five times the usual number suffer with problems of their immune systems - which leads to allergies. So they tend to pay for their precocious ability.

page 12.

More recent studies from Europe and America are examining genetic factors which may shed light on a possible biochemical explanation for schizophrenia.[19] The symptoms include confused thinking, disturbed perception such as hearing voices, and a preoccupation with illogical ideas and fantasies - is this indicative of right brain dominance?

One interesting finding is connected with circumstances of children whose parents were not considered mentally ill. What characterised their parents was that they habitually gave children confusing and negative messages - this to me is highly suggestive of switching! This disordered communication was a strong predictor of a group of children who later developed schizophrenia. 18.5% of the children who were monitored over a long period developed the illness against a parental background of that type! A child of a schizophrenic parent is now thought to be six times more at risk than the normal to succumb to the disorder.

So, it would appear that the medical fraternity have stumbled across switching without really being aware of just what they have found. They have the money and the means for detailed brain research; we have the ability to spot and correct the problem but without a full comprehension of what we are actually doing. Now would be the time for closer cooperation and a more intimate liaison between all the professions concerned.

page 13.

CLOSING REMARKS:

Most people will admit that no paper is ever complete, and if this modest offering stimulates more questions than answers then so much the better. Knowledge can only come with an interchange of concepts, ideas and hypotheses, the proof will follow to show whether one is right or not. I sincerely hope that my endeavour will not go to waste but, trigger off some debate and I would be grateful to receive any criticisms, corrections and topics for future study. Moreover, I do fervently believe that we are fast approaching the time when a well-ordered view of the wondrous thing we call the human brain will be revealed, just as soon as we are able to chart the byways of the mind.

page 14.

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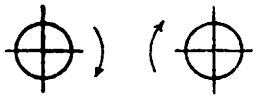
APPENDIX A.

Switching Questionnaire – Part 2

Which is your dominant (most preferred)	R	L	both	unsure	test
075 Hand					
076 Eye					
077 Foot					
078 Ear					
079 Face Appraisal					

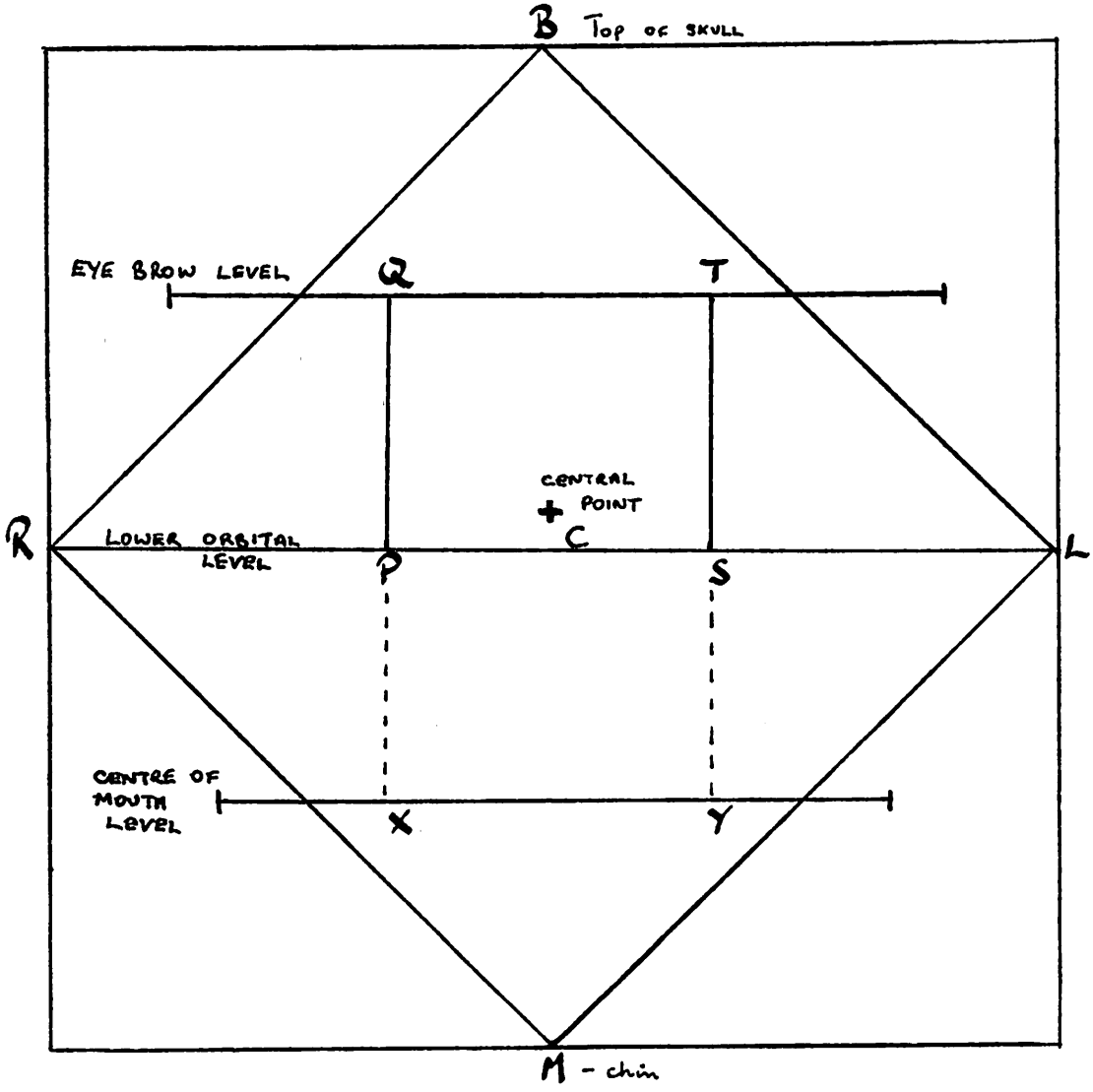
Tests	Tick Box (+)	Tick Box (–)
080 Gen Electromag Screen (5 fingers on torso)		
081 Bilat TL TO K27		
082 Swop Hand Test		
083 Hand TL Umbilicus		
084 CV – GV		
085 AUX K27 – TII		
086 Crossed K27		

	(+)	R	(–)	L
087 SP21 TL Head turn to negate				
088 WIM –– S with appropriate x crawl Homolat				
089 Ionisation LCD (+) RKU (-)				
090 X crawl indication			Homolat	

Ocular Lock	(+)	(–)		(+)	(–)
			095 Centering		
			096 Hyoid		
			097 Cloacal		
			098 Gait		
091 Bladder			099 Gen unilat weak		
092 Nasal Tap			100 Cranial		
093 Shock Absorber			101 TMJ		
094 Psoas Turn In			102 CATS		

page 16.

APPENDIX B.



Measurements:-

1. C to R.
2. C to 1.
3. C to B.
4. C to M.
5. P to Q.
6. S to T.
7. P to X.
8. S to Y.

APPENDIX C. (i).

LOWLANDS CHIROPRACTIC CLINIC
HARROW ENGLAND

NAME	NO.
D.O.B.	DATE:

SWITCHING QUESTIONNAIRE – PART I

		0	1	2	3	4	5
001	Is there a left-handed tendancy in your family						
002	Do you have poor concentration						
003	Do you have difficulty judging distance						
004	Do you have a poor memory						
005	Is your attention span short						
006	Do you have difficulty keeping track of time						
007	Are you forgetful						
008	Can you identify where sound comes from						
009	Do you confuse left and right						
010	Have either of your parents had learning problems or hyperactivity						
011	Are you by nature punctual						
012	Do you have to ask people to repeat or explain because you are not sure what they said						
013	Do directional signs confuse you						
014	Do you confuse plus and multiplication signs or minus and division signs						
015	Can you do intricate functions with one hand as easily as with the other						
016	Do you have occasional short span memory						
017	Have any of your brothers/sisters had learning problems or hyperactivity						
018	Do you get lost when trying to follow directions to a location						
019	Do you sometimes say just exactly the opposite of what you wanted to say						
020	Do you ever go to do something and when you get there you've forgotten what you went to do						

APPENDIX C. (ii).

		0	1	2	3	4	5
021	Have you suffered difficulty with any aspect of learning						
022	Do you tire easily while attempting to read						
023	Do you ever have double vision						
024	Do you lose you place on the page often						
025	Do you read or talk with a degree of hesitation						
026	Have you ever or do you now have any speech impediment, stammer, stutter or hesitancy						
027	Have you suffered any behavioural problems						
028	Do you tend to write or print backward or reverse your letters						
029	Do you find you must go back over a word, sentence or phrase to get a meaning out of it						
030	Is you handwriting and/or spelling poor						
031	Do you see words or letters transposed or backwards						
032	Is your reading ability below your mathematical ability						
033	Do you have difficulty adding numbers or working out change						
034	Do you find difficulty doing jigsaws						

Birth and Assoc. Problems

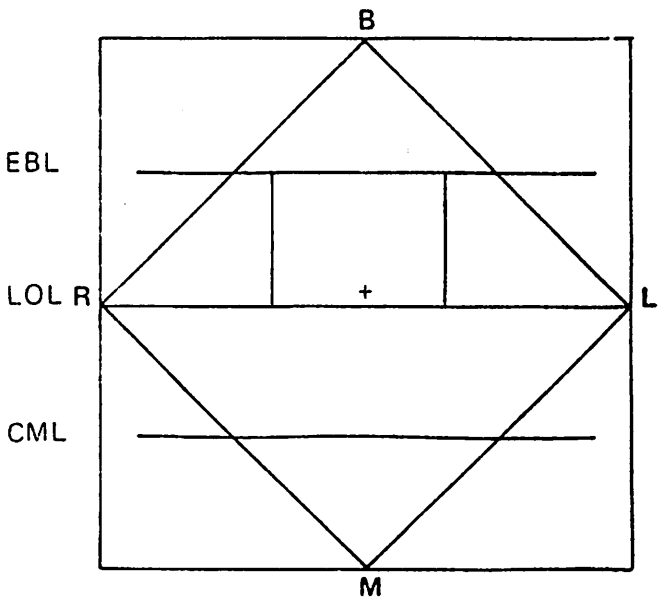
		Yes	No	Don't Know
035	Were you – Premature			
036	~~[illegible]~~ Low birth weight			
037	Forceps			
038	Breech			
039	Caesarian			
040	Breast fed			
041	Bottle fed			
042	Have you had any serious injury (fall, fracture) or emotional shock as a child			
043	Details –			
044	Any other illness / conditions			
045	Did you suck your thumb as a child			
046	If so which Left ☐ Right ☐ Don't Know ☐			
047	Were you an early walker			

APPENDIX C. (iii).

	0	1	2	3	4	5
048 Is your walking uneven or disorganised						
049 Are you good at sports						
050 Is your posture good						
051 Do you knock things over						
052 Do you have most of your problems on one side						
053 Do you suffer pains that shift around your body						
054 Did you crawl as a child						
055 Do you have difficulty doing jigsaws						
056 Do you have disturbances of sleep						
057 Do you tend to run into things even though you try to avoid them						
058 Is your balance good						
059 Do you get sea sick, car sick						
060 Are you blind/impaired vision in one eye						
061 Are you deaf/hard of hearing in one ear						
062 Do you develop any kind of problem when you run						
063 Do you have poor co-ordination ability						
064 Do you have difficulty with tasks such as tying shoelaces, threading needles etc.						
065 Do you have difficulty with ball games						
066 Are you a messy eater						

Face Appraisal

067
068
069
070
071
072
073
074



" The Left Anterior or Right Posterior Phenomena of Muscle Weaknesses "

By Dr. Elmer J. Cousineau, D.C.

Abstract:

The correction of extremity subluxations produced the phenomena of muscle weaknesses being switched from one side of the body to the corresponding muscle on the other side of the body. This resulted in a uniformity of muscle weaknesses known as left anterior or right posterior.

Introduction:

In 1980 your author wrote a paper for the ICAK Summer Meeting entitled "Left Brain and an Extremity Lesion " (1). The indicator for Left Brain disconnect from Right Brain was the inability to maintain muscle strength while using the memory-recall faculty, as in counting or adding numbers. Your author discovered that following correction of a radial subluxation of a pronated elbow, the left brain faculty was restored without ensuing muscle weakness. Another confirming test was corrected, and that was the ensuing weakness to a previously tested strong muscle, when the left eye was closed, but not when the right eye was closed. But the most remarkable discovery, was the "switching" of muscle weakness from one side of the body to its corresponding muscle on the other side of the body. This phenomena resulted in a pattern of muscle weakness known as "Left-Anterior - Right Posterior".

"Left Anterior - Right Posterior" (Contd.)..... page 2

"Switching" .

The correction of the elbow subluxation known as "Tennis Elbow" would switch the accompanying weakness of the right lower trapezius muscle to the left , while the right would now test strong. (1)

The correction of the right medial calcaneal subluxation (2) would immediately switch any weakness of the right abdominal to the left side of the body, and of a weak left quadratus lumborum to the right side of the body.

Left Anterior or Right Posterior

Thosemuscles that tested weak on the anterior or lateral surface of the body or of the arm or the leg were always on the left side of the body.

Those muscles that tested weak on the posterior of the body or on the medial surface of the arm or of the leg would be weak on the right side.

The following table lists the pattern of uniformity of muscle weaknesses that resulted:

"Left Anterior - Right Posterior" (Contd.)..... page 3

Left Anterior	Right Posterior
Sternocleidomastoideus	Neck Extensors
Pectoralis Major Clavicular	Upper Trapezius
Pectoralis Major Sternal	Middle Trapezius
Deltoid and Coracobrachialis	Anterior Deltoid
Psoas Major and Iliacus	Quadratus Lumborum
Fascia Lata Femoris	Adductors
Piriformis and Gluteus Medius*	Gluteus Maximus
Lower Trapezius *	Posterior Tibialis
Abdominals	Hamstrings
Diaphragm	Popliteus
Sartorius - Gracilis	Gastrocnemius - Soleus
Subscapularis	Supraspinatus
Teres Major	Infraspinatus - Teres Minor
Anterior Tibialis - Peroneus	Latissimus Dorsi - Triceps
Quadriceps	Sacrospinalis

Note: The (*) items do not fit the pattern, but could be caused by another fault coexisting: (3)

Left gluteus medius and posterior left atlas.

Left gluteus maximus and an Axis Body Left

Conclusion:

The correction of extremity subluxations, such as the "Tennis Elbow" or the "Posterior-Medial Calcaneal" will switch muscle weakness from one side of the body to the other, while the former weak muscle will now test strong.

This switching of muscle strength resulted in the uniform pattern of muscle weakness known as "Left Anterior-Right Posterior ".

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" Multiple-Point Therapy Localization/Challenge, or The Summation of Stimuli, "

By Dr. Elmer J. Cousineau, D.C.

Abstract:

An exploration of the use of Multiple-Point Therapy Localization/Challenge as a useful tool in Applied Kinesiology. Setting the parameters and contraindications in its use.

Introduction:

This paper is an expansion of the very fine outline written by Dr. John V.N. Bandy, D.C. in his research paper submitted in the Collected Papers of ICAK for the summer of 1982 entitled " Two Point Therapy Localization/Challenge ".

Body Responses as Indicators:

a strong muscle weakens

a weak muscle strengthens

The Points or Variables:

Any stimuli added to the body that changes its response from strong to weak, or from weak to strong, is called a Point. The following Points may be listed:

(1) Moving the head:

- as in chin up or chin down (Occipital-Atlantal)
- rotating the cervicals as in head rotation.

Multiple Point TL/C (Contd.) ..... page 2

The Points or Variables (Contd.)

(2) Moving the Eyes:

Right or Left as in Ocular Lock

Right Inferior or Left Inferior

as in E.I.D. or Eyes Into Distortion

(3) Light or the Absence of it:

Eyes Open, Eyes Shut, Eyes Open (Light Change)

Testing in a Darkened Room (Pituitary)

(4) Adding a Substance to the Body:

Putting a Nutrient on the Tongue

Placing different Allergens on the body

(5) Adding Color or Sound:

The Color Pink, indicating Hypoglycemia (1)

Different Pitches of Sound

(6) Moving a Joint to the limits of its motion:

Pronation or Supination of the Elbow (2)

Chin Up or Chin Down (Occipital-Atlantal Motion)

(7) Stressing a Joint:

As in the Challenge with Force applied in opp. directions.

(8) Touching the body:

Over a joint, an organ, a reflex point, a pulse of an Acupuncture Meridian, or its Beginning or End Points.

With the Patient's own hand, dorsal or volar surface.

With the patient's finger tips (Meridian End Points)(3)

With the Testing Doctors fingers (Meridian End Points)

The Test Result:

As each successive point is added to the patient's body, the indicator muscle changes from strong to weak, and then back to strong again, as the points accumulate.

Example: I have seen as many as ten different points added by the patient using a finger from each of his hands, the fingers of another person, plus the fingers of the testing doctor, with substances added to the body, and light added or the room darkened. Each successive Point added changed the indicator muscle from strong to weak, then back to strong again. (4)

Conclusion:

Therapy-Localization and the Challenge is a valuable tool in Applied Kinesiology to locate those substances, stresses and limits to the range of motion of the patient's body parts, in promoting health or disease. They must be used with understanding, and the conclusions must be based upon the total points used in determining what is needed for the patient's recovery.

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CANNABINOID TOXICITY SYNDROME (CTS) : ITS RELATIONSHIP TO PERSISTENT "NEUROLOGIC DISORGANIZATION", SHORTLIVED THERAPEUTIC RESPONSIVENESS, AND CHRONIC ILL HEALTH. METHODS OF DRUG ("POT") DETOXIFICATION.

BRENT W. DAVIS, D.C.

ABSTRACT. The damaging effects of Cannabis consumption are briefly described from an overview of exhaustive, current research findings. Relationships between cannabinoid toxicity, "neurologic disorganization" and chronic ill health are hypothesized. Methods of treating active or latent Cannabis-toxic patients to enhance therapeutic responsiveness and reduce chronic biomechanical instability are discussed.

INTRODUCTION

Despite the fact that thousands of articles on marihuana have appeared internationally in medical journals over the last 15 years, few physicians ever consider Cannabis-related health problems on a day to basis. (Cannabis and cannabinoids will be used somewhat interchangeably in this paper, although technically, Cannabis contains some 60 cannabinoids, delta 9-THC being the most psychoactive.)

That active marihuana consumption can be damaging to several physiological processes is widely documented in research literature (1-5). The concept that marihuana can exert persistent damaging effects in the human organism (other than genetic) long after its use has stopped (latent effects), however, has generally not been recognized, and is a primary consideration of this paper.

Cannabinoid Toxicity Syndrome (CTS) is a symptom complex observed clinically over several years and defined by this author to help explain a constellation of health disorders which very possibly occur in a considerable percentage of the population between fifteen and thirty-five years of age.

Individuals detected suffering from Cannabis toxicity have repeatedly used

Cannabis in the past, although they may not have been heavy users. Also, they may be current users of the drug at the time they are seeking medicial care. Two types of Cannabis toxicity will be described: the acute and latent stages. The latent stage is particularly interesting due to the fact that it can manifest after (sometimes years after) marihuana use has been completely stopped.

General practitioners tend to overlook the issue of how marihuana consumption (past or current) may be affecting patients' health because the whole subject of Cannabis use has been clouded on the one hand by academic controversy and on the other hand by emotional appeals of both individuals favoring its recreational use and those vehemently opposed to the drug. In an authoritative work on health effects of marihuana, Heath et al. note that "each time sound data have been presented which indicate that the drug might be injurious to health, there have been rebuttals, the critics claiming the study lacked adequate controls. Typical arguments have been that subjects participating in the study used drugs in addition to Cannabis sativa or that the pathology would have developed in the absence of Cannabis use"(3).

Further, it is important to note that some holistc health practitioners assume that because they emphasize in their practice the importance of healthy life styles, they are not treating the type of individual that would be a candidate for CTS. A cursory review of the statistics of marihuana use among adolescents and young adults over the last two decades should dispel this notion on the basis of the sheer volume of individuals that have used or are using the drug, if nothing else.

Before defining the syndrome, general information will be briefly presented relating to the prevalence of marihuana use, the metabolism of Cannabis, its toxicology and pharmacology.

In the interests of the burgeoning numbers of chronically ill, it is hoped

that this paper will encourage a greater awareness of Cannabis' related health problems (especially of Latent Cannabinoid Toxicity) and wider clinical appreciation of the marihuana detoxification measures suggested here.

OCCURENCE OF CANNABIS USE

Trend studies of Cannabis use in U.S.A., Canada, Australia and Norway show that marihuana consumption has dramatically increased in the last 15-20 years. "For example, only 6.7% of students in Canada in 1968 but 31.7% in 1979 used Cannabis. Annual household surveys in the U.S.A. showed that the percentage of young people (aged 12 to 17) who smoked Cannabis increased from 15% in 1971 to 22% in 1977. ...Currently there are some signs of stabilization in rates of use. The latest studies of high school seniors by Johnston et al. showed no increase in use in 1980 and 1979 over 1978..."(Smart, R.G.(5)).

Nicholi (6) reports that "recent surveys indicate that approximately 60% of high school seniors have smoked marihuana and approximately the same percentage of college students. ...Within the college age group, the 18 to 25 year olds, some **21 million use the drug with about 40% of this group having used it a minimum of a hundred times"** [emphasis mine].

PHARMACOLOGY, TOXICOLOGY AND METABOLISM OF CANNABIS AN OVERVIEW

A tremendous number of experimental studies have demonstrated the potentially far reaching negative effects of Cannabis on experimental animals and humans. Adverse findings suggest, and in some cases prove, that Marihuana(7):

1. Diminishes growth and body weight.
2. Causes chromosomal aberrations and mutagenicity.

3. Impairs synthesis of macromolecules.
4. Depresses T-lymphocyte, B-lymphocyte and macrophage activity.
5. Seriously disturbs male and female endocrine balance.
6. Causes CNS dysfunction, altering cognitive-perceptual and psychomotor activities.
7. **decreases gastric acid secretion, causing hypochlorhydria.**
8. Invades and becomes sequestered in fatty tissues and organs throughout the body. Due to its strong lipophilicity, its noxious influence could persist for long periods of time.

- 97% of blood born delta 9-THC is bound mainly to protein, especially Low Density Lipoproteins.
- Radiolabelled THC accumulates particularly in the lungs, liver, kidney, heart gut, spleen, brown fat, and mammary glands. THC also accumulates in several endocrine glands such as the adrenal cortex, thyroid, and pituitary. There is a high uptake by the tissues, common for highly lipophilic compounds (Harvey,D.J.(2)).
- Cannabis has marked lipophilic properties which aid its rapid passage accross lipoprotein membranes in the lung and in the vascular system. Cannabinoids appear to disrupt the structure and function of biological membranes (Mellors, A. (1)).
- THC has excitatory as well as depressant effects on spinal pathways of the rat and of polysynaptic reflexes of the nonhuman primate. ...The ambivalent effects of THC are also observed on EEG records which display patterns of desynchronization under effects of the drug" (Nahas, Gabriel G. (2)).
- THC reduces the number of lymphocytes and uptake of nucleic acids by lymphocytes in the adrenal medulla. THC has reportedly caused decrease in the weight of the thymus in experimental animals. (Albert, et al.(4)).

- In man Cannabis s moking suppresses circulating levels of (FSH) and (LH) accompanied by a decrease in the amount of Testosterone. Cannabis appears to act on the testes directly. (Nogawa, T. et al. (4)).
- In female primates it is likely that THC directly suppresses hypothalamic/pituitary activity (Smith, C.G. et al.(4)).
- Leukocyte and sperm cell nuclear chromatin is altered under the effects of Cannabis (Issidorides, M.R. et al.(4)).

METABOLISM

-At the time of peak psychoactivity, brain concentrations of THC are quite low estimated at less than 1% of the intravenous administered dose (Harvey,D.J.(2)).
- In experimental animals THC non competatively inhibits brain MAO by interacting with a lipophilic moiety of the enzyme or its microenvironment.
(Schurr, A. et al. (4)).
- THC is extensively metabolized by liver enzymes [working to change it into metabolites which the body would hope to more easily handle] (Wall,M.E. et al.(4)).

EXCRETION

In humans, about 70% of the dose of "pot" is excreted during the first week. The pattern that emerges from studies on the metabolism, distribution and excretion of THC is similar to typical lipophilic drugs, which are rapidly taken up by the tissues, particularly fat. "Although metabolism is both rapid and extensive, elimination is mainly governed by slow release of the drug sequestered in deep body compartments" (Harvey, D.J. (2)).

CHARACTERIZATION OF CANNABINOID TOXICITY SYNDROME (CTS)

CTS is a pan symptomatic disorder. Due to Cannabinoids ability to infiltrate,

sequester, and negatively influence diverse areas of the human organism, they have the potential of disrupting the body's homeostatic mechanisms, especially at the level of the CNS. Once this has happened, physiological and psychic breakdown by attrition can occur from any number of chronic stressors which are encountered by individuals living in modern industrial societies. The mental predisposition and constitutional or genetic inheritance will govern which systems or organs break down in a given case. Therefore, specific mechanisms establishing the causal relationship between marihuana consumption and manifestation of any particular disorder are hard to define.

Nevertheless, several signs have been observed in clinical practice by this writer which indicate that a given patient may be experiencing CTS. Over the last five years, Dr. Davis has treated at least 50 previously refractory cases (suffering from diverse complaints) the success of which principally depended on recognizing and helping the patient diminish Cannabis toxicity. In most cases, the patient had made no association whatsoever between marihuana consumption and the onset of orthopedic and other complaints. As far as patient interview and physical examination can establish, none of the patients presented for examination under the immediate influence of drugs, although some had consumed marihuana within the previous 24 hours. The characterization of CTS in this paper is a composite of factors seen in several model cases.

CTS can include effects of acute intoxication which have been widely described in medical literature (Brill, H. et al. (2)). Generally speaking,however, there is a small likelihood of seeing acutely intoxicated patients in general practice. Non habituated patients that have previously scheduled office visits (and who would show obvious signs of intoxication) are apt to refrain from drug use prior to seeing their doctor. Clinically, therefore, CTS primarily involves:

1. chronic users habituated to Cannabis who experience acute episodes of

dis-ease, presumably by passing the endpoint of the body's ability to compensate to the noxious influence of a persistent chemical stressor.

2. Periodic users that may not have consumed the drug for several days or weeks, and

3. Individuals who relate that they have stopped smoking "pot", and may not have consumed it for months or years.

Interestingly, the latter frequently exhibit persistent weaknesses in the body's compensatory and homeostatic mechanisms. These weaknesses lead to chronic physical complaints that most physicians would dismiss as being psychosomatic or ephemeral dyspathies. Diagnostic signs in the latter, the latent stage of CTS - designated as Latent Cannabinoid Toxicity (LCT) - are subtle and can easily evade detection. (Psychiatric tests do identify numerous psychopathological effects of Cannabis consumption which are believed to be reversible, disappearing after drug use has stopped.) The possibility of identifying latent effects of past Cannabis use is significant in light of the findings of a recent, authoritative WHO report ((5)p. 41):

> "Because of the sequestration of cannabinoids, THC or its biologically active metabolites could theoretically accumulate in fatty tissues during chronic or intermittent administration (Jones,1980). This accumulation would not be measurable by determinations of blood levels of cannabinoids, and has not, as yet, been demonstrated in human tissue samples. In humans, cumulative behavioral or physiological effects have not been deomonstrated under conditions of controlled administration of up to three months duration, although the simultaneous development of tolerance may have masked this phenomenon (Jones, 1980). In animals, cumulative toxicity ...has been observed at doses relevant to those consumed by human chronic users. The possible occurence of cumulative toxicity in humans, therefore, is a question that should be examined.

B. W. Davis, CTS / Drug Detoxification

GENERAL SIGNS OF CTS

1. Deep seated and persistent tendency toward "neurologic disorganization" or "switching" as defined and demonstrated in the field of Applied Kinesiology (8).
2. Loss of musculoligamentous tone and inability to maintain therapeutic musculoskeletal manipulative corrections (even with nutritional support.)

SIGNS OF THE ACUTE STAGE OF CTS

1. Profound anxiety experienced by the patient over loss of previous relatively normal musculoskeletal integrity, attended by severe pain, spasm and the feeling that weight-bearing structures (pelvis, lumbar spine, neck/shoulders) will "give out" or collapse.
2. Moderate as well as severe (and occasionally bizarre) antalgic positions, in the most extreme cases resembling a combination of Intervertebral Disc Syndrome antalgia and torticollis.

SIGNS OF THE LATENT STAGE OF CTS (aka Latent Cannabinoid Toxicity - LCT)

1. Months or years after stopping marihuana use, the patient may remark that he has begun to notice subtle deficiencies in his cognitive processes.
2. Transient/mild or deep sense of depression, mood swings, apathy.
3. Poor attention span - tendency to confuse sides of the body and proper execution of verbal orders in muscle testing.
4. Similarity to and increased propensity of association with chronic ill-health syndromes such as: functional hypadrenia, blood sugar handling stress, Candida albicans allergy, immunodepression, fatty acid metabolism problems, etc.

B. W. Davis, CTS / Drug Detoxification

CASE REPORT

Acute CTS - Mr. R., a 28 year old male Caucasian, came to my office as a new patient on a Monday. He was heavy set, about 6'2" tall, 230 pounds. He was extremely distressed about an injury he had sustained two weeks previously while working at his janitorial job. He had never had back problems before. After he had finished mopping, his low back and shoulder muscles began to tighten. Without abating, the spasms continued to worsen in the next week such that he assumed an antalgic position that made him twist in deformity. In my office, he literally began to weep, asking what was going to happen to his body - would he be paralyzed.

On temporary disability leave, Mr. R. had visited a neurologist and an orthopedist. He had been prescribed bedrest the previous week, and Fiorinal/Codeine III (30 mg. Codeine/tablet) to relieve pain and anxiety. He took the medication several days with no relief. In fact, the Saturday before seeing me, he took "at least 6 pain pills". He felt he was still getting worse, so on Sunday he took nothing. His condition continued to worsen on Sunday.

Since this case was reminiscent of CTS, I asked Mr. R. if he was presently using or if he had ever used recreational drugs. He related that he had used "pot" for years and that he had smoked a lot of it the previous week to relax his muscle spasms. He had smoked several "joints" on Saturday and Sunday to "handle it." I told him I had seen many cases where marihuana had caused orthopedic problems, and mentioned that he would have to stop if he wanted the pain to go away.

I asked Mr. R. if he could lie on the examining table. He said he was in too much pain. No strong indicator muscles could be found for weight-bearing Applied Kinesiological muscle testing.

Following a procedure that I had seen work several times before in both acute Cannabis induced muscle spasm and in chronic musculoskeletal instability, I recommended that he take 1 rounded teaspoon of a powdered Basil preparation I had made up with each meal and before bed, and return the next day.

To my astonishment, Mr. R.'s antalgia and anxiety had almost entirely disappeared. He was then able to undergo routine AK diagnostic workup and treatment. He said he would continue the Basil and stop smoking "pot". He called on Thursday to tell me he felt quite well. He reportedly had not used any marihuana since his first visit with me, and said that he would not use it again. He did not return to the office.

It is well documented that chronic users of marihuana tend to persist in drug use even when they have identified unpleasant side effects from such use (H. Brill, G.G. Nahas (2)). It is significant, then, that recognition by the physician of drug-related orthopedic problems, and adjunctive use of Basil to detoxify cummulative effects of Cannabis, can bring such dramatic relief of symptoms that in some cases it is a strong enough object lesson to induce cessation of marihuana altogether.

Since Mr. R.'s case, several musculoskeletal and other complex conditions have been greatly helped by using a tableted preparation of highly bioactive species of organically grown Basil (9).

EXPERIMENTAL RESEARCH EVIDENCE SUPPORTING CTS

The varying and potentially far reaching negative biochemical and psychic influence of cannabinoids cannot help but encourage one to try to discover if Cannabis consumption is causally associated with many of the chronic ill-health syndromes seen so frequently in practice today, problems which could very possibly be centrally mediated, i.e. at the brain level. Experimental evidence does support this possibility.

Evidence in Support of "Neurologic Disorganization", "Occular Lock" and "Switching".

- W.W. Just et al. (1) report : "Brain autoradiographs [using labelled cannabinoids] showed that gray matter was more heavily labelled than white matter. Apart from this gross distribution, however, some brain structures contained higher levels of radioactivity that gray matter in general. Most of these structures are involved in the processing of visual and acoustic information and in motor co ntrol [emphasis mine] ...The visual pathway displayed high concentrations of radioactivity..."

It was further noted that six hours after introduction of labelled THC into the live animal, brain levels of THC, in general, diminished, but the midbrain and medulla oblongata maintained rather high levels of radioactivity.

- Robert G. Heath et al. (3) report that in non-human primates that showed lasting EEG changes consequent to moderate or heavy smoking of marihuana, consistent brain ultrastructural changes were observed. Pathologic changes were: (1) in the morphology of the synapse; (2) in the volume density of the rough endoplasmic reticulum; (3) in the nuclei, characterized by the presence of a large number of intranuclear inclusions. Greatest pathology was noted in the septal region, the hippocampus and amygdala.

- P. Etevenon (3) reports that permanent subcortical EEG changes can be observed in limbic structures and sensory thalamic nuclei of monkeys that have undergone 3 months of exposure to marihuana via a "smoking machine".

C.J. Hillard et al. (4) remark that THC has an extremely high affinity for brain synaptosomal membranes and "exerts a wide range of effects on membrane associated systems as well." For example, THC has been shown to affect neurotransmitter uptake systems and membrane-bound enzyme systems which "suggest that a mechanism of THC action could be a primary alteration in the physical properties of the phospholipid bilayer of the membrane which secondarily affects membrane associated macromolecules."

- Gabriel G. Nahas (2) states: "Acute as well as chronic manifestations of cannabis intoxication will result from nanomolar [10^{-9}M] concentrations of this [drug] and of its many metabolites, sustained in vital organs for hours, days or years. Neutral fat deposits represent a major storage buffer compartment for THC and limit exposure of the brain and other tissues to low but sustained concentrations of the drug." The fact that activity of the drug can be achieved

with such a small concentration likely indicates that its action is on or close to receptor sites (Maureen Bronson et al. (4).

There is ample evidence to suggest that cannabinoids (and by extension, other noxious lipophilic substances) could chemically induce "neurologic disorganization" over time by sequestering strategically in the CNS.

That Cannabis can aggravate tendencies toward schizophrenia is well established in psychiatric literature. There is clear evidence in A.K. between the relationship of "neurologic disorganization" and schizophrenia - a further tie between disruption of fundamental mechanisms and invasive chemical substances in marihuana.

EVIDENCE LINKING ACTIONS OF CANNABINOIDS AND FUNCTIONAL HYPOADRENIA

- Orthostatic hypotension was induced in human experimental subjects by marihuana inhalation (10).
- The number of lymphocytes in the adrenals is diminished under the influence of THC, and could possibly initiate adrenal autoimmune dysfunction.
- Cannabinoids selectively sequester in the adrenals, and may persist over time.
- Blevins and Regan (1) point out that some cannabinoids and their metabolites have chemical structures that resemble cholesterol. This has strong implications with respect to the interactions between the cannabinoids and all membranes of and within the cell, and could represent an additional source of stress on the adrenals during steroid synthesis.

MARIHUANA AND PREDISPOSITION TO FUNGAL INFECTION:

- Introduction into the lungs of Aspergillus by contaminated marihuana cigarettes has been reported by different authors (eg.F.S. Tennant (5) and may not be an uncommon occurence. The pathway of entry of airborn pathogens via the lungs is

fascilitated due to Cannabis' considerable destructive effects on lung macrophages and general physiology. This could weaken the body's resistance to opportunistic organisms such as Candida.

MARIHUANA IN RELATIONSHIP TO FATTY ACID METABOLISM

- With reference to cannabinoids, D.J. Harvey (2) states that" the exact nature of the material accumulated in the tissues is largely unknown, although ...studies ...do indicate that a substantial portion is THC itself. The 11-hydroxy metabolite has been found in brain and other tissues including fat. ... A substan tial portion of the drug, particularly at later times, appears to be in the form of fatty acid conjugates..."

MANAGEMENT OF CTS

Well trained holistic chiropractic physicians already have at their disposal the tools necessary for managing CTS. The obvious prerequisite to management is identifying the problem.

1. Patients with unusual or chronic symptoms should routinely be asked "have you ever or are you now using recreational drugs - marihuana?
2. If CTS does exist, the physician must have a strategy to try to mobilize sequestered drug residues by increasing circulation of biological fluids. Musculo-skeletal manipulation and cranial-sacral respiratory therapy are ideal for this purpose.
3. Channels of elimination should be kept open. A high roughage diet helps move toxic bile out of the body more quickly.
4. Specific nutritional supplemental therapy should be initiated. Conventional vitamins and minerals can be helpful, but in this author's opinion, cannot approach the effectiveness of bioactive Basil (9).

BASIL & DRUG DETOX - POSSIBLE MODES OF ACTION

In the course of Dr. Davis' research in medicinal plant therapy and ethnomedicine, it was discovered that Basil was used in ancient India and Egypt, among other places, to detoxify Hashish (a potent concentration of marihuana.) This led the author to try to empirically evaluate its usefulness in clinical practice. Several years of application have confirmed its utility. As a therapeutic plant its usefulness is specific against Cannabis, but it should also be utilized in the case of sequestration of other lipophilic, noxious chemicals, when AK reflex testing or independent clinical knowledge dictate.

The chemical composition and action of Basil is complex. Basil contains: camphor, the essential oils, thymol, eugenol, and others, sesquiterpenes, caffeic acid, polysaccharides, vitamins and minerals (11).

- In homeopathic medicine, camphor has proven to be an extraordinary remedy to revive patients whose vital energy has fallen to a dangerously low level. It can also relieve severe muscle spasm.
- Thymol is a highly antiseptic agent. Basil shows antibacterial and antifungal activity (13) and exhibits fungitoxicity to species of Aspergillus (12). Basil has anthelminic (antiwormal) properties (14). It is also an anti-oxidant;(15) which is very significant considering membranal damage that occurs from marihuana.
- Basil possesses anxiolytic properties (allays anxiety) and is helpful when there is a confused state of mind and difficulty in concentrating (16).

General Medicinal Properties of Basil (17,18):

gastric antispasmodic	aromatic
stomachic tonic	diaphoretic
expectorant	stimulant
	carminative

Perhaps Basil's strongest therapeutic force comes from the fact that it is also able to penetrate fatty tissue, but in a constructive way. Chemically, the

isoprene and phenylpropanoid constituents of the essential oils of Basil are quite similar to the cannabinoids in Marihuana. Basil's essential oils might solubilize cannabinoid residues, and since Basil does accelerate G/I eliminative function, it would help the body rapidly discharge sequestered toxins that had been released.

For millenia in India, Basil has always been planted around temples. It is respected as a holy herb.

CONCLUSION

In addition to finding and correcting somatic problems, one of the great joys of healing is the ability to help restore the patient's mental order and harmony of spirit. To this end, and ancient remedy - the benevolent herb, Basil - makes a unique contribution.

SUMMARY

1. CTS is a constellation of health disorders more prevalent than one might assume.
2. A.K. procedures give physicians particular insight into identifying and successfully treating patients with toxic problems from Cannabis that very likely could be missed or misdiagnosed.
3. Administration of tableted bioactive species of Basil (9) to individuals in both the acute and latent stages of CTS can bring dramatic improvement.
4. The possibility that CTS can serve as a model for deleterious effects of lipophilic noxious chemical substances other than cannabinoids should be considered.

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BALANCING IONIZATION

by

Sheldon C. Deal, D.C.

ABSTRACT: Herein lies a method of balancing positive and negative ions by the use of mineral supplementation. A distinction is made whether the problem is due to too many positive ions or not enough negative ions; which was previously treated as the same problem. Or it could be the other way around, meaning too many negative ions or not enough positive ions. This balancing is accomplished by using four (4) different kinds of minerals which include two (2) types of calcium and two (2) types of potassium.

HISTORICAL:

It was Dr. George Goodheart who first made us aware of ionization as it pertains to applied kinesiology by his famous example of chronic clonic tonic intermittent toricollis.(1) He stated that if he had the patient breathe in through one nostril only for one hundred or more times it would afford the patient a period of relief from this devastating condition. This was based on the conclusion that the right nostril specialized in positive ions and the left nostril specialized in negative ions. The treatment was very affective but of short duration because when the patient resumed breathing through both nostrils the preponderance of one ion or the other was lost. At that time there were comments; such as, it was not by accident that the human body was designed with two nostrils rather than one. When the comment was made that we would look funny if we only had one nostril the person was reminded that we would not look funny if everybody only had one nostril instead of two, because we would not know it any other way.

In the field of otolaryngology it has been shown by instrumentation that the nasal cycle changes approximately every 20 minutes(2) meaning that we receive a perponderance of our air we breathe in through one nostril for 20 minutes and then it changes over to the other nostril for 20 minutes ect., ect. This would explain why we all have had the experience of having one nostril occluded during an episode of acute rhinitis only to find that suddenly, with no apparent explanation, the occluded side opens up and the previous patent side becomes occluded. This research also showed that the amount of air passing through the nostril was not dependent on nor in porportion to the size of the lumen of that nostril.(3) This same instrumentation showed that positive ions came through the right nostril and that negative ions came through the left nostril. Thus it became established that the turbinates of the right nostril form an ionization chamber specializing in positive ions and the turbinates of the left nostril form an ionization chamber specializing in negative ions.(4)

OBSERVATIONS:

The above data is a good basis for why it is important for us to have a balance of positive and negative ions in our body to start with. There are many conditions in our world where we are exposed to a predominance of either positive or negative ions. Such as a weather front moving through the area where we live which is preceeded by an abundance of positive ions and succeeded by an abundance of negative ions, or being around electrical equipment or internal combustion engines which gives off an abundance of positive ions. If we have a balance of ions in our body to start with then we are not bothered by a temporary exposure to a perponderance of one kind

or another of ions. But if we have an imbalance of positive or negative ions to start with and then we are exposed to a condition such as above where there are a perponderance of one kind or another of ions. Then we become further imbalanced as the original condition becomes exaggerated.

Another interesting observation in the field of personanology is that people who are predominately negative in their habits, attitudes and personality have a larger opening of the left nostril and people who are predominately positive in their habits, attitudes and personality have a larger opening of the right nostril. The idea is that we need a balance in our lives and therefore we should have equal sized nostrils.

In applied kinesiology it has been established that if a patient breathes in through the left nostril and out through the right nostril and this weakens a previously strong indicator muscle, that patient is low in positive ions. An interesting observation in this patient is that they will therapy localize with the palms against the body only. If the condition is reversed, meaning that breath in through the right nostril and out through the left nostril weakens a previously strong indicator muscle that patient is low in negative ions and will therapy localize only with dorsum of the hand against the body.

So for therapy localization purposes only, it is important to establish whether or not there is an ionization problem in the patient. I have had a few patients who were low in negative and positive ions and hence would neither therapy localize palms up or palms down!

When you fix this kind of patient that other doctors have failed on, you are a hero. The obvious advantage here, is if you will establish ionization first in your patient then you do not have to therapy localize everything twice, meaning once palms up and once palms down.

I have had some remarkable success with patients who remarked to me that their symptoms came only when it rained or that they felt particularly elated or particularly depressed at the beginning of a storm or at the end of the storm, or that weather changes always made a difference in how they felt, just by checking and correcting ionization.

As mentioned earlier the original correction for this condition was to have the patient breathe in through one nostril only according to which side they showed a need for. More recent investigation shows that breathing in through the right nostril only, activates the left brain and thus is conducive for stressing left brain activities and vice versa, meaning that breathing in through the left nostril only activates the right brain and is conducive for stressing right brain activities.(5) The catch to all this is that it has a temporary effect only.

It was Dr. John Stoutenburg who established in the early 1970's that the taking of calcium would provide positive ions and that the taking of potassium would provide negative ions. The big advantage being that now the correction would stay fixed.

One time when I had presented the above evidence in a lecture at the University of California at Davis Medical School, I was asked why did that w

since calcium and potassium were both positive ions. My answer was that since calcium had a valance of plus two and potassium had a valance of plus one, that calcium was twice as positive as potassium and potassium was twice as negative as calcium and thus the difference was a relative one. To date I have not found a better answer and so I still use that same explanation.

CURRENT OBSERVATIONS:

Since I do alot of work with nutrition in my office and I have been exposed to the work of Dr. Herschel Robertson from Higgensville, Missouri, I became aware that there is a difference between having too many negative ions or not enough positive ions, which previously was treated as the same condition. Or vice versa, that there is a difference between having too many positive ions or not enough negative ions which also was previously treated as the same condition.

This can be established kinesiologically by having the patient breathe in through one nostril only and testing your indicator muscle and then having the patient breathe out through one nostril only and testing your indicator muscle. Whereas before this was all one test. Now we can establish if the condition is due to too many positive ions (breathe in through the right nostril only) or is the condition due to too few negative ions (breathe out through the left nostril only). Perhaps the condition is due to too many negative ions (breath in through the left nostril only) or it could be due to too few positive ions (breath out through the right nostril only).

It has been established that one form of a particular mineral has a positive reaction in the body whereas another form of the same mineral has a negative reaction in the body. It was on this basis

that I established which form of the mineral to use by breaking down the ionization testing into the above four (4) parts. By following these methods I found that too many positive ions would respond to potassium gluconate, but would not respond to potassium citrate for example. If found that too many negative ions would respond to calcium gluconate, but would not respond to calcium lactate for example. I also found that too few positive ions would respond to calcium lactate, but would not respond to calcium gluconate for example. And finally I found that too few negative ions would respond to potassium citrate, but would not respond to potassium gluconate for example. I keep saying for example here because there are other forms that will work.

For the purposes of learning this phenomenon and using these principles in your office I have devised the following chart:

NASAL IONIZATION AND MINERAL BALANCE

Condition	Indicator muscle changes when patient breaths	Corrected by:
Excess Negative Ions	In through the left nostril	Positive Calciums Calcium Oxide Calcium Carbonate Calcium Gluconate
Deficient Positive Ions	Out through the right nostril	Negative Calciums Calcium Lactate Di Calcium Phosphate
Excess Positive Ions	In through the right nostril	Positive Potassiums Potassium Oxide Potassium Carbonate Potassium Gluconate
Deficient Negative Ions	Out through the left nostril	Negative Potassiums Potassium Citrate Potassium Aspartate

CONCLUSION:

We now have a kinesiologycal method of more precisely balancing the ions in the body and the minerals used to do so not only bring about a lasting effect, but also greatly help to balance the patient's chemistry. We previously knew that the acid or negative calcium lactate was preferred if the urine Ph was over 6.4 and that the alkaline or positive calcium gluconate was preferred if the urine Ph was under 6.4. So now we have another piece of the jigsaw puzzle to help us determine kinesiologically which calcium to use.

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Trigger Point Therapy

Mark S. Diener D.C.

Abstract: There are many different approaches to the diagnosis and treatment of the trigger point complex. Travell, Nimmo and Pruden all demonstrate varying tecniques in their treatment. This paper is concerned with management of a trigger point with Applied Kinesiology.

It has been a pleasure to be an associate of Dr. Goodhearts for over three years now. During these years I have learned many invaluable bits of information. I learned that if you don't ask a question you won't get an answer. The following is an answer I received with respect to trigger point treatment.

Many of us are aware of the trigger point complex. It is an area of soft tissue irritation that has the capability of referring pain, numbness and or tingling to another area of the body. There are many charts designating these areas. Trigger points will therapy localize, will respond to treatment and will stop localizing when fixed.

Many patients come to your office with complaints into an area that after a comprehensive history and work-up comes up normal. One example is a complaint of pain in the RT arm and forearm, upon examining the teres major and minor muscle areas on the Right Scapular area the pressure produced pain into the areas of complaint. After several treatments the patient was better but still had pain with driving and sleeping on that side at night. I talked with Dr. Goodheart about the patient and he said to treat that point with pressure, and the proper torque which challenges out, until the point stops referring. After approximately ten minutes the points did stop referring. He is doing well and if the points act up he treats the point by lying on a golfball on the point until it stops referring.

In concluding trigger points will Therapy Localize. They will challenge out with a direction of torque to use while treating the point with adequate pressure. The Therapy Localizing will be abolished upon successful treatment of the point. The most important point

is to treat the point until the referral stops. Between treatments the patients can augment the response by treating the points at home once a day until the point stops referring.

The Use of a Temperature Thermister with Trigger Point Treatments

Mark S. Diener D.C.

The following paper deals with trigger point therapy and the case of a temperature thermister to monitor a patients progress.

Many patients experience referred pain from a trigger point area yet upon examination you are unable to induce the symptoms they relate to you. A good majority of the complaints concern the upper extremities and many are brought on by sleeping when pressure is put on the trigger point area. The thermister will allow you to monitor the area of symptomatology even though with pressure you cannot reduplicate the patients symptoms.

Place a thermister on the area of the patients complaint and after the temperature reading has plateaued seek out a trigger point. When an active point is found the temperature will usually go up, in some cases it will decrease. The point will therapy localize, it will show a torque challenge to give while the pressure is being administered. The point may not refer any symptoms at all but the temperature will change. The treatment is ended when the temperature starts returning to the original temperature.

The technique has proven invaluable in those patients with unexplained symptoms that follow "trigger point" like history that you are unable to reproduce with pressure on a trigger point. Thermography can also be used, the thermister is just easier to work with. This recommendation of Dr. Goodhearts to measure has helped with many tough patients.

THE DURAL TORQUE AND THE POSTERIOR SPHENOID

LOUIS F. DONNER D.C.

ABSTRACT: THIS PAPER SUGGESTS AN ALTERNATIVE TREATMENT OF THE DURAL TORQUE, AND INTRODUCES A CRANIAL FAULT WHICH IS NAMED THE POSTERIOR SPHENOID.

THE TREATMENT OF THE DURAL TORQUE INCLUDES A HIDDEN INTERNAL FRONTAL BONE ROTATION, AND A DROPPED METATARSAL HEAD ON THE SIDE OF THE GREATER FOOT TURN IN.

THE POSTERIOR SPHENOID IS FOUND BY BILATERAL POSAS WEAKNESS, CONFIRMED BY BILATERAL THERAPY LOCALIZATION TO THE NASO-SPHENOID, RESPIRATORY CHALLENGE. CORRECTED BY ADJUSTING THE NASO-SPHENOID POSTERIORLY ON INSPIRATION, THE COCCYX POSTERIORLY ON INSPIRATION AND CORRECTING A DROPPED METATARSAL HEAD.

THE DURAL TORQUE WILL BE CONSIDERED FIRST.

THE DURAL TORQUE IS DIAGNOSED AFTER THE TECHNIQUE DESCRIBED BY GOODHEART.[I] THE DURAL TORQUE IS SUGGESTED BY THE FOOT TURN IN TEST, IN WHICH ONE FOOT TURN IN MORE THAN ITS OPPOSITE WITH THE PATIENT SUPINE.

A STRONG INDICATOR MUSCLE IS FOUND. THE PATIENT IS THEN BLOCKED IN THE SUPINE POSITION WITH ONE BLOCK UNDER THE ACETABULUM ON THE SIDE OF THE GREATER FOOT TURN IN AND THE OTHER UNDER THE OPPOSITE GLENOID. THIS WILL CAUSE THE STRONG INDICATOR MUSCLE TO WEAKEN. WHICH, IS CONSIDERED DIAGNOSTIC OF A DURAL TORQUE ON THE SIDE OF THE GREATER FOOT TURN IN.

WITH THE PATIENT STILL SUPINE AND ON THE BLOCKS, THE FRONTAL BONE ON THE SIDE

DURAL TORQUE AND POST SPHENOID # 2

OF INVOLVEMENT OR GREATER FOOT TURN IN IS THERAPY LOCALIZED. THE THERAPY LOCALIZED FRONTAL BONE CAUSES A STRENGTHENING OF THE PREVIOUSLY WEAK INDICATOR MUSCLE. THE THERAPY LOCALIZATION IS DISCONTINUED CAUSING A RE-WEAKENED INDICATOR MUSCLE. THE MALAR SURFACE OF THE ZYGOMATIC BONE ON THE SAME SIDE IS CHALLENGED, CAUSING THE WEAKENED INDICATOR MUSCLE TO BECOME STRONG. THIS IS CONSIDERED DIAGNOSTIC OF A HIDDEN INTERNAL FRONTAL BONE ROTATION, AS IT WILL NOT REVEAL ITSELF UNTIL THE PATIENT IS ON THE BLOCKS. THE INTERNAL FRONTAL BONE ROTATION IS THEN TREATED BY THE USUAL METHOD. THIS CAUSES THE WEAKENED INDICATOR MUSCLE TO BECOME STRONG.

WITH THE PATIENT STILL SUPINE ON THE BLOCKS AND THE INDICATOR MUSCLE STRONG, THE PATIENT THEN RE-THERAPY LOCALIZES THE CORRECTED FRONTAL BONE. THE INDICATOR MUSCLE REMAINS STRONG. THE METATARSAL HEADS ON THE SIDE OF INVOLVEMENT, (GREATER FOOT TURN-IN SIDE) ARE CHALLENGED AS A GROUP, WHILE THE THERAPY LOCALIZATION OF THE FRONTAL BONE IS MAINTAINED. THIS WILL CAUSE A WEAKENING OF THE STRONG INDICATOR MUSCLE. THE METATARSAL HEADS ARE THEN CHALLENGED INDIVIDUALLY. ONE METATARSAL HEAD (USUALLY THE SECOND), CHALLENGE WILL CAUSE A WEAKENING OF THE STRONG INDICATOR MUSCLE. THIS IS DIAGNOSED AS A DROPPED METATARSAL HEAD AND ADJUSTED WITH THE PHALANGEO-METATARSAL BREAK TECHNIQUE OF JANSE, HOUSER AND WELLS.[II] THAT IS, THE DOCTOR HOLDS THE HEEL OF THE FOOT IN THE PALM OF ONE HAND AND PLACES THE THUMB OF THE SAME HAND ON THE PHALANGEAL METATARSAL JOINT. WITH THE OTHER HAND, HE GRASPS THE TOE OF THAT JOINT, THEN THE TOE IS DRAWN OVER THE CONTACT THUMB AND GIVEN A SHARP DOWNWARD SNAP.

THE PATIENT IS THEN ASKED TO WALK A SHORT DISTANCE AND THE DURAL TORQUE IS RE-EXAMINED. IT WILL HAVE RETURNED ON THE SAME SIDE AS PREVIOUSLY FOUND. THE HIDDEN

INTERNAL FRONTAL BONE IS THEN RETREATED. THE METATARSAL HEAD IS THEN RECHALLENGED AND RETREATED.

A SMALL STYLE #3128, METATARSAL PAD IS TAPED TEMPORALLY UNDERNEATH THE OFFENDING METATARSAL HEAD. THE PATIENT IS THEN ASKED TO WALK, REPLACED ON THE BLOCKS AND RE-CHALLENGED. THE CHALLENGE WILL BE NEGATIVE AS WILL BE THE FOOT TURN-IN TEST.

THE METATARSAL PADS COME IN VARIOUS SIZES. THE CORRECT SIZE CAN BE DETERMINED BY TAPING THE PAD UNDER THE OFFENDING METATARSAL. THE PAD THAT IS TOO LARGE WILL CAUSE A WEAKENING OF THE STRONG INDICATOR MUSCLE. I FIND THAT THE SIZE USED 99% OF THE TIME IS THE SMALL (13/16"THICK).

THE METATARSAL PADS MAY BE OBTAINED FROM:

WOLVERINE LEATHER AND FINDINGS
8306 WEST DAVISON AVENUE.
DETROIT, MICH. 48238
TELEPHONE: #313-933-2424

THE PROPER METATARSAL PAD IS THEN GLUED INTO THE PATIENTS SHOE BY THE FOLLOWING METHOD.

1. A SMALL DOT OF YELLOW WATER SOLUABLE PAINT IS PUT ON THE AFFENDING METATARSAL HEAD WITH A COTTON SWAB.

2. THE PATIENT IS THEN ASKED TO CURL THE TOES DOWN WHILE PUTTING ON THE SHOE AND KEEPING THEM CURLED UNTIL THE SHOE IS ON. THIS IS SOMETIMES QUITE DIF-

FICULT.

3. THE PATIENT IS THEN ASKED TO STRAIGHTEN THE TOES AND STAND UP PLACING THE WEIGHT ON THAT FOOT.

4. THE SHOE IS REMOVED. A SMALL YELLOW MARK SHOULD BE OBSERVED AT THE POINT OF CONTACT OF THE SOLE OF THE SHOE.

5. A DAMP CLOTH IS USED TO WIPE THE PAINT FROM THE BOTTOM OF THE FOOT.

6. THE PROPER METATARSAL PAD IS THEN GLUED WITH HOT GLUE OR ANY QUICK DRYING GLUE, EXACTLY IN THE CENTER LINE OF THE DOT, BUT SLIGHTLY POSTERIOR OR HEEL-WARD FROM IT. THE FRONT OF THE METATARSAL PAD JUST TOUCHING THE EDGE OF THE DOT.

THE METATARSAL PADS MAY BE USED IN CONJUNCTION WITH SCAPHOID PADS. WHEN THEY ARE INDICATED. NO PROBLEM HAS BEEN OBSERVED AS YET.

THE NUTRITIONAL SUPPLEMENT I USE WITH THE DURAL TORQUE IS PHOSPHORIC ACID AS SUPPLIED BY THE STANDARD PROCESS LABORATORIES AS PHOSFOOD.

THE TECHNIQUE I USE TO DIAGNOSE THE NEED FOR PHOSFOOD WITH THE DURAL TORQUE IS:

THE DURAL TORQUE IS DIAGNOSED IN THE STANDARD METHOD. THE STRONG INDICATOR MUSCLE IS THEN WEAK. A DROP OF PHOSFOOD IS THEN PLACED ON THE TONGUE. THE WEAK INDICATOR MUSCLE THEN BECOMES STRONG.

DURAL TORQUE AND POSTERIOR SPHENOID #5

THE DOSAGE USED IS 10 DROPS IN WATER (6-8 OZ.) THREE TIMES A DAY FOR A WEEK. THEN 10 DROPS ONCE A DAY UNTIL THE BOTTLE IS USED UP. THEN A WEEK OR TWO WITHOUT TO SEE IF THE NEED COMES BACK.

THIS DOES NOT SEEM TO CORRECT THE STRUCTURAL FAULTS. THEY NEED TO BE TREATED AS DESCRIBED. NOR DOES THE CORRECTION OF THE STRUCTURAL FAULTS REPLACE THE NEED FOR THE NUTRITION. THEY SHOULD BE USED TOGETHER.

NOW TO CONSIDER THE POSTERIOR SPHENOID.

IT IS WELL KNOWN IN APPLIED KINESIOLOGY THAT THE BILATERAL PSOAS WEAKNESS IS CAUSED BY AN OCCIPITAL FIXATION. THIS IS NOT DISPUTED. HOWEVER, DIFFICULTY IN CORRECTING THIS FIXATION ON A PERMANENT BASIS GRADUALLY LED ME TO THE CONCLUSION THAT AN UNKNOWN PROBLEM WAS PRESENT THAT WAS INTERFERRING WITH ITS CORRECTION. I BELEIVE THIS TO BE THE POSTERIOR SPHENOID.

THE POSTERIOR SPHENOID MAY BE FOUND IN SEVERAL WAYS:

FIRST: THE BILATERAL PSOAS IS WEAK IN THE CLEAR. BILATERAL INDEX FINGERS ARE USED TO THERAPY LOCALIZE SIMULTANEOUSLY AT EACH SIDE OF THE NASO-SPHENOID NEAR THE GLABELLA. THIS WILL STRENGTHEN THE BILATERAL PSOAS. THE PATIENT IS THEN ASKED TO TAKE A BREATH AND HOLD IT. THIS WILL CAUSE A WEAKENING OF THE BILATERAL PSOAS.

THE POSTERIOR SPHENOID IS CHALLENGED BY PRESSURE STRAIGHT POSTERIOR TOWARD THE OCCIPUT. THIS WILL CAUSE THE INDICATOR MUSCLE TO CHANGE STRENGTH. THE PRESSURE

DURAL TORQUE & POSTERIOR SPHENOID #6

IS THEN REAPPLIED WHILE THE PATIENT IS THEN ASKED TO TAKE A DEEP BREATH AND HOLD IT. THE MUSCLE AGAIN CHANGES STRENGTH. THIS IS CONSIDERED A POSITIVE CHALLENGE. THE DOCTOR'S THUMBS ARE THEN USED ON THE NASO-SPHENOID BILATERALLY TO MOVE THE SPHENOID POSTERIORLY WHILE THE PATIENT BREATHES IN. THIS IS REPEATED 4-5 TIMES. THE PATIENT IS THEN RE-CHALLENGED.

THE PATIENT THEN IS ASKED TO TURN INTO THE PRONE POSITION. THE COCCYX IS THERAPY LOCALIZED BILATERALLY. CARE MUST BE TAKEN HERE TO FIND THE EXACT SPOT OR IT WILL NOT THERAPY LOCALIZE. THIS CAUSES WEAKNESS IN A STRONG INDICATOR MUSCLE. THE PATIENT TAKES A BREATH AND HOLDS IT. THIS RESTORES STRENGTH TO THE INDICATOR MUSCLE. THE COCCYX IS THEN CONTACTED BILATERALLY, BY THE DOCTOR'S THUMBS AND BROUGHT POSTERIORLY ON THE PATIENTS INSPIRATION. THIS IS REPEATED 4-5 TIMES. THE COCCYX IS THEN RE-THERAPY LOCALIZED. IT SHOULD BE NEGATIVE.

THE PATIENT IS THEN ASKED TO TURN SUPINE. THE BILATERAL NASO-SPHENOID IS AGAIN THERAPY LOCALIZED WHILE THE METATARSAL HEADS ARE CHALLENGED AS A GROUP. ON THE OPPOSITE FOOT INVOLVED IN THE DURAL TORQUE. THIS WILL CAUSE A STRONG INDICATOR MUSCLE TO GO WEAK. THE METATARSAL HEADS ARE THEN CHALLENGED INDIVIDUALLY. ONE METATARSAL HEAD (USUALLY THE SECOND) WILL CAUSE WEAKNESS OF THE STRONG INDICATOR MUSCLE. THIS IS DIAGNOSED AS A DROPPED METATARSAL HEAD. THE METHOD I USED TO TREAT IT IS AGAIN THE PHALANGEO-METATARSAL BREAK TECHNIQUE OF JANSE, HOUSER AND WELLS.[II]

TO CONFIRM THAT THE DROPPED METATARSAL HEAD IS CONNECTED TO THE NASO-SPHENOID. THE PATIENT IS ASKED TO GET UP AND WALK AROUND THE TABLE. THIS CAUSES WEAKNESS OF THE BILATERAL PSOAS AND THE POSTERIOR SPHENOID TO RETURN. THIS IS RETREATED

DURAL TORQUE AND POSTERIOR SPHENOID #7

AS DESCRIBED ABOVE. A SMALL SIZED METATARSAL PAD IS TAPED TEMPORARILY UNDERNEATH THE OFFENDING METATARSAL HEAD. THE METATARSAL HEAD IS THEN CHALLENGED WHILE THE BILATERAL NASO-SPHENOID IS THERAPY LOCALIZED. THE CHALLENGE WILL BE NEGATIVE. THEN THE PATIENT IS ASKED TO WALK AROUND AND LIE SUPINE ON THE TABLE. THE BILATERAL PSOAS WILL NOT RETURN.

SECOND: FIND AN INDICATION OF BILATERAL PSOAS WEAKNESS ON THE TEMPERO-SPHENOIDAL LINE, BUT THE PSOAS MUSCLES ARE STRONG IN THE CLEAR. THE BILATERAL NASO-SPHENOID IS THERAPY LOCALIZED ON A 51%ER BASIS AND THE BILATERAL PSOAS BECOMES WEAK, THEN STRENGTHENS ON INSPIRATION. THE METHODS OF TREATMENT REMAIN THE SAME.

THIRD: THIS IS WHEN THERE IS NO APPARENT DURAL TORQUE, AND/OR NO APPARENT POSTERIOR SPHENOID. I HAVE FOUND THAT BY THERAPY LOCALIZING THE OCCIPUT BILATERALLY AND BY TESTING A STRONG MUSCLE NOT INVOLVED WITH A OCCIPITAL FIXATION, AN OCCIPITAL SUBLUXATION CAN BE FOUND WHICH RESPONDS TO INSPIRATION. THIS IS ADJUSTED BY CONTACTING THE SOREST POINT ON THE OCCIPUT AND THRUSTING TOWARDS THE GLABELLA ON INSPIRATION. THIS WILL USUALLY REVEAL THE POSTERIOR-SPHENOID AND THE DURAL TORQUE WHEN THEY ARE PRESENT. THEY ARE TREATED BY METHODS PREVIOUSLY DESCRIBED.

FOURTH: I HAVE ALSO FOUND INSTANCES WHERE THE PATIENT WOULD NOT SHOW THE POSTERIOR SPHENOID UNTIL A BILATERALLY WEAK PSOAS WAS FOUND IN THE CLEAR, SOMETIME THROUGH THE TREATMENT OF THE PATIENT. THIS WOULD THEN REVEAL THE POSTERIOR SPHENOID AND IN TURN THE DURAL TORQUE.

THE NUTRITIONAL SUPPLEMENT USED WITH THE POSTERIOR SPHENOID ALSO SEEMS TO BE

DURAL TORQUE AND POSTERIOR SPHENOID #8

PHOSPHORIC ACID AS SUPPLIED BY THE STANDARD PROCESS LABORATORIES PRODUCT: PHOSFOOD.

THE TECHNIQUE USED TO DIAGNOSE THE NEED FOR PHOSFOOD WITH THE POSTERIOR SPHENOID IS:

THE PATIENT IS SUPINE ON THE TABLE. THE PSOAS MUSCLES ARE TESTED. THEY ARE EITHER WEAK IN THE CLEAR OR ON A 51%ER BASIS WITH BOTH INDEX FINGERS ON THE NASO-SPHENOID. A DROP OF PHOSFOOD IS PLACED ON THE PATIENT'S TONGUE. THIS WILL CAUSE THE PSOAS MUSCLES TO AGAIN BECOME STRONG.

THE DOSAGE I USE IS THE SAME AS IN THE DURAL TORQUE. I.E. 10 DROPS IN WATER. (6-8 OZ) THREE TIMES A DAY FOR A WEEK AND THEN 10 DROPS ONCE A DAY UNTIL THE BOTTLE IS USED UP.

AS IN THE DURAL TORQUE, THIS DOES NOT SEEM TO CORRECT THE STRUCTURAL FAULTS. THESE NEED TO BE TREATED AS DESCRIBED. NOR DOES CORRECTION OF THE STRUCTURAL FAULTS REPLACE THE NEED FOR NUTRITION. THEY SHOULD BE USED TOGETHER.

THE PATIENT IS THEN UPON FINISHING THE REST OF THE TREATMENT ASKED TO PUT ON HIS SHOES, AND TO REMAIN STANDING FOR A LEAST 2-3 MINUTES WHILE DRESSING. THEN THE WEIGHT BEARING IS CHECKED BY TESTING AT LEAST THREE DIFFERENT MUSCLES. THE INDICATOR MUSCLES SHOULD REMAIN STRONG.

SUMMARY:

THE TREATMENT OF THE DURAL TORQUE BY CORRECTING THE HIDDEN FRONTAL BONE RO-

TATION. THE ELEVATION OF THE METATARSAL HEAD, BY MANIPULATION AND MAINTAINING IT WITH A METATARSAL PAD ON THE SIDE OF THE GREATER FOOT TURN IN. TREATING THE POSTERIOR SPHENOID BY PRESSING POSTERIOR-WARD ON THE BILATERAL NASO-SPHENOID WITH INSPIRATION. CORRECTING THE COCCYX BY BRINGING IT STRAIGHT POSTERIOR WITH THE BILATERAL THUMBS ON INSPIRATION. ELEVATION OF THE INVOLVED METATARSAL HEAD BY MANIPULATION AND MAINTAINING IT WITH THE METATARSAL PAD ON THE OPPOSITE FOOT, APPEAR NOT ONLY TO BE RELATIVELY SIMPLE BUT EFFECTIVE AS WELL.

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AKSP BARIOUS POST GRADUATE ADVANCED COURSES.

II. CHIROPRACTIC PRINCIPLES AND TECHNIQUE 2ND EDITION. J. JANSE D.C., R.H. HOUSER D.C. AND F.F. WELLS D.O.D.C. PAGE 605.
PHALANGEO-METATARSAL BREAK TECHNIQUE. 2ND PARAGRAPH ILLISTRATION PAGE 606 FIGURE 150-A.

THE METATARSAL HEADS

LOUIS F. DONNER D.C.

ABSTRACT: THE METATARSAL HEAD DROP AS ANOTHER CAUSE OF BILATERAL MUSCLE WEAKNESS, WHEN THE MUSCLES OF THE KNEES, THIGH AND PELVIS ARE BILATERALLY WEAK IN THE CLEAR, THE METATARSAL HEADS MAY BE INVOLVED.

IF ANY WORK HAS BEEN DONE ON THE METATARSAL HEADS IN APPLIED KINESIOLOGY, I AM UNAWARE OF IT.

AFTER TREATING THE DURAL TORQUE AND THE POSTERIOR SPHENOID BY ADJUSTING THE DROPPED METATARSAL HEADS INVOLVED, IT OCCURED TO ME THAT OTHER MUSCLES COULD BE INVOLVED WITH THE METATARSAL HEADS. I HAVE FOUND THIS TO BE TRUE REGARDING THE MUSCLES OF THE THIGH, PELVIS, KNEE, ABDOMEN AND LOWER BACK ON A REMARKABLY CONSISTANT BASIS. THESE ARE IN ADDITION TO THE OTHER CAUSES OF BILATERAL WEAKNESSES.

THE INVOLVEMENTS FOUND WHEN THE MUSCLES ARE BILATERALLY WEAK IN THE CLEAR, TESTED SEPERATELY ARE:

QUADRACEPS (RECTUS FEM)	1ST METATARSAL HEAD
TENSOR FASCIA LATA	4TH METATARSAL HEAD
SARTORIUS	2ND METATARSAL HEAD
HAMSTRINGS	5TH METATARSAL HEAD
PSOAS	2ND & 4TH METATARSAL HEAD
PILIFORMIS, GLUTEUS MED, MIN. & ADDUCTORS	5TH METATARSAL HEAD
GLUTEUS MAXIMUS	5TH METATARSAL HEAD
RECTUS ABDOMINUS	3D METATARSAL HEAD
POPLITEUS	1ST METATARSAL HEAD
QUADRATUS LUMBORUM	2ND METATARSAL HEAD

METATARSAL HEAD DROPS #2

THESE METATARSAL HEAD DROPS ARE ADJUSTED BY THE PHALANGEO-METATARSAL BREAK TECHNIQUE OF JANSE, HOUSER AND WELLS.[I] DEPENDING ON RE-OCCURANCE, I MAY USE THE METATARSAL PAD TO BRACE UP THE METATARSAL HEAD BY GLUEING IT IN THE PROPER PLACE IN THE SHOE. USING THE TECHNIQUE AS FOLLOWS:

1. A SMALL DOT OF YELLOW WATER SOLUABLE PAINT IS PUT ON THE OFFENDING METATARSAL HEAD WITH A COTTON SWAB.
2. THE PATIENT IS THEN ASKED TO CURL THE TOES DOWN WHILE PUTTING ON THE SHOE AND KEEPING THEM CURLED UNTIL THE SHOE IS ON. THIS IS SOMETIMES QUITE DIFFICULT.
3. THE PATIENT IS THEN ASKED TO STRAIGHTEN THE TOES AND STAND UP PLACING THE WEIGHT ON THAT FOOT.
4. THE SHOE IS REMOVED. A SMALL YELLOW MARK SHOULD BE OBSERVED AT THE POINT OF CONTACT OF THE OFFENDING METATARSAL HEAD AND THE INSIDE OF THE SOLE OF THE SHOE.
5. A DAMP CLOTH IS USED TO WIPE THE PAINT FROM THE BOTTOM OF THE FOOT.
6. THE PROPER METATARSAL PAD IS THEN GLUED WITH HOT GLUE OR ANY QUICK DRYING GLUE. CENTERED EXACTLY IN THE CENTER LINE OF THE DOT, BUT SLIGHTLY POSTERIOR OR HEEL-WARD FROM IT. THE FRONT OF THE METATARSAL PAD JUST TOUCHING THE EDGE OF THE DOT.

THE METATARSAL HEADS MAY BE INVOLVED UNILATERALLY OR BILATERALLY.

SUMMARY:

THE METATARSAL HEADS ARE INVOLVED IN BILATERAL MUSCLE WEAKNESSES OF THE KNEES, THIGHS, PELVIS, ABDOMEN AND LOW BACK.

METATARSAL HEAD DROPS #3

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PRIORITY MECHANISM & SPINAL FIXATIONS

LOUIS F. DONNER D.C.

ABSTRACT: BY PRIORITIZING TREATMENT AND SEEKING INDICATORS THAT THERAPY LOCALIZE, CHALLENGE AND/OR ARE WEAK IN THE CLEAR, IT IS POSSIBLE TO FIND FIXATIONS THAT WERE PREVIOUSLY UNKNOWN.

I WAS GREATLY IMPRESSED WITH TWO PAPERS WRITTEN ON BODY PRIORITIES. THESE ARE "BODY PRIORITIES AS DEMONSTRATED BY A DENTAL SPLINT"[I], BY SHELDON DEAL D.C.N.D. AND "THE BODY KNOWS---ASK IT",[II] BY MICHAEL ALLEN D.C.N.D. AND SHELDON DEAL D.C. N.D. YOU ARE REFERRED TO THESE FINE PAPERS FOR THEIR RATIONALE AND TECHNIQUE.

I USUALLY START BY POSTURE ANALYSIS, THEN BLOOD PRESSURE, SITTING, LYING, STANDING. THE PATIENT THEN LIES SUPINE AND IS CHALLENGED FOR THE DURAL TORQUE AND POST SPHENOID, CHALLENGED FOR THE HYOID AND SP-21. AT THIS TIME, I RE-CHECK THE FINDINGS THAT I TREATED THE LAST TIME I SAW THE PATIENT OR WHEN I EXAMINED HIM. I DO THIS IN THE EXACT SEQUENCE OR ORDER THAT I FOUND THEM IN, THE PREVIOUS TIME.

AS A GENERAL RULE, I FIND THAT THOSE FINDINGS HAVE ALL HELD THEIR TREATMENT AND RESPOND NEGATIVELY TO THE MUSCLE TEST, POSITIVE THERAPY LOCALIZATION AND/ OR CHALLENGE THAT WAS USED TO DIAGNOSE THEM ON THE PREVIOUS VISIT.

IF ONE FINDING HAS NOT RETAINED ITS THERAPY AND IS AGAIN POSITIVE AS REVEALED BY THE TEST THAT REVEALED IT THE PREVIOUS TIME, I USE THAT AS A STARTING PLACE FOR THE BEGINNING OF THIS TREATMENT. IF I DO NOT FIND THIS SITUATION, I PROCEED TO PALPATE THE TEMPORAL-SPHENOIDAL LINE AND SEARCH FOR A MUSCLE OR PAIR OF MUSCLES WEAK IN THE CLEAR. I USE THE WEAKNESS IN THE CLEAR, POSITIVE THERAPY LOCALIZATION, OR CHALLENGE, AS THE BODY LANGUAGE INFORMING ME THAT THIS PARTICULAR

PRIORITY MECHANISM & SPINAL FIXATIONS #2

SITUATION IS READY TO BE TREATED NOW. I THEN USE THAT AS THE BEGINNING OF MY SEQUENCE. I TREAT THAT SITUATION AND CONTINUE TO SEARCH AND TEST THE TEMPORAL-SPHENOIDAL LINE INDICATED MUSCLES UNTIL I FIND ANOTHER MUSCLE OR PAIR OF MUSCLES WEAK IN THE CLEAR. TREAT THOSE, AND RE-TEST UNTIL I FIND ANOTHER IN THE CLEAR SITUATION. IF I DO NOT FIND ANYTHING, I THEN TEST INDICATED POSTURAL MUSCLE DEVIATIONS, CATAGORY I, II, ILLIO-CECAL VALVE, CRANIAL FAULTS, FEET, GAIT, PITCH, ROLL, YAW, TILT, ETC. UNTIL I FIND SOMETHING THAT SHOWS A POSITIVE THERAPY LOCALIZATION OR CHALLENGE AND TREAT THAT SITUATION. AFTER THAT, I GO BACK TO THE TEMPORAL-SPHENOIDAL LINE, TEST THE MUSCLES INDICATED UNTIL I FIND AN "IN THE CLEAR SITUATION". IF I DO NOT FIND ONE, I CONTINUE TO EXAMINE AND SEARCH OTHER POSSIBILITIES UNTIL I FIND SOMETHING POSITIVE TO TREAT. TREAT THAT, AND GO BACK TO THE TEMPORAL-SPHENOIDAL LINE AGAIN AND KEEP RETURNING TO THE TEMPORAL-SPHENOIDAL LINE UNTIL I CAN ACCOUNT FOR, AND TREAT, ALL OF THE INDICATORS PRESENT, (TEMPORAL-SPHENOIDAL LINE AND POSTURAL EXAMINATION).

THE KEY TO MY TREATMENT IS TO FIND ALL OF THE MUSCLE WEAKNESSES IN THE CLEAR. DOING THINGS THIS WAY, I HAVE UNCOVERED A GREAT DEAL OF BILATERAL MUSCLE WEAKNESS THAT I WAS UNAWARE OF BEFORE, AND AS A RESULT, MANY MORE FIXATIONS THAN I THOUGHT EXISTED. I FIND MANY MORE FIXATIONS THAN I DO SUBLUXATIONS IN THIS MANNER. THESE FIXATIONS ARE: (BILATERAL MUSCLES WEAK IN THE CLEAR).

HAMSTRINGS	ATLANTO-OCCIPITAL OR THORACIC 7-8-9
BILATERAL STERNO CLIEDO MASTOID	CERVICAL DORSAL
INFRASPINATUS	CERVICAL DORSAL
CORACOBRACHIALIS	CERVICAL DORSAL
SUPRASPINATUS	CERVICAL DORSAL
SUBSCAPULARIS	THORACIC 1-2-3

PRIORITY MECHANISM & SPINAL FIXATIONS #3

LATISIMUSS DORSI	THORACIC 2-3-4
PECTORALIS MAJOR CLAVICULAR	THORACIC 4-5-6
PECTORALIS MAJOR STERNAL	THORACIC 6-7-8
SATORIUS	THORACIC 8-9-10
QUADRACEPS (RECTUS FEMORIS)	THORACIC 9-10-11
PSOAS AND ILLIACUS	THORACIC 10-11-12
QUADRATUS LUMBORUM	LUMBAR-1-2-3

FINDING THE SAME FIXATION ON BILATERAL WEAKNESS ON A REPEATED BASIS, WITHOUT SEQUENCE CHANGE, IS PROBABLY INDICATIVE OF NUTRITIONAL SUPPORT NEED.

SUMMARY:

THE PRIORITIZING OF TREATMENT AND THE USE OF THE "WEAK IN THE CLEAR" CHALLENGE, OR THERAPY LOCALIZATION, REVEALS FIXATIONS OF THE SPINE WHEN THEY WERE NOT EVIDENT PREVIOUSLY.

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EVALUATION OF THE ACTIVATOR INSTRUMENT EFFECTIVENESS IN SACROILIAC JOINT OSSEOUS SUBLUXATIONS

By Daniel H. Duffy, D.C.

ABSTRACT: Twenty cases of sacroiliac joint subluxations were adjusted by the activator instrument. None of the twenty cases responded to this form of manipulation when assessed by therapy localization and challenge.

Protocol for this short study was to use therapy localization, challenge and rib head tenderness as indicators required to be present in order to be included.[1] Fourteen patients showed evidence of posterior ishium subluxations and six showed posterior ilium subluxations. Interestingly enough, two of the posterior ilium patients showed a long leg on the side of involvement and both had "sciatica", with pain radiating down the lower limb and into the foot. Leg length by itself is not pathognomonic of the sacroiliac joint position and must be checked by challenge technique in every case, regardless of other subjective palpatory signs etc. This writer often finds a posterior ilium on the long leg side when the patient is in the adrenal exhaustion stage and the sartorius and gracilis muscles are weak in the clear.

Following diagnosis as to the proper line of drive etc., the activator was used twenty times on each subluxation and the patient rechecked for therapy localization and challenge. No responses were obtained on any of the patients. This short study proposes that the activator is not effective in the correction of the category two, osseous subluxation of the sacroiliac. Both patients with radiating pain were successfully relieved by the sacroiliac adjustments and required no intervention into lumbar disc adjustment.

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SPLIT BRAIN ACTIVITY AND HEART FUNCTION. A CASE STUDY VALIDATION OF A NEW TECHNIQUE BY ENDOCARDIOGRAPHIC TRACING

By Daniel H. Duffy, D.C.

ABSTRACT: Tapping of the sternum and posterior thoracic area during proper hemispheric brain activity has been demonstrated to have immediate, demonstrable effects on physical parameters (range of motion etc.) and to negate the effects of reversible split brain dysfunction involving tapping of the cranium. This paper demonstrates the effect of this technique on the endocardiograph.

Goodheart has shown that activation of the left and right hemisphere by counting and humming produces muscle strength changes in selected cases. He has developed in-depth procedures to negate these dysfunctions. Tapping of the cranium on the side of brain activity producing muscle-strength-changes during activation of the opposite side has been found to negate the original dysfunction, however this in turn has been found to be reversible by reversing the side of tap and brain activity. This new procedure of tapping the sternum etc., negates the reversibility of the original procedure. Effects are demonstrable on the endocardiograph as seen in figure 1.

A 63 year old female patient with the complaints of sudden onset weakness, dizziness, nausea and subsequent migraine showed weakness of the left psoas muscle only during left brain activity which which responded to the usual cranial tap and was reversible. At this point an endocardiographic tracing was performed. A smooth baseline was established on the tricuspid, aortic and pulmonary positions and a tracing of all valves made. The patient was then tapped on the right side of the sternum and associated posterior thoracic area and another tracing was immediately taken of the mitral area. Elapsed time between tracings was less than four minutes and all settings remained the same on the recording instrument. While leaving the office the patient made an unsolicited comment of having a greatly improved sense of well being and did well in the days following the treatment. The change in the mitral valve area is obvious and offers clinical proof that the heart does indeed act like a "second brain".

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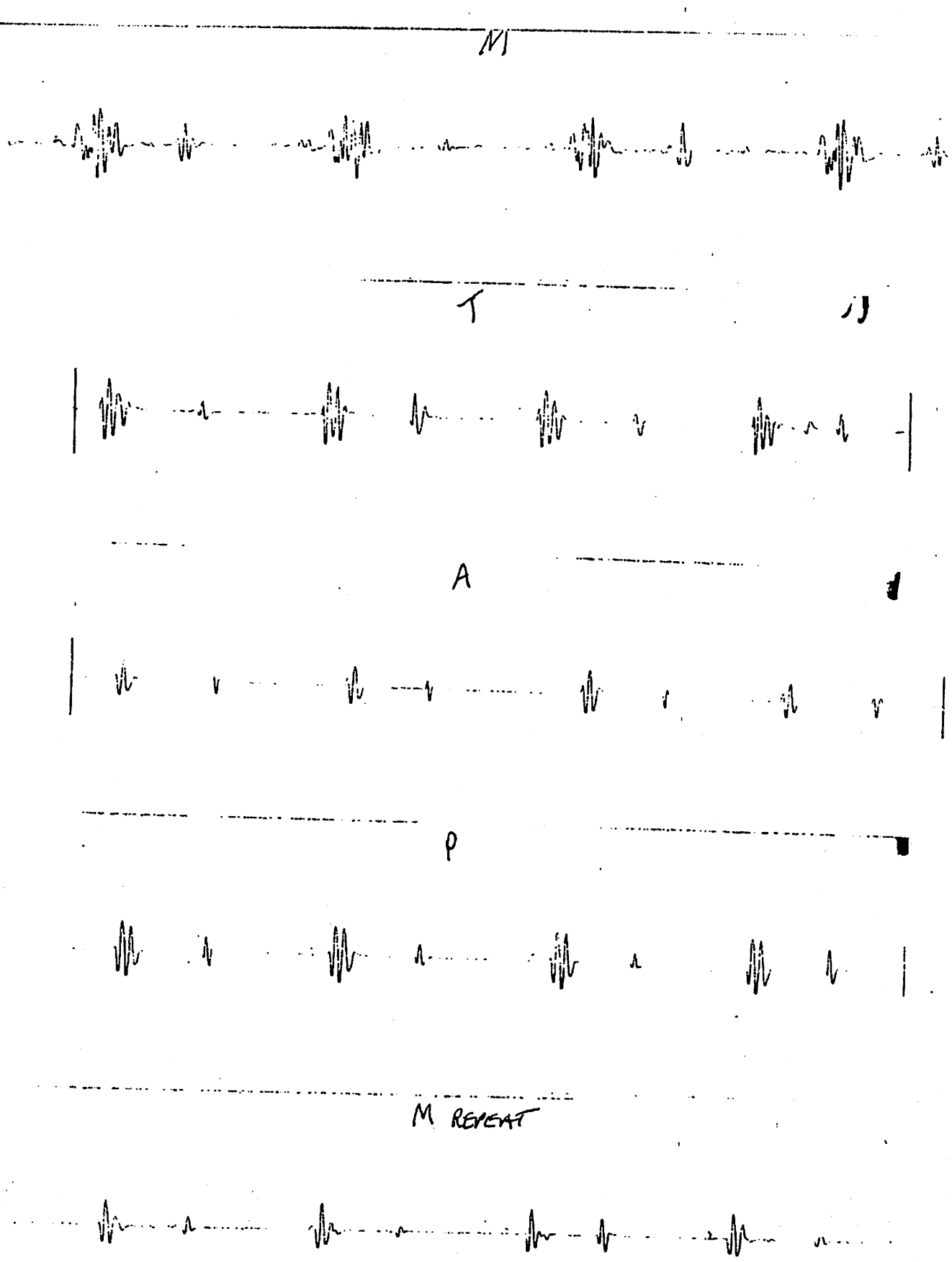

FIGURE 1

TESTING CONSISTENCY AMONGST EXPERIENCED APPLIED KINESIOLOGISTS

By David P. Engel, D.C.,D.I.C.A.K.

ABSTRACT

An effort to evaluate inter-examiner consistency of commonly tested muscles revealed a random pattern of results. This paper describes the testing format and offers some suggestions in explanation of the results.

INTRODUCTION

On several occasions I have observed a curious discrepancy. Two or more doctors testing the same muscle on the same patient will observe different results. This phenomenon has been frequently repeated at seminars, workshops and when examining a referral from another applied kinesiologist. The confusion which often clouds one's observations and techniques at meetings may explain the discrepancy under these circumstances and testing a patient in different offices at different hours, etc. may offer further explanation for referral testing inconsistencies. Not so easy to explain, though, is when two or more experienced applied kinesiologists observe markedly different results when testing the same patient in a quiet, undisturbed atmosphere where most of the contributing variables are eliminated. In this research project, I sought to carefully evaluate the presence of this phenomenon and offer possible explanations for its existence.

MATERIALS AND METHODS

On October 13, 1984, at Drs. Goodheart and Schmitt's seminar on Chiropractic Diagnosis in Detroit, Michigan, I requested the aid of five experienced applied kinesiologists for this project. The six of us each tested a series of commonly tested muscles on the same subject. With the exception of myself, who tested the subject first, none of the doctors were aware of the results of any other doctor's tests. The testing, requiring fifteen minutes, took place in an undisturbed and relatively quiet corner of the seminar meeting room at a lecture break. The subect arose and walked after each testing series to avoid fatigue and all the testing was performed during the Bladder Meridian Horary period to avoid excessive energy shifting. All muscle tests were performed in the same sequence on a standard height portable table with the patient supine (Table one). The doctors evaluated each muscle by grading its testing strength in one of the following four categories:

a. "strong" or totally intact

b. mostly "strong", exhibiting slight "weakness"

c. mostly "weak", but with some strength noted

d. "weak" or turned off

I observed for any variation in inter-examiner testing technique. The subject was asked to subjectively evaluate the amount of testing pressure exerted by each doctor. (Table Two)

RESULTS

Of all the twelve muscles tested, all examiners agreed upon three. All but one agreed upon an additional five muscles. Of the five muscles with single dissentions, examiner #1 was responsible for three of them and examiner #6 for the other two. Interestingly, these examiners were noted as using the hardest and lightest pressures respectively. Of all the tested muscles, only the left and right Sternocleido-mastoideus received more than one evaluation (in these cases two) of being totally weak, yet received impressions from three other examiners as being strong. Cross referencing muscle evaluations by each examiner and comparing examiner testing pressures tempts one to extrapolate many conclusions. In my opinion, companion studies with possibly tighter controls are needed before more conclusive observations are made.

DISCUSSION

I believe we should consider the many possible contributing factors to the observed inconsistancy. For example, I've heard it stated that a doctor who finds a frequency of weakness in a particular muscle should be checked for that weakness him/herself. For example, on a day of observing Dr. Gary Klepper in Dr. Goodheart's office, which I had done on several previous occasions, I noticed something unusual.

Dr. Klepper found several cases of Thoraco-Lumbar fixation patterns (indicated by weak Lower Trapezius muscles) which he treated throughout the day. I don't recall if Dr. Klepper found that pattern on me as we exchanged treatments at the end of the day, but I found the fixation on him. Was the apparent fixation in Dr. Klepper responsible for the unusual frequency of that phenonenon in his patients on that day? Just how does a doctor's energies, attitudes, health or experiences affect what he or she finds with each patient? Is the testing inconsistency observed in this project a reflection of varying levels of expertise or some entirely different factor? Perhaps we should, in further evaluation of this phenonenon, appreciate each physician's unique contributions to patients and how this distinctive quality each individual has may also affect what we find in our patients.

CONCLUSIONS

The results of this study invalidate a concept which has been almost universally accepted (based upon the frequency and variety of forms with which I've heard it expressed). That concept is as follows: greater testing pressure is directly proportional to the number of "weak" muscles found, otherwise known as "if you didn't find it weak, you're not testing hard enough".

ACKNOWLEDGEMENTS

Dr. George Goodheart has inspired me to be a careful observer. Dr. Lance West has emphasized the importance of caring for people as opposed to just treating them. Dr. Charles Rybeck's input and ideas greatly aided in this project's interpretation. Dr. Alan Beardall's observations and insights inspired this research. To all of these I owe my deepest gratitude.

TABLE ONE

EXAMINER #	PECTORALIS MAJOR STERNAL		PSOAS MAJOR		SARTORIUS		STERNO CLEIDO MASTOID		RECTUS FEMORIS		PECTORALIS MAJOR CLAVICULAR	
	LEFT	RIGHT	L	R	L	R	L	R	L	R	L	R
1	a	c	a	b	a	c	a	b	a	a	b	a
2	a	a	a	a	a	b	a	a	a	a	a	a
3	a	a	a	a	a	a	b	d	a	a	a	a
4	a	a	a	a	d	a	d	a	a	a	a	a
5	a	a	a	a	a	d	d	a	a	a	a	a
6	a	a	b	a	b	a	a	d	a	a	a	d

TABLE TWO

TESTING PRESSURE OF EXAMINERS

EXAMINER #			
1 = 4	2	5	3 = 6

HEAVIER (TESTING PRESSURE) LIGHTER

TABLE ONE

EXAMINER #	PECTORALIS MAJOR STERNAL		PSOAS MAJOR		SARTORIUS		STERNO CLEIDO MASTOID		RECTUS FEMORIS		PECTORALIS MAJOR CLAVICULAR	
	LEFT	RIGHT	L	R	L	R	L	R	L	R	L	R
1	a	c	a	b	a	c	a	b	a	a	b	a
2	a	a	a	a	a	b	a	a	a	a	a	a
3	a	a	a	a	a	a	b	d	a	a	a	a
4	a	a	a	a	d	a	d	a	a	a	a	a
5	a	a	a	a	a	d	d	a	a	a	a	a
6	a	a	b	a	b	a	a	d	a	a	a	d

TABLE TWO

TESTING PRESSURE OF EXAMINERS

EXAMINER #			
1 = 4	2	5	3 = 6

HEAVIER (TESTING PRESSURE) LIGHTER

MULTI ORGAN CHALLENGE TECHNIQUE

BY KENNETH S. FEDER, D. C.

Abstract:

This paper will present a technique to be used when multiple organ/gland involvement exists and reveals a priority organ.

After having utilized techniques involving two organ therapy localization to determine the major organ or priority organ with satisfactory results, there were many occasions where I was unable to determine a priority organ/gland which would negate muscle weakness.

The following technique has been helpful in determining the priority system when multiple organ therapy localization is required:

Procedures:

1. Determine muscle weakness by standard muscle testing procedures.
2. TL to the NL to determine if NL involvement exists (should not affect muscle weakness.)
3. Continue to hold original TL while patient uses other hand to try to negate original weakness (if no strengthening occurs, move on to Step 4.)
4. If no change in original muscle weakness occurs after individual contact of both suspected organs/glands; then have the patient continue with two separate hand contacts on the suspicioned organs while the doctor challenges the third suspicioned reflex point to attempt to negate the original weakness by finding the true

priority organ/gland. The challenge is performed by a tapping of the alarm point or neurolymphatic points. This procedure involves a trail and error searching for the priority organ, but once the organ/gland has been determined, desired results can be achieved.

Treatment:

Once the priority system has been discovered, normal treatment utilizing AK procedures should be employed.

To nutritionally challenge - have the patient simutaneously therapy localize to two different organs suspected of being influenced by a third priority organ and determine which additional organ nutritional component will negate the therapy localization.

Conclusion:

The above treatment system has afforded me a useful technique in controlling the recidivism of certain patients' complaints once the priority organ influence has been discovered and treated.

STUDENT SURVEY

Avery H. Ferentz, D.C.

ABSTRACT: A survey of eligible attendees, licensed practitioners or students at healing arts college, at an ICAK 100 hour course given in New York City by Dr. Avery H. Ferentz. Various data was collected.

The Questionnaire given:

(I) For Students

1) Age
2) Sex
3) School Attending
4) What semester are you in?
5) Did you have chiropractic care before going to school?
6) If so was it Applied Kinesiology (AK)?
7) Do you currently get chiropractic care?
8) If so is it AK?
9) Have you had AK in school yet?
10) Have you taken any other ICAK approved classes?
11) What was your first exposure to AK?

(II) For Licensed Practitioner:

1) Age
2) Sex
3) What license do you hold?

4) What school did you graduate from?

5) What year did you graduate?

6) What state do you practice in?

7) Have you taken any other ICAK approved classes?

8) Do you use AK in your practice?

9) If so, how long have you used it?

10) What was your first exposure to AK?

The results of the survey are as follows:

Student Responses: 38 Polled

20 Males (53%)
18 Females (47%)

Ages	# of Students	Percentage
20-25	13	(34%)
26-30	13	(34%)
31-35	9	(23%)
36-40	1	(3%)
41-45	0	(0)
46-50	1	(3%)
51-55	1	(3%)

Schools:

New York Chiropractic College	35	(92%)
Los Angeles College of Chiropractic	1	(3%)
National College of Chiropractic	2	(5%)

Semester in School	# of Students	Percentage
1	2	(5.)
3	2	(5%)
4	8	(21.)
5	8	(21.)
6	1	(3%)
7	10	(27%)
8	4	(11%)
9	1	(3%)
10	2	(5%)

Chiropractic Care:

Those that received chiropractic care before going to school Numbered 34 (89%), of which only 9 (26%) received chiropractic AK care. Those currently receiving chiropractic care number 36 (95%) of which 24 (66%) received chiropractic AK care. Only 3 students (8%) had already taken AK in school (as it is offered in 9th semester), while 3 other students have audited the course.

Previous ICAK approved AK classes were taken by 12 (32%) of the students.

First exposures to AK were as follows:

From a friend at school	14	(37%)
From an intern at school clinic	7	(18%)
From their own chiropractor before school	10	(27%)

Touch for Health demonstrator	2	(5%)
From a relative who is a D.C.	3	(7%)
School AK club	1	(3%)
In a nutrition class	1	(3%)

Licensed Practioners Responses: 18 Polled

14 Males (78%)

4 Females (22%)

Ages	# of Students	Percentage
26-30	9	(50%)
31-35	7	(40%)
36-40	1	(5%)
65-70	1	(5%)

All attendees in this group are licensed as chiropractors.

Of those attending: 16 were chiropractors

1 was a dentist

1 was an osteopath

The areas they practice in are:

New York	8	(44%)
New Jersey	4	(22%)
Massachusetts	3	(18%)
Pennsylvania	2	(11%)
England	1	(5%)

Schools graduated from:

New York College of Chiropractic	4	(23%)
Columbia College of Chiropractic	1	(5%)
National College of Chiropractic	3	(17%)
Logan College of Chiropractic	1	(5%)
Life College of Chiropractic	1	(5%)
Northwestern College of Chiropractic	1	(5%)
Palmer College of Chiropractic	3	(23%)
Sherman College of Chiropractic	1	(5%)
Adio Institute of Chiropractic	1	(5%)
University of Maryland Dental School	1	(5%)
Northern College of Physiotherapy (England)	1	(5%)

Years of Graduation:

1951	1	(5.)
1977	1	(5.)
1978	1	(5.)
1980	2	(11.)
1981	4	(23.)
1982	4	(23.)
1983	5	(28.)

14 practitioners stated that they used AK in their practices. While ICAK approved classes had been attended previously by 12.

First exposures to AK were listed as the following:

In school	9	(50.)
Personal Chiropractor before school	2	(17.)
Touch for Health Class	1	(5.)
Fellow Chiropractor	4	(23.)
At a seminar for another chiropractic technique	1	(5%)
Fellow DDS	1	(5.)

Although the size of the sampling for this survey is rather small (56 people) it does provide a base to which future samples can be added. One observation made from this data is that of the 56 students polled 19 of them (42.) knew about AK before ever coming to chiropractic school. This is significant in light of how new a technique AK is.

[1] ICAK BYLAWS CONCERNING [illegible]

ARTICULAR AND HOLOGRAPHIC CRANIAL FAULTS

by

Terry L. Franks, D.C.

ABSTRACT: This paper discusses the diagnosis of holographic cranial faults and the most common primary type of cranial fault.

If vertebra can be holographically subluxated and need to be mechanically corrected, it would seem reasonable for the same situation to occur with cranial bones. By combining Dr. Goodheart's concept of a holographic subluxation which he describes as an interosseous fault[1], with Dr. Beardall's hand moding for diagnosis[2], this writer has been able to differentiate articular cranial faults and holographic cranial faults.

The hand mode for an osseous articular cranial fault, as taught by Dr. Beardall, is placing the tip of the index finger on top of the middle of the first knuckle of the thumb. The hand mode for a holographic subluxation is placing the pad of the little finger against the pad of the thumb. By combining these two modes simultaneously, we now have the indicator for holographic cranial faults.

The paper presented in 1984 at the summer meeting of the fronto-sphenoid lesion as a primary fault[3], appears to frequently have a holographic counterpart. As the cranium is compressed medially either in utero or in childbirth, numerous changes take place. The two most significant alterations appear to be (1) the jamming of the fronto-sphenoid suture and (2) a holographic bulging unilaterally or bilaterally at the frontal eminances; or a midline bulge at the metopic suture on the frontal bone. If holographic correction is not made when needed, the body will adapt with a reoccuring series of articular cranial,

muscular, spinal, and TMJ changes. Primary frontal sphenoid cranial faults and primary holographic frontal cranial faults have been unresponsive to respiratory correction, resonance tapping and TMJ correction. Their need for specific mechanical correction is the same as found in the spine. A system indicating both proper sequence and the primary fault is critical when working in this area.

As our knowledge expands, the source problems of structural, chemical, and emotional imbalances are getting simpler. The traditional complexity of cranial therapy is being replaced by fundamental patterns which are correctable by basic chiropractic techniques.

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Scars: a Case Study

Bert T. Hanicke, D.C.

ABSTRACT: This paper is a report of a patient that demonstrated the relationship between poorly – healed scars and symptoms at a location distant from the scar.

The effects that scars have on the human body have been known in classical acupuncture for hundreds of years, but most comments were about scars across meridian pathways and how they interfere with energy movement along the meridian.

In 1979, Dr. William Khoe, M.D.[1.] reported about a German physician, F. Huneke, M.D. who had treated patients by injection of procaine into the scars wherever found. This produced many remarkable improvements in pain throughout the body.

In 1983 George Goodheart[2.] reported a method of treatment of scars which can be used by Applied Kinesiology doctors and does not involve injection of a substance into the scar, but rather the use of spray – stretch technique.

My own study of scars was initiated after reading Dr. Khoe's article and further reinforced by Dr. Goodheart's publication. I use a procedure containing aspects of both methods.

THE CASE: A middle – aged female patient presented herself at our office complaining of intractable low back pain and right leg sciatica of six months' duration. She had been treated with muscle relaxants and non-steroid

anti-inflammatory medications with no relief. She then was treated for two months by competent standard chiropractic care with little improvement. She was then refered to my office and an Applied Kinesiology examination was performed. She was treated with standard Applied Kinesiology (muscle balancing, categories, etc.) . At this time she experienced some relief for the first time, although still only partial. During the examination a large abdominal scar was noted extending from the xiphoid process to the pubes, the result of an earlier gall bladder surgery. Although the scar was several years old, it still looked angry with red and bluish areas throughout. The patient was scheduled for an acupuncture analysis. Readings were taken along the length of the scar with a Dermatron. These were found to vary between low, high, and normal at different points along the scar. The abnormal areas were treated electrically until the readings were near normal. After the scar treatment the patient reported greatly decreased pain and more comfort on movement, even before any other treatment was given. Using a combined approach of treating the scar with the acupuncture unit and standard Applied Kinesiology the patient improved rapidly to complete alleviation of pain.

The patient's husband commented that her scar "looked different, like it finally was healed" two days after the treatment, spontaneously and with no mention from his wife of the type of treatment being used. I have seen this rapid completion of healing on many other patients for whom I have treated scars.

Treatment of scars can be accomplished by electrical current, normalizing both hyper- and hypo-tonic areas, by spray-stretch technique, and/or massage. I have found electrical current of low intensity and spray-stretch technique best for office treatment. With severe involvement I have the patient massage the scar daily with a vitamin E ointment such as Tocophoderm by Nutri-Dyn.

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DIAGNOSIS AND TREATMENT

OF

MENINGEAL TORQUE

BY

CHRISTOPHER L. HARRISON, D.C.

DIPLOMATE

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ABSTRACT

This paper deals with the meningeal coverings of the brain and of the spinal cord. The author deals with the fundamental anatomy of the meninges and conveys some new ideas regarding their physiology, including his own.

The author shares a technique that he has developed to assist in diagnosing the situation wherein the meninges are torqued.

Effective theraputic manipulations are given and valuable clinical insights are offered so that the reader may add to his/her theraputic regimen armamentarium.

The meninges have been, are now, and in all liklihood will continue to be a very important aspect of theraputic manipulative therapy. There appears to be a surge of inquest into the physiological and pathophysiological manifestations of the meninges in the present day literature for both the allopathic and the natural healing arts. Serious study of the meninges within the osteopathic profession can be traced back to Sutherland in the early 1900s with his book, The Cranial Bowl, being published in 1939. DeJarnette, has carried the Sutherland legacy forward into the Chiropractic profession which has been the breeding ground of present day cranio-meningeal-sacral theraputics in the Chiropractic profession. With the advent of Dr. George Goodheart and Applied Kinesiology, this legacy has not only been handed down to AK practioners but has been refined, streamlined and made more practical in clinical use.

It is not the purpose of this paper to bore the reader with long boring descriptions of the antatomy of the meninges, however, I thought it best to incorporate some facts that are pertinent to the subject of meningeal theraputics.

In the cranial vault the dura mater is composed of two layers: the inner layer is composed of dense connective tissue which is lined on its inner surface by a single layer of flat cells. The outer layer , which actually forms the periosteum of the inner cranial bones, while similar to the inner layer is much richer in blood vessels and nerves. The important thing to remember here is that the spinal meninges do not have this periosteal connection as do the meninges covering the brain. The vertebrae have their own periosteum and the inner layer of the cerebral dura corresponds to the spinal dura mater. In other words, the spinal

dura has one layer as compared to two layers in the cerebral dura. This one layer is the outermost layer of the spinal meninges and is not contiguous with the spinal canal of the vertebrae, thereby affording a freer movement of the cord and its meninges within the canal. The spinal dura contains more elastic tissue than the inner layer of the cerebral dura but is quite similar otherwise. The outer surface of the spinal dura is covered with a single layer of flat cells and is separated from the periosteum by the epidural space. [1].

We are all aware of the traditional concepts of meningeal function, ie, that of forming a liquid cushion for protection of the central nervous system as well as a mechanical wall protective device. When one considers the biochemical makeup of the cerebro spinal fluid, one could assume a nutritional responsibility of the meningeal apparatus as well.

It is the protective function, especially the mechanical part that I choose to deal more directly with. The meningeal structures of the spine, most notably the dura can be likened to a ski boot of a skier. Certainly there is a restrictive function of the boot, whereby it prevents gross movements that would be injurious to the skier. However, it does allow some movement within the boot so that the muscles and articulations of the foot and ankle can function enough to allow the skier balance and control for his run. Here, it is good to remember that the skier can move the ankle in 360 degrees of motion but is restricted by the boot so that harmful movements are prevented. So, too with the dura of the spine, the cord can be moved by trunk-spinal motion in 360 degrees of movement but movement that would be injurious to the cord is prevented by the restriction of the dura. This protective restriction is dynamic. This dynamicity presents a confusing clinical picture unless the observer is well aware of how this mechanism works.

Apparently, different sections of the dura can restrict spinal movement while other sections can relax all at the same time. It is this property of elasticity and plasicity along with it tremendous strength and holding power that makes the dura both dynamic and clinicly confusing. I think we sometimes get into the trap of visualizing the dura as one long mass of tight, super strong, tough, unyielding tissue, when in reality it is both this and a dynamic plastic, yielding substance that can protect its master, the spinal cord,at varying levels with varying modes of protection (restriction and tolerance). It should be suffice to say that the dura is free from cervical 3 to sacral 2 with a filum terminale attachment to the coccyx.

Any movement therefore, can initiate the dura to protect the cord, if that movement is indeed insultive to the cord.

I have incorporated two diagnostic indicators to assist in monitoring dural stress.

1. A general muscle indicator can be used when the insulting position to the cord is accenuated. A common example of this is a patient who is a typist and is asked to bend the head and neck forward and as often the case, the cord is offended due to her extensive hours in this position. In this case, any muscle indicator will weaken when tested.
2. A specific therapy localization to the distal coccyx can also be used to show dural stress. In some cases this does not show up in the clear and if the patient is asked to assume a position that is offensive to the cord and this inititates dural stress then the therapy localization will be positive.

A large percentage of patients with positive meningeal tests have one thing in common: an inner conflict stress syndrome. I have found this type of stress to be where the patient feels that he or she is up against

the wall, so to speak, with no solution to the problem so they have to just live with it and no end is in sight. A prime example would be a strongly religious Catholic or Mormon with traditional views of the family who is considering splitting up with their spouse and must consider, the children, the church, friends, family and their own personal needs. Either way they go will in one sense be a losing situation for them. Work conflicts, are very powerful in producing meningeal stress, especially when the person needs the job but can't get along with fellow employees or the boss. Here, in Silicon Valley where government contracts mean life or death to a business and where getting the product to market before the next generation of equipment hits the market, produces tremendous "deadline" stresses on the employees. We see many of our patients face the conflict of having to choose between their work or their families and personal life. Another example of meningeal stress in a patient is that of a 16 year old male, apparently, very healthy, a weight lifter, well nourished, very few weak muscles, but with a very acute low back pain syndrome. There was no history of trauma. He made no mention of any stress in the history but upon the second visit I asked him if he had some conflict going on. At first he said no, and when I said I think that you do, he turned around and looked at me as if to say how do you know that? He went on to say that he had just been released from prison on murder charges, was placed on probation and would be sentenced the following week, which could be several years. This is the type of "up against the wall" conflict that we see produce meningeal stress almost every day. It is as if the body turns against itself or perhaps better said; tightens itself up when it finds that it is in conflict with itself and there is no answer to the problem.

TREATMENT

The patient is placed on their side with their back

in the fetal position. The underwear is pulled down just enough to expose the rectum, thereby covering the genitalia. The doctors middle finger is inserted into the rectum cephal and posterior to make contact with the coccyx. Once a firm contact on the coccyx is made,instruct the patient to bring both lower extremities posterior as far back as possible with the knees locked. You will see that locking the knees is very important due to the pulling of the hamstrings and the fascia on the posterior knees and thighs. Simultaneously have the patient extend the trunk as far posterior as possible with the head in extension. Actually the position is almost the same as that of opisthotonos, which is as you know,meningeal tetany seen in meningitis and tetnus. The patient should hold the position for 30 seconds while the doctor holds the coccyx with a very firm pressure posteriorward. Then have the patient return to the fetal position and the doctor maintains posterior pressure for 30 seconds in the fetal position. I usually have the patient perform three extensions and three flexions with a firm pressure on the coccyx throughout the entire procedure.

The finger inserted into the rectum can be diagnostic to a certain extent. The degree of tightness that you find in extension and flexion will advise you of the degree of tension in the lower meningeal area, however, I question whether a tightness on your finger will monitor all meningeal tightness for reasons explained above. However, at times, when the patient flexes the head and neck forward, there is a tremendous tightness felt on the inserted finger. It is interesting to note that we have placed the patient in the gait position and occasionally find a tightening in one of the gait positions and this has mildly correlated with the Isogai-short step gait that Dr. Goodheart has informed us about. We have had the patient rotate the head and side bend the head and have never felt any change in the tension of the inserted finger. However, when the neck

is moved,tension can often be felt on the finger but not necessarily so. Now here is an interesting point. If the meningeal structures are firmly attached to the foramen magnum and to the upper cervicals how can dural torque in the sacral area possibly cause cranial distortion other that occipital misalignment ? This would seem to dispel the theory that the meninges themselves cause the cranials and sacrum to reciprocate in misalignment except for the occiput. Now, I am not saying that they don't but with the firm dural attachment to the foramen magnum it is inconceivable to think that the coccyx for instance can misalign the sphenoid via the meningeal torque. This idea correlates with the fact that I have never felt any tension change with just head rotation and side bending without neck movement.

One of the most significant situations that you will see respond to meningeal therapy is the intervertebral disc syndrome. How many times have you seen patients with acute disc syndromes that responded slowly or poorly to therapy ? Usually with a meningeal, they respond almost immediately and I am convinced that many disc cases are the result of meningeal pressure on the vertebrae. Hemorrhoids, respond well to the release of meningeal tension. It appears that meningeal stress in the coccyxgeal area produces congestion in the hemorrhoidal veins and we have seen numerous cases where the hemorrhoids actually retract upon taking the inserting finger from the rectum. When performing cranial adjustments, you will find that by first working with the meningeals many of the faults will release or at least correct with only slight effort.

It is my sincere wish that you use the aforementioned information and utilize this most powerful tool in your quest to assist our fellow humans back to optimum health. It has been both a pleasure and a privilege to communicate with you.

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Some of these basic ideas and hypotheses were extracted from "Spinal Stressology" by Lowell E. Ward, with permission.

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ALLERGY TESTING IN INFANTS AND CHILDREN

John T. Hughes, D.C.

Abstract: An AK method of testing infants and young children for allergy to different substances is presented.

Infants and small children frequently have problems due to allergies.

We recognize that different subjects show different degrees of apparent muscle weakness to certain substances when ensalavated. We are all familiar with the weakening of the latissimus dorsi in response to sugar, in most subjects. We also know that the pectoralis major sternal division is weakened by various substances; however I feel that the bilateral weakening of the pectoralis major clavicular division , while strong individually, is our best indicator of allergies.

This bilateral test has been difficult in infants and small children, therefore we use the following method of surrogate testing for children who are too young to test on their own.

First, we make sure the Doctor and the Mother or surrogate have intact bilateral pectoralis major claviculars. Then we have the child make skin contact with the surrogate and test the bilateral pectoralis major clavicular of the surrogate. If they stay strong we can place suspected substances such as milk, choclate,wheat,etc in the mouth of the child and retest the surrogate.

If the initial test weakens the surrogate, it indicates a greater degree of allergy. In this case, we make a partial correction sufficient to bring strength to the bilateral PMC of the surrogate. We then follow the above proceedure of placing the suspected substance into the mouth of the child and retesting the surrogate.

If the child is too young to cooperate by touching the Mother, we use one of our support personel or preferably the Father, if avaliable, to hold the baby in order to make contact with the Mother. Make certain that the person holding the baby also has intact bilateral PMC's.

This proceedure is not only for our information but also to impress the Mother with the effect the substance ,being tested, has on the child.

Cooperation from the parents on dietary matters is more likely to follow.

Reference: Goodheart 1970 Research Manual

FINDING ADRENAL PROBLEMS VIA PUPILLARY REFLEX, POSTURAL BLOOD PRESSURE, KOENIGSBURG'S AND MUSCLE TESTING

by: ALEX P. KARPOWICZ, D.C.
D., I.C.A.K.

ABSTRACT

This paper is a follow up of my summer 1984 Research paper titled "Validation of Office Urinalysis Procedures and Treatment Procedures". In discussing the urinary test, in particular the Koenigsburg test, with Dr. Walter Schmidt, he felt that a verification of the accuracy of screening tests used to determine adrenal problems would be of significant importance. The Koenigsburg test of course is a Urinary test used to determine whether hypo or hyper adrenia may be present in patients. The normal being 17 to 25, above that to 60 being mild to severe hypo-adrenia below 17, especially below 10 being severe hyper-adrenia. The majority of my previous works had come from the information in Dr. Walter Schmidt's book "Common Glandular Disfunctions in the General Practice".

DISCUSSIONS

The criteria for more exactly determining that a patient has adrenal problems consisted of the Koenigsburg Sodium secretion test, postural blood pressure, the pupillary reflex and of course the associated muscle weakness. The blood pressure is taken from a sitting to standing position

and it should rise 4 to 10 mm of Hg. If it didn't rise or was below that (Dropped), it was indicative of a hypo-adrenic problem, whereas if it rose greater than 10 mm. Hg. a hyper-adrenic problem. In the normal pupillary reflex the pupils stay constricted for 30 seconds to light, if they dialated or vasilated in that period of time that was Paradoxial Pulmonary Reflex found in hypoadrenia. If it was a normal pupillary reflex we find this in Hyper adrenia. The associated muscle weakness in Applied Kinesiology are the Sartorius, the Gracilus, and the Soleus and Gastronemius. The muscle that we primarily tested because of frequency of involvement was the Sartorius. We tested or checked 142 Patients. Sixteen patients had a normal sodium level, 44 patients had a normal blood pressure when standing, 27 patients had a normal pupillary reflex and 77 patients had a normal muscle. Therefore out of 142 patients we found that 126 patients showed an abnormal sodium level, 98 patients showed an abnormal postural blood pressure, 115 patients showed an abnormal pupillary reflex, and 65 patients showed a Sartorius muscle weakness which clearly indicates a high degree of abnormality found in most patients when using these tests. Out of the 126 patients who showed an abnormal Koenigsburg test, 33 patients showed an abnormal postural blood pressure,

pupillary reflex and a related Saratorius muscle weakness showing all four positive on the four (4) various screening devices.

CONCLUSION

I think some interesting statistics developed out of this, first of all the importance of having the sodium test or Koenigsburg Test run on patients was self evident. Eighty nine percent of all patients showed an abnormal sodium level, which we previously said is indicative of an adrenal problem. Of the 126 patients who had an abnormal Koenigsburg test, 69 per cent of them also showed an abnormal postural blood pressure test. Eighty one per cent also showed a paradoxal pupillary reflex, 46 per cent showed an associated adrenal muscle weakness. Also noteworthy is that the 26 per cent of the 126 patients with the abnormal Koenigsburg had all four screening devices positive, which I think bears out the importance of using all tests and guaging the results of the total picture rather than picking any one test as a final determining factor. Also of interest is the fact that less than 50% of the patients that had an abnormal Koenigsburg test showed a related Sartorius muscle weakness. Granted they possibly had all the other adrenal related muscles been tested the percentage

would be somewhat higher but in my experience the great majority of times the muscle weakness shows in the Sartorius muscle and the purpose of this was simply to simplify things. Because of our stress related society, the dietary habits of the American people, and the general declining health of our populace that we find so much of an adrenal problem in our patients should be of no surprise. The fact that we have screening devices that apparently are so accurate in helping qualify people as having an adrenal problem I think makes the Koenigsburg test a vital part of armament in solving health problems in patients.

Lastly, but certainly not least of all is the thought of Dr. Goodheart who has said on repeated occassions not to use the muscle tests as the beginning and end but as a screening device and to correlate our Applied Kinesiology with other accepted Diagnostic tools to more accurately and definitely ascertain the proper diagnosis and subsequent treatment for our patients.

DR. GARY N. KLEPPER
CHIROPRACTOR

DIRECT MENINGEAL TRACTION THERAPY

ABSTRACT A technique is described which is of great value in the treatment of problems related to intense longitudinal meningeal compression. Rationale, parameters for measuring need, and specifics of application are discussed.

Introduction

An understanding of meningeal biomechanics has long been the basis for many of the techniques used in the chiropractic and osteopathic professions for correction of musculoskeletal problems. First described by Sutherland(1), alteration of tensions in the dura mater was one of the objectives of his cranial techniques. Introduced into the chiropractic profession and developed into a practical system of application by DeJarnette(2), attention to meningeal biomechanics became the heart of the system known as Sacro-Occipital Technique, which is the basis for much of the structural corrective work done by practitioners of Applied Kinesiology. Recent work by Dr. George Goodheart on dural torque and its correction(3) has done much to make objective the analysis of this phenomenon and has dramatically increased our understanding of the various factors contributing to its persistance and of how various types of health problems are manifestations of its existence. Other ICAK members have made significant contributions in this field, such as Dr. Terry Franks' observations on primary cranial faults(4),

and Dr. Alan Beardall's categorization of the multiple factors involved in the dural torque syndrome(5).

Inspired partially by the exceptional text on meningeal biomechanics by Dr. Alf Breig(6), our friend Dr. Lowell Ward developed a system known as Stressology which employs some new concepts which originated with him(7). Among the techniques employed in the Stressology system is a method of rapidly releasing microadhesions within the dural mechanism. This particular adjustment I have found at times to be of great value, and the purpose of this paper is to describe its rationale, diagnostic parameters suggesting its need, and the specifics of its execution.

Causes of Meningeal Tension

Many factors can contribute to the creation of longitudinal tension in the dura mater. The text by Breig describes the exact manner of how various space-occupying lesions of the spinal column and cord exert pathological tension in the various meningeal elements(8). Additionally, high velocity trauma such as the common whiplash injury can create a momentary intense tractional force on the dura mater which exceeds its physiological capability to elongate and thus damage the fibers of the dura mater, if at the time of the injury a force exceeding the tensile strength of the spinal ligaments and muscles is exerted in a vector causing an elongation or traction effect. This generally resolves into a general or local fibrosis of the dura which shortens it, inducing a significant longitudinal compressive tension in the dura mater, which can create a reduction in spinal length significantly below normal values(9)

if adequate extraspinal soft tissue defenses are insufficient to balance this compressive effect(10).

Another cause of meningeal tension, described by Goodheart, is spinal torque. By reducing the ability of spinal motor units to undergo normal counterrotation during flexion of the spine, spinal torque compromises the ability of the spine to avoid excessive hyperelongation during flexion, and thus pathological longitudinal meningeal tension is superimposed on the physiological meningeal tension which normally occurs during flexion of the spinal column(11).

In addition to the tension caused by space-occupying lesions, post-traumatic fibrosis, and biomechanical torque, there is evidence that the meningeal mechanism reacts to all stresses and acts as a repository of unreleased stress forces be they mechanical, biochemical, or emotional(12).

Recognizing Meningeal Tension

Regardless of the cause of longitudinal tension in the dura mater, the effects are the same. These can range from generalized defensive tension in the paraspinal muscles to intervertebral disc compression or even brain stem deformation.

The major concern here, however, is to recognize significant meningeal tension which would not respond to the usual untorqueing maneuvers.

One method of recognizing meningeal tension is by the presence of typical lesions which correlate with the presence of longitudinal meningeal tension. These include multiple levels of spinal fixations, especially recurrent fixations, intervertebral

disc compression, anterior coccyx, and multiple cranial faults, especially recurrent cranial faults. The diagnosis of a number of these faults all existing concurrently is a strong indication for the presence of extreme longitudinal meningeal tension, especially if the diagnosed lesions have been previously treated.

The methods suggested by Ward for examining the meningeal structures for undue tension involve the use of physiological indicators. One method is the simple palpation of tension in the anal sphictor by inserting a gloved finger and grading the tissue resistance noted. Anal tension generally shows a linear relationship to meningeal tension.

The preferred method described by Ward is what he calls synchronous testing. This name is derived from the fact that the spine, pelvis, cranium, and meninges function together as a single synchronous unit. Synchronous testing was described in my paper on the accelerator-defender concept which was published last year(13).

Synchronous Meningeal Testing

Synchronous meningeal testing consists of stressing an area of the dura, and noting a reactive contraction response which is seen as a shortening of the leg length and a posterior tilt of the base of the occiput on the same side.

The first step is to determine whether the left or right side is the side of dominant stress in the meningeal mechanism. Radiographically, this is seen on the A-P films as the side of decreased pelvic drop or increased pelvic lift on the sitting radiograph as compared to the standing radiograph(14). Physiologically, this is tested with the patient prone, and is

seen as the side that shows a contraction response by a shortening of the leg and a posterior tilt of the occiput when the anterior dura is challenged by a stress created by pulling the entire spine into extension as in the Cobra posture used in Hatha Yoga while pressure is held by the doctor on the coccyx, then releasing back to the neutral prone position to make the observation. For example, if in the prone position a left short leg is seen, and after the spinal extension challenge to the anterior meninges the left leg is seen to be shorter than before, and the occiput is more posteriorly tilted on the left than before, this is indication of a left stress dominant condition. This is because, as you recall, the anterior meninges is always the stress accelerator side of the meninges, and whichever side (left or right) functions in synchrony with the anterior meninges is the side of stress dominance.

Next, the posterior or defensive meninges is challenged by getting the patient into a hands and knees position with the lumbar spine arched up towards the ceiling and the head flexed to the chest, in order to stress the posterior dura by flexing the spine, again while tension is maintained by the doctor's contact on the coccyx. Then the patient is quickly returned to neutral prone position and again the leg length and occiput monitored. The contraction here should be on the side opposite that observed in the extension challenge, and is the side opposite stress dominance.

Now that the side of stress dominance is known, it is necessary to denote whether the side of meningeal tension needing

release is primarily the accelerator meninges (meninges on the stress dominant side), or primarily the defense meninges (meninges on the side opposite stress dominance).

This differentiation is made by noting the response to lateral flexion challenge of the dura. Again, with the patient prone and tension held on the coccyx to immobilize it, laterally flex the patient to the left, return to neutral and check leg length and occiput. This stresses the right-sided dura, and reaction is monitored by noting degree of contraction on the left. The degree of reaction on the left is proportional to the degree of pathological tension in the right side of the dura.

Next, repeat the test, only laterally flexing to the right, and noting the contraction response involving leg length shortening and posterior occipital tilt on the right side as a test of the degree of pathological tension in the dura on the left side.

Any contraction response which is pronounced (more than several millimeters) is an indication of the need for release in the involved meninges. If the pathological tension is noted primarily in the meninges on the stress dominant side, then an accelerator meningeal release is needed. If the pathological tension is primarily on the side opposite that of stress dominance, then an overdefense meningeal release adjustment is needed.

Release Procedure

The meningeal release adjustment consists of a traction applied to the coccyx by an intrarectal contact while traction is applied to specific areas of the dura by use of specific leg and

neck positions.

To release a area of the dura, first put slack into that area by flexing the head toward the point of tension, then traction the involved area by flexing the head away from the point of tension. For instance, to release the anterior dura, first flex the neck then fully extend it.

Release is concentrated in the lumbar and cauda equina area by doing the head movements while the legs are flexed with the knees drawn up to the chest till an increase in tension is noted at the coccygeal contact. Release is concentrated in the cervico-cranial area by doing the head movements with the legs fully extended. Release is concentrated in the thoracic region by holding the head in each position while the legs are pulled down and up, in order to mobilize the dura over the apex of the thoracic kyphosis.

To perform a full accelerator meningeal release adjustment, have the patient lie on their side with the head and neck completely off the end of the adjusting bench. Contact the coccyx with an intrarectal contact, and have your assistant grasp the patient´s ankles and draw the knees up toward the chest by flexing the patient´s hips and knees. In this position, the doctor flexes the patient´s head and neck fully forward than backwards twice to release the anterior meninges, then laterally flexes the patient´s head and neck fully toward the stress dominant side then away from the stress dominant side twice in order to release the dura on the stress dominant side. This releases the lumbar and cauda equina level of the dura. Next,

the head is held completely in flexion while the assistant pulls the legs fully into extension then returns them to flexion twice. These leg movements are repeated with the head and neck in extension, then laterally flexed toward the stress dominant side, then laterally flexed away from the stress dominant side. This releases the thoracic dura. Lastly, the legs are held in full extension by the assistant, and the head movements are repeated, with the head and neck first fully flexed then fully extended twice, then laterally flexed toward stress dominant side then away from stress dominant side twice. This releases the cervico-cranial dura and completes the adjustment.

To perform an overdefense meningeal release adjustment, the procedure is identical except for the order of the head movements. Here, the overdefense dura is targeted by first bringing the head and neck into extension then into flexion, and by first laterally flexing the head and neck ~~toward~~ away from the stress dominant side then ~~away from~~ toward it. Simply remember the rule to first put slack into, then stretch the area of meninges being targeted.

As experience with this technique is accumulated, it can be made more specific by only releasing the levels of primary tension, such as the cranial area only or the cauda equina area only.

Cautions and Contraindications

During the thoracic release phase of the adjustment, if the pathological tension is extreme, it will be very difficult to move the patient's legs. The assistant should bring the legs down into extension only a few inches into the zone where extreme

resistance is noted in the leg movements and extreme increase in tension is monitored at the coccyx. Forcing the legs past that point has the potential of inducing compression fracture at the thoracic apex.

This adjustment can be very uncomfortable for the patient. The doctor must talk to the patient before performing the adjustment, letting them know the rationale for the adjustment, and informing them that it may be quite painful.

If the meningeal tension is due primarily to emotional stress, this must be released before performing the adjustment or severe physical and emotional reactions can be expected.

Summary

In spite of the cautions and general lack of gentleness involved with this technique, its extreme benefits make it the technique of choice in many instances. On many occasions it has led to prompt symptomatic resolution when other techniques have failed, including those administered by other very competent doctors. I can only recommend its serious consideration by all doctors attempting to resolve problems related to longitudinal meningeal tension, as these problems are ubiquitous and can be severe.

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HOLOGRAPHIC MANDIBLE SUBLUXATIONS

David W. Leaf

Goodheart has theorized that a bone can subluxate, bend, within itself creating symtoms. He has demonstrated that the mandible can bend as to increase or decrease the angle formed by the ramus and the body. It has been found that the mandible will also bend so as increase or decrease the width. It appears that these subluxations can be determined from an analysis of the muscles found to be hypertonic.

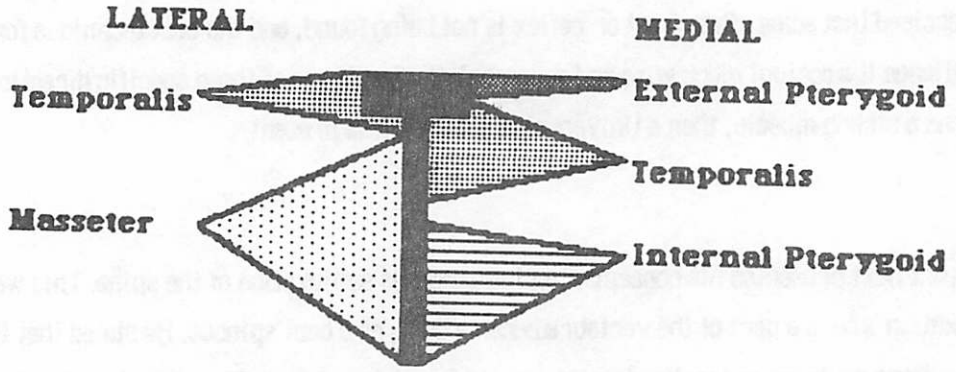
The first research by Goodheart that led to the concept of holographic subluxations was the discovery of the Universal cranial fault. This fault appears to be created at birth by forces against the occipital bone. The bone is bent within itself. This fault can be found by placing the patients fingers on both sides of the occiput and testing a strong muscle for weakening. Care must be exercised that some other fault or reflex is not being found, and the best technique for this is to challenge the occiput clock wise and counterclockwise. If one of these specific directions also weakens a strong muscle, then a Universal cranial fault is present.

Goodheart next presented his concept of an intraosseous subluxation of the spine. This was a subluxation where a part of the vertebra became bent, as a bent spinous. He stated that these subluxations could cause localized tenderness and symptomotology. The procedure for finding them was to place one finger of one hand on the transverse process and a finger from the opposite hand on the spinous process and test a strong muscle for weakening. He later added a procedure where the finger of one side of the body was used to touch the vedrtebral part on the opposite side of the body. This was due to polarity changes in the bone that occur when the bone is compressed. The treatment procedure was to test the two parts of the vertebra that were found on testing by either approximating or separating them. Once again a strong muscle is tested for weakening. When the direction is found that causes the strong muscle to weaken, a strong thrust is applied to the bone. Any localized tenderness will be immediately relieved.

In 1983, Goodheart demonstrated this phenomenom in relation to the mandible. The mandible was tested with the patients fingers touching along the ramus and forward on the body close to the symphysis menti. If a strong muscle was found to weaken, the ramus and mandible were challenged as to increase or decrease this angle. Once the direction was found, a strong thrust was applied to the mandible in this direction.

The mandible is an 'L' shaped bone. The major muscles of mastication attach to the ramus or to the body close to the angle of the jaw. The jaw joint is like a hinge. If you bend your hand into extension as far as possible and then bend your fingers at a ninety degree angle to your hand, you will have a rough facsimile of the jaw. Keeping your fingers locked into position, move your hand in flexion and extension. Try pushing against your fingers with the other hand. Changes in the angle between your palm and your fingers would represent the changes found in the original holographic mandible subluxation.

Examining the attachments of the muscles on the mandible led to the supposition that another type of holographic subluxation might exist. In this case, the masseter is counter-balanced by the force of contraction of the medial pterygoid. The temporalis, if hypertonic, would fight against itself leaving the masseter - medial pterygoid contractions as the main forces that could cause a medial or lateral imbalance within the ramus of the mandible.



MANDIBLE A - P VIEW

Patients are routinely being examined for holographic mandible subluxations when imbalances in the muscles of mastication are found. A video report on these findings will be presented at the I. C. A. K. meeting, and a follow up report on the statistical findings will be presented in the next Collected Papers.

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NUTRIENT TESTING EVALUATION

David W. Leaf

ABSTRACT : Patients were evaluated in a double blind study to determine if the findings of Triano, in regards to the efficiency of lingual and skin testing of nutrients, could be substantiated.

Triano, in Journal of Manipulative and Physiological Therapeutics, has reported the findings of a research project, funded by the International College of Applied Kinesiology, that sheds serious doubt on the efficiency of the testing of nutrients in relation to muscle weakness. This project was undertaken to determine if the findings of Triano were valid for another muscle-organ complex.

The testing of pharmaceutical agents sublingually is well documented as a valid method of absorption. Erdle, Schultz, Wetzel and Gross have shown that g-strophanthin can be measured in plasma concentrations fifteen minutes after sublingual administration. Cunningham has reported on hypotension after sublingual absorption of nitroglycerine. Klepzig reported in 1979 that molsidomine effected angina patients within ten minutes of a sublingual dosage, and Fallon, and others, reported nitroglycerine changing heart rates in less than two minutes after sublingual administration. Delayed absorption of nitroglycerine due to dry mouth has been discussed by Robbins. Gale, Gallon and Porter have recently written on the sublingual absorption of lorazepam as an improved method of absorption. The absorption of estradiol sublingually has been researched in both France and the United States with studies published on the positive effects of the administration in postmenopausal women.

Triano chose as his target muscle the latissimus dorsi. Teachings in Applied Kinesiology relate this muscle to the pancreas. Triano further chose four glandular substances for testing. In retrospect, it appeared to the author that the choice of four protein substances for testing was a poor choice as they would all contain amino acids that could be required for function by any other structure in the body.

For this study, the teres minor muscle was chosen. This muscle, supposedly associated with the thyroid, was picked for three reasons:

1. limited physiological functions
2. ability to determine which nutrients were necessary for proper functioning of the gland.
3. ease of testing
4. physiological parameters, subjective and objective, that could be utilized for patient evaluation

PROCEDURE

Four nutrients were chosen:

1. Niacinamide
2. Vitamin E
3. Inositol
4. Thyroid extract

The first three substances were considered to be inert as to their ability to effect thyroid function.

These substances were labeled A, B, C and D, and were sent with a questionaire of symptoms that are commonly found associated with imbalances of the thyroid (see figure 1).

The participants were instructed to test patients for a weakness of the teres minor. If one was found, they were to test the nutrients on the skin and then sublingually and record their findings. The patient was to then fill out the questionairre and if the doctor had biofeedback thermomter equipment, to record the patient's axillary temperature.

FINDINGS

A total of 388 persons were tested. The results of the skin tests were random in that 11% responded to niacinamide, 8% to Vitamin E, 9% to inositol, 8.5% to thyroid extract and 64% to no substance. The results were examined for changes based upon number of symptoms marked or tmperature readings that were recorded. In both cases, no changes over 2% in the above reported findings were found.

These findings were then broken down on the basis of the number of symptoms recorded on the symptom list. Those patients that marked less than four symptoms showed no significant difference for testing on any of the nutrients. The results were 21% for niacinamide, 24% for vitamin E, 23% for inositol, 26% for thyroid extract and 7% for no response. Totals are over 100% due to multiple results by some patients. Those patients that marked over 20 symptoms (4) tested positive to all nutrients when tested sublingually. Those patients that marked more than three and less than twelve symptoms proved to be most significant. The results were niacinamide 3%, Vitamin E 5%, inositol 4%, thyroid 92% and no response in 3%. The results for the group of thirteen to sixteen responses were niacinamide 13%, Vitamin E 15%, inositol 11%, thyroid extract 91% and no response in 1%. The patients with seventeen to twenty responses were found to show niacinamide to respond in 28%, Vitamin E in 22%, inositol in 24% and thyroid extract in 100%.

Those patients that were examined for axillary temperature, 114 of the total, showed that 96% of those with more than four symptoms marked had temperatures below 97.8, with an average of 97.2. Those patients that marked over 15 symptoms had temperatures averaging 97.0.

DISCUSSION

As is evident, there was a variable relationship between the introduction of the thyroid extract and the response of the teres minor muscle. When compared to the number of symptoms that the subject recorded, as the symptoms increased above four the testing sublingually proved to correlate. The skin testing proved to be random and without correlation to symptom number. It is interesting to note that in those patients that marked over forty percent of the symptoms other responses than thyroid extract were found and the number of 'false positives' increased in relation to the number of symptoms marked. In the four subjects marking over seventy percent of the symptoms, all nutrients tested positive.

CONCLUSIONS

1. Sublingual testing may be used in patients exhibiting subclinical and clinical symptoms of nutrient deficiencies.

2. Skin testing of nutrients gives random findings.

3. 'false positive' findings are found in those patients who have many symptoms. In these cases, the doctor's judgement should be trusted more than the strengthening of a weak muscle after the sublingual testing of a substance.

TERES MINOR SYMPTOM SURVEY

Please mark and of the following symptoms that you have had or are currently having over the past three months.

INSOMNIA	INCREASE IN WEIGHT
NERVOUSNESS	DECREASE IN APPETITE
CAN'T GAIN WEIGHT	FATIGUE EASILY
INTOLERANCE TO HEAT	RINGING IN THE EARS
HIGHLY EMOTIONAL	SLEEPY DURING THE DAY
FLUSH EASILY	SENSISTIVE TO COLD
NIGHT SWEATS	DRY OR SCALY SKIN
THIN, MOIST SKIN	CONSTIPATION
INWARD TREMBLING	MENTAL SLUGGISHNESS
HEART PALPITATIONS	COARSE HAIR, FALLS OUT EASILY
INCREASED APPETITE	MORNING HEADACHES
PULSE RATE FAST AT REST	SLOW PULSE BELOW 65
EYELIDS OR FACE TWITCH	INCREASED FREQUENCE OF URINATION
IRRITABLE OR RESTLESS	IMPAIRED HEARING
CAN'T WORK UNDER PRESSURE	NO AMBITION

TESTING

AGE: SEX:

Mark with a check any strengthening of a weak teres minor:

Substance	A	B	C	D
Skin				
Sublingual				

AXILLARY TEMP:_____

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A REPORT ON INTENSIVE TEACHING

David Leaf

The author and George Goodheart have been teaching Applied Kinesiology in an intensive manner using video tapes and class notes. This paper discusses the ability of the students to learn more material than previously deemed possible. The results of this indicate that Applied Kinesiology can be taught so that at least twice as much material can be assimilated by the students in a week-end course than was originally planned in the original 100 hour basic course.

After teaching the original 100 hour course in Applied Kinesiology, five times, Leaf determined that there must be a better way to organize the material. The material was originally re-worked for a course in Montreal, and with the help of Barbara McQueeney, the material was reorganized in 1981 and 1982 into a logical and systematic presentation. The original intent was to present material that interrelated. Leaf began preparing class notes for the teaching of Applied Kinesiology in 1979. These were expanded over the next five years to cover all of the topics in Applied Kinesiology. Recognizing the excellent books prepared by Walther, the notes were designed to briefly and succinctly present each topic.

In March 1984, the author and George Goodheart presented the first seminar under the new format. This format revolves around video tape projected, by a ten foot video projector, of each topic including actual testing of muscles and treatment of conditions found. A complete packet of notes was handed out for each student. These notes covered each topic that was covered in the seminar so that only notes of clarification needed to be added by the student. Workshop time was used after every two to three topics. Goodheart remarked during the seminar that it was the first time that he had seen everyone working during a workshop. On Saturday and Sunday, the following topics were presented:

1. Basics of Applied Kinesiology

2. Discussion and testing of the following:
    a. Psoas
    b. Hamstrings
    c. Gluteus Maximus
    d. Quadriceps
    e. Sartorius
    f. Tibialis Anterior
    g. Tibialis Posterior
    h. Peroneus longus and brevis
    i. Peroneus tertius

j. Flexor hallicis longus & brevis

k. Gastrocnemius

l. Soleus

3. Structural Procedures

a. Aerobic - Anaerobic testing

b. Reactive muscle testing

c. Fascial Technique

d. Strain Counterstrain

e. Ligament Interlink

f. Skin involvement

g. Treatment of scars

4. Foot/Ankle Problems

a. foot subluxations

b. adjusting procedures

c. Taping procedures

5. Gait Imbalances

a. Gait testing

b. Stride Length

6. Analysis of Gait

a. Muscles of walking

b. Visual analysis of gait imbalances

7. Effects of Pterygoid contractions on leg range of motion

It must be pointed out again, that all of these topics were workshopped by the students.

In October, 1984, the author's presented their second seminar on cranial and TMJ problems. The following topics were covered:

1. Basic Applied Kinesiology procedures

2. Cranial anatomy

3. Sutural faults including cruciate

4. Inspiration - expiration assist faults

5. Sphenobasilar flexion - extension faults

6. Temporal bulge - Parietal descent

7. Glabella Fault

8. Universal Fault

9. Frontal Faults

10. Cranial Muscles

11. Temporomandibular Joint

    a. importance
    b. muscles
    c. reflexes
    d. examination
    e. visual analysis of imbalances
    f. treatment

12. Neurological Tooth

13. Hyoid Imbalances

Before the class and after the completion of the course, a test was given to the students. They were instructed to answer as many questions as they could in a five minute period. The following are the results of that test.

The test was handed out at the beginning of the cranial seminar and the students were asked to code the test with any two letters and one number. They were to write down the code and use it the next day when they retook the test.

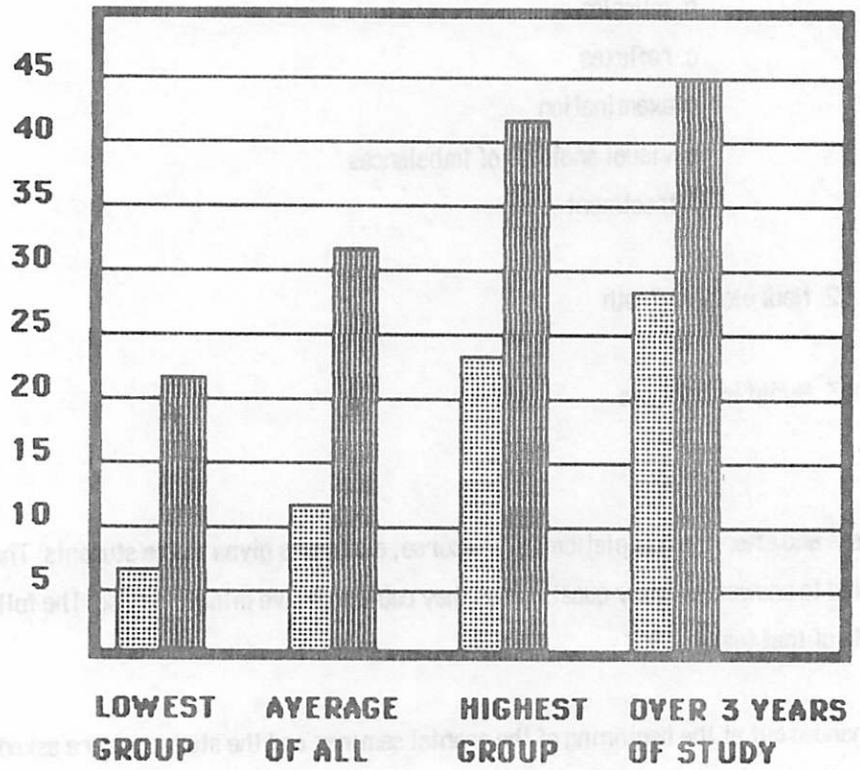
The class totaled 124 students of which 97 completed both sections of the test. On the first test, the students were to record the number of months that they had been studying and/or using Applied Kinesiology. The range was from 0 months to 15 years with an average of 14 months.

The results of the first test showed an average score of 12 correct responses. The second test results showed this rise to 32 correct answers.

When the improvement in the lowest original scores was examined, it was found that those who had scored less than ten on the first test, an average of 7, improved to an average of 21.5.

The test scores of those who originally scored over twenty correct responses, an average of 24, improved to 42.9 correct responses after the test.

Further evaluation of the results show that the highest original test score was 38 by a student with 36 months of study. The students who had been studying Applied Kinesiology for over three years scored an average of 29 correct responses on the first test and 45 responses on the second test. It should be noted that the students who had no prior experience in Applied Kinesiology originally scored 7 responses and after the test scored 21.5 responses, almost equal the original test scores of those attendees that had over three years of study.



It is the authors opinion, that this method of teaching needs close scrutiny and possible adoption as at least an alternative teaching method to those already being employed by the teaching diplomates.

ALLERGIES AND THE CHRONIC HYPO-ADRENIC PATIENT

Nancy L. McBride, D.C.

ABSTRACT: The relationship of allergy factors and their association with the patient suffering with chronic hypo-adrenia is discussed.

During a seminar in Los Angeles, Calif. in 1984, Dr. Walter Schmitt, diplomate I.C.A.K., reviewed the testing of patients for allergies using food substances he had assembled in a kit. I was concerned about the possibility of rancidity with grains especially and spoilage of other items in the kit. I asked Dr. Schmitt about the usage of the testing kit available from Seroyal Products. Dr. Schmitt said he had not used this product but didn't think it was as substantial for testing as real food stuffs. A few weeks later I had a conversation with my Seroyal distributor John Kail. He spoke of many reports of very good results from many of his Doctor clients who were using the test kit The kit contained 81 small vials of liquid ortigen solution. The substances ranged from a wide variety of trees and plants, food groups, grains, pollens, dust, grasses, molds, insect and animal factors, and the microbiotic group #76-81 having to do with various blood diskrasias.

Rather than test a patient for all 81 items individually, I divided the items into what I considered to be logical groups, For example all of the air borne tree items in one group, all the air borne plant, grass and pollen items in one group, all the similar foods in one group, such as all the animal proteins in one group. We narrowed 81 items down to 12 groups of dropper bottles.

When we encounter a patient who demonstrates clear signs of hypo-adrenia (positive Ragland's test , chronic weak adrenal group muscles, adrenal N-L indicators positive for an extended period of time, etc), and the patient is taking the recommended adrenal support products, and following the recommendations for diet and stress management, then we get suspicious that this patient may be constantly being irritated by an allergy that the body cannot overcome hence fatigued adrenals. Many times when one of these patients contacts the adrenal N-L any strong indicator muscle will become weak and very often not even a double adrenal N-L contact makes any difference. In other words the patient can contact both adrenal N-L simultaneously and a strong indicator muscle will weaken. With the patient contacting either one or both adrenal N-L reflex points and a strong indicator muscle weakness prevalent we begin to narrow down which if any of the 12 aforementioned groups makes a change in the muscle test. In other words cross indexing the ortigen against the adrenal N-L reflex. If sniffing the ortigen substance in the vial causes the strong indicator muscle which was weakened by T-L of the adrenal N-L reflex to become strong again, we hold that ortigen vial aside for testing of all the groups later. After the patient has sniffed all the 12 vials, we then pull out from the kit the individual members of each group that produced a positive response with the adrenal T-L. We then have the patient sniff the individual members of each vial group. Whenever a food item that

can be eliminated from the diet gives a positive test response, we recommend that the patient remove that item from the diet for a period of three weeks. At the end of that time this item can be reevaluated after the patient has reintroduced the item back into the diet. All other items which cannot be easily avoided are given to the patient in a homeopathic type ortigen remedy to be taken orally by the patient supplied through the office from Seroyal products. Seroyal also produces a glandular support in liquid form from freeze dried glandular substances. Depending on the individual patient and the number of items which have been positive on testing I make a judgement about whether to support the patient with the glandular support while they are detoxifying the allergen with the ortigen. I ahve used this method on over 100 patients and I am thrilled with the results but most importantly so are the patients and the feed-back I receive is only positive. One patient who had been badly stung with insects years ago had a slight flare-up much like many patients do when placed on other homeopathic remedies but her general response was remarkable especially the elimination of her PMS symptoms. Many times when we tell a patient what foods they are"allergic" to after the testing has been completed they remark - but I eat that all the time!

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HOMEOPATHIC SUPPORT FOR THE CHRONICALLY ILL PATIENT

Nancy L. McBride, D.C.

ABSTRACT: Many patients have repeated occurrences with disease processes that may be due to hidden remnants of other problems or diseases encountered many years prior; such as streptococcuc infections, scarlet fever, herpes progens, poison oak or ivy, mononucleosis, and a score of others. I believe that when the "disease energy" is still in the body it can be the source of disease "flare-up".

Although I am very much a novice in the field of Homeopathy I have experienced the effect of taking homeopathic remedies myself and with very positive results. About two years ago I was treating a patient who on her case history report indicated that in her youth she had had mononucleosis. This patient had chronic hypo-adrenia and chronically weak liver indicators. I checked the liver N-L refles eliciting a weak pectoralis major sternal division weakness and tested her with one 30x mononucleosis pellet on her tongue and the weakness was abolished. I then removed the pellet, rinsed her mouth and rechecked her against the N-L for the adrenals and again the same response. The weakness was again abolished. After the patient had finished a small bottle of 30x mono pellets at4 4x/day. I kept her off any for a week although the adrenal N-L response was no longer positive. I then rechecked her for the next x I could get which was 200X and against her now intact adrenal N-L T-L. I felt that if she did not need to go to the next X that possibly putting a pellet on her tongue would

weaken her now intact adrenal reflex. When it did not I asked her to take the whole vial at 4 pellets 4 x/day. In the meantime her symptoms of severe fatigue virtually disappeared.

On another occassion we checked a chronically unstabile low back with repeated piriformis. gluteus maximus and medius weakness against herpes progens 30x by placing one pellet on the tongue and the weakness was eliminated. The patient was asked to take 4 pellets 4x/day for one bottle of 250 pellets. The patient was free of low back pain within one week but is now taking 200X pellets. On another occasion we treated a patient with recurrent bladder infections. I asked her if she had ever been treated for a strep infection and of course she had. Of the three strains of strep homeopathic remedies she cleared a bladder alarm point reflex with one pellet of streptococcus on her tongue of 30x. Within three days the pain in her bladder tissue which had been there for months was gone. Of course the patient was also given support nutrients to support chronic infection. I have used this method on over 25 patients with very successful patients especially when I was at my wits end. All of the remedies mentioned were ordered from Standard Homeopathic Pharmacy, Los Angeles. and are from Hylands formulas.

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LINGUAL ASCORBIC ACID TESTING: A METHOD OF IDENTIFYING INEQUITIES IN NUTRIENT DISTRIBUTION

Kerry M. McCord, D. C.

Abstract: A statistical review of Lingual Ascorbic Acid Tests performed in a "not-so-classical" fashion on over 1,000 patients suggests Lingual Ascorbic Acid testing as a viable method of identifying nutrient distribution inequities. Measurements were made and recorded over the past three years from tests simultaneously performed on right and left sides of the tongue. This provided an opportunity to observe the potential difference in the oxidation-reduction reaction between L-ascorbic acid and 2,6 dichlorophenol-indophenol from side to side.

As Dr. George Goodheart began to discuss concepts of right and left brain function,[1] hypothalamic/pituitary influence on body chemistry,[2] and dural torque phenomena[3] and their relationships to nutrient identification and distribution, I became interested in the concept of selective distribution of nutrients. It occurred to me that an easily accessible method of monitoring a disturbance in distribution was Lingual Ascorbic Acid testing.[4,5] We began performing Lingual Ascorbic Acid tests simultaneously on the right and left sides of the tongue and observed evidence of selective distribution of tissue Vitamin C.

Below is a statistical review of testing performed on 1,112 patients over the past three years.

For the purpose of this discussion, 19 seconds and below was used as the outer limit of normal.

Normal (i.e., adequate tissue Vitamin C) 701

Abnormal (i.e., inadequate tissue Vitamin C) 411

That is, 37% of all patients tested exhibited inadequate tissue Vitamin C.

In measuring inequitable distribution of Vitamin C as measured by simultaneous Lingual Ascorbic Acid Tests on right and left sides of the tongue, patients with distribution discrepancies equaled 219:

113 patients with a 5-9 second discrepancy

53 patients with a 10-14 second discrepancy

21 patients with a 15-19 second discrepancy

23 patients with a 20-29 second discrepancy

9 patients with a 30+ second discrepancy

(patients with less than a 5 second discrepancy were not counted in this review.)

These figures translate into relatively significant percentages as 19.7% of all patients tested were found to manifest an inequitable distribution of tissue Vitamin C when measured simultaneously on right and left sides of the tongue.

9.5% of the total patients exhibited discrepancies at 10 seconds and above

4.7% at 15 seconds and above

2.9% at 20 seconds and above

.8% at 30 seconds and above

The patients tested often manifested some major structural or neurological disturbance, such as a simple Category 2 or a hypothalamic/pituitary or dural torque problem. The correction of these and other faults tended to normalize the discrepancies seen in bilateral Lingual Ascorbic Acid testing.

CONCLUSION:

Using the Lingual Ascorbic Acid Test in the manner previously described appears to be a viable means of measuring a distribution inequity relative to Vitamin C. One might deduce that if Vitamin C is unequally distributed, suggesting some functional disturbance affecting the neurochemical mechanisms that are responsible for distribution, then other nutrients may also be unequally distributed.

The results of this study suggest that simultaneously measuring the oxidation-reduction reaction between L-ascorbic acid and 2,6 dichlorophenol-indophenol on each side of the patient's tongue may provide information regarding the relative integrity of the structural and neurological mechanisms associated with distribution of the various chemical agents necessary for normal cellular function.

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CBAKI--COMPUTERIZED BIOMAGNETIC APPLIED KINESIOLOGIC INSTRUMENT

by Galen D. Methvin, D.C.

The purpose of this paper is to describe CBAKI in sufficient detail as to it's diagnostic capabilities so that the reader may determine it's desirability for use in the individual practice as well as research.

DESCRIPTION:

The CBAKI unit consists of a computer section, a computer-generator interface, an electromagnetic field generator and a linear motion sensor.

The computer section involves off-the-shelf components from Apple IIE, Corvis 16 megabyte hard disc drive, a 100-200 megabyte random access tape storage, a single floppy disc, two printers, an optional telephone modem, program for operating CBAKI, an office management program integrated with the CBAKI program and optional additional software for word processing, etc.

The computer-generator-sensor interface simply allows communication/control functions to be exchanged among computer, generator and sensor.

The electromagnetic field generator section provides the extraneous magnetic fields necessary to access the body's internal computer and cause leg length variances directly analogous to muscle strength variances associated with applied kinesiologic findings.

The linear motion sensor converts linear leg movements into voltage output that is monitored by the computer and interacts in a measurable, predictable and consistent manner with the operating program for CBAKI.

The computerized biomagnetic applied kinesiologic instrument (CBAKI) functions as a diagnostic unit for structural faults of the skull, spine and pelvis as well as any other joint one may wish to diagnose. The information gained is the existence/location of the structural fault,

the direction of thrust required for appropriate treatment, and, subsequent to treatment, re-examination with CBAKI reveals the accuracy/completeness of the treating physician's effort. (Treatment is, of course, repeated until the CBAKI indicates a complete setting of that joint for that point in time, should the post-check so require.)

Performance of the above test-treatment-test sequence, assuming no substantial delay in patient movement/dressing time should consume an average total of about ten to twelve minutes where one CBAKI serves an entire office.

The CBAKI performs one test every 1/4 to 1/2 second on the average and is capable of some 2060 tests, should they be required. As a practical matter, it is quite rare that more than a few hundred tests will be required on any patient and the routine number is more nearly a hundred or fewer. The more difficult the case and/or the more nearly well (stabilized) the patient, the greater is the number of tests required in order to find what to do for the patient that day. The program for CBAKI allows the setting of depth of diagnostic acumen for each patient and will automatically follow those instructions until informed to do otherwise by the operator.

A technician handles all the CBAKI operations while the physician concerns himself/herself with treatment as indicated to be appropriate by the instrument. Light emiting diodes (LEDS) are lit up by the computer on the spinal section in either red or green color. The light, when lit, designates within approximately one fourth of an inch the level of the spine which must be contacted for treatment in the case of a posteriority. Red denotes posteriority. Contact should be made with the thenar eminence approximately one fourth of an inch below the green light which denotes an anteriority. The technician will, of course, be required to use an

appropriately colored marker to mark the skin exactly adjacent to the lighted LED when the CBAKI has completed it's diagnostic run through.

A print out of the skull and pelvic structural faults may be made and/or it will be shown on the monitor screen. In a multi-station unit set up (a monitor and terminal in each treatment room), the information could be called to the screen from memory when the physician enters the patient's identification prior to treatment.

All this means, of course, that the technician will place the patient on the instrument, attach the linear motion sensor, place the generator units on the appropriate areas, key in patient identification, turn on the unit, mark the skin of the patient, (unit automatically shuts off when function is complete), remove the generator, remove the sensor, assist the patient to his/her feet and instruct the patient to go to the appropriate treatment area (if treatment is not performed on the CBAKI unit). Following indicated treatment, patient is recycled through the diagnostic procedure to check on the accuracy and completeness of the physician's treatment. Once said treatment is complete to the satisfaction of CBAKI, a notation is electronically encoded on the patient's record that whatever subluxations were originally noted have now, for this moment, been adequately treated.

On occasion several (up to four in my personal experience) adjustive efforts have been required in the same direction on the same bone in order to achieve what the body's internal computer defines as "acceptable correction."

A hand held (or strap on) unit is presently used for pelvic and extremity structural fault testing.

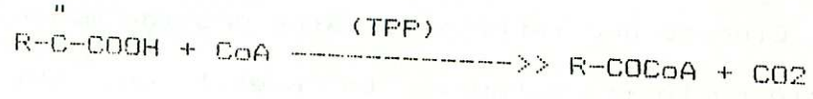
AMINO ACID TESTING

BY
EVAN MLADENOFF B.Sc.,D.C.,
I.C.A.K. DIPLOMATE

ABSTRACT: 5 subjects who underwent extensive laboratory amino acid investigation were examined via the Deal amino acid screening procedure. Discussion of data distortion, comparison of results and questions regarding further detailed investigation of applied kinesiology amino acid testing are raised. This presentation concludes that there is a high correlation between laboratory amino acid testing and applied kinesiology testing.

The citric acid cycle is the body's chemical energy generation sequence for chemical respiration and oxidative phosphorylation This chemical sequence produces carbon dioxide and bound hydrogen atoms which lead to an electron transfer sequence that produces ATP. ATP is the phosphate energy carrier used for operations of many carbohydrate and amino acid metabolism steps. If the citric acid cycle is abnormally limited in rate or capacity, the patient may exhibit symptoms of chronic tiredness, fatique, and need for excessive sleep, and possible mental depression.

Thiamine is of importance because the coenzyme form, thiamine pyrophosphate, is an obligatory participant in two key steps in the citric acid cycle. These steps are virtually identical except for the chain length of the substraight keto acid. They are the formation of acetyl-CoA from pyruvate and the formation of succinyl-CoA from alpha-ketoglutarate:



the reactions are quite complex; the enzymes are multi-enzyme complexes, and require the participation of thiamine pyrophosphate (TPP) and lipoic acid. Inadequate levels of thiamine could greatly diminish the citric acid cycle's ability to provide for adequate ATP.

Deficiencies in the uptake of essential amino acids may be caused by: incomplete digestive proteolysis of dietary protein, poor or competitive absorption of amino acids through the intestinal mucosa and into the blood stream, or by the patient fasting or consuming a very low protein diet. Data that would indicate incomplete digestive proteolysis may be occuring includes an abnormal Heidelberg pH gastrogram and disordered assimilation of zinc and manganese as seen via hair mineral analysis. Zinc and manganese are proteolytic enzyme activators. Complete proteolysis of food protein requires the secretion of pancreatic enzymes into the duodenum and the secretion of peptidases in intestinal mucosa. To be operative, these enzymes must be exposed to the correct pH (not too acidic), and most are activated by zinc and manganese.

When methionine, valine and isoleucine are deficient, their common metabolite, methylmalonyl-CoA, may be deficient in supplying the citric acid cycle with succinyl-CoA. When leucine and phenylalanine are deficient, their common metabolite, acetyl-CoA, may be deficient in supplying the citric acid cycle with a component needed for citric acid synthesis. Lysine, tryptophan and threonine also contribute to the supply of acetyl-CoA. Glucose and fatty acids also provide major inputs into the citric acid cycle via pathways to acetyl-CoA, but laboratory analyses are not available to access the chemistry of these pathways.

Besides inputting to the citric acid cycle at various points, essential amino acids also lead to other necessary compounds such as neurotransmitters and hormones. Tryptophan leads to serotonin (a neurotransmitter) and to NAD (a co-enzyme). Phenylalanine leads to tyrosine and thence to adrenal hormones and catecholamine, and via thyroglobulin it leads to the iodated thyroid hormones. Methionine leads to taurine, a component of bile acid, and to cysteine, which is needed for synthesis of many sulphur containing proteins like insulin. Histidine is needed for maintenance and growth of tissue. It is also the precursor of histamine, a vasodilator secreted by cells in the liver, lungs and stomach. Histidine is catabolized to form glutamic acid.

All of the essential amino acids are used in the body to synthesize proteins. Some of these protein are enzymes which serve as catalysts that enable chemical reactions to occur throughout the body. Another use of amino acids is the synthesis of antibody proteins (immunoglobulin). The essential amino acids are also the source of "non essential" or intermediate amino acids which are also used to build body proteins. Amino acid deficiencies may occur as a result either of deficient essential precursors or due to insufficient dietary uptake. Here are a few examples:

cystine (from methionine in dietary protein)

tyrosine (from phenylalanine and dietary protein)

taurine (from cystine/cysteine)

glycine (from threonine and dietary protein)

serine (from threonine and glycine, dietary protein, and formed endogenously via glycolysis)

phosphoserine (via glycolysis)

glutamic acid (from alpha-ketZoglutaric acid that is aminated by amine groups from dietary source amino acids)

glutamine (from glutamic acid and dietary protein)

aspartic acid (endogenous formation and dietary protein)

asparagine (from aspartic acid and glutamine and dietary protein)

Materials and Methods

Deal (1) has presented a method of evaluating patients for amino acid deficiency. The protocol he established to determine the primary priority indicator is determined by the following sequence:

1) First ensure that the acupuncture system is clear. Perform any necessary balancing procedures so that no alarm point or pulse points will therapy localize.
2) The meridian end point will therapy localize in the clear.
3) Inspiration will make the indicator muscle in step #2 go strong.
4) Pinching does not alter the indicator muscle in step #2.
5) Eyes left or right does not change the indicator muscle in step #2.

According to Deal, when the above criteria have been met, this is an indicator of amino acid deficiency. Five patients, all members of the same family, were selected to participate in this investigation. The year before (Summer of 1982) they had travelled to the centre for Bio-Ecologic Medicine (1611 Hicks Rd., Rolling Meadows, Illinois 60008, (312) 934-1100) to under go extensive amino acid laboratory investigation. The laboratory investigation included serum analysis, urine protein analysis, hair analysis, vitamin

serum analysis. A sample of the data report is included as an indication of the detailed laboratory investigation. The five test subjects were examined as described by Deal such that all alarm points and pulse points would not therapy localize. Then all twenty four meridian end points were therapy localized and tested.

RESULTS

Subject #1, female age 8, laboratory findings revealed deficient phosphoethanolamine (trace) but normal ethanolamine, subnormal asparagine, normal aspartic acid and normal glutamine. The triamine level appears to be only marginally adequate. The uptake of several essential amino acids is below the optimum range: leucine is cautionary low; isoleucine is deficient, and methionine is cautionary low. Cystathione is low, hydroxylysine is high, Gamma-aminobutyric acid is notably elevated in the urine.

The suggested supplementation program for Subject #1 relevant to the above laboratory findings were as follows:

Amino acids - valine, leucine and isoleucine; phosphatidyl choline; perhaps a low dose of glutamic acid.

Vitamins - pyridoxine or pyridoxal phosphate, thiamine and phosphorylated thiamine, B2 and B3, biotin is deficient per the blood vitamin assay therefore supplementation is suggested.

Minerals - no minerals are positively indicated.

Applied Kinesiology testing revealed positive therapy localization of the following acupuncture points:

right GB1 - L-phenylalanine right and left Li20 - L-lysine

right CX1 - L-valine right Hi1 - L-serine

right TH1 - L-tyrosine right Si1 - L-leusine

right GB44 - L-aspartic Acid

Subject #2, female age 48, laboratory findings revealed a deficient uptake of essential amino acids as indicated by urine spillage in less than normal amounts. The following are low or deficient - leucine, isoleucine, phenylalanine, threonine, and tryptophan, with respect to anomalous metabolism of methionine, taurine is deficient although methionine, cystathionine and cystine are all within normal limits. Laboratory diagnoses include:

a) subnormal uptake of five essential amino acids, probably caused by incomplete digestive proteolysis of dietary protein

b) deficient taurine per urine data

c) deficient assimilation of magnesium into enzyme protein.

The suggested supplementation program for Subject #2, relevant to the above laboratory findings, were as follows:

Amino acids: balanced mixture of all essential amino acids, also taurine (500 mg/day).

Vitamins: B6 as pyridoxal phosphate, thiamine, riboflavin, niacinamide, vitamins K, A, and E, folic acid with B12.

Minerals: magnesium, zinc, manganese, iron, some copper.

Diet: no colas and no beverages containing phosphoric acid. A moderate protein diet is appropriate.

Applied Kinesiology: Testing revealed positive therapy localization of the following acupuncture points:

right and left K1 - L-cystine

right and left Li20 - L-lysine

right and left LI1 - L-threonine

right and left HT1 L-serine

Subject #3, male age 7, laboratory findings revealed: with respect to deficient uptake of amino acids as indicated by urine spillage in less than normal amounts the following are low or deficient: valine, leucine, isoleucine, phenylalanine, threonine, lysine, tryptophan, methionine and histidine. The appearance of gamma-aminobutyric acid in the urine is anomalous because other usually confirmatory evidences of retarded amino transfer are not present. Therefore, all of the nine essential amino acids are indicated to be deficient per the urine data.

The suggested supplementation program for Subject #3, relevant to the above laboratory findings, were as follows:

Amino acids: balanced mixture of all essential amino acid, also, tyrosine, serine, cystine, aspartic acid, glycine, glutamic acid, and phosphatidyl choline.

Vitamins: Thiamine, pyridoxal, riboflavin, niacinamide, vitamins A and E, Vitamin C (Ca ascorbate)

Minerals: Magnesium, calcium, zinc, manganese

Diet: no colas and no beverages containing phosphoric acid. A moderate protein diet is appropriate.

Applied Kinesiology testing revealed positive therapy localization of the following acupuncture points:

left Li1 - L-alanine left TW 1 - L-tyrosine

right Si1 - L-leucine right K1 - L-cystine

left GB: - L-phenylalanine right Liv14 - L-glutamic acid

Subject #4, female age 10, laboratory findings revealed: 1-methyl-histidine is in the upper 1/3 of its normal range while all essential amino acid valves and most catabolites of the essentials are cautionary low or deficient. This indicates the probability of retarded methyl group transfer. Deficient uptake of essential amino acids as indicated by urine spillage in less than normal amounts, the following are low or deficient: valine, leucine, isoleucine, phenylalanine, threonine, lysine, methionine, tryptophan and histidine. The co-enzyme activity of pyridoxal phosphate is subnormal as indicated by higher than normal urine spillage of kynurenic acid.

The suggested supplementation program for Subject #4, relevant to the above laboratory findings, were as follows:

Amino Acids: balanced mixture of all essential amino acids plus tyrosine, cystine, aspartic acid, serine, glutamic acid and phosphatidyl choline.

Vitamins: folic acid, B6 as pyridoxine or pyridoxal phosphate, thiamine, riboflavin, niacinamide, vitamins A and E, vitamin C (Ca ascorbate)

Minerals: magnesium, zinc and iron, as indicated by the ferratin result.

Diet: no colas or beverages containing phosphoric aid. A moderate protein diet is appropriate.

Applied Kinesiology testing revealed positive therapy localization of the following acupuncture points:

left TW23 - L-cysteine left Si19 - L-glutamine

right Cx1--L-valine

right Cx2--L-isoleucine

left St45-L-tryptophan

left Liv14 - L-glutamic acid

right Si1-L-leucine

left K1-L-cystine

Subject #5, male, age 51, laboratory findings revealed: With respect to either retarded transfer of amine groups in amino acid metabolism or generalized aminoacidurias, the following are excessive: ornithine, alpha aminoadipic acid, phosphoserine, alanine, beta aminoisobutyric acid, cystine, aspartic acid, valine, tyrosine, leucine, beta alanine; glutamic acid is cautionary low. There is a reduced function of the urea cycle with argininuria and notable ornithinuria, possible hyper-beta-alanemia, hypervalinemia or hyper-beta-aminoisobutyric acidemia which reduces tubular reabsorption of taurine, (urine taurine is notably excessive).

The suggested supplementation program for subject #5, relevant to the above laboratory findings, were as follows:

Keto acids: alpha-ketoglutaric acid (Ca-buffered); a 2/3 citrate, 1/3 citric acid mixture as an additional supplement is optional.

Vitamins: B6 as pyridoxal phosphate, thiamine, riboflavin, lipoic acid, folic acid with B12, vitamin C, vitamins K, A and E

Minerals: manganese, magnesium, lithium, cobalt (copper?)

Diet: maintain water intake such that 24 hr. urine control volume exceeds 2000 ml. A relatively low-protein diet for 2-3 months with a 24 hr. limitation of 1.5g/kg body weight. Restrict intake of beef, pork, chicken, turkey, duck, rabbit,

and salmon. No colas or beverages containing phosphoric acid, no alcoholic beverages.

Applied Kinesiology testing revealed positive therapy localization of the following acupuncture points:

Si1	- L-leucine	K1	- L-cystine
HT1	- L-serine	Sp21	- L-histidine
Liv14	- L glutamic acid		

DISCUSSION

The comparison of lab to applied kinesiology results are summarized in chart form. The reader will note that there are areas of complete agreement of amino acid deficiency as determined by both methods of testing and that there are areas that there is a great amount of discrepency. However, the lab tests that were performed on the test subjects were performed at least one year old prior to the applied kinesiology testing the specific nutritional recommendations listed in the results had been instituted for no less than 6 weeks. These 2 facts alone should have distorted the resultant data. In fact, applied kinesiology tesing detected some common deficiencies and perhaps even detected newer deficiencies or excesses that could have been transient due to the nutritional intake of specific amino acids.

It is this author's belief that applied kinesiology testing reflects the functional status of the body. For example, the stomach meridian may contain an excess amount of energy shortly after eating meals. This is a transient excess energy that is a desired function of the body. Electronic meridian diagnosis may not be sensitive enough to detect this transient functional excess but applied kinesiology

testing is. Perhaps this explains some of the unusual applied kinesiology amino acid testing results that were found, remembering that the data could be distorted due to the time factor.

The minute details of laboratory investigation as seen by the sample data is indicative of the highly refined and accurate nature of amino acid analysis. The supposition of Deal that meridian beginning/end point positive therapy localization is indicative of levorotary amino acid deficiency could be suspect. In some of our test subjects there was weakness of only the right or left meridian beginning/end point. Deal didnot indicate whether a bilateral weakness would correlate to be a levorotary amino acid deficiency.The question that arises is, is a right sided involvement only indicative of dextrorotary deficiency and a left sided weakness indicative of a levorotary deficiency ??? Another question that arises after observing the data is, is this an alpha or a beta levorotary weakness OR is it an alpha or a beta dextrorotary weakness ?

In Deal's original paper there was no data representing a determination of any excess amino acid levels via applied kinesiology testing. Therefore, is a hypertonic meridian beginning/end point indicative of an excess amino acid situation and if it is, is it an alpha or a beta form, is it levorotary or dextrorotary ??? Many complex possibilites exist. The logistics of determining the above possibilities may be impossible due the fact that nutritional companies may not be able to manufacture alpha - aminoadipic acid, hydroxylysine etc. etc. to be available for applied kinesiology testing.

On the other hand, Voll has identified and correlated specific acupuncture points which correspond to specific anatomical locations, physiological functions, chemical functions etc.(see diagram #1 +#2) Deal's hypothesis - applied kinesiology evaluation of the meridian begining/end points as an indicator of amino acid deficiencies - is valid and requires further investigation to evaluate amino acid excesses, and determination of the various forms of amino acids. The ideal investigation would be to have patients tested for serum and urine amino acid profiles in the depth as listed in our sample data and then have the subject immediately tested in a modified Deal applied kinesiology amino acid testing profile.

It is this author's opinion that a detailed laboratory amino acid profile and a detailed investigation of the meridian beginning/end point hypothesis will produce a more significant correlation between laboratory investigation and applied kinesiology testing than was exhibited in this presentation.

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A CLINICAL LABORATORY STUDY OF HAIR COPPER LEVELS WITH 24 HOUR URINARY COPPER EXCRETION LEVELS

Richard A. Mowles, D.C., DICAK

ABSTRACT: This paper will involve a clinical laboratory study evaluating iron and copper levels through Hair Trace Mineral Analysis with 24 hour urinary copper excretion levels. The relationship of iron and copper in Applied Kinesiology has important clinical significance. Objective methods in the evaluation of those two minerals can be of great benefits in clinically evaluating these patients with Applied Kinesiology.

INTRODUCTION

There has been quite an upheaval in research concerning copper and iron with Applied Kinesiology.[1,2,3,4,5] These two minerals play a powerful cornerstone in balancing the biochemical homeostasis within the body. In the previous literature we have seen correlative patterns between these two minerals and neuromuscular findings with Applied Kinesiology.[6,7,8,9] The correct diagnosis of a problem can lead to a more positive treatment regimen for that patient.

Copper plays a powerful role with iron in the body. The objective evaluation of copper and iron levels in the body leads to monumental significance with understanding electron poising problems, retrograde lymphatic problems, right and left brain problems, and aerobic muscle problems. These aforementioned patterns can be very significant in stabilizing a patient with Applied Kinesiology techniques.

There is very little correlation between the blood levels of minerals and the tissue levels of these same minerals as evaluated through Hair Trace Mineral Analysis. The blood tends to be a stable medium only to change at advanced stages of pathology. This has been shown in regards to iron and sub-clinical iron deficiency.[10]

It has been mentioned in the literature that, "the only way to diagnose copper toxicity is by measuring a 24 hour urinary copper excretion. Urinary copper output is often several times above the upper limits of normal. This is a very significant clinical observation which has been made by numerous clinicians utilizing hair analysis in their practices but which has not yet been subjected to controlled scientific studies and to our knowledge has not yet been published."[11]

This author felt a clinical laboratory study between the levels of copper and iron with the Hair Trace Mineral Analysis in comparison with 24 hour urinary copper excretion levels was warranted.

PROCEDURES

This study commenced on June 1984 and ended in November 1984. There were 25 randomly picked subjects involved in the study which consisted of 16 females and 9 males. The subjects main health complaints ranged from fatique and depression to spinal problems such as low back or neck pain. The subjects were evaluated utilizing laboratory procedures which consisted of Hair Trace Mineral Analysis, (HTMA), [12] done by Atomic Absorption Spectrophotometry, and 24 Hour Urinary Copper Excretion.[13] Laboratory values were obtained at the initial onset of the study and every 2½ months for the six month evaluation period. The subjects were seen clinically on a frequent basis and treated with standard Applied Kinesiology techniques as taught by the International College of Applied Kinesiology.

At the onset of treatment, the subjects were questioned as to the possibility of hair contamination with copper. This is very important in that many swimming pools are treated with chemicals which extract copper salts from the pipes through which the water is circulated during purification and treatment.[14] These subjects were also requested at the onset of treatment not to take any nutritional supplements unless as directed in the study.

RESULTS

The results of this study are presented in Table I and Table II. Table I shows the HTMA levels of copper and iron as well as the 24 hour urinary copper excretion levels. Each column represents the 3 periods of evaluation during the study. Table II shows the patterns and number of subject for that pattern at that evaluation period in the study. The high and low HTMA levels of iron and copper as well as the high and low ranges of 24 hour urinary copper excretion levels are considered "normal". Anything outside this range is considered high or low and categorized accordingly. The number of subjects at each evaluation period are categorized into patterns of HTMA iron and copper levels and this is correlated with the 24 hour urinary copper excretion levels. This information is presented in Table II.

In Table II, Category A showed the most significant pattern. In this category, the HTMA levels of iron was low and copper was high. In repetitive evaluations, this pattern showed high correlation with urinary copper excretion. On the second evaluation period, this correlation was noted most significantly, where the HTMA levels of copper and the urinary copper excretion levels were equal in correlation. This high correlation probably reflects active elimination or mobilization of copper from the tissues and represents a true copper toxic pattern. This urinary copper excretion pattern is most prevalent when the HTMA iron levels are low and the HTMA levels of copper are high. Seven of these subjects showed this Category A pattern throughout the six month study.

Category B subjects showed a normal HTMA level of copper and iron and all had normal urinary copper excretion levels. There were only three subjects who held this pattern throughout the six month study.

Category C subjects showed normal HTMA levels of iron and high copper with only one showing a high urinary copper excretion level. None of these subjects held this Category throughout the study. When the HTMA level of copper was elevated, there was a corresponding decrease in the HTMA iron levels. The fact that the urinary copper excretion levels were significantly correlative may have some relationship to the HTMA levels of iron and copper initially.

Category D subjects showed low HTMA levels of copper and iron and all had normal urinary copper excretion levels. None of these subjects held this pattern throughout the study. Two of these subjects established normal HTMA copper and iron levels. There was no decrease in urinary copper levels noted when the HTMA levels of copper were below normal.

Category E subjects were represented by normal HTMA levels of iron and low copper. None of these subjects held this category throughout the study. During the course of the study this category reverted into another category by the third evaluation. No change in the urinary copper levels was noted in this case.

Category F subjects were represented by low HTMA levels of iron and normal levels of copper. This category had little significance since there were no subjects seen with this pattern except in the second and third evaluation period.

DISCUSSION

This study has evaluated a diagnostic correlation of the HTMA levels of copper and iron with urine copper excretion values. The study has shown a definitive interaction between the tissue levels of copper and urine excretion.

TABLE I

Subjects	HTMA Iron (2.1-4.5ppm)			HTMA Copper (1.5-3.3ppm)			Urinary Copper Excret. (26-64mcg/24 hr.)		
1	1.00	1.50	2.10	12.00	14.60	9.50	74	82	67
2	2.30	1.50	1.50	3.20	6.80	10.00	28	92	115
3	3.60	3.20	3.10	1.80	2.10	2.80	36	29	34
4	1.40	.70	.90	8.60	18.50	16.00	84	100	82
5	1.50	1.80	2.30	5.40	2.10	2.40	52	42	50
6	2.40	2.00	1.80	6.10	9.80	15.00	60	78	88
7	1.00	2.00	4.00	1.00	1.40	2.70	29	35	44
8	.80	.90	.90	20.00	14.00	11.00	110	118	69
9	4.00	3.10	2.00	3.20	4.10	8.60	44	34	48
10	3.80	3.10	2.60	3.10	3.40	3.80	52	40	37
11	.50	.60	.70	16.00	22.00	15.00	58	98	82
12	4.40	3.80	3.70	1.20	1.50	2.00	32	29	34
13	1.80	.70	.80	11.00	16.00	14.00	115	94	76
14	2.10	1.00	1.10	3.50	6.80	5.40	62	78	62
15	1.00	.80	.80	18.00	22.00	25.00	92	115	100
16	2.10	2.20	1.80	.80	.90	1.70	29	37	46
17	.80	1.00	1.80	.90	.90	9.00	46	62	88
18	1.30	.80	.60	10.00	14.50	20.00	177	97	80
19	3.20	2.80	3.20	2.50	3.10	3.00	34	50	29
20	1.00	1.00	.80	.90	1.00	8.50	48	58	54
21	4.20	2.20	1.00	1.80	1.60	.80	34	40	38
22	.80	2.50	3.00	.90	2.50	3.10	37	32	47
23	.90	1:10	.90	25.00	40.00	18.00	99	120	82
24	2.70	1.60	.90	4.10	5.60	7.00	42	49	56
25	3.20	2.20	1.50	5.60	5.30	8.60	82	68	94

TABLE II

Initial Evaluation	
Category A 9 HTMA Low Iron, High Copper	7 High Urinary Copper Excretion
Category B 6 HTMA Normal Iron, Normal Copper	All with normal Urinary Copper Excretion
Category C 4 HTMA Normal Iron, High Copper	1 High Urinary Copper Excretion
Category D 4 HTMA Low Iron, Low Copper	All with normal Urinary Copper Excretion
Category E 2 HTMA Normal Iron, Low Copper	All with normal Urinary Copper Excretion
Category F 0 HTMA Low Iron, Normal Copper	Not applicable
2nd. Evaluation - 2 months into Study	
Category A 12 HTMA Low Iron, High Copper	11 High Urinary Copper Excretion
Category B 5 HTMA Normal Iron, Normal Copper	All with normal Urinary Copper Excretion
Category C 2 HTMA Normal Iron, High Copper	1 High Urinary Copper Excretion
Category D 4 HTMA Low Iron, Low Copper	All with normal Urinary Copper Excretion
Category E 1 HTMA Normal Iron, Low Copper	All with normal Urinary Copper Excretion
Category F 1 HTMA Low Iron, Normal Copper	All with normal Urinary Copper Excretion
3rd. Evaluation - 5 months into Study	
Category A 14 HTMA Low Iron, High Copper	10 High Urinary Copper Excretion
Category B 6 HTMA Normal Iron, Normal Copper	All with normal Urinary Copper Excretion
Category C 3 HTMA Normal Iron, High Copper	1 High Urinary Copper Excretion
Category D 1 HTMA Low Iron, Low Copper	All with normal Urinary Copper Excretion
Category E 0 HTMA Normal Iron, Low Copper	Not applicable
Category F 1 HTMA Low Iron, Normal Copper	All with normal Urinary Copper Excretion

This interaction is most significant when HTMA levels of iron are low and copper is high. In a previous paper this pattern was seen in the HTMA, but the correlation with urinary copper excretion helps support the findings and establish a working diagnosis.

This study also showed a frequent clinical situation where the copper and iron HTMA levels are normal or low only to have the HTMA levels of copper to rise while iron levels go down with treatment. As this pattern is exhibited the corresponding rise in urinary copper excretion is seen. This would help support an interesting hypothesis in that the body compensates biochemically to stress. The copper is put away in the tissue and thus with appropriate treatment this toxic element is available to be either utilized or eliminated from the body. With the elimination of copper, there is an interaction with iron causing levels to lower and increased levels of copper to spill over in the urine.

In certain clinical problems where the need for iron is apparent but therapy does not give results, a HTMA would be warranted. In this situation if a high copper low iron HTMA pattern is seen, and a 24 hour urinary copper excretion is performed and high levels are seen, then the underlying iron problem could be properly understood and treated.

If the patient is being treated and the initial HTMA showed no apparent problem but response to treatment is slow or none at all, then a HTMA and urinary copper study is warranted. The treatment may be guiding the system towards homeostasis, however the buried toxicity of an element has to be corrected before that homeostasis is achieved. The diagnostic usefulness of HTMA to evaluate copper and iron with 24 hour urinary copper excretion can give the physician an understanding

into the reasons treatment may make a patient's subjective symptoms worse, however objective evaluation shows increased response to treatment.

CONCLUSION

In the utilization of Applied Kinesiology, changes in the biochemical environment would reflect in neuromuscular patterns that change. Applied Kinesiology tells you that there is a problem but not the nature of the problem. When a physician's tools are correlated with laboratory procedures, then these tools can be most efficiently utilized to enable the patient's problems to be eliminated and health maintained. This study has attempted to validate a clinical laboratory analysis to be utilized in the understanding of those difficult neuromuscular changes that are seen by the physician utilizing Applied Kinesiology.

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A CLINICAL STUDY INVESTIGATING THE DIAGNOSTIC SCREENING AND APPLICATION OF THE TRACE MINERAL, MOLYBDENUM, IN APPLIED KINESIOLOGY

Richard A. Mowles, D.C., D.I.C.A.K.

Abstract: The trace mineral, Molybdenum, is discussed and it's importance in certain Applied Kinesiology patterns. A screening test for Molybdenum is described using Ammonia and Formaldehyde.

A CLINICAL STUDY INVESTIGATING THE DIAGNOSTIC SCREENING AND APPLICATION OF THE TRACE MINERAL, MOLYBDENUM, IN APPLIED KINESIOLOGY

In man and other mammals, molybdenum is an essential trace mineral. Molybdenum deficiencies have been associated with esophageal cancer, tooth decay, sexual impotency, and in some cases of gouty arthritis.[1,2,3] In the human biochemistry, it is a necessary component of three enzymes - xanthine oxidase, aldehyde oxidase and sulfite oxidase. These enzymes play a crucial role in the oxidation of fats and in the metabolism of purines.

The enzyme, xanthine oxidase, serves a role in several important biochemical pathways within the body. One pathway is where this enzyme mobilizes iron stores within the liver, where most of the iron is stored.[4] The second pathway is where this enzyme helps to bind up nitrogen in the body and turn it into uric acid and urea to be eliminated as urine through the kidneys.[5] If the nitrogen is not bound up due to inadequate amounts of xanthine oxidase, then the body could build up toxic levels of ammonia.

The literature cites significant facts about the inhibitory effects that molybdenum has upon the mineral, copper. Sheep and cattle grazing on molybdenum rich pastures may develop a copper deficiency.[6] It is also cited that molybdenum and copper seem to have a "balancing" relationship where they may interact or even share or compete for common enzyme systems. High copper intake increases molybdenum excretion and a copper overload may be corrected by administering molybdenum.[7]

In Applied Kinesiology, techniques that may require iron supplementation and/or where iron supplementation fails to give

therapeutic results such as in electron poising problems, retrograde lymphatic problems and aerobic muscle problems, the support therapy of molybdenum supplementation may be indicated. The enzyme, xanthine oxidase, in which molybdenum is a component, may be inadequate and this would impair iron mobilization from the liver. It hae also been shown through Applied Kinesiology research the strong interaction between iron, folic acid, and copper.[8] This is significant where the copper is high and iron is low. Molybdenum may be necessary to counteract the high copper aud allow iron levels to rise.

An interesting situation was observed by this author in a patient who presented clinically with a retrograde lymphatic problem. The patient had shown this pattern on several previous observations and was treated with standard Applied Kinesiology procedures, including supplementation with iron. This particular patient revealed a pattern of high tissue copper with low tissue iron on Hair Trace Mineral Analysis. On the third office visit, the patient was examined and showed a return of the previously treated retrograde lymphatic pattern and this time molybdenum was supplemented which permanently neutralized the pattern. There was no return of the need for iron supplementation and corresponding changes were seen in this particular patient's blood chemistry and Hair Trace Mineral Analysis comparative studies. The implications and importance of molybdenum with iron/copper inbalances in Applied Kinesiology procedures warranted a clinical study by this author.

Since there is no definite laboratory test to determine

molybdenum levels and the clinical significance of hair concentrations of molybdenum are unknown as of this study, an investigation into Applied Kinesiology screening procedures was warranted.

This author has hypothesized two possible methods of screening for a possible need for molybdenum in Applied Kinesiology. The first method is based upon the fact that molybdenum makes up the enzyme, xanthine oxidase. This enzyme helps to bind up excess nitrogen in the body. If the excess nitrogen is not bound up, then excess ammonia can build up in the body. Theoretically, if a patient sniffs some ammonia and a strong muscle goes weak, then a possible deficiency of xanthine oxidase could exist in the body of which molybdenum is a component. A second method of screening for molybdenum deficiency is founded on the fact that molybdenum is also incorporated into another enzyme, aldehyde oxidase. Most fragrances biochemically have an aldehyde base. Theoretically, the inhalation of a substance such as formaldehyde (diluted down with distilled water to avoid toxicity) would make a weak muscle strong if molybdenum is needed by the body. Since these two hypothesized methods of screening could give the investigator an indication that molybdenum supplementation was a possibility, their incorporation into a study was warranted.

PROCEDURE

This study started September 1984 and terminated in November 1984. There were 14 subjects involved in the study, consisting of 8 males and 6 females. All of these subjects had been screened with

with standard AK diagnostic methods and showed clinical patterns of retrograde lymphatic impairement that may require the need for iron supplementation. All of these subjects were screened and showed no clinical need for vitamin A supplementation. This was to keep the number of variables that may enter into this study as minimum as possible.

Four of these subjects had a diagnosed past history of iron deficiency anemia, in which iron was prescribed by the attending physician.

The subjects were evaluated utilizing Hair Trace Mineral Analysis, (HTMA),[9] and standard Applied Kinesiology procedures as taught by the International College of Applied Kinesiology.

The subjects were screened for possible molybdenum deficiency by the inhalaxion of ammonia from a small vial, and also by the inhalation of a 50/50 mixture of formaldehyde and distilled water. After each procedure, a change in muscle strength was noted aud recorded.

The subjects were also tested with molybdenum and/or iron to evaluate changes in muscle strength for their respective muscle patterns that presented itself clinically. These results were noted and recorded.

The subjects were treated by other means taught in AK for the retrograde lymphatic pattern. This included manipulative procedures and reflex therapy to the pectoralis minor muscle such as neurolymphatic reflexes and origin/insertion technique. However, emphasis was placed on those subjects who showed a clinical need for iron supplementation.

The protocol for preparation and nutrient supplementation with

these subjects was standard as in a previous study by this investigator.[10]

These subjects were evaluated 2 times during the course of the study with AK methods of diagnosis and with HTMA studies focusing on hair copper and iron levels. The subjects were treated with AK techniques and those who required supplementation, received either iron, molybdenum, or iron and molybdenum.

RESULTS

The results of this study is presented in Table I which is a total of all findings recorded in this study. The two columns under each listing designate the initial evaluation and the 2nd. evaluation period. All of the subjects showed a retrograde lymphatic pattern initially. At the 2nd. evaluation period, 7 of the subjects showed a persistent retrograde lymphatic pattern.

Table II is a summarization of patterns with iron and copper HTMA levels to the need for supplementation with iron, molybdenum or iron and molybdenum. These findings are also correlated to those subjects AK evaluation in the inhalation of ammonia and formaldehyde. The most significant pattern in Table II is the 1st. pattern where the HTMA shows low iron and high copper levels. Most of these subjects showed a need for both iron and molybdenum supplementation. This becomes even more significant in the 2nd. evaluation, apparently due to the mobilization of copper from the tissues and it's interactions with iron and/or molybdenum. The need for iron supplementation by itself, was most noted in the 3rd. pattern where the HTMA shows normal iron and copper levels. This pattern throughout the study showed the most

TABLE I

Subjects	Retrograde Lymphatic		HTMA Iron (2.1-4.5ppm)		HTMA Copper (1.5-3.3ppm)		Ammonia Inhalat.		Formaldehyde Inhalat.		Iron Suppl		Molybdenum Suppl.	
1	+	+	1.5	1.0	7.3	10.8	+	+	+	+	+	+	+	+
2	+	-	2.2	1.6	4.2	4.8	+	+	+	-	+	-	-	-
3	+	-	2.6	.9	10.0	16.0	-	+	+	+	-	+	+	+
4	+	+	2.2	.8	3.2	12.0	-	+	+	+	-	-	+	+
5	+	-	2.9	3.0	3.2	3.1	+	-	-	-	+	-	-	-
6	+	+	1.9	1.0	5.6	9.8	-	-	+	-	+	+	+	+
7	+	+	.9	1.0	16.0	18.0	+	+	+	-	+	+	+	-
8	+	-	2.5	2.8	3.1	2.9	-	-	-	-	+	-	-	-
9	+	+	1.2	1.8	7.6	5.8	-	+	-	-	+	-	-	-
10	+	-	2.1	.9	4.5	7.8	-	+	+	+	+	-	+	+
11	+	+	1.0	1.5	6.8	6.0	+	-	+	+	-	-	+	+
12	+	-	2.0	2.4	3.5	3.0	-	-	-	-	+	-	-	-
13	+	-	3.8	.8	2.6	11.0	-	-	+	+	+	+	+	+
14	+	+	3.1	2.4	3.2	3.7	-	-	-	-	-	-	-	-

UNDER EACH CATEGORY THERE ARE TWO COLUMNS THAT DESIGNATE THE INITIAL EVALUATION AND THE 2ND. EVALUATION IN THIS STUDY.

+=this means the finding is present with Applied Kinesiology examination and diagnostic procedures.

-=this means the finding is not present with Applied Kinesiology examination and diagnostic procedures.

TABLE II

This Table is a summarization of findings from Table I according to Mineral Patterns and correlation to supplement support with Molybdenum, Iron, or Molybdenum and Iron together to neutralize AK findings. This table also has the corresponding mineral patterns correlating with ammonia and formaldehyde screening procedures.

Minerals Tested positive		Ammonia positive	Formaldehyde positive
Low Iron, High Copper HTMA levels, Initial Evaluation - 6 Subjects			
Molybdenum	1	1	1
Iron	2	0	0
Molybdenum and Iron	3	2	3
Low Iron, High Copper HTMA levels, 2nd. Evaluation - 10 Subjects			
Molybdenum	3	2	3
Iron	1	1	0
Molybdenum and Iron	4	2	3
No need for supplements	2	1	0
Normal Iron, High Copper HTMA levels, Initial Evaluation - 3 Subjects			
Molybdenum	0	0	0
Iron	1	1	1
Molybdenum and Iron	2	0	2
Normal Iron, High Copper HTMA levels, 2nd. Evaluation - 1 Subject			
Molybdenum	0	0	1
Iron	0	0	0
Molybdenum and Iron	0	0	1
Normal Iron, Normal Copper HTMA levels, Initial Evaluation - 5 Subjects			
Molybdenum	1	0	1
Iron	2	1	0
Molybdenum and Iron	1	1	1
No need for supplements	1	0	0
Normal Iron, Normal Copper HTMA levels, 2nd. Evaluation - 3 Subjects			
Molybdenum	0	0	0
Iron	0	0	0
Molybdenum and Iron	0	0	0
No need for supplements	3	0	0

response to standard AK therapy with minimum supplementation. Iron supplementation is most effective in this pattern where copper is not high.

The results of the evaluation for the scrdening methods utilizing the inhalation of ammonia and formaldehyde showed that formaldehyde was more correlative with a need for molybdenum than ammonia. Both of these substances showed a positive reaction (a detectable change in muscle strength by manual testing) where there was a need for iron and/or molybdenum.

DISCUSSION

This study attempted to study the trace mineral, molybdenum, and its usefulness in Applied Kinesiology. The study shows that molybdenum is very useful where copper is elevated with inhibitory effects towards iron. This must be due to molybdenum and its antagonistic effect towards copper or it could be due to molybdenum's motilization of iron from the liver. This investigator feels it is a combination of both.

The scbeening tools for molybdenum show that the inhalation of formaldehyde correlates more closely to a molybdenum deficiency than the inhalation of ammouia. This investigator feels that there are many facxors related to the catabolism of ammonia within the body. Another investigator has seen a pattern of a need for folic acid supplementation when ammonia weakens a strong indicator muscle.[11] This particular phenomena needs further investigation.

Even though the study confined it's boundaries on those subjects showing a retrograde lymphatic pattern kinesiologically, this investigator hypothesizes the results of this study to be applicable in other AK situations where iron supplementation is a possibility. This would also be another area that would warrant a clinical investigation.

CONCLUSION

This paper has focused upon a clinical study examining the screening and utilization of the trace mineral, molybdenum, in Applied Kinesiology. Molybdenum is another "piece of the puzzle" in the comprehension of iron and copper interactions within the body. Supplementation of this trace mineral can be very useful in clinical situations where high copper and low iron patterns affect the neurological homeostasis of the body. Those difficult patients that fail to respond could be lacking one important link in responding to treatment.

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HOLOGRAPHIC THERAPY LOCALIZATION AND INTEROSSEOUS ADJUSTMENT LISTINGS BY ALPHABETICAL SYMPTOMS AND VERTEBRAL LEVELS

H. Louis Obersteadt, D.C.

ABSTRACT After receiving a list of symptoms and challenges for Holographic Subluxations from Dr. Goodheart, I started applying the techniques on difficult patients that had not responded completely to the standard applied kinesiology approach, and this definitely made many great changes. However, it was taking longer to find the complaint on the list that Dr. Goodheart had supplied than it was to make the correction. I decided that I was probably not alone in this matter and proceeded to develop the following list for your convenience.

The interosseous vertebral holographic subluxation described by Dr. Goodheart in the 1982 Research Manual will therapy localize when contacting the spinous process and transverse process at the same time causing a strong muscle to weaken or a weak muscle to be strong. The vertebral challenge is pushing the S.P. and T.P. in a line of drive opposite to each other or in line of drive toward each other. In most cases you will find the line of drive in opposite directions.

The alphabetical listings are read as follows: Intermittant claudication L1 Left, L4 Left. This means that the spinous process is contacted on the left side and challenged to the right, while the transverse process is contacted on the left and challenged P to A on the left of L1 vertebra and do the same to the L4 vertebra. The vertebral listings are the same. For example, page 13, Thoracic (T11), Right appendicitis L1 Left. This means to contact the S.P. on the Right and T.P. on the Right and separate them. Also, you need to do the opposite direction in this case to L1. However, it is best to challenge and find the best vector and adjust in that direction.

Holographic Subluxation
Alphabetical Symptoms

-A-

AMENNORRHEA -	L4 Right
ANEMIC HEADACHE -	C4 Right
ANKLE (Left) PAIN -	L2 Right
ANKLE (Right) PAIN -	L5 Right
APPENDICITIS -	T12 Right, L1 Left
ARTERIOSCLEROSIS IN LEGS -	L1 Right, L4 Right T5 Right, T7 Right, T11 Right Calf of Leg - L4 Right Side of Leg - L1 Right
ASTHMA -	T1 Left, T3 Right All asthma patients show C6 Right
ASTHMATIC ATTACK -	T1 Left

-B-

BALL OF FOOT -	T12 Left
BLADDER (Inflamed) -	L1 Left
BLADDER(Nervous) -	L2 Right
BOWEL, LAZY (Goes every other day) -	T5 Right, T7 Left, L2 Left
BREAST (Hard) -	T3 Right (2 to 4 for upper and lower breast)
BREATH (Short of) -	T1 Left
BRONCHIAL (Large) TUBES (To Clear) -	T3 Right
BRONCHIAL PERSPIRATION -	T1 Left
BRONCHITIS (Shortness of Breath) -	C6 Left, T1 Left inferior, T3 Right

-C-

CALCIUM IN THE SYSTEM	T5, T7, T11
CHEEK BONE PAIN -	C5 Right
CLOGGED NOSE -	C5 Right
CONSTIPATION (Doesn't Feel Like Moving) -	L3 Right, T12 Right
CONSTIPATION (Feels Like Moving, But Can't) -	T6 Left, T12 Right, L4 Right
CONSTIPATION HEADACHE -	L4 Right, T6 Left, T12 Right
COUGH (Dry) -	T1 Left (T.P. to left superior)
CRYING OR FEAR -	T6 Left, T9 Left, T12 Right, L4 Left

Holographic Subluxation
Alphabetical Symptoms

-D-

DECREASE HORMONES -	T9 Left
DIABETES -	T8 Right, T9 Left
Hunger Pangs	T5 Right
Itch	T10 Right
Sweating	T1 Left
Urination	L1 Left
Vaginal Itch	T10 Right
DIAPHRAGM -	T8 Right
DIARRHEA -	L2 Right, T7 Left, L2 Left
DIZZINESS -	C4 Left
DIZZINESS (Constant from HBP) -	C2 Right plus side slip

-E-

ECZEMA -	T5 Right, T10 Right, T12 Right, T9 Left
EDEMA -	T9 Right (for adrenals, T10 Left, L1 Right
ENLARGED HEART -	T2,3,4 Left, C3 Left
ERECTION -	L4 Left
Blood Supply To Penis	T11 Left
Desire	L2 Left
Nervous Tolerance	T6 Left
No Emission	L2 Left
Premature Ejaculation	L2 Right
EYEBALL PAIN (Behind Eye) -	L3 Right (L3 anterior IAW GJG)

-F-

FACIAL NEURALGIA -	C5 Right
FALLOPIAN TUBES (Infantile) -	L4
FEAR OF CRYING -	T6 Left, T9 Left, T12 Right, L4 Left
FEVER -	T6 Left, T10 Right, T12 Right, L2 Left
FINGERS NUMB & STIFF -	C7 Right
FOOT (Ball of) -	T12 Left

-G-

GALL BLADDER (Inflamed) -	T9 Right
GALL BLADDER (Nervous)-	T9 Right
GALL STONES -	T4 Left
GAS -	T4 Left, T6 Left, T9 Right, T12 Right, L2 Left
GLABELLA PAIN -	C4 Left
GLAUCOMA -	T6 Left, T7 Right, T12 Right

-H-

HARD BREASTS -	T3 Right (2 to 4 for upper and lower breasts)
HEAD BURN (Top of) -	T6 Right, T12 Left
HEAD (Pressure on top of) -	C4
HEADACHE	
Anemic	C4 Right
Constipation	L4 Right, T6 Left, T12 Right
High Blood Pressure	C2 Right, L5 Left or Right (see sideslip)
Liver	C2 Right, C4 Left, T4 Right, T5 Right, T7 Left
Menstrual	L4 Left, C4 Left
Migraine	C4 Left
Neurasthenic (Head Contents Shake)	C1 Left, T6 Right
Occipital	L2 Right
Occipital from HBP	L2 Right
Renal (Left Top of Head)	T12 Left, L1 Left
Renal (Right Top of Head)	L2 Right
Rheumatic	C2 Right, C4 Left, T6 Left, T12 Right
Sagittal	C2 Left, C4 Left
HEART CONDITIONS	T2 Left, T4 Left, C3 Left
Enlarged Heart	T2 Left, T3 Left, T4 Left, C3 Left
Heart Block	T3 Left, C7 Right
Heaviness In Heart	T2 Left
Hypertension	C7 Left, C2 Left, C3 Left, C4 Left
Left Side of Heart Pain	T2 Left
Nervous Heart	T2 Left, T3 Left, T4 Left, T6 Left, T9 Right, T12 Right, C3 Left, (T6 Left is Major)
Normal Circulation and Heartbeat	C3 Left
HEARTBURN -	T5 Right
HEMOGLOBIN (Low) -	T8 Left
HERNIA ON LEFT -	L1 Left, L2 Right, T10 Left
HERNIA ON RIGHT	L1 Left, L3 Left, T12 Right
HIGH BLOOD PRESSURE HEADACHE -	C2 Right, L5 Left or Right
HIGH PULSE RATE -	T3 Right
HIVES -	T5 Left, T12 Right
HORMONES (Decrease) -	T9 Left
HOT FLASHES -	L4 on the left (HT)
HUNGER PAINS (Diabetes) -	T5 Right
HYPERTENSION	C6 Left, C2,3,4 Left

-I-

INDIGESTION -	T4 Left, T5 Right, T7 Left, L2 Left
INFANTILE FALLOPIAN TUBES -	L4
INFLAMED BLADDER -	L1 Left
INTERMITTANT CLAUDICATION -	L1 Left, L4 Left
ITCH (Diabetes) -	T10 Right

-J-

-K-

-L-

LARGE BRONCHIAL TUBES (To Clear) -	T3 Right
LAZY BOWELS (Goes Every Other Day) -	T5 Right, T7 Left, L2 Left
LEFT ANKLE PAIN -	L2 Right
LEFT OVARY -	L2 Right
LEFT SIDE HEART PAIN -	T2 Left
LEUKEMIA	
Increase Hemoglobin	T8 Left
Lymph	T1 Left
Prevent Frequent Attacks	L5 Right
Reduce Spleen & Liver Swell	T7, T9
Tissue Repair	T5
LIVER HEADACHE -	C2 Right, C4 Left, T4 Right, T5 Right, T7 Left
LOW HEMOGLOBIN -	T8 Left
LOW PULSE RATE -	T3 Left

-M-

MENSES	
Frequent	L4 Left
Heavy Flow	L4 Left
Pain	L4 Left, T6 Left
MENSTRUAL HEADACHE -	L4 Left, C4 Left
METATARSALS -	L1 Right
MIGRAINE -	C4 Left
MISCARRIAGE -	L4 Left

-N-

NECK STIFF -	C3 Left
NERVOUS BLADDER -	L2 Right (HT)
NERVOUS HEART -	T2 Left, T3 Left, T4 Left, T6 Left, T9 Right, T12 Right, C3 Left, (T6 is major)
NERVOUS STOMACH -	T6 Left, T12 Right
NEURALGIA (Facial) -	C5 Right
NEURASTHENIC HEADACHE (Head contents shake) -	C1 Left, T6 Right
NO PULSE -	T2 Right
NORMAL CIRCULATION AND HEART BEAT -	C3 Left
NUMB & STIFF FINGERS -	C7 Left
NOSE (Runny)	See below
NOSE (Clogged)	C5 Left

-O-

OCCIPITAL HEADACHE -	L2 Right
OCCIPITAL HEADACHE FROM HBP -	L2 Right
OVARIES -	L3 Right
OVARY (Left)-	L2 Right
OVARY (Right) -	L3 Left

-P-

PAIN UNDER HEART -	T4 Left
PERSPIRATION -	T1 Left
PLEURA (Right) -	T1 Left, T3 Right, T12 Right
POISON IVY, OAK, Etc. -	T9 Left, T5 Right, T10 Right, T12 Right
PRESSURE ON TOP OF HEAD -	C4
PULSE RATE (High) -	T3 Right
PULSE RATE (Low) -	T3 Left
PULSE (No) -	T2 Right

-Q-

-R-

RASH -	T5 Right, T9Left, T10 Right, T12 Right
RENAL HEADACHE	
(Left Top of Head) -	T12 Left, L1 Left
(Right Top of Head) -	L2 Right
RHEUMATIC HEADACHE -	C2 Right, C4 Left, T6 Left, T12 Right
RIGHT ANKLE PAIN -	L5 Right
RIGHT OVARY -	L3 Left
RIGHT PLEURA -	T1 Left, T3 Right, T12 Right
RUNNY NOSE	C4 Left (C 4 T.P. on left adjust to inferior)

-S-

SAGGITAL HEADACHE -	C2 Left, C4 Left
SCALP TENDER -	C4 Left
SHORT OF BREATH -	T1 Left
SHORTNESS OF BREATH (Bronchitis)	C6 Left, T1 Left, T3 Right
*SIDE SLIP -	Always adjust side slip first - This is figured by leg and gluteal fold
SKIN TROUBLE (Poison ivy, oak,)	T9 Left, T5 Right, T10 Right, T12 Right
STIFF NECK -	C3 Left
STIFF & NUMB FINGERS -	C7 Right (usually in A.M.)
STOMACH	
Nervous -	T6 Left, T12 Right
Sour	T5 Right, T7 Left, L2 Left
STUFFY NOSE -	C5

-T-

THROAT TICKLING -	C6 Left
THROAT TIGHTENING -	T1 Left
TICKLING IN THROAT	C6 Left
TIGHTENING IN THROAT -	T1 Left
TINNITUS -	C4 Left, T6 Left, T12 Right, Side Slip
TOP OF HEAD BURNS -	T6 Right, T12 Left

-U-

URINATION (Diabetes) -	L1 Left
URINE	
Complete Block	L1 Right
Copious	L1 Left
Partial Block	L1 Right
UTERINE PROLAPSE -	L2 Right

-V-

VAGINAL DISCHARGE -	L4 Left
VAGINAL ITCH (Diabetes) -	T10 Right, L4 Left
VERTIGO (If Patient Lies On One Side Only, Usuall Right)	C4 Right (Challenge Left inferior)
VOICE LOSS -	C5 Right, C6 Left, T5 Right

-W-

WOMB (Inflamed) -	L4 Left

-XYZ-

POSTURAL

1. Right long leg, right gluteal fold inferior, L5 Left posterior.
2. Left long leg, left gluteal fold inferior, L5 Right Posterior.
3. Legs even, left gluteal fold inferior, sacral tubercule right, sacral alae left. Reverse if right gluteal fold is low.
4. Right leg longer, right gluteal fold superior, D5 SP Right, TP Left.
5. Left leg longer, left gluteal fold superior, D5 SP left, TP Right.
6. Right leg longer, gluteal folds even, L5 TP Right Superior, SP Left inferior. Sacral alae superior, sacral tubercule right inferior. Reverse if left pattern exist.

CERVICAL (C1)

LEFT Neurosthenic Headache (head contents shake) T6 Right

CERVICAL (C2)

RIGHT High Blood Pressure Headache L5 Left or Right (see side slip)
LEFT Hypertension C2, C3, C4, C7 Left
RIGHT Liver Headache C4, T7 Left, T4, T5 Right
RIGHT Rheumatic Headache C4, T6 Left, T12 Right
LEFT Sagittal Headache C4 Left

CERVICAL (C3)

LEFT Enlarged Heart T2, T3, T4, & C3 Left
LEFT Heart Condition T2, T4, C3 Left
LEFT Hypertension #7Left, C2, C3, C4 Left
LEFT Nervous Heart T2,T3, T4,T6 (T6 is major)Left, T9, T12 Right
LEFT Normal Circulation & Heart Beat
LEFT Stiff Neck

CERVICAL (C4)

RIGHT Anemic Headache
LEFT Dizziness
LEFT Glabella Pain
LEFT Hypertension C2,C3, C7 Left
LEFT Liver Headache C2, T4, T5 Right, T7 Left
LEFT Menstrual Headache L4 Left
LEFT Migraine
LEFT Pressure on Top of Head
LEFT Rheumatic Headache C2, T12 Right, T6 Left
LEFT Runny Nose, S.P. varies
LEFT Sagittal Headache L2 Left
LEFT Scalp Tender
LEFT Tinnitus T6 Left, T12 Right & side slip
RIGHT SUPERIOR - Vertigo if patient lies on one side only, usually right.

CERVICAL (C5)

RIGHT Cheek Bone Pain
RIGHT Facial Neuralgia
RIGHT Stuffy Nose (HT)
RIGHT Voice Loss C6 Left, T5 Right

CERVICAL (C6)

RIGHT	Asthma (all asthma patients show), T1 Left, T3 Right
LEFT	Bronchitis
LEFT	Tickling in the throat
LEFT	Voice Loss C5 Right, T5 Right

CERVICAL (C7)

RIGHT	Heart Block T3 Left, C7 Right
LEFT	Hypertension C2, C3, C4,
RIGHT	Numb & Stiff Fingers (often in A.M.)

THORACIC (T1)

LEFT	Asthma (all asthma patients show C6 Right), T3
LEFT	Asthamatic Attack
LEFT	Bronchial Perspiration
LEFT	Bronchitis (shortness of breath)
LEFT	Diabetes (sweating) T8, T9, T10, L1, T5, L4
LEFT	Dry Cough, tightening of throat
LEFT	Leukemia (for lymph)
LEFT	Perspiration
LEFT	Right Pleura T3, T12 Right
LEFT	Shortness if Breath
LEFT	Tightening of Throat (Dry Couth)

THORACIC (T2)

LEFT	Enlarged Heart T3, T4, C3 Left
LEFT	Heart Conditions T4 Left, C3 Left
LEFT	Heaviness In Heart
LEFT	Left Side of Heart Pain
Left	Nervous Heart, T3, T4, T6 (T6 is major), T9, T12 Right, L3 Left
RIGHT	No Pulse

THORACIC (T3)

RIGHT	Asthma (all asthma patients show C6 right) T1 Left
RIGHT	Bronchitis (shortness of breath) C6 Left, T1 Left
LEFT	Enlarged Heart T2, T4, C3 Left
RIGHT	Hard Breasts (2 to 4 for upper and lower breast)
LEFT	Heart Block C7 Right
RIGHT	High Pulse Rate
RIGHT	Large Bronchial Tubes (to clear)
LEFT	Low Pulse Rate
LEFT	Nervous Heart T2, T4, T6 Left, T9, T12 Right, C3 Left
RIGHT	Right Pleura T1 Left, T12, Right
RIGHT	Left leg longer left gluteal fold superior
RIGHT	Right leg longer, right gluteal fold superior

THORACIC (T4)

LEFT	Enlarged Heart T2, T3, C3 Left
LEFT	Gall Stones
LEFT	Gas L2, T6 Left, T9, T12 Right
LEFT	Heart Condition T2, C3 Left
LEFT	Indigestion T5, Right, T7 Left, L2 Left
RIGHT	Liver Headache C2, T5 Right, C4, T7 Left
LEFT	Nervous Heart T2, T3, T6 (T6 is major) Left, T9, T12 Right C3 Left
LEFT	Pain under heart

THORACIC (T5)

RIGHT	Arteriosclerosis In Legs T7, T11, C1, C4 Right Calcium in the system, T7, T11
RIGHT	Diabetes (Hunger Pains) T8, T9, T10, C6, L4, T1
RIGHT	Eczema T10, 12 Right, T9 Left
RIGHT	Heartburn
LEFT	Hives T12 Right
RIGHT	Indigestion
RIGHT	Lazy Bowel (goes every other day) T7 Left, L2 Left Leukemia (for tissue repair)
RIGHT	Liver Headache C2, T4 right C4, T7 Left
RIGHT	Rash T9 Left, T10, T12 Right
RIGHT	Skin Trouble (poison ivy, oak, etc.) T9 Left, T12, right
RIGHT	Sour Stomach T7, L2 Left
RIGHT	Voice Loss C5 Right, C6 Left

THORACIC (T6)

LEFT	Constipation (feels like moving, but can't) L4 Right, T12 Right
LEFT	Crying or Fear T9, L4 Left, T12 Right
LEFT	Erection (nervous tolerance)
LEFT	Fever T10 Right, T12 Right, L2 Left
LEFT	GAS T4, L2 Left, T9, T12 Right,
LEFT	Glaucoma T7, T12 Right
LEFT	Menses (pain) L4
LEFT	Nervous Heart T2, T3, %4 Left, T9, T12 Right, C3 Left
LEFT	Nervous Stomach T12 Right
RIGHT	Neurasthenic Headache (head contents shake) C1 Left
LEFT	Rheumatic Headache C2, T12 Right, C4 Left
LEFT	Tinnitus C4 Left, T12 Right, side slip
RIGHT	Top of Head Burns T12 Left

THORACIC (T7)

RIGHT	Arteriosclerosis in Legs L1, L4, T5, T11 Right
	Calcium in the System T5, T11
LEFT	Diarrhea L2 Right, L2 Left
RIGHT	Glaucoma T6 Left, T12 Right
LEFT	Indigestion T4, C2 Left, T5 Right
LEFT	Lazy Bowel (goes every other day) T5 Right, L2 Left
	Leukemia to reduce spleen and liver swelling
LEFT	Liver Headache C2, T4, T5 Right, C4 Left
LEFT	Sour Stomach T5 Right, L2 Left

THORACIC (T8)

RIGHT	Diabetes T1, T5, T9, T10, L4
LEFT	Diaphragm
LEFT	Increase Hemoglobin - Leukemia
LEFT	Low Hemoglobin

THORACIC (T9)

LEFT	Crying or Fear T6, L4 Left, T 12 Right
LEFT	Decrease Hormones
LEFT	Diabetes T8, C1, T10, L4, T1
LEFT	Eczema T5, T10, T12 Right
RIGHT	Edema (for adrenal) T10 Left, L1 Right
RIGHT	Gall Bladder (inflamed)
RIGHT	Gall Bladder (nervous)
RIGHT	Gas T4, T6, L2 Left, T12 Right
	Leukemia - To reduce spleen and liver swelling
RIGHT	Nervous Heart T2, T3, T4, T6 (T6 is major) left, T12 Right, C3 Left
LEFT	Rash T5, T10, T12 Right
LEFT	Skin Trouble (poison ivy, oak etc.) T5, T10, T12 Right

THORACIC (T10)

RIGHT	Diabetes (Vaginal itch, itch) T8, T9, L5, T5, L4, T1
RIGHT	Eczema T5, T12 Right, T9 Left
LEFT	Edema T9 Right (for adrenals)L1 Right
RIGHT	Fever T6 Left, T12 Right, L2 Left
LEFT	Hernia on Left L1 Left, L2 Right
RIGHT	Rash T5, T12 Right, T9 Left
RIGHT	Skin Trouble (poison ivy, oak etc.) T5, T12 Right, T9 Left

THORACIC (T11)

RIGHT	Arteriosclerosis in Legs L1, L4, T5, T7 Right
	Calicum in the System T5, T7
LEFT	Erection (blood supply to penis)

THORACIC (T12)

RIGHT	Appendicitis L1 Left
LEFT	Ball of Foot
RIGHT	Constipation (feels like moving but can't) L4 Right, T6 Left
RIGHT	Constipation (doesn't feel like moving) C3 Right
RIGHT	Crying of Fear T6, T9, L4 Left
RIGHT	Eczema T5, T10, T9 Left
RIGHT	Fever T6 Left, T10 Right, L2 Left
RIGHT	Gas T4, T6, L2 Left, T9 Right
RIGHT	Glaucoma T6 Left, T7 Right
RIGHT	Hernia on Right L1 Left, L3 Left
RIGHT	Hives T5 Left
RIGHT	Nervous Heart T2, T3, T4, T6 (T6 is major)Left, T9 Right C3 Left
RIGHT	Nervous Stomach T6 Left
RIGHT	Rash T5, T10 Right, T9 Left, T5, T10 Right
LEFT	Renal Headache (Left top of head)
RIGHT	Rheumatic Headache C2, T6 Left
RIGHT	Right Pleura T1 Left, T3 Right
RIGHT	Skin Trouble (poison ivy, oak, etc.)
LEFT	Top of Head Burns T6 Right

LUMBAR (L1)

LEFT	Appendicitis T12 Right, C1 Left
RIGHT	Arterosclerosis in Legs L4 right, T5, T7, T11 Right
RIGHT	Arterosclerosis (side of lower legs)
RIGHT	Complete Urine Block
LEFT	Copious Urine
RIGHT	Edema T9 Right (for adrenals) T10 left
LEFT	Hernia on Left L2 Right, T10 Left
LEFT	Hernia on Right L3Left, T12 Right
LEFT	Inflamed Bladder
RIGHT	Metatarsals
LEFT	Renal Headache (left top of head)
LEFT	Urination - Diabetes (also see Diabetes)
RIGHT	Urine partial block

LUMBAR (L2)

RIGHT	Diarrhea T7 Left, L2 Left
LEFT	Erection (desire)
RIGHT	Erection (premature ejaculation)
LEFT	Erection (no emission)
LEFT	Fever T6 Left, T10 Right, T12 Right
LEFT	Gas T4, T6 Left, T9, T12 Right
RIGHT	Hernia on Left L1 Left, T10 Left
LEFT	Indigestion T4, T7 Left, T5 Right
LEFT	Lazy Bowel (goes every other day) T5 Right, T7 Left, T2 Left
RIGHT	Left Ankle Pain
RIGHT	Left Ovary
RIGHT	Nervous Bladder
RIGHT	Occipital Headache
RIGHT	Occipital Headache from High Blood Pressure
RIGHT	Renal Headache (right top of head)
LEFT	Sour Stomach T5 Right, T7 Left
RIGHT	Uterine Prolapse

LUMBAR (L3)

RIGHT	Constipation (doesn't feel like moving)
RIGHT	Eye Pain (behind eyeball) L3 Anterior IAW GJG
LEFT	Hernia on right L1 Left, T12 Right
RIGHT	Ovaries
LEFT	Right Ovary

LUMBAR (L4)

RIGHT	Amennorrhea
RIGHT	Arteriosclerosis in legs L1, T5, T7, T11 Right
RIGHT	Arteriosclerosis (calf of legs)
RIGHT	Constipation (feel like moving by can't) T6 Left, T12 Right
LEFT	Crying or Fear T6, T9 Left, T12 Right
LEFT	Diabetes
LEFT	Erection (desire) C2 Left, (blood supply to penis) T11 L, (nervous tolerance) T6 Left, (premature ejaculation) L2 Right, (no emission) L2 Left.
LEFT	Hot flashes (HT)
	Infantile Fallopian Tubes
LEFT	Inflamed Womb
LEFT	Intermittent Claudication
LEFT	Menses (frequent)
LEFT	Menses (Heavy flow)
LEFT	Menses (Pain)
LEFT	Menstrual Headache
LEFT	Miscarriage
LEFT	Vaginal Discharge

LUMBAR (L5)

LEFT &
RIGHT High Blood Pressure Headache (see side slip), C2 Right
RIGHT Leg Pain Right
RIGHT Leukemia (to prevent frequent attacks)

RIGHT Right leg longer and right gluteal fold inferior.
LEFT Left leg longer and left gluteal fold inferior.
RIGHT (superior) Right leg longer, gluteal fold even (S.P. left inferior). Sacral alae to superior. Right sacral tubercule, right inferior. Reverse if left pattern exists.

SACRUM

Legs even, left gluteal fold inferior. Sacral tubercule right, sacral alae left.

Legs even, right gluteal fold inferior. Sacral tubercule left, sacral alae right.

FURTHER EXPLANATION OF SURROGATE TESTING AND THERAPY LOCALIZATION

By: Paul T. Sprieser, B.S., D.C.

Abstract: A view of the surrogate testing with the concept of cellular resonance. A specific vibratory pattern that may be similar in frequency to all humans.

The use of the surrogate test has raised many questions as well as many eyebrows in its application to Therapy Localization.

I will attempt to explain this phenomena by use of the Resonant Memory Theory.

For those of us who have used this Applied Kinesiology Technique, we would usually explain it to the patient in a very simplistic manner. "If I were to hold an electric wire and you were to hold my hand you would also receive the shock" is usually an explanation that may suffice for the lay public by not for the scientific community.

METHODS:

First, let me explain for those of you who are not that familiar to this A.K. technique exactly what is done.

Let's start with the fact that the technique is usually only used with children who are to young to muscle test by themselves, or perhaps a patient who is so dibilitated with a disease such as Mutiple Sclerosis or other neuromuscular disorder that they are too weak to test.

This technique may be applied in a different variety of ways.

1. The surrogate is tested to find a stable indicator muscle that can be used to test. The area in question is then contacted by the surrogate and then the intact indicator muscle is now tested. If the muscle weakens we have a positive T.L. (Therapy Localization).

2. Another method is the surrogate will contact the area in question (example, the knee joint), patient's knee is flexed on the table. The surrogate indicator muscle is then tested to make sure that the area that is being contacted is free of any findings.

Patient is then instructed to contact (Therapy Localize) the area in question (example, the ileocecal valve). The surrogate indicator muscle will then weaken if there is a positive (T.L.) or dysfunction present.

3. A third method is to verify that the surrogate that will be used to test is free of any findings and that includes muscle weakness. The patient will then contact the surrogate body at some point that is convient so the surrogate can be tested. The areas in question are then examined and will show positive for what the patient actually has wrong!

All of the above methods make it quiet difficult to explain scientifically how this can effect the surrogate's body.[1]

However, if we take into consideration the evidence that especially in the sensory system specific nerve endings or areas respond to specific frequencies or vibration patterns such as the holographic image.

Current research shows that specific neural response to sensory feedback from both touch and proprioceptors of joints effect specific nerve receptors in the brain. This can very easily be

seen in the sensory system of sight and hearing, although this is not a new theory.

Dr. Paul Weiss in 1936 had a theory on selectivity in fibers connection (Radio Broadcast Modle) based on resonance effects involving diffuse morphological interconnection with impulses specificity and selective neuronal and end-organ attunement. Like a radio pickup, the "resonance principle" provided selective responce in the presence of diffuse nonselective synaptic connection.[2]

What is meant by "Resonance is the property whereby any vibratory system responds with maximum amplitude to an applied force having a frequency equal or nearly equal to its own."[3]

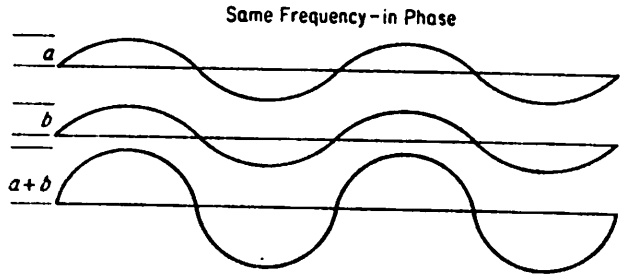
Webster's dictionary defines resonance as: a vibration of large amplitude in a mechanical or electrical system caused by a relatively small periodic stimulus of the same or nearly the same period as the natural vibration period of the system.[4]

"Resonance occurs when the frequency of an applied force equal one of the natural frequencies of an occillator."[5]

It was also stated that "all rigid structures have resonant frequencies."[6] This would apply to all skeletal structures.

The next statment applies to the oscillation and frequency can also be applied from this physics principle to neural physiology. "When periodic impulses are given to a string at frequencies other then those of its fundamental frequency and overtones, hardly any response occurs, similar to the (All or Nothing Law). This situation then, is like pushing a child's swing at a frequency different from its natural one, which produces oscillations of negligable amplitude. All rigid structures possess characteristic natural

frequencies though their vibrations may be more complex in character then those of a stretched string, and these vibrations can be excited by a stimulus of the proper frequency. The traditional example of a goblet shattering when a violin is played with the right frequency is an illustration of resonance (Fig. 1).[7]



(FIG. 1)

Examples of this phenomena are sound frequencies which stimulate only specific nerve endings in the cochlea of the ear to produce what is interperted as high or low sounds.

Sound, as you know, also travels in wave lenghts the human ear can discern levels from 20 Hz to 20,000 Hz. The lower and slower the resonance or vibration the lower the sound and the higher the vibration the higher the sound.

The ability of the human ear to differentiate difference in sound is because of the three dimensions of sound waves have.

The three dimensions are, a). Intensity (loudness), b). Quality or Frequency wave (pitch), c). Purity (timber).[8]

These sounds and tones are perceived by the human ear because they cause specific stimulation of the hair like receptors in the cochlea of the ear that respond to a specific wave lenght or resonance

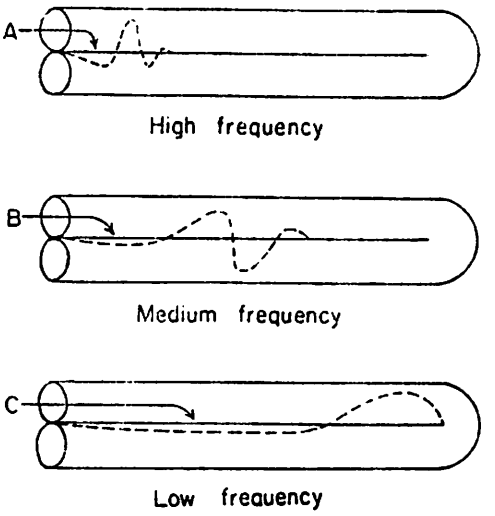


Figure 19-4. Diagrammatic representation of "traveling waves" along the basilar membrane for high, medium, and low frequency sounds.

(Fig. 19-4)

Guyton: Pattern of vibration of the Basilar Membrane for different sound frequencies. Note in Figure 19-4 the different pattern if transmission for sound wave of different frequencies. Each wave is relatively weak at the onset but becomes strong when it reaches the portion of the basilar membrane that has a natural resonance frequency. At this point the basilar membrane can vibrate back and forth with such great ease that the energy in the wave is completely dissipated. Consequently, the wave ceases at this point and fails to travel the remaining distance along the basilar membrane. Thus, a high frequency sound wave travels only a short distance along the basilar membrane before it reaches it resonant point and dies out, a medium frequency sound wave travels about halfway and then dies out and finally, a very low frequency sound wave travels the entire distance along the membrane (Figure 19-5).[9]

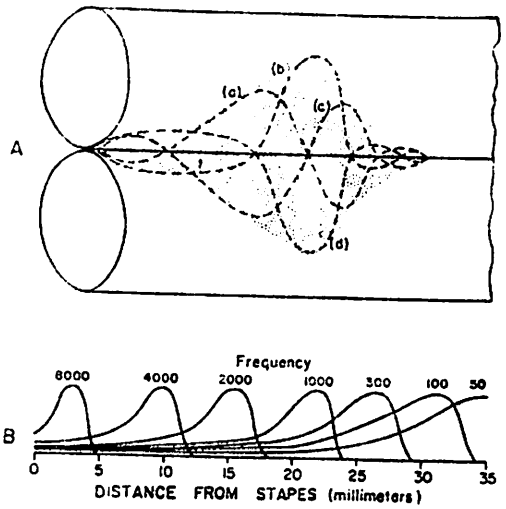


Figure 19–5. (A) Amplitude pattern of vibration of the basilar membrane for a medium frequency sound. (B) Amplitude patterns for sounds of all frequencies between 50 and 8000 per second, showing the points of maximum amplitude (the resonance points) on the basilar membrane for the different frequencies.

(Fig. 19-5)

Color to the cones of the eye most likely stimulates specific receptor for certain colors, because each color would have a different wave length or vibratory pattern (resonant pattern).

"It will become evident that three different types of pigments are present in different cones, thus making these cones selectively sensitive to the different colors, blue, green, and red. The absorption characteristics of the pigments in the three types of cones show peak absorbancies at light wavelengths, respectively, of 430, 535, and 575 millimicrons. These are also the wavelenghts for peak light sensitivity for each type of cone, which begins to explain how the retina differentiates the colors.[10]

The more intense the stimulus, the more rapid the firing of the visual field.

The frequency of wave length the chart shows the color and visable light represent only a small part of the Electromagnatic

wave length from 700 Hz to 400 Hz or stated (4.3×10^{14} to 7.5×10^{14}).

Red is 650-700 Hz., Orange 600-650 Hz., Yellow 560-600 Hz., Green 500-560 Hz., Blue 440-500 Hz., Violet 400-440 Hz., (Figure 23-5).[11]

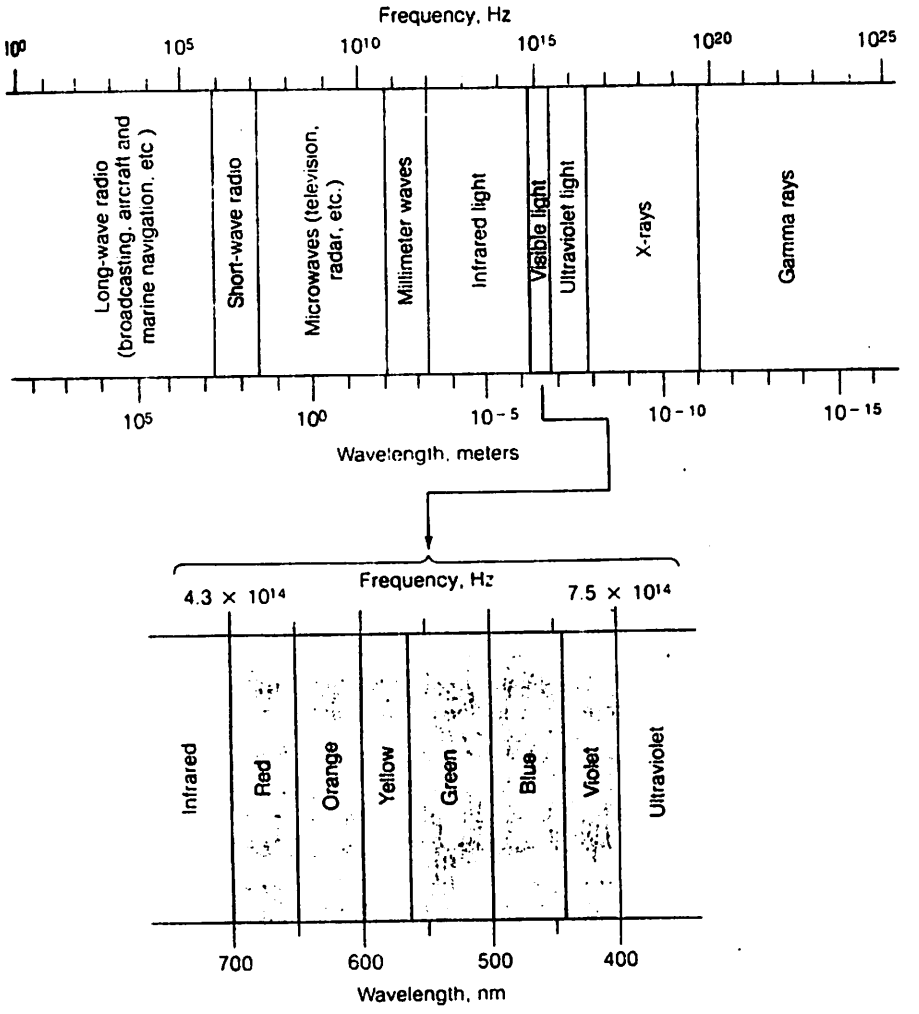


FIG. 23–5 The electromagnetic wave spectrum. The boundaries of the various categories are not sharp. (1 nm = 1 nanometer = 10^{-9} m.)

(Fig. 23-5)

So color can be preceived because of the specific stimuli to the retinal field or to specific cells in the visual cortex that resonate to that specific frequency of light.

With sight if the two color are of equal intensity then the color that is preceived will be a combination rather then the basic color example red and yellow will combine and be preceived as orange.

Taste and Smell would also have specific vibratory patterns due to the molecular difference of the sent or taste and the stimuli to specific endings of the olfactory and taste bud system and perhaps specific cells in the various regions of the brain, such as the limbic system.

Detection of different sensations of taste by the taste buds: Since each taste bud responds to mutiple primary taste stimuli, it is difficult to understand how a person perceives the different primary taste sensations independently of each other. However, a theory attempting to explain this is the following. Some area in the nervous system presumably is capable of detecting the ratios of stimulation of the different types of taste buds, (figure 20-1).[12]

TABLE 20–1. Relative Taste Indices of Different Substances

Sour Substances	*Index*	*Bitter Substances*	*Index*	*Sweet Substances*	*Index*	*Salty Substances*	*Index*
Hydrochloric acid	1	Quinine	1	Sucrose	1	NaCl	1
Formic acid	1.1	Brucine	11	1-propoxy-2-amino-4-nitrobenzene	5000	NaF	2
Chloracetic acid	0.9	Strychnine	3.1	Saccharin	675	$CaCl_2$	1
Acetyllactic acid	0.85	Nicotine	1.3	Chloroform	40	NaBr	0.4
Lactic acid	0.85	Phenylthiourea	0.9	Fructose	1.7	NaI	0.35
Tartaric acid	0.7	Caffeine	0.4	Alanine	1.5	LiCl	0.4
Malic acid	0.6	Veratrine	0.2	Glucose	0.8	NH_4Cl	2.5
Potassium H tartrate	0.58	Pilocarpine	0.16	Maltose	0.45	KCl	0.6
Acetic acid	0.55	Atropine	0.13	Galactose	0.32		
Citric acid	0.46	Cocaine	0.02	Lactose	0.3		
Carbonic acid	0.06	Morphine	0.02				

From Derma: *Proc. Oklahoma Acad. Sc.*, 27:9, 1947; and Pfaffman: Handbook of Physiology, Sec. I, Vol. 1, p. 507, 1959. Baltimore, The Williams & Wilkins Co.

(Fig. 20-1)

We can assume that this premise will also hold true for smell, because of their interconnection. The resonant pattern of the substance and its activity on the taste buds or nasal receptors causing them to respond in a specific way.

Each taste will cause a specific index index of response to the sensory cells in the specific area and to specific neuronal sites within the central nervous sustem.

The Skin can respond to many types of stimuli such as touch, temperature, vibration, pain, etc. This is due to specific neural receptors found in the skin that will resonate or respond to specific stimuli.

TYPES OF RECEPTORS:

1. Pain use free nerve endings of the skin.
2. Deep pressure (touch or pressure) Pacinian corpuscles.
3. Tactile sensitivity are Meissner, Merkels, Raffini, Krause, Glomar endings.

The Meissner Corpuscles are abundant in those parts of the body which are relatively hairless such as the palms and soles.

There are specific frequency or resonant patterns that certain receptors will respond to some examples are:

1. Touch threshold of eye is 2 grm/m.m.^{2}
2. Fingers 3 gm/m.m.^{2}, Tip of finger 5 gm/m.m.^{2}
3. Sole of foot 25 gm/m.m.^{2}

Pain has specific threshold for response.

1. Back of hand 100 grams/m.m.^{2}
2. Sole of foot 200 grams/ m.m.^{2}
3. Finger tip 300 grams / m.m. ^{2}

The skin can also detect vibration in resonant frequencies between 10 and 2,000 Hz.

Detection of vibration: All the different tactile receptors are involved in detection of vibration (Resonance though different receptors detect different frequencies of vibration). Pacinian Corpuscles from 400 to 500 Hz/sec, and transmit over Beta type A

nerve fibers, which can transmit more than 1000 impulses per second. Low frequency vibrations up to 80 cycle per second stimulate other receptors specially Meissner Corpuscles.[13]

The skin shows selectivity by causing inhabition of surrounding region. This noted (Figure 9-4) and is stated that furthermore, where as primary cutaneous afferent fibers do not display lateral inhabition neurons of the dorsal column nucli, VPL nucleus.[14]

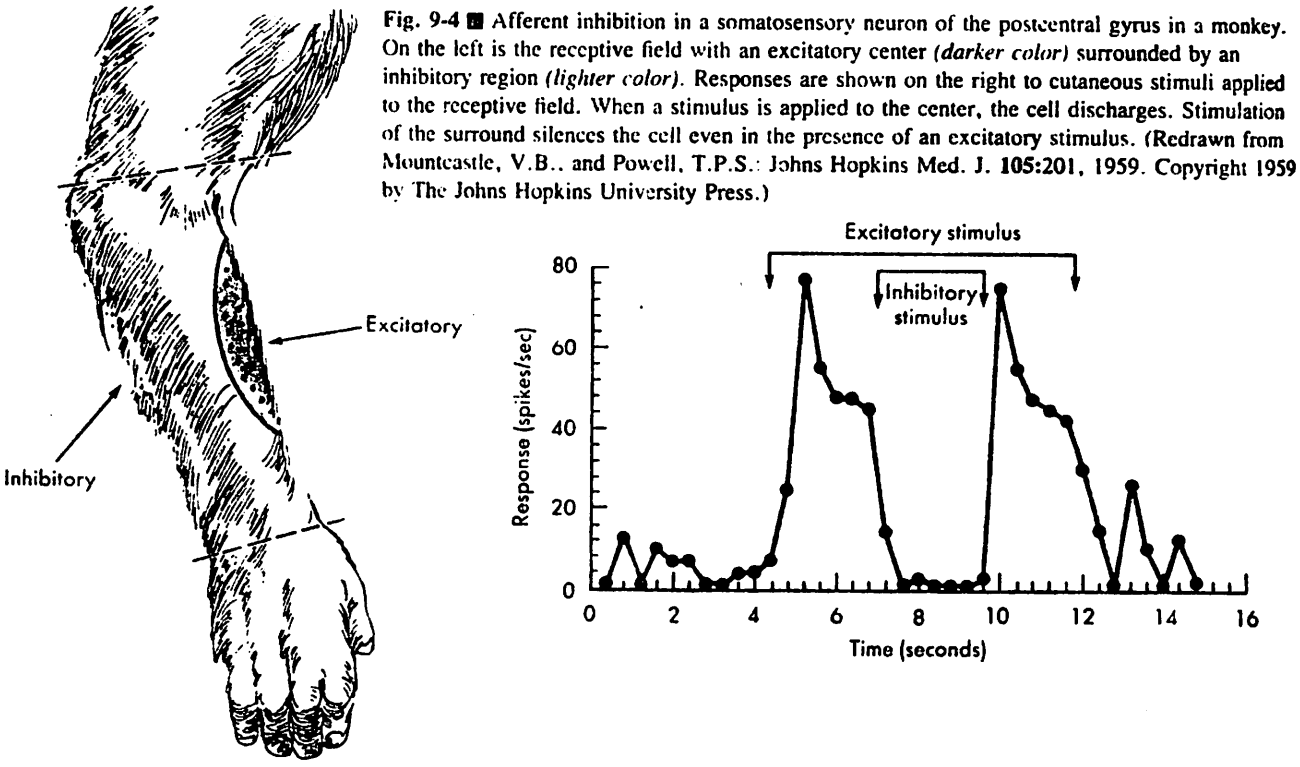


Fig. 9-4 ■ Afferent inhibition in a somatosensory neuron of the postcentral gyrus in a monkey. On the left is the receptive field with an excitatory center *(darker color)* surrounded by an inhibitory region *(lighter color)*. Responses are shown on the right to cutaneous stimuli applied to the receptive field. When a stimulus is applied to the center, the cell discharges. Stimulation of the surround silences the cell even in the presence of an excitatory stimulus. (Redrawn from Mountcastle, V.B., and Powell, T.P.S.: Johns Hopkins Med. J. **105:201**, 1959. Copyright 1959 by The Johns Hopkins University Press.)

(Fig. 9-4)

Many cells in the somastosensory cortex exhibit a dramatic direction selectivity (Figure 9-5) that is, they respond when a cutaneous stimuli, such as a brush, is moved in one direction across the skin but not for the reverse or orthogonal directions. This analogous to the direction and orientation selectivity seen first in the geniculocortical system amoung cortical neurons.[15]

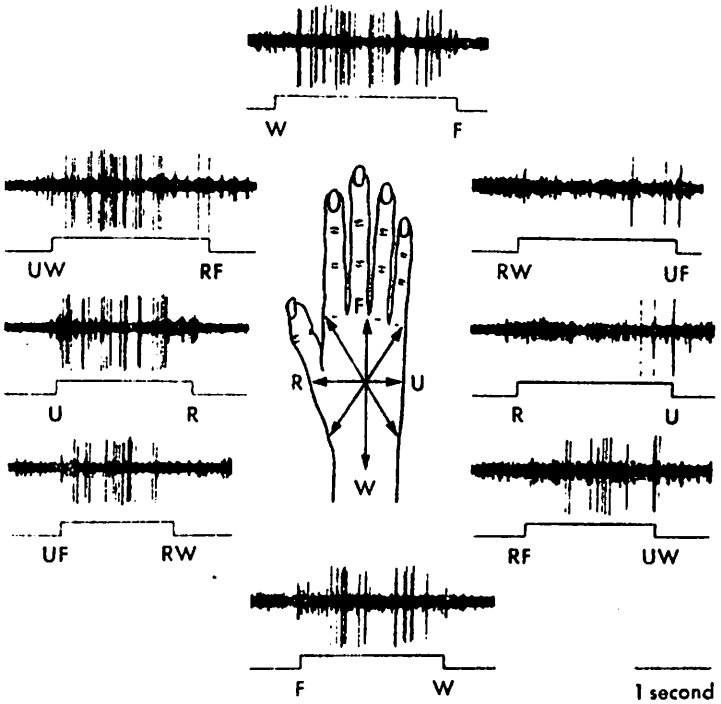


Fig. 9-5 ■ Direction selectivity in a somatosensory neuron of the postcentral gyrus in a monkey. The neuron is most responsive to cutaneous stimuli moving along the axis *UW* to *RF* and least responsive along the axis *RW* to *UF*. (From Costanzo, R.M., and Gardner, E.P.: J. Neurophysiol. 43:1319, 1980.)

(Fig. 9-5)

Many touch sensitive neurons in the dorsal column system respond tonically to touch or pressure.

Specificity and Selectivity can been seen in this section titled "Kinesthesia"-Neurons sensitive to joint position or angle have also been studied thoughout the dorsal column system. They generally exhabit tonic responce, that encode joint angle in the frequency of firing (Figure 9-6A). As Figure 9-6B shows some of these cells increase firing with increased flexion, and others inccreased rease firing with increased extension. These replica are in the appropriate transducer-containing tissue.[16]

The brain presumably has little difficulty determining which joint has been stimulated, a spatial resolution is of less concern in Kinesthesia that is the frequency code that is used analyze joint angle.

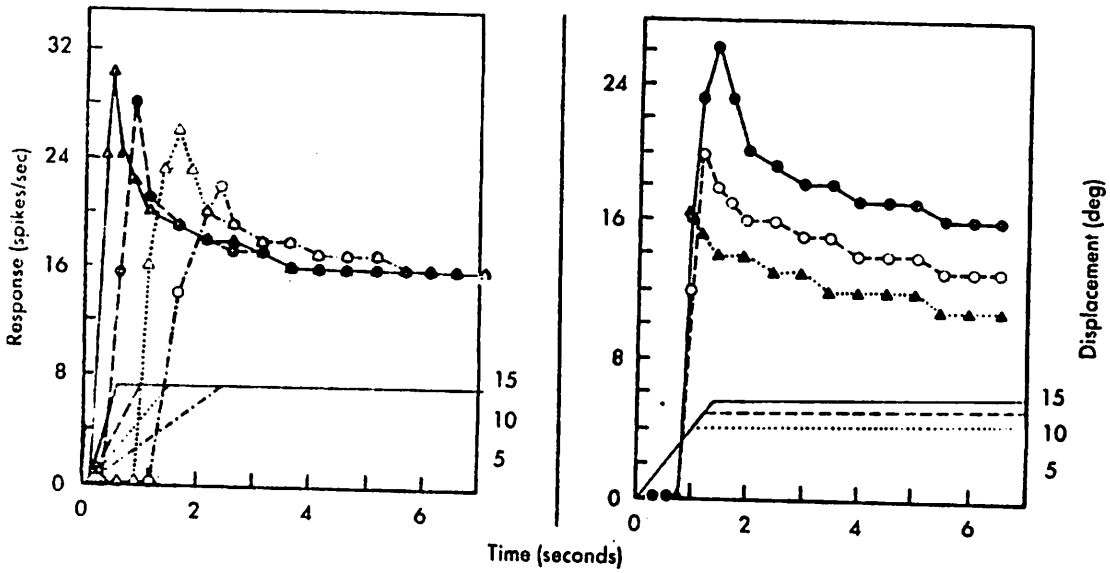


Fig. 9-6 Response properties of kinesthesia-sensitive neurons. A, Responses of primary afferent fibers to flexion of the knee of a cat. As shown, the magnitude of the sustained response encodes the extent of flexion, whereas the rate of flexion determines the rate of rise of the response.

(Fig. 9-6A and 9-6B)

SOMATOTROPIC ORGANIZATION:

The tertiary fibers maintain a precise and regular spatial relationship amoung themselves. An orderly map of the body, or homunculus, thus exists for the somatosensory cortex.

Despite the somatotropic organization, there is no modality mixing within single cells at any level of the system for the dorsal funiculi of the spinal cord to the somatosensory cortex; that is the cells respond either to touch or to joint change but never to both.

All the evidence shown for touch or Kinesthesia sensory feed back showed that there is specificity to particular signals exists.[17]

That it is quite possible that a resonant pattern exists for all areas of the body with specific receptors for that particular signal.

Muscles have also been found to respond to specific electrical stimulus that correspond to the length of that specific muscle.

In research tape #76 Dr. Goodheart speaks about this phenomena that complex wave forms generated with patterns of interference which form a hologram. This was related to the use of a Hertz Frequency Unit called the MYOMATIC developed by Tom Wing.

To quote Dr. Goodheart from this tape. "Frequency of a muscle is proportional to its length. If we use a Hertz frequency much longer then the muscle itself in terms of numbers. The trapezius muscle 2 to 6 Hz units or the Quadrecepts would be 18 to 24 Hz units. If you attempt to use the same time of treatment of an obvious muscle weakness the muscle does not respond. But if you use the right frequency it does."

"They transplanted the biceps to the gastrocnemius site of the opposite limb and gastrocnemius into biceps position. Then when nerve energy is fired at the appropriate level, the cervical level for the biceps and the sacral-lumbar region for the gastrocnemius. When you stimulate the cervical level, which would normally fire the biceps, but it has the gastrocnemius in place. The gastrocnemius doesn't fire, but the biceps lower down does and when you do the same nerve firing to the lumbar area the gastrocnemius fire up above. Indicating the muscles have wave lengths.[18]

This again shows the specificity does exist within the nervous system and also a hologramic memory for what should occur.

CONCLUSION:

I believe this specific resonance or vibratory pattern can be

seen in almost every area and cell of the body. This concept can be carried foward to the idea that each region, organ, muscle, joint, in the body including the specific nuclei receptors, may be able to give off a specific resonant frequency that is similar in each human and is common to all humans for that specific region of therapy localization.

If this specific resonant frequency pattern is correct and there is a similarity in these resonant holographic patterns in the various neural centers of the brain; which current research seems to bear out these findings.

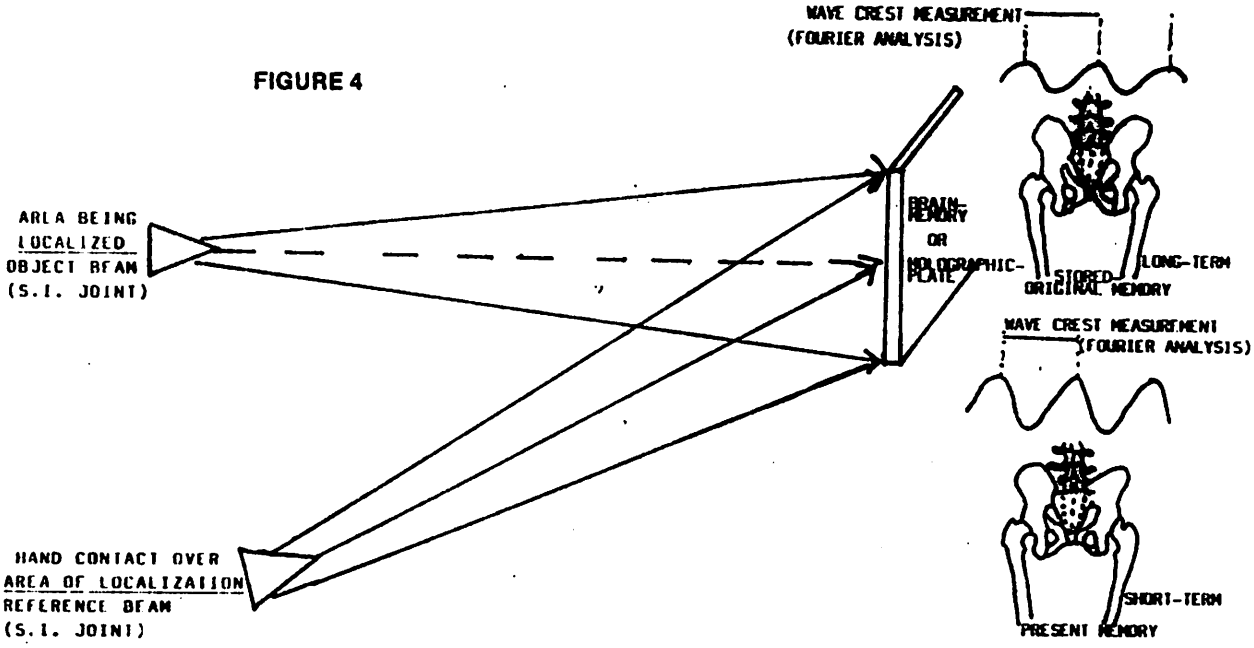
It would stand to reason when we have the patient contact the surrogate or visa versa. The similarity in resonant pattern can be recognized by the surrogate's nervous system allowing us to get the same therapy localization as if we were testing the patient directly.

Let me see if I can give you an illustration of this premise. Take for example my revised paper on Holographic Memory and Therapy Localization (figure 4). The sacroiliac joint and the over lying skin may have a normal resonant pattern of 475 Hz per second and the palm of the hand may have a resonant pattern of, let say 225 Hz per second. These two frequencies are theoretical you understand, but if it is true all humans may have a similar resonant frequency pattern for this area.

Now then the surrogate therapy localizes the patients sacroiliac joint his body compairs the normal S.I. joint frequency of 475 Hz per second to what exists at the present time an for example the patient has a Category # 2 P.I. on the left side. The sacroiliac joint frequency may now register 530 Hz per second resonant

frequency. Since the nervous system will due a Fourier Transform in the hologramic concept it compairs and measures the height and width of the wave crest and can recognize the difference between 475 Hz per second perfect sacroiliac joint to the now present 530 Hz per second subluxated sacroiliac joint and we get the positive therapy localization in the surrogate.[19]

FIGURE 4



CNS OR BRAIN

PERFORMS THE FOLLOWING:

A. FOURIER ANALYSIS OF WAVE CREST OF SENSORY FEED BACK FROM HAND AND S.I. JOINT BEING THERAPY LOCALIZED. (COMPARES HOLOGRAPHICALLY)

B. THE STORED ORIGINAL MEMORY (LONG-TERM) TO THE PRESENT MEMORY (SHORT-TERM).

C. IF THE MEMORY OR IMAGES DON'T COMPARE (MATCH UP) WE GET A POSITIVE THERAPY LOCALIZATION-WEAKNESS OF INDICATOR MUSCLE.

(Fig 4)

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NIGHTSHADE SENSITIVITY - PATIENT INFORMATION SHEET

Robert P. Radtke D.C.

Abs. Testing for nightshade sensitivity is an important part of any screen for food sensitivity.

Members of the solanacea family of plants have long been recognized in both European and Asian traditions as potentially toxic. The role of nightshade plants has recieved attention in recent years as a source of arthritic symptoms. Our experience has been that many other symptoms are produced in response to their toxic alkaloids. These alkaloids act as cholinesterase inhibitors, as do many insecticides. It was discovered that in cattle as little as 100 grams of dry Solanum malacoxylon would result in an increase in blood calcium and phosphate approximating 50% of normal values within a period of 48 hours. Soft tissue calcification is reported endemic in all areas where livestock feed on this species.

In a recent conversation with Dr Childers, I shared the observation that mental and gastrointestinal symptoms were much more frequently observed as acute responses to ingestion. In his own case he reported that his diverticulitis symptoms had remitted by eliminating the nightshades. Other health improvements associated with the diet include, skin rash, sluggish mind mental depression, poor endurance, aches and pains in muscles and joints, headaches, and diarhea.

In our experience, tomatoes provoke the most acute symptomatic responses. When testing for sensitivity, initially only tomatoes are screened for sake of expediency. If a positive response is noted, potato, eggplant, green pepper, paprika and tobacco are evaluated at the next appointment (where relevant). Be aware that in the extremely reactive patient, even paprika which is widely used, can be problematic. Once a sensitivity has been identified through lingual testing, we will send the patient home with the handout appearing on the following page.

Summary

Thorough abstinence from nightshades may be required to achieve a good response from the symptoms produced by their ingestion. Patient education is vital. Use of the handout helps establish credibility in the diagnosis and patient compliance is more likely. This is important because 6 to 12 months cooperation may be needed in the resolution of chronic joint disability. In addition, the handout has a recipe for a substitute in tomato dishes. Please reproduce it and use it in the best of health.

A Diet to Stop Arthritis; The Nightshades and Ill Health, Somerset Press, Norman F. Childers, is available by writing the author at 3906 N.W. 31st. Place, Gainesville, Fl. 32606

Arthritis Can Be Eased by Eliminating Nightshades, was exerpted from a news clipping. It was written by June Roth

ARTHRITIS CAN BE EASED BY ELIMINATING NIGHTSHADES

Years of testing and study have led Norman Childers, a professor of horticulture at Rutgers University, to believe that most arthritic problems may be controlled by not using or eating the nightshade plants. Childers bases his statements on more than 100 years documented research with the white potato and tobacco, and more recently with studies that include the tomato, eggplant, and all kinds of peppers except black pepper. He has written a book with case histories, showing how people have been helped by the avoidance of these plants. "A Diet to Stop Arthritis: The Nightshades and Ill Health," published by Somerset Press in Somerville New Jersy, shows how to eliminate the nightshade plants from the life of the arthritic person.

The nightshade plants are all closely related members of the solanecea family of plants, some of which are highly toxic- such as the deadly black nightshades. Popular edible members of this family are potatoes, tomatoes, and all peppers.

Childers is a well known scientist and makes no rash claims in this book. He states that although the nightshade plants are very important and widely popular, less than ten percent of the population is sensitive to them. More than 5,000 people in the United States, Canada, the Carribean and Europe participated in the study of nightshades upon which the book is based. More than 72 percent have recoverd completely or shown marked improvement,

depending on the developemental stage of arthritis in the back, neck, chest or limbs. The book includes a small section of menus and recipes that involve tomato substitutes. Here are several recipes from Childers.

CARATO (Substitute for tomato)

4lbs. fresh carrots

2 large beets

1½ cup water

2 lemons

Cut carrots and beets into sections and cook in water at high temperature in a covered pot for 10 minutes. Add juice of the two lemons and mash with a potato masher, or use a food processor. Use this mixture as a tomato substitute in your own recipes and in those that follow. Makes about 8 cups sauce.

CATSUP

1 cup carato, 2 T. water, ½ t. salt, 5 T. vinegar, 1 T. honey, ¼ t, onion powder

Combine all ingredients in blender and blend at low speed for 60 seconds. Makes 1½ cups.

CARATO SAUCE

6 cups Carato, 1 cup water, ¼ cup olive oil, 1 large onion, chopped, ½ cup Parasan cheese, 2 T. oregano, 2 t. garlic powder, 1 t. basil, ½ t. salt, ½ t. black pepper, juice from one lemon.

Cook at high temp. 30 min. in a large pan, stirring frequently.

IDENTIFICATION AND CLASSIFICATION OF POSTURAL DISTORTION PATTERNS

Robert P Radtke D.C.

Abstract- Distortion in the anterior to posterior postural dimension creates, aggravates, and perpetuates the most commonly observed patient complaints. Clinical manifestations are extremely varied, the best theraputic responses are obtained by dealing with the entire complex.

Postural distortions may be divided into two broad catagorys. One, rotational patterns including spinal torque and scoliosis, and second is a distortion complex involving multiple aberations in the anterior to posterior dimension. It has been described in part as a forward head posture. In total it represents an anterior to posterior collapse encompassing the skull, TMJ and all elements of the postural mechanism. Patient complaints will vary tremendously. Often even within one patient a progression of symptom displays will emerge as the complex pattern unwinds. Combination patterns with both rotational and anterior to posterior aberations are frequently observed as well. In one respect the A. to P. collapse displays as an exaggeration in the three primary curves in the spine. This is best visualized on the lateral postural analysis. A plumb line through the external auditory meatus will fall ahead of the mid humerus. There will be an increase in the thoracic kyphosis and in the lumbar lordosis. The pelvis will be flexed forward on the hips.

Michael W. Darnell R.P.T. described a proposed chronology of

of events for forward head posture in the Sept. 1983 issue of The Journal of Crainiomandibular Practice. In a discussion of the role of masticatory dysfunction he reports:

During the past few years, much attention has been focused on the effects of head, neck, and shoulder posture on mandibular position, and consequently on masticatory dysfunction. When the occiput is bent backwards the mouth opens and the mandible retracts. When the mouth closes with the occiput in this backward bent position the suprahyoid and infrahyoid muscles will come under increased tension. The position of the scapula, to which the omohyoid is attached, will also influence the length/tension relationship of the hyoid muscles.

As the mandibular condyle assumes a retro position in the joint, the superior head of the lateral pterygoid becomes stretched. By reflex action, this stretch may lead to premature contraction, causing the disc to become anteriorly displaced. As the mandible's position and the hyoid's length/tension relationship is changed, the occlusal contact pattern, the arthrokinematics of the temporo-mandibular, and the position of the hyoid bone wil likewise be changed. If the hyoid bone is elevated and mouth breathing compounds the situation, the tongue may assume a lowered position, and abnormal swallowing patterns can result.

An altered mandibular position can create a vicious cycle, causing an altered occlusal contact pattern and altered temporo-

mandibular joint mechanics. This cycle may lead to many symptoms involving the masticatory system. The rest position of the mandible is that point at which equilibrium should exist between the muscles that elevate the jaw and those that depress it. The activity of these muscles is related to that of the neck and trunk muscles as well as to the direction of gravitational forces acting upon the system. An imbalance in any of these muscles may thus have widespread effects."

In discussing the A. to P. collapse beyond the head and jaw, familiar symptom constellations will appear as our investigation moves caudad. Recurrent extension faults are seen at the upper cervical area, including fixation and subluxation patterns in the osseous structures. In the soft tissues, hypertonus, trigger points, and myofascial contracture are seen. Involvement with the greater and lesser occipital nerves is possible. The nucleus of the trigeminal nerve extends as far down as the third cervical, thus allowing involvement of the fifth crainial nerve (trigeminal neuralgia).

As the head moves further forward, contraction in the cervical extensor muscles increases. This accelerated compressive force over time generates the frequently observed patterns of spondylosis seen in the lower cervical spine. Dr. Goodheart has discussed this as the hidden cervical disc complex. Thoracic outlet syndromes are frequently observed in this context. The lower cervical spine extends while the upper thoracic segments flex forward thus producing a "dowager's hump" appearance.

When the upper dorsals move forward, the ribs impact into the sternum producing readily palpable (and painful) enlargements at the costo-sternal articulations (Tietze's syndrome). Be aware that these compressive forces also generate similar proliferative changes at the angle of Louis. The medial ends of the clavicle will suffer in the same fashion as the shoulders roll forward and the pectorals contract. As the thoracic cage as a whole descends on the abdominal contents, hiatal hernia and diaphragm involvements will present as the chief complaint. This crowding of the abdominal contents interferes with digestive function by restricting normal motility of the gut.

Increase in the lumbar lordosis creates, aggravates and perpetuates the problematic low back or sciatica. As the base of the sacrum follows the lumbar spine forward into extension, the inominate will subluxate posterior into a classic catagory two configuration. The pelvis as a whole will flex forward on the hips. As this occurs, any "excess baggage" tends to spill over the belt line and further compound the increasing lordosis in the lumbar spine.

With flexion present in the pelvis, the knees lock into an extended position. Pulling tight soreness is observed in the hamstrings under these conditions. Recall that the posterior tibial muscles are often found weak and unsupportive of the medial plantar arch with the hidden cervical disc syndrome. Foot pronation, arch pains, and knee problems are both symptoms and causes in this

breakdown in the postural mechanism.

CONCLUSIONS

It has been useful in practice to assign the patient's chief complaint to a context involving a particular distortion pattern. That is, as a component of a rotational or A. to P. distortion (or both). In describing findings to the patient it is helpful to describe a symptom as only a part of, or the tip of an iceberg. Having done this in advance, patients are prepared to accept therapy applied to areas and systems remote from the area of chief complaint. Also, when retracing of old symptoms occurs, these problems are understood in a meaningful context.

This model has use for the doctor as well. If a weak indicator muscle has been cleared, it may return by increasing the original distortion pattern, either torque (use blocks or a gait challenge) or by increasing the A. to P. distortion. In a sciatic pattern, dropping the lumbar spine forward or tilting the pelvis into flexion with a prone subject will often precipitate hidden weakness. This allows further processing of the distortion and a better resolution. The converse is also true. Untorquing or flattening out the spinal curves will often clear a weak indicator, thus giving the doctor a theraputic direction.

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Enhancing the Educational Experience of Applied Kinesiology
(developing an effective workshop)

Marc S. Rosen, D.C.

Abstract:

A design for the workshop portion, of an introductory series of lectures, is presented as a means of enhancing the educational experience of applied kinesiology.

The workshops explore the clinical assessment of function, through instrumentation, physical examination procedures, laboratory tests, and the dynamic evaluation of biomechanics.

Participants actually perform these procedures, in conjunction with those unique to, or characteristic of the practice of applied kinesiology.

A traditional seminar format has therefore been modified, so as to become an exercise in making, then correlating clinical observations/findings.

Educational Experience of A.K.
Rosen ... page one

Introduction:

For the most part, the study of applied kinesiology, is an endeavor that remains separate and distinct from the academic environment, of institutions that grant degrees in the healing arts.[1]

For students who are enrolled in these institutions,(typically chiropractic colleges),the investigation of applied kinesiology is oftentimes postponed until "after graduation". As it represents additional demands upon an already limited time and financial resource..

Unfortunately, a "first year" practitioner is usually in no better position, as far as available time and funds are concerned.

Still, many students do find their way clear, and develop a considerable competence with applied kinesiology, prior to or shortly after graduation.

The academic neglect, or de-emphasis of the functional aspects of illness,(or of a heritage of natural health care),has prompted many students to seek educational avenues, that do address the issue of functional health problems,(in terms of their clinical assessment/management).

Chiropractic, osteopathy, homeopathy, classical acupuncture, naturopathy, and ayurvedic medicine were founded upon theoretical laws and principles,(that were thought to govern the human condition).[2,3,4,5,6,7]

Without a conceptual basis, the "orthodox", or allopathic diagnosis and treatment of disease tends to be of a mechanical and technological orientation.

Characteristics, while vital to the efficacy of "crisis care", poorly confront the challenge of functional illness.

The authors experience of the healing arts, perhaps one common to many others, suggests "orthodox" psychosomatic labels, and the routine use of therapies that only further disorganize the causal neuroendocrine disorganization.*

* the reader is refered to discussions of neurologic disorganization and functional endocrine problems, contained within references 12,19

Introduction: (cont.)

A state that defys detection, and correction, by the invasive, expensive technology that appears to be the growing emphasis of the allopathic approach.[8]

If the need to investigate the functional nature of a health problem does exist, and if alternate methods of diagnosis and treatment are to be mastered, then where might a student receive clinical guidance in this regard.

The workshop portion of an applied kinesiology seminar, can be easily modified so as to simulate a clinical setting.

Under the supervision of a teaching diplomate, seminar participants can be guided through making, then correlating a wide variety of observations. All in an attempt to better understand the functional status of the areas under investigation,(usually the topics for the lecture portion of the session).

Previous papers,(appearing in the Collected Papers), that have dealt with "hands-on" workshops, or other educational aspects of applied kinesiology, are those referenced through numbers 9,10,& 11.

Discussion:

Upon the request of a group of students from the Western States Chiropractic College, the author traveled to Portland to deliver a series of five lectures on applied kinesiology.

The syllabus was self designed, and will be presented as a separate paper, on a later date.

Each ten hour session included a minimum of three hours of workshop. The lecture-workshop format was patterned after a college curriculum. Where traditionally, both basic and clinical science is taught through specified hours in the lecture room, and then in a laboratory.

In order that the series might have the benefit of a "purpose", it's theme or emphasis was that; the art of clinical observation and the study of applied kinesiology are inseperably one in the same.

Discussion: (cont.)

When instruction in the art of observation, preceeds a lecture on scientific and clinical significance, a students mind is free to simply observe. Thereby developing the visual, motor, and palpatory skills necessary to assess the functional status, of a wide variety of living structures and processes.

By specifying what and how certain observations are to be made, the workshop is a potential source of unbiased data. As participants are recording observations/findings without any preconceived notions as to their meaning or validity.

It is reasonable, however, to question the reliability of inexperience.

In total, three individual workshops will be outlined. It would be foolish to assume that their form is complete, or even adequate.

Additions, deletions, and/or corrections will be considered as needed. This will, possibly, enhance the educational experience of applied kinesiology.

Proposed Workshop #1

Under investigation are the basic analytical/diagnostic "tools" employed through applied kinesiology.

To properly apply these "tools", the workshop also addresses the art of muscle testing.

Students work in pairs. The first observations to be made, are of their partners posture.[12] With each student observing the same area, at the same time, beginning with the ankles, then moving sequentially toward the occiput, posture,(structural balance), is visualized from the posterior, lateral, and from the anterior.

As each area of musculo-skeletal anatomy is inspected, the muscles responsible for characteristic distortions, are loudly related to the class. Other relevant correlations,(muscle-organ association, muscle relation to a pelvic fault etc.), are also

Discussion: (cont., proposed workshop #1)

verbally presented as students are "walked" through a postural analysis.

After examiner and subject roles have been reversed, subjects assume a supine position on the very uncomfortable seminar tables.

A drawing of the Temporal Sphenoidal Line is placed on the subjects chest. For this exercise, the author personally palpated each students T.S. line, therby offering both an opportunity to experience and to observe a palpatory search for T.S. nodules.[13]

When T.S. line palpation has been completed, the pectoralis major clavicular and hamstrings are tested.[14] Again, each student experienced and witnessed the authors manual tests.

Using a strong indicator, prone subjects are instructed to therapy localize the fifth lumbar. A positive or negative response is noted.[15]

Irregardless of the nature of the response,(to therapy localization), the fifth lumbar is challenged by directional pressure to it's spinous process.[16] Should a challenge identify a subluxation, a respiratory assist is applied.

So that the exercise might simulate an actual clinical situation, the nature of the subluxation and the direction of correction are expressed aloud.

At this point the lovett relationships, along with the theoretical mechanisms of therapy localization and the vertebral challenge were reviewed/restated.[17]

While the remainder of the class continues to test muscles, palpate T.S. lines, perform postural analysis, and subject the upper cervical/lumbar regions to therapy localization and challenge, groups of six witnessed a demonstration of the applied kinesiology management of spinal fixations.[18] Only the thoracic spine was dealt with. All analysis and corrections were simulated.

The workshop portion of session four was in part, devoted to the tarsal tunnel syndrome, with small groups observing a demonstration, then examining a partner.

Discussion: (cont.)

Proposed Workshop #2

It is this style workshop that was the subject of the introduction.

Students were asked to come equipped with a pen light, stethoscope, and sphygmonometer. A list of the procedures to be followed and the observations to be made appeared on "the board", in the seminar room.

Working in groups of four,(so that at least three pupils would be observed, chests auscultated etc.), students were instructed to:

- observe the pupillary response to light (19)

- record the systolic blood pressure sitting, supine, then standing (19)

- ascultate the heart, comparing the relative intensity of the first and second heart sounds,(at the pulmonic valve) 19

- perform a postural analysis for the presence of genu valgus, an elevated iliac crest, or internal/external rotation of the lower extremity

- test the sartorius and psoas muscles

- using an intact indicator muscle, such as a tensor fascia lata, retest the indicator with a lead square placed over the subjects lips

- measure vital capacity with a spirometer 20

No other information was given, although the exercise was prefaced with the assurance that the findings would be discussed in detail.

The findings were recorded on whatever paper was available, then surveyed via a "show of hands". This proved to be a totally inadequate means of gathering data. It was an unsuccessful attempt to reveal possible correlations between the postural suggestion of a muscle weakness, the actual response of those muscles to a manual test, and with functional measures of associated organs.

A lecture on the biologic response to environmental stressors,

Discussion: (cont., proposed workshop #2)

the general adaptation syndrome, and the etiology and assessment of functional hypoadrenia [21,22,23] followed the workshop.

The various physical exam findings, and their proposed physiological mechanisms could be easily related to, as the student had already observed the phenomenon, refered to in the lecture.

Proposed Workshop #3

This exercise in making then correlating clinical observations was a continuation of/expansion upon workshop #2.

A written procedural outline was provided, along with a data sheet upon which the findings could be recorded for collection and analysis.

The workshop was scheduled for the second day of the session, so that students could bring with them a first morning urine sample, (collection containers were distributed).

The authors hotel room was converted to a laboratory. Groups of six students measured urinary specific gravity, calcium and chloride excretion. [24]

When not performing the laboratory tests, postural analysis, motion palpation, ranges of motion, muscle tests-including use of the "51%'er" phenomenon, therapy localization and challenge of the ileo-cecal valve[25] and sacro-iliac joints[26] was performed according to the written procedural outline.

Under investigation was:

- urinary excretion of calcium, as a parallel of serum calcium, was there any correlation between this finding and the postural pattern or actual test weakness of calcium related muscles

- the levator scapula through it's parathyroid association[27] if unilaterally weak would influence upper cervical biomechanics, is there an evidence of this upon motion palpation and does this correlate with calcium excretion or postural patterns of levator imbalance

Discussion: (cont., proposed workshop #3)

- urinary specific gravity as a reflection of toxicity due to functional liver/kidney involvement, would a low specific gravity correlate with weakness of the liver and kidneys associated muscles, and, if an ileo-cecal or colon involvement was a common contributor to this visceral dysfunction, would their therapy localization and muscle weakness also be reflected in the specific gravity 28,29

- urinary chlorides as an indicator of various stages of functional hypoadrenia,(thanks Rich),would chloride excretion correlate with sartorius weakness, a category two, and possibly with altered sacro-iliac biomechanics that might also be evaluated through motion palpation

- a functional relationship between a pattern of bilaterally weak pect. major claviculars, a temporal bulge/parietal descent and gastric HCL levels, as this relates to calcium metabolism

The discussion that followed this exercise, stressed the correlation of findings observed during manual muscle testing, with those of laboratory evaluation, motion palpation, therapy localization, and postural analysis, in addition to applied kinesiologies emphasis on understanding functional interrelationships of physiology.

As with the second workshop, the mood was one of intensity, and enthusiasm. From the nature of the questions raised, it was clear that this was a creative experience for all concerned.

Summary:

Three separate workshops, to be held in conjunction with an applied kinesiology lecture series, are presented as a means of developing the art of clinical observation/correlation. A situation that is, in fact, the essence of applied kinesiology.

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OBSERVATIONS ON YAW PATTERN

by

Mario A. Sabella, D.C.

Abstract: A clinical study involving observable correlation of PRY and gait patterns. Other factors considered in this paper include occiput fixation, switching, dural torque and acupuncture meridian involvement.

INTRODUCTION

Neurologic disorganisation has, for a long time, been accepted in Applied Kinsiology, as a major source of irritation to normal body function. Amongst other things it may lead to recurring subluxations and confused afferent proprioceptive impulses. This area of involvement is particularly important when it comes to dealing with such problems as learning difficulties and general behavioural problems. PRY, gait, dural torque, switching, proprioception problems are some of the clinical manifestations linked up to neurological disorganisation. Several clinical studies had been conducted by various practitioners, including the author, dealing with one or more of the enumerated factors.

Various studies, made outside the chiropractic profession, have indicated that the generalised postural reflex is situated in

the neck. This concept is a very old one and in an account written in 1845, Longet described a gait disorder (as severe as that following cerebellectomy) as a consequence of surgical interference with the neck musculature. Occasional clinical reports have also emphasized a role for neck muscle proprioception in postural and occulomotor reflexes. In the early 20th century, systematic analysis of postural reflexes in decerebrate cats led to the description of a class of "tonic neck reflexes", in which neck movement leads to predictable changes in limb position. Earlier experiments concerning the origin of tonic reflexes in the decerebrate cat suggested that receptors underlying the reflex were located in deep neck structures, probably in the intervertebral joints. During electrophysiological experiments it was noted that many fibers entering upper cervical dorsal roots appeared to come from small muscles lying in close proximity to the neck intravertebral joints (Abrahams, 1980).

To examine the matter further, Bakker and Richmond have reconstructed the receptor organization of the atlanto-axial and C3-C4 joints by examination of silver stained serial sections of decalcified cat necks. The small muscles that invest the bone have been found to contain dense accumulations of muscle spindles with calculated spindle densities as high as 500/g (hind leg muscles

contain about 5-20 spindles/g). These spindles were not randomly distributed, but confined to specific regions of the muscles, frequently in arrays extending the full length of the muscles. Many golgi tendon organs are also present in these small muscles, often in juxtaposition to the spindles.

From these experiments and many others reported by several researchers it seems obvious that any disturbance in the upper cervical complex, be it from atlanto-axial or atlanto occipital malfunctions could have far reaching repercussions on the entire system. This state of affairs may manifest itself in the form of neurological disorganization, leading, in time, to more complex functional disturbances.

During the course of general practice procedures I have, for some time, noticed that many patients showed involvement of gait reflexes, requiring stimulation of the known acupuncture reflexes on the feet. Some patients presented with a disturbance of all the known gait reflexes and stimulation of the acupuncture reflexes did not have a lasting effect. Further investigation revealed that these patients had a yaw pattern and exhibited a bilateral weakness of the psoas muscles, indicating an occiput fixation. In addition those patients showed a positive therapy localisation to several

acupuncture pulse points, indicating involvement of two or more meridians.

Based on the above information I decided to conduct a clinical study to establish the correlation between those factors, in addition to switching and dural torque.

EVALUATION METHODS

Patients selected for this study were those exhibiting positive gait involvement affecting all patterns. The total number of patients recorded was 44. Prior to the evaluation major areas of involvement were corrected (switching, subluxations, cranial faults, etc.). The factors considered were: gait, pitch roll and yaw, occiput fixation, switching, dural torque and acupuncture pulses. A summary of the results is tabulated herebelow. Meridians were not evaluated individually, but rather therapy localisation was done on each pulse point and the positive recorded. It is therefore possible that either or both of the meridians on that pulse could have been involved.

RESULTS

		Number of patients
Yaw occiput		44
Occiput fixation		44
Gait reflexes		44
Switching		17
Torque		7
Meridian pulses - # positive TL=	2	2
	3	8
	4	19
	5	12
	6	3

The results show a definite correlation between occiput fixation, yaw (occiput) and acupuncture pulse points. There is no significant associated pattern with switching or dural torque. When the occiput was corrected. (in the fashion of a fixation), all indicators, except switching and dural torque, if present, became negative. In some cases there was a residual positive therapy localisation to one or two acupuncture pulses requiring the standard meridian correction procedures.

CONCLUSION

It may be concluded from the above that the existence of a YAW pattern has a profound effect on gait and that an occiput fixation is a manifestation of that pattern. Furthermore an involvement in the occipito-atlantal area creates a major disturbance of the acupuncture meridian system, which demonstrates the futility of carrying out any diagnostic or therapeutic procedures in this area before correcting the neurological involvement.

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THE DOUBLE CRUSH PHENOMENON

Julius L. Sanna, M.S.,D.C.

Abstract:

Have you ever wondered why a cervical adjustment will relieve a carpal-tunnel syndrome, a "tennis elbow" or a scalneius anticus involvement? The double crush phenomenon explains why; but before we can discuss this subject we must review axoplasmic flow within the axon.

The axon is filled in its center with axoplasm.[1] The movement of axoplasmic components from the cell bodies to the terminals was first demonstrated in 1948. A wide variety of transported materials have been identified (enzymes, proteins, phospholipids, glyco-proteins, etc.). Recently the important trophic action that nerves have striated muscles was shown to be related to axoplasmic transport. The movements of particles through the nerves are not driven by diffusion, but require metabolically generated energy. Important to manipulative therapy, this local energy flow requirement implies a demand for a continuous blood and fluid replenishment.[2]

With the present state of knowledge, the impact that nerve compression, stretching, angulation or other deformations may have on the neurochemistry of axonal transport is not known but can reasonably be inferred to be significant. In view of the trophic actions which neurons have on cells they innervate, subtle changes in the transported materials may have a profound influence on the state of well being of the innervated tissues as well as the neuron itself. We can see at least a possibility that the basis of some of the therapeutic effects of manipulation may lie in axoplasmic transport events. (3)

In LANCET, August 18, 1973, Drs. Upton and McComas stated that a study was done of 115 patients with carpal-tunnel syndromes or lesions of the ulnar nerve at the elbow. In 81 of the cases there was electrophysiological evidence, sometimes supported by clinical symptoms, of associated neural lesions in the neck. Compression of the median nerve at the wrist or ulnar nerve at the elbow causes reduced axoplasmic flow within the axon. If this compression is slight it would not result in clinical symptoms. However, when compounded by a further axoplasmic block due to a cervical lesion, clinical symptoms are produced. Therapy should be directed to the neck and the wrist (or elbow).

Procedure:

Joint evaluation of the extremity should included examination of the spinal nerve root innervation at the associated spinal level by therapy localization. If the relationship does in fact exist a joint evaluation of the spinal region should also be included (range of motion, orthopedic, neurological and muscle testing).

Challenge can then be applied to determine the method of choice in therapy.

Conclusion:

If an individual has a predisposition to a problem in the wrist, elbow, shoulder, foot, knee or hips due to a deficient axoplasmic flow, a spinal trauma could create another axoplasmic block along the same nerve pathway and be responsible for the appearance of symptoms.

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THE CLOROX TEST:

A SCREENING TEST FOR FREE RADICAL PATHOLOGY

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

Abstract:

The use of sniffing the household bleach, Clorox, is discussed as a screening test for free radical problems initiated by the inability of the body to neutralize the free radical OCl^-. Generalized muscle weakness induced by sniffing Clorox may be neutralized by one or more of the following: 1) taurine, 2) niacin or niacinamide, 3) selenium, 4) vitamin E, 5) certain essential fatty acids, and/or 6) vitamin C. The biochemical mechanisms behind the neutralization of the Clorox sniff test are discussed for each of the six substances listed, including the use of aspirin as a screening test for the need for essential fatty acid supplementation. The concept of free radical pathology in the etiology and perpetuation of a number of symptoms is covered, including pain and inflammatory conditions, autoimmune processes, and Candida Albicans allergy syndrome. In addition, the concept of pseudo-infection syndrome is introduced in relation to free radical pathology at the local tissue level and in relation to the Clorox sniff test as a screening tool to be used in apparent infectious processes, especially those of the recurring type.

The immune system has a number of ways of dealing with our body's foreign invaders, such as bacteria, etc. One of these methods of dealing with foreign bodies is the production by white blood cells of highly

oxidizing substances known as free radicals. There are numerous free radicals, such as super oxide (O_2^-), singlet oxygen (O^-), peroxides (such as H_2O_2) and a family of compounds which contain chlorine (Cl^-). One of these oxidizing agents is the substance OCl^-. In general chemistry, OCl^- is known as hypochlorite.

In the chemistry of the body, this OCl^- ion is a highly oxidative, very short-lived (i.e., 1/10,000 second) free radical produced by white blood cells. It is designed as part of the immune system to oxidize bacteria and other foreign bodies which are potential invaders of the body defenses. OCl^- and other free radicals may be thought of as chemical bullets which the white blood cells fire in order to destroy bacteria and other invading organisms which might adversely affect the body.

Because some of these chemical bullets may go astray, the body produces many chemical "bullet-proof vests" to protect the body's own tissues from destruction from these chemical free radical bullets. The OCl^- is one of the most powerful of all the oxidizing free radicals. There are a variety of chemical "bullet-proof vests" which may quench or neutralize the OCl^- free radical. One of these, the so-called amino acid, taurine, is a specific quencher for OCl^-. Other substances which act as quenching agents (chemical "bullet-proof vests") for free radicals produced by white blood cells include a variety of anti-oxidents, such as vitamin E, vitamin C, the enzymes super-oxide dismutase and catalase, selenium (as found in glutathione peroxidase), vitamin A, certain essential fatty acids, and niacin. Niacin, or niacinamide, in the form of NADH, is able to act as a supplier of hydrogen ions as reducing equivalents to neutralize the effect of the free radical oxidization through the enzyme glutathione reductase which is riboflavin, or FAD, dependent.

A number of disease processes may result when OCl^- or other free radicals are produced by the body in the presence of a lack of free radical-quenching anti-oxidants. These disease processes are known as "free radical pathologies." Free radical pathologies may be initiated by the immune system reacting in a normal fashion by shooting its chemical bullets in a situation where the body is unable to neutralize these chemical bullets at the tissue level. This can allow an oxidization reaction to take place at the cell membrane level of the body's own tissues, resulting in tissue injury or destruction by the body's normal immune substances, the free radicals.

Free radical pathology may be stopped at several junctions. First, as will be discussed in this paper, there may be a deficiency of free radical quenchers. This deficiency can allow a free radical pathology process to begin which results in inflammation, tissue injury, and destruction. Secondly, when the free radical oxidizing agent reaches the cell membrane, it will convert essential fatty acids in that cell membrane into their respective prostaglandins.[1] The effect of a free radical at the cell membrane level will be modulated by the balance of the essential fatty acids in the cell membrane and in the surrounding tissues. The presence of arachidonic acid will lead to production of the PG_2 series, which are generally inflammatory in nature. If there are more PG_2 precursors present in cell membranes, etc. than PG_1 and/or PG_3 precursors, an inflammatory process may result as a result of oxidation of essential fatty acids in the cell membrane. On the other hand, if there are adequate supplies of essential fatty acid precursors for the PG_1 family and the PG_3 family, in general, oxidizing agents at the cell membrane will produce these two series of prostaglandins, which have a general anti-inflammatory

effect. This is the second point at which free radical pathology may be blocked in the body, as was discussed in a previous paper.[1]

OLFACTORY TESTING WITH CLOROX

The common household bleach Clorox contains 5.25% of sodium hypochlorite (NaOCl). (Similar bleaching agents contain similar percentages of NaOCl and may be used as an alternative to Clorox.) The OCl^- portion of sodium hypochlorite is the same chemical formula as the free radical OCl^- which we have been discussing.

A patient presented in our office carrying a small bottle of Clorox which he brought from home, with the history of having had a severe reaction of a musculoskeletal problem (back pain, carpal tunnel syndrome, neck pain) after close contact and exposure with Clorox over a prolonged period of time. The patient thought that perhaps Clorox the been affecting his problem, and brought the substance in for us to test. During the preceeding week, we had been studying the tape[2] and technical memorandum[3] produced by Jon Pangborn, Ph.D., regarding the OCl^- free radical. The information in this tape and this paper were interesting, but having listened to them, we had not decided on any appropriate method of testing. It was a fortuitous and serendipitous observation on our patient's part regarding his contact with Clorox and his interest in having us test the substance.

It became immediately clear that Clorox may be an excellent test substance for identifying the body's ability to handle the free radical OCl^-. During the course of treatment, this patient was asked to sniff the fumes from the Clorox bottle. These are not particularly irritating to the nasal passage, although this is a substance which should never be ingested orally. The patient was seen to show immediate and total muscle

weakness (inhibition) upon the sniffing of the fumes from the Clorox bottle.

Dr. Pangborn's work had discussed taurine as a specific free radical quencher for OCl^-. The substance taurine was placed on the patient's tongue, and as it was being insalivated, the patient was again asked to sniff the Clorox fumes. Sniffing the Clorox fumes with taurine insalivated resulted in no muscle weakness. In other words, the taurine blocked the total body muscle weakness (inhibition) which was produced by the sniffing of the Clorox fumes. We asked the patient if we could keep the bottle of Clorox which he had brought, to which he agreed, and we proceeded to test the sniffing of Clorox on multiple patients thereafter. If the patients were found to weaken on sniffing of the Clorox fumes, taurine was tested. Initially several patients were found to have the weakening effect of Clorox blocked by oral insalivation of taruine. Although a very high percentage (approximately 1/3) of our patients, showed a weakening response to Clorox, many of them did not show a neutralization by taurine of the weakening effect of Clorox.

Other substances were then investigated with oral insalivation to determine what might also block the weakening effect of Clorox sniffing. At this point (January, 1985) the following substances have been found as potential blocking agents to the weakening effect produced by Clorox: 1) taurine, 2) niacin or niacinamide, 3) selenium, 4) vitamin E, 5) certain essential fatty acids (EFA), and 6) vitamin C (ascorbic acid). The following discussion will take up each of the first five of these substances individually and identify how a variety of alternatives have been discovered which confirm our understanding of the use of the Clorox test as a viable clinical tool in identifying patients who may be overly sensitive to OCl^- and/or lack the essential free radical quenching substances in the body which will allow the production of free radical pathology by OCl^-.

1) TAURINE AND METHIONINE METABOLISM

Taurine is called an amino acid by many people, although it is not a normal amino acid because it does not contain a carboxyl (-COOH) group, but rather a sulfonic acid group ($-SO_3H$). It is produced from cysteine in the body, which is in turn produced from the metabolism of methionine. See Figure 1. The essential factors in the production of taurine from methionine and cysteine are shown in Figure 2 and are summarized in Figure 3. Notice that folic acid and B-12 are necessary in the recycling of methionine from homocysteine and that magnesium and B-6 are essential in the metabolism of methionine to cysteine; B-6 is further essential for the metabolism of cysteine to taurine. These chemical pathways are described by Dr. Pangborn[2,3], and in most general biochemistry texts.

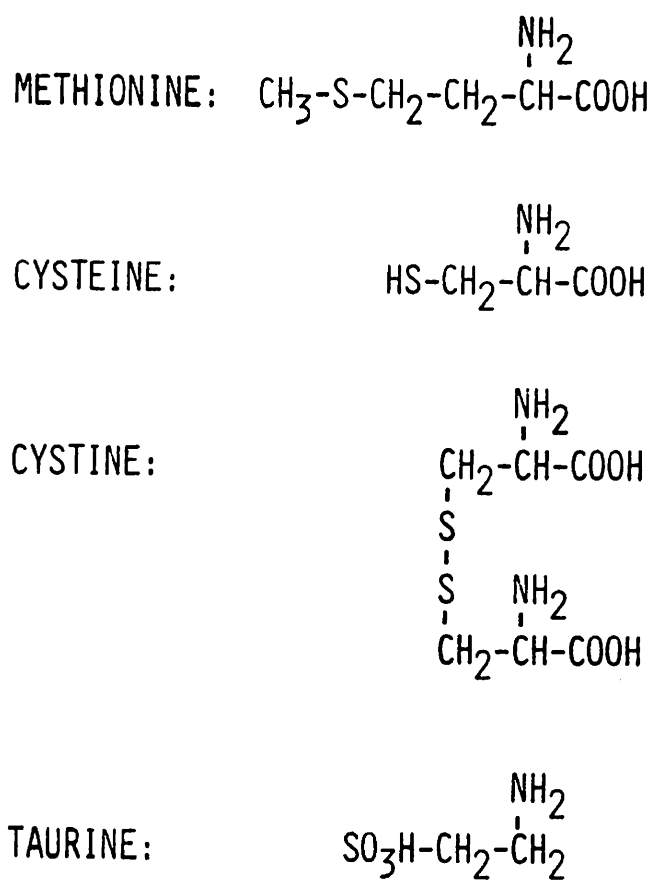


Figure 1

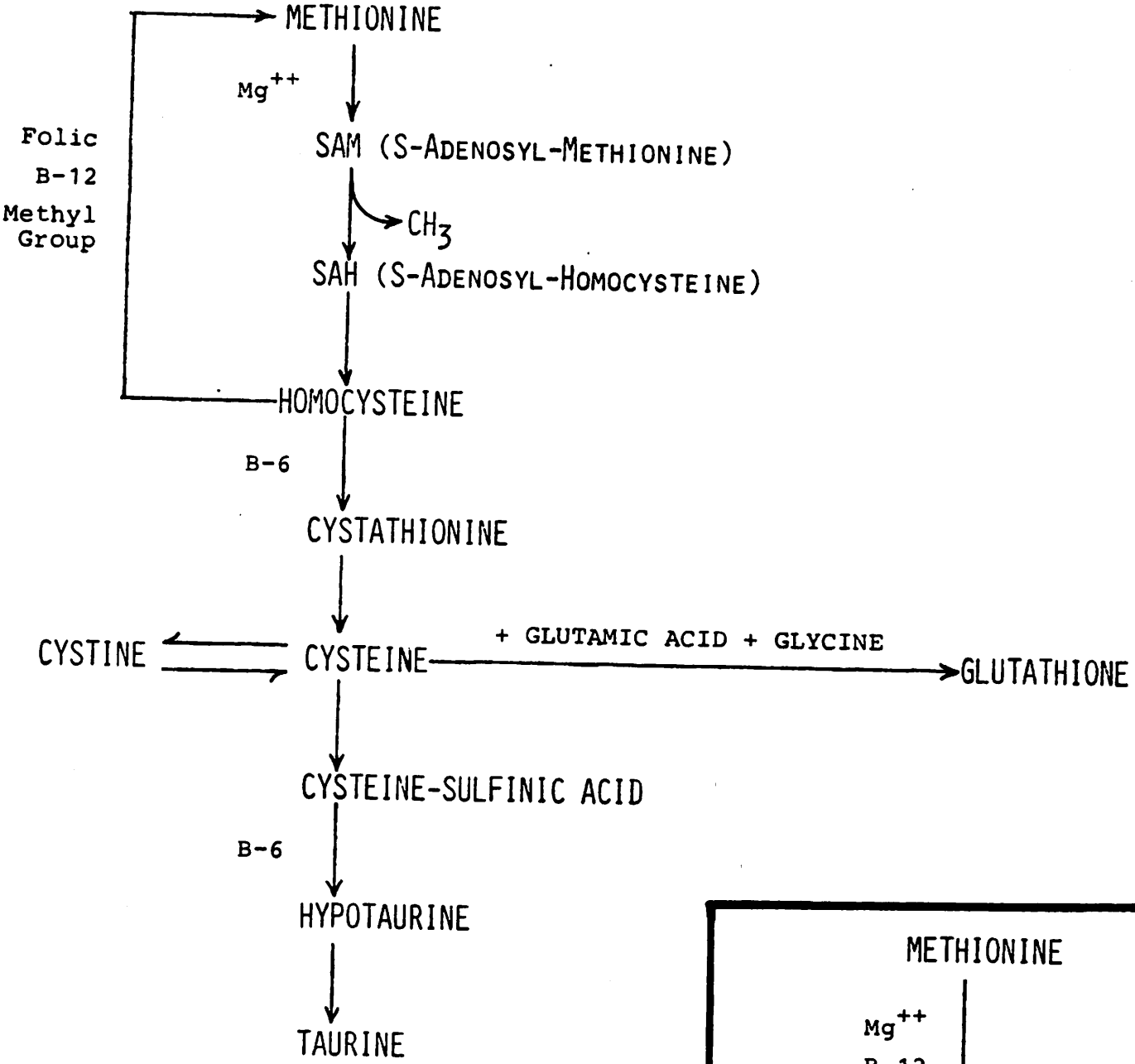


Figure 2

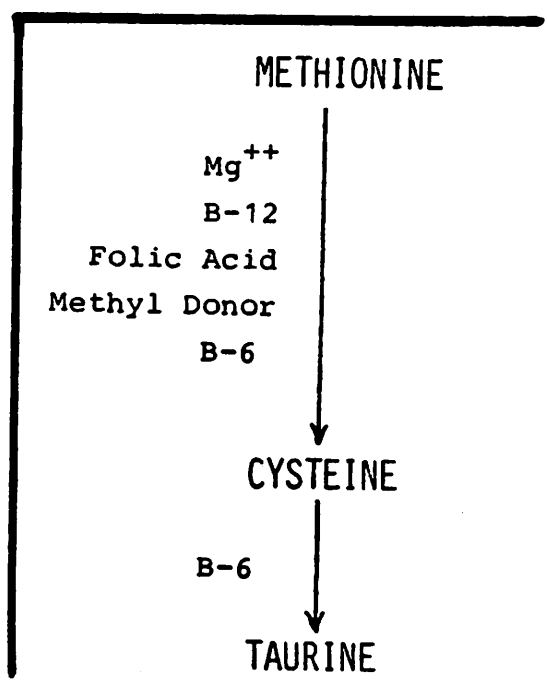


Figure 3

Patients who show a strengthening response on the ingestion of taurine and/or a blocking response by taurine of the weakness induced by sniffing Clorox will oftentimes have a problem in this metabolic pathway.

Methionine is an essential amino acid. In giving up its methyl group, it becomes a methyl donor and may be converted eventually into cysteine. (See Figure 2.) Cysteine is then further converted into taurine. All these amino acids are sulfur-containing. (See Figure 1.) If taurine shows a strengthening response and/or a neutralization of the weakening produced by sniffing Clorox, further testing should involve the response to cysteine and methionine. If methionine strengthens the patient, it is important to then check for the co-factors which are necessary for the conversion of methionine eventually into cysteine. This includes folic acid and B-12 and B-6, and magnesium, as is seen in Figures 2 and 3. If cysteine strengthens the patient, it is necessary to test for the co-factors essential for the conversion of cysteine into taurine. This involves the testing again of B-6.

One can literally pick apart the metabolism of the sulfur-containing amino acids by testing the amino acids individually, and identifying where strengths and weaknesses occur, and then further by testing the vitamin co-factors involved in the metabolism of methionine through taurine at each step along the metabolic pathway. When muscles are weak in the clear, it is a simple procedure of testing methionine, cysteine, and taurine against the weak muscles as a screening technique, and then further testing folic acid, B-12, B-6, and magnesium against the weak muscles in the clear to identify which substances create strength. When a substance creates strength of a muscle which is weak in the clear, that same substance is then held in the mouth while the patient sniffs Clorox

to identify its effect on blocking the weakness otherwise induced by the Clorox.

Rarely is it found necessary for a patient to be supplemented by methionine, cysteine, or taurine. Very commonly, it is found essential to supplement a patient with folic acid and/or B-12 and/or B-6. In these cases, for example, in a B-6 requirement, a patient will have weak muscles which will show to strengthen on oral insalivation of B-6, and the neutralization of the weakening effect produced by Clorox by B-6. This patient then requires supplementation with B-6. Other patients may actually require methionine, since it is a somewhat commonly deficient amino acid, or, sometimes, cysteine. More commonly, the essential factors involved in the faulty metabolism of taurine are the vitamin co-factors listed above.

There is a very important tangent in relationship to the potential availability or unavailability of cysteine in this group of patients. Cysteine, along with glutamic acid and glycine, must be available to the body for the production of the essential tripeptide, glutathione. (See Figure 2.) Glutathione is the central component of the electron poising system[4,5,6,7] and any process which interferes with glutathione production, such as the restriction of cysteine (and/or glutamic acid and/or glycine) can interfere with this essential, fundamental system of self-regulation of the body chemistry. More will be said on this subject in a future paper.

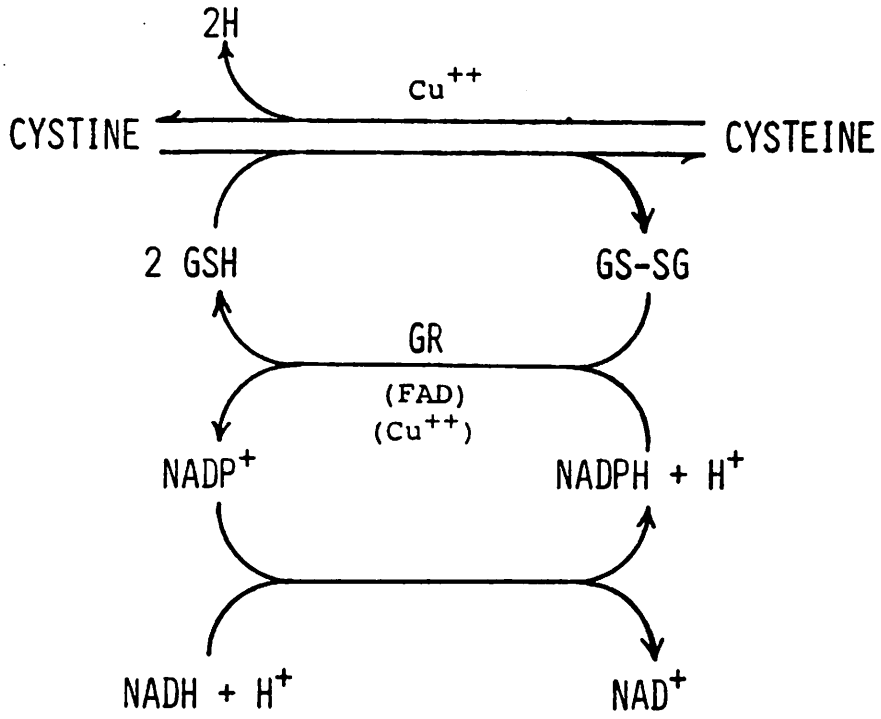
2) NIACIN AND/OR NIACINIMIDE

OCl^- is a highly oxidizing substance. In patients with free radical pathology, there is generally a lack of anti-oxidant availability throughout the body which results in the body becoming generally over-oxidized. In an over-oxidized patient, the amino acid cysteine will be converted to its oxidized form of cystine. Note in Figure 1 that cystine is merely two cysteine molecules coupled at their sulfur end. Oxidation of

cysteine results in cystine. Reduction of cystine results in cysteine. In the patient with free radical pathology it appears that it is very common for cysteine to be immediately converted into cystine, thereby resulting in an unavailability of cysteine for conversion into taurine. (See Figure 2.) If the patient's tissues become over-oxidized, as often results in free radical excesses, much cysteine may be converted into cystine as soon as it becomes available to the body, either from oral ingestion or from conversion from methionine.

Note in Figure 4 that the reaction reconverting cystine to cysteine involves the conversion of reduced glutathione (GSH) to oxidized glutathione (GS-SG) and the co-enzyme NAD (nicotine adenine dinucleotide). The conversion of NADH into NAD is called a transhydrogenation reaction. Notice also in Figure 4 that this reaction requires an enzyme called glutathione reductase (GR) and that this enzyme is copper-dependent and riboflavin (FAD)-dependent. This glutathionine reductase/NADH reaction should look familiar as part of the electron poising system, as discussed by Isaacs[4] and in previous papers by this author.[5,6,7] (See Fig. 5.)

Many patients who will fall into the over-oxidized category, due to free radical pathology or other metabolic imbalances, will be in danger of an overconversion of cysteine into cystine. If there is a problem with an excess or deficiency of copper, the availability of niacin, or glutathione-reductase,there may be overproduction of cystine in relation to cysteine. The level of riboflavin also affects this pathway since glutathione reductase enzyme is dependent on the riboflavin containing coenzyme FAD, or flavin adenine dinucleotide. Riboflavin deficiency can result in an inability of cystine to be converted back into cysteine. An over-oxidized patient and/or one with a copper, niacin, or riboflavin problem may be unable to convert cystine back into cysteine for eventual conversion to taurine. This becomes a viscious cycle in these patients who are then unable to neutralize OCl^- and they get stuck in a trap where



GSH = REDUCED GLUTATHIONE

GS-SG = OXIDIZED GLUTATHIONE

GR = GLUTATHIONE REDUCTASE
(RIBOFLAVIN (FAD) DEPENDENT)
(COPPER (Cu^{++}) DEPENDENT)

Figure 4

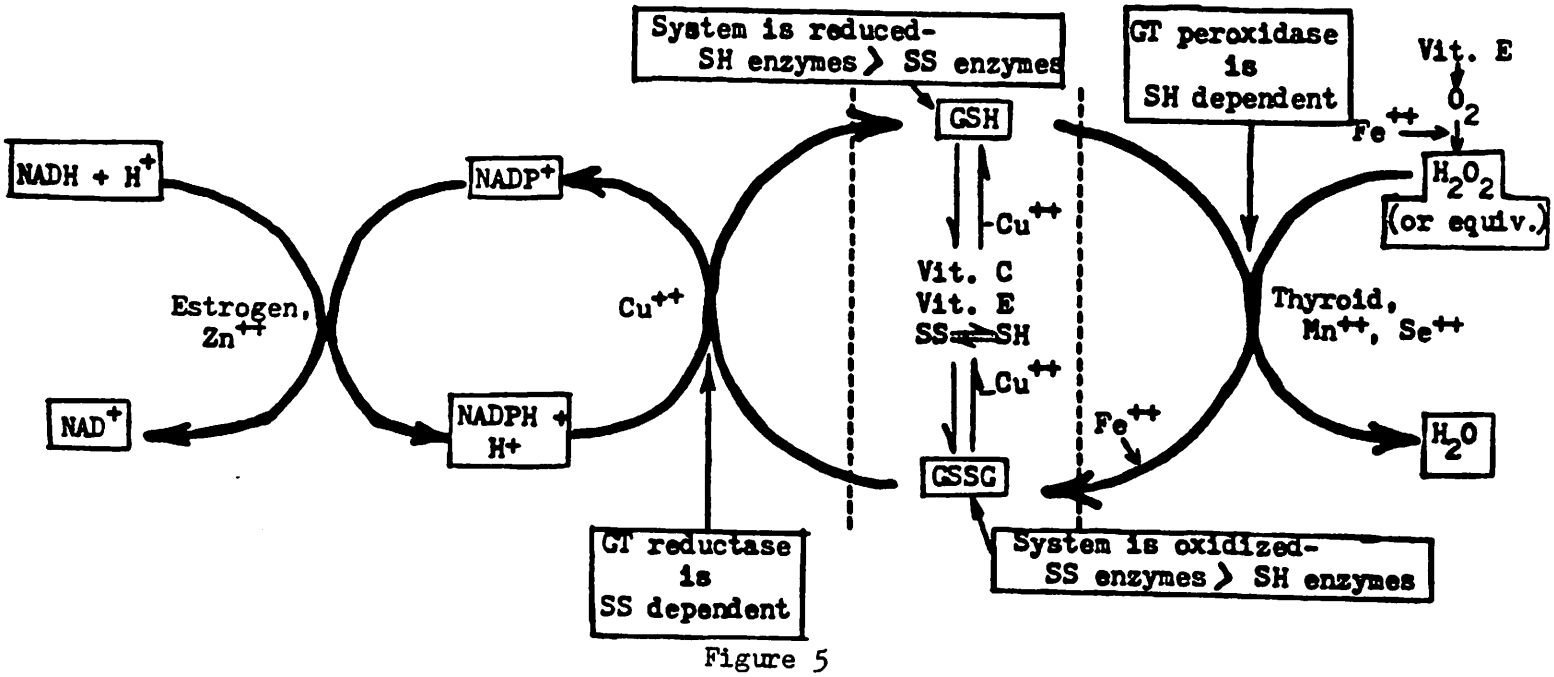


Figure 5

the OCl^- creates a state of oxidation at the tissue level which cannot be neutralized by taurine and which further compounds the problem by creating a conversion of all available cysteine into cystine, making the restriction of taurine even more dramatic.

These patients will often respond with strengthening to niacin or niacinamide and either or both of these substances will also neutralize the weakening effect of sniffing Clorox when they are held in insalivation simultaneously with the Clorox testing. Further, in this same reaction, if riboflavin is deficient, it is possible that the patient may also have an inability to reconvert cystine into cysteine for eventual production into taurine. Therefore, if niacin strengthens weak muscles in the clear, and neutralizes the weakening effect of Clorox, one should also check riboflavin as a possible factor necessary in this reaction, although we rarely see this present. Note that riboflavin and niacin together are found in the vitamin substance known as Complex G (from Standard Process Laboratories.) Some patients with free radical pathology will respond very nicely to Complex G and may have a variety or other vitamin G deficiency symptoms.[1]

The availability of copper is also essential for the glutathione reductase reaction to take place, as well as potential need for vitamin C. The need for copper is rarely seen in the area of the country in which this author practices. More often, patients are seen who are copper toxic.

At the present time, it is felt that some of the severe effects of a toxic copper body burden are due to a stimulation of this enzyme pathway converting cysteine into cystine and compounding whatever symptoms the patient may otherwise have which are related to free radical pathology and overoxidation. The need for zinc and/or manganese

to balance out a copper toxicity are also seen in some patients with the free radical pathology of OCl^-. Zinc and/or manganese supplementation may also be necessary in an effort to bring copper into balance and neutralize the effects of OCl^- induced free radical pathology. It is interesting to note in this regard that zinc and manganese are also necessary (as is copper) for the production of the free radical quenching enzyme, superoxide dismutase at various sites in the body.

3) SELENIUM AND GLUTATHIONE PEROXIDASE

For the transhydrogenation reaction involving glutathione reductase to take place as described by Figure 4, it is essential that there be adequate available glutathione, as well as adequately available glutathione peroxidase activity to carry out the next step in the reaction of the glutathione reductase transhydrogenation reaction. Again, this system of modulating the electron flow of oxidation and reduction is known as the electron poising system.[4,5,6,7] (See Figure 5.)

The electron poising system reaction is summarized in Figure 5. Note that for the operation of glutathione peroxidase it is essential for there to be adequate activity of selenium, as well as thyroxine, manganese and iron. Selenium has a great reputation as an anti-oxidant. It works as an anti-oxidant through the glutathione peroxidase pathway. Many patients have a deficiency of selenium, and selenium will be seen to strengthen weak muscles in many patients with OCl^--induced free radical pathology. This is really nothing more than a further step from what was previously discussed in relationship to niacin and niacinamide in the glutathione reductase reaction. That is, in the body, these reactions are linked together and do not operate independently, but in a chain. In addition, however, selenium in the form of glutathione

peroxidase is available in cell membranes throughout the body to act as a quencher of free radical activity, particularly as a neutralizer of peroxidation reactions and peroxides produced by free radical oxidation.

The patients who show generalized muscle strengthening from selenium as a screening device will often then be seen to have the selenium further neutralize the weakening effects of the Clorox sniff. This reaction brings into play all the factors which are involved in the electron poising system which have been discussed by this author and by Isaacs in previous papers, as previously referenced. The reader is referred to these papers for a complete discussion of the electron poising system. In relationship to the Clorox test, however, the use of selenium is a very commonly found factor in neutralizing the Clorox reaction and OCl^--induced free radical pathology.

It is important to note at this point that selenium deficiency has been epidimologically related to cardiovascular disease[8] and multiple sclerosis (M.S.).[9] It is a reasonable assumption that MS and other neuropathies may be initiated or aggravated by unchecked free radical pathology which attacks the myelin sheaths of these patients. Since employing the Clorox sniff test we have seen positive reactions (i.e., generalized muscle weakening in response to Clorox) in each neuropathy patient seen. This includes two cases of MS, one case of amyotrophic lateral sclerosos (ALS), one case of muscular dystrophy (MD), and one case of cerebellar and brain stem degeneration of unknown origin as well as in many chronic pain patients.

4) VITAMIN E AND 6) VITAMIN C

Vitamin E and vitamin C are well known anti-oxidant substances. Vitamin E is available circulating in the body and also is present in cell membranes to act as an anti-oxidant to neutralize the effect of free radical oxidizing reactions and to control the rate of oxidizative reactions in the body. A vitamin E deficiency may result in the inability of the body to quench free radicals oxidizing effects. That is to say, inadequate vitamin E may lead to inadequate anti-oxidant ability, which will allow free radical oxidation to occur unimpeded, and therefore create problems in various locations in the body.

Vitamin E in either high dosage or low dosage may on occasion strengthen weak muscles throughout the body and neutralize the weakening effect of sniffing Clorox. The effects of high dosage vitamin E and low dosage vitamin E were discussed in a previous paper.[1] Vitamin E in these patients will strengthen a variety of weak muscles (vitamin E related and non-vitamin E muscles alike) and, when insalivated, neutralize the weakness induced by sniffing Clorox.

On occasion, vitamin C may also be found to be useful as an anti-oxidant in neutralizing the Clorox weakening reaction. Vitamin C is also an essential factor along with copper in the activation of glutathione for its electron poising function (Figure 5) and its deficiency may result in the inability of the transhydrogenation reaction in Figure 4 to function properly.

5) ESSENTIAL FATTY ACID METABOLISM

All cell membranes in the body contain a variety of fatty acids. A number of these fatty acids are essential fatty acids and precursors

to the prostaglandin local tissue hormones. Oxidation of fatty acids which reside in cell membrane and which circulate in the body as free fatty acids, phospholipids and cholesterol esters, leads to their conversion to prostaglandins. The ability of prostaglandins to enhance or dampen an inflammatory process is well known.[1]

The availability of fatty acids in cell membranes and other places in the body is directly proportional to their ingestion in the diet and their storage in the tissues. Excessive availability of arachidonic acid will lead to the production of the inflammatory prostaglandin-2 (PG_2) series upon oxidation of arachidonic acid, which is present in cell membranes and other areas of the body.

Oxidation of other essential fatty acids in cell membranes, etc. will lead to the formation of the anti-inflammatory prostaglandin-1 (PG_1) series from linoleic acid, gammalinolenic acid, and dihomogamma-linolenic acid. Likewise oxidation of those fatty acids which lead to the prostaglandin 3 (PG_3) series will also generally have an anti-inflammatory effect. These fatty acids are derived from alpha-linolenic acid and are also available directly in the diet from eicosapentaenoic acid (EPA) which may be ingested from fish oils. A balance of all essential fatty acids is necessary for the conversion of the EFA into prostaglandins in the proper proportion. All too commonly, there is an excessive availability of arachidonic acid and a relative deficiency of the precursors to the PG_1 and the PG_3 series, resulting in the production of the inflammatory PG_2 hromones from arachidonic acid. This occurs upon the oxidation of cell membranes of the fatty acids which reside there as well as the oxidation of the other sources of fatty acids in the body.

Free radical pathology can be perpetuated by free radicals, such as OCl^-, coming into contact with the cell membrane and oxidizing the fatty acids that are in the cell membrane. If excessive amounts of the PG_2 series are produced (due to the excess availability of arachidonic acid), inflammatory process will be enhanced rather than dampened. This inflammatory process will cause a resulting increase in number of white blood cells migrating to the area, which will cause increased release of free radicals, such as OCl^- which will further oxidize fatty acids in the cell membrane which are already out of proportion and which are already leading to inflammatory process and will lead to further inflammatory process, creating a very severe viscious cycle. This is the type of process which has been observed in patients with the most severe types of auto-immune disease, such as systemic lupus erythematosous, multiple sclerosis, amyotrophic lateral sclerosis, rheumatoid arthritis, and so on. This same process can occur in a local area, resulting in a local neuritis, neuralga, bursitis, tendinitis, etc.

Patients who show a prostaglandin problem which is due to an essential fatty acid imbalance have been found many times to respond with strengthening to the oral insalivation of aspirin. Aspirin is a prostaglandin inhibitor. If aspirin strengthens a patient's weak muscles in the clear, it implies that there is a problem with prostaglandin production in the body secondary to a problem with essential fatty acid balance in the body. Aspirin, however, has not been seen to neutralize Clorox weakening response.

If aspirin is found to strengthen the muscles which were weak in the clear, however, the appropriate essential fatty acid precursors to the PG_1 and/or PG_3 series will also be found to strengthen the weak muscles.

Substances used to strengthen weak muscles when aspirin is found to strengthen weak muscles are evening primrose oil (linoleic and gamma-linolenic acids), linseed oil (alpha linolenic acid), and fish oils (EPA and docosahexanoic acid). One or more of these substances will strengthen weak muscles in any patient who also has shown weak muscles to be strengthened by aspirin. Further, oral insalivation of the appropriate EFA will also block the Clorox weakening response from sniffing, even though aspirin did not block the Clorox sniff reaction. Some patients with free radical pathology will require the appropriate essential fatty acid to block the viscious cycle of free radical pathology-induced inflammation, as previously discussed. This may be the sole factor involved, or may be in conjunction with any of the aforementioned categories of substances which were found to neutralize free radical pathology induced by OCl^- and observed by Clorox weakening response.

PSEUDOINFECTION SYNDROME

Occasionally patients are seen who show all of the signs of a local infection such as a pharyngitis, sinusitis, other upper respiratory tract infections, or urinary tract infection, but culture of a specimen of the area is totally negative. This has always been a curious observation, especially when local pus and inflammation are observed, but for lack of better knowledge, it has always been assumed that the negative culture was the result of lab error or improper collection of the specimen. In light of the present understanding of free radical problems, a new hypothesis of the mechanism of symptom production in these patients will be presented.

The symptoms and signs of an infectious process depend on the local action of the immune response. Without appropriate immune response, an infection may be present with no outward signs or symptoms, but when the immune response kicks in, both the doctor and patient know the patient is sick. The immune system causes a local defense response with the migration of white blood cells to the area which release free radical oxidizing agents to attack the invading organism. This results in an inflammatory process with the formation of pus (white blood cells, dead bacteria, products of tissue inflammation, etc.) and other outward signs of infection.

Suppose that a very minor immune response results from the presence of some invading organism, and the white blood cells respond with free radical release and phagocytosis and quickly destroy the bacteria, etc., but with the result that some local tissue destruction has also taken place due to a deficiency of free radical quenchers. The inflammatory response causes a migration of more white blood cells, with the subsequent release of more free radicals which cause continued inflammation in spite of the fact that the invading organisms are already conquered. The area of apparent infection is now free of infection, but the local process of inflammation is the same as if there is an active infection. A culture will be negative since there are no longer any invading organisms present, but both the doctor and the patient think there is an infection ongoing when in fact the signs and symptoms are from ongoing free radical pathology.

Two variations of this process have been observed. One is in the patient who never recovers completely from an apparent local infection or who recovers extremely slowly in spite of antibiotic therapy and natural therapies combined. The other pattern involves the patient who shows

apparent recurrent infections in a local area -- as soon as he or she recovers from one, another one sets in. Tissue damage inflicted by the first immune response in these patients leaves them with a lowered local resistance and sets the stage for more, future infections.

In the latter group, therapy directed at beefing up the thymus and lymphatic system is also ineffective. In fact, this approach may actually be detrimental to the patient. By improving the immune system function in light of the inability of the patient to quench the free radicals released by a stronger immune system, the patient's anti-oxidant system may become further compromised and lead to prolongation of the symptoms or the development of yet another process such as auto-immune disease, allergy and hypersensitivity syndromes, and the like. A strong immune response in the presence of anti-oxidant depletion coupled with the use of oral antibiotics which destroy friendly, gastrointestinal flora can set the stage for the Candida albicans allergy syndrome which has become so popular lately.

The Clorox sniff test will result in a weakening response in these patients. Patients with a sluggish immune system have occasionally been seen to strengthen when sniffing Clorox although this is rare. It is important in the recurrent "infection" patient to identify the response to the Clorox test and take the appropriate measures to insure that a short term gain does not result in a long term loss.

CONCLUSIONS

OCl^- is a potent oxidant in the body. Its over-production is discussed by a number of authors. However, in this paper, we have discussed not the fact that OCl^- is over-produced, but the fact that there is a deficiency of some of those anti-oxidant substances and free

radical quenching substances which are normally produced by the body. These anti-oxidant and free radical quenching substances are derived from normal nutrients and are dependent on vitamin and mineral co-factors for the production of the anti-oxidant, free radical-neutralizing reactions. Deficiencies or imbalance in the vitamin, mineral, amino acid, and essential fatty acid intakes and metabolisms in the body can lead to serious consequences of free radical pathology, either at the local or systemic level.

These free radical pathologies may be short-lived, and transient in nature. However, it is our experience that most patients who have chronic and recurrent problems, especially involved with inflammation and pain and occasionally involved with infection, will show a weakening response to Clorox, implying the presence of an on-going free radical pathology of OCl^- type. The rapid screening test of sniffing Clorox and identifying muscle weakness, accompanied by screening for those substances of the six categories listed which may neutralize this response has resulted in very gratifying responses in our patients, both in the office setting and the follow-up of clinical responses in their symptom patterns. If Clorox is found to weaken a patient, a rapid screening using 1) taurine, 2) niacin or niacinamide, 3) selenium, 4) vitamin E, high or low dose, 5) aspirin or essential fatty acids, and/or 6) vitamin C (ascorbic acid) has led to appropriate supplementation for the patient and has led to many gratifying results in many perviously very difficult and non-responsive problems.

SUMMARY OF PROCEDURE FOR CLINICAL APPLICATIONS

1) Patient sniffs Clorox.

2) If strong muscles weaken, test weak in the clear muscle(s) with the following substances in the mouth:

A) Taurine

1) If taurine strengthens, test with

a) cysteine - if cysteine strengthens:

1) test with B-6

b) methionine - if methionine strengthens, test with:

1) B-6

2) folic acid

3) B-12

4) Magnesium

B) Niacinamide (or niacin)

C) Selenium

D) Vitamin E

1) low dose (e.g., Standard Process Labs)

2) high dose (e.g., 100 IU or higher)

E) Aspirin - if aspirin strengthens, test with:

1) evening primrose oil

2) linseed oil and/or fish oils (eicosapentaenoic acid)

3) other essential fatty acid products

F) Vitamin C

3) Test each positive testing (i.e., strengthening) substance in 2)A- 2)F against the sniffing of Clorox

4) Supplement these substances which <u>both</u> strengthen weak muscles and neutralize the weakening effect of Clorox.

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THE DIAGNOSIS AND TREATMENT OF AUTO IMMUNE DISEASES THROUGH THE USE OF APPLIED KINESIOLOGY, NUTRITION, AND HOMEOPATHY.

By Sheldon Sinett, B.A., M.A., D.C.

ABSTRACT

This paper will examine the diagnosis of Auto Immune Diseases by the application of the patient's blood on his tongue and evaluation of changes in muscular strength. It will also deal with the treatment using applied kinesiology, nutrition and homeopathy on the muscular skeletal system.

INTRODUCTION

Auto Immune Diseases were hard to diagnose and harder to get favorable results.

Last year, my son Todd, age 14, developed severe shortness of breath upon physical exertion. He had difficulty running for more than 30 seconds at one time. Todd has had a bronchial condition for the past four years. When his resistance was low he would have coughing spells that would keep him up all night. As hard as I would try, I was unable to alleviate his problem.

I recalled Dr. Goodheart[1] speaking of allergy reactions. He would ask the patient to put a drop of his own blood on his tongue. The patient would show a change in muscular strength if there was an allergic reaction. I remembered reading Dr. Schmitt and Dr. Mowles' article "An Applied Kinesiological Approach to Candida Albicans."[2] I then speculated that perhaps Todd was allergic to himself. I had Todd put a drop of his blood on his tongue, there was an immediate change of muscular strength. I used the vitamins that Drs. Schmitt and Mowles recommended in their paper, along with homeopathic remedies, diet and applied kinesiological techniques to negate Todd's muscular weakness. In a matter of two weeks, Todd was able to participate in athletics and enjoy a complete uninterupted nights sleep once more. This lead me to do this examination on all difficult patients exhibiting auto-immune symptoms.

EXAMINATION AND TREATMENT

1. Check bilateral PMC, PMS, infraspinatus muscles for strength.

2. Put drop of patient's blood on his tongue. Check for weakness on previously strong muscles.

3. Use Nestatin powder on tongue along with blood to see if muscles test strong.

4. If step 3 is positive, rinse mouth. Put another drop of blood on tongue and use Seroyal candidia albicans 30x, 60x, 100x, 200x or 500x,[3] one of which will negate weak muscles.

5. Rinse mouth, put new drop of blood on tongue. Check for muscular weakness. Use Wildwood Botanics Imu Stem 1 and Hobson's homeopathic remedies[4] to check which remedy will negate weak muscles. Important: after strengthening occurs with homeopathic remedy rinse mouth and use another drop of blood on tongue. The following is a list of homeopathic remedies:

Adaptosode
Adaptosode R.R.
Biosode O.S.
Detoxosode O.S.
Detoxosode for Alcohol
Detoxosode for Allergens
Detoxosode for Antigens
Detoxosode for bacteria
Detoxosode for chemicals
Detoxosode for dentals
Detoxosode for food additives I
Detoxosode for food additives II
Detoxosode for Free radicals
Detoxosode for fungi yeasts
Detoxosode for insecticides
Detoxosode for metals
Detoxosode for nematodes
Detoxosode for parasites
Detoxosode for pollution (air)
Detoxosode for pollution (environs)
Detoxosode for radiation
Detoxosode for rickettsia
Detoxosode for tobacco
Detoxosode for viruses
Candidia albicans 30x
candidia albicans 60x
candidia albicans 100x
candidia albicans 200x
candidia albicans 500x

6. Rinse mouth, put drop of blood on tongue. Check for muscular weakness. Use vitamins which negate muscular weakness.

Thyme
Antronex
Biotin
Vitamin E
Zinc

7. Do applied kinesiology examination with new drop of blood on tongue. Make necessary corrections.

8. Hobon's remedies are prescribed 1t 2x daily for 12 days. Imu-Stem prescribed 5-10 drops in 1/4C water 3-6x daily for 12 days. Vitamins prescribed one 3x daily.

9. Diet for Candidia. Candidia control or low carbohydrate diet. See "Yeast Connection."[5]

CONCLUSION

By using the patient's blood we are able to get a true picture of auto immune invovement which was previously disguished. By using applied kinesiology procedures along with homeopathy, vitamins, and diet we were able to get favorable responses in difficult cases.

NOTES

Goodheart, George J. Personal Communication, 1978.

Schmitt, Walter H., Mowles, Richard A. Summer Meeting, International College of Applied Kinesiology, 1984.

Wildwood Botanics, Box 60397, Pasadena, CA.
Hobon, 3200 Polaris Drive, Las Vegas, Nevada 89102.

Yeast Connection. Wm. C. Crook, M.D. Professional Box, Jackson, Tennessee.

APPLIED KINESIOLOGY SCIENCE

By

John F. Thie, D.C.

Founding Chairman of the International College of Applied Kinesiology

Chairman of the Certified Teachers of Applied Kinesiology

Pasadena, California

Abstract

Discussion of why the science of applied kinesiology is not readily accepted as scientific. The discussion raised questions as to what terminology should be utilized in discussing applied kinesiology with others engaged in scientific endeavors. The author suggests that members of the ICAK should utilize the words technics, comment, editorial, theory, hypothesis, and laws in discussion of applied kinesiology within and outside the group, for better understanding and communication.

APPLIED KINESIOLOGY SCIENCE

COMMENT

John F. Thie, D.C.

In the 20 years that I have been associated with George Goodheart and his development of applied kinesiology, much has been said about research and the scientific basis of applied kinesiology. There is an unsaid hope that somehow applied kinesiology could be called scientifically proven. The difficulty in most of the discussions that I have been involved in is semantic, that is, the communication gets blocked because the same understanding of the words are not held by all the parties. Part of the problem lies in the generally accepted (by public thinking) idea, that modern medicine is scientifically proven and the methods utilized by the medical profession are part of the scientific methods of pure science and that applied kinesiology is not. This is, in my opinion, partly because the popular media often utilize the words science and medicine in the same phrase - "medical science has discovered a new cure for AIDS".... new medical scientific breakthrough in artificial heart"..etc, are what we all read. We know that if a big lie is told often enough and with enough emphasis, people in general will begin to believe it and then as the general public, both of the educated and less educated, accept this as if it were fact and rely on it, no one challenges the use of the words, even though the educated when challenged with admit the falsity of the statement.

This is because understanding between people is what everyone is seeking not the correct use of words according to dictionaries. It is, in various acadmenic and other communities, which have specific special meanings to words. According the some in the medical field, the word science means "the systematic observation of natural phenomena for the purpose of discovering laws governing those phenomena and the body of knowledge accumulated by such means." Then, applied science is the application of discovered laws to the matters of everyday living. Whereas "pure science is concerned solely with the discovery of unknown laws relating to particular facts". Then, research could be classified into research for the purpose of utilizing the findings of science for particular purposes of everyday living and, second, the discovery of unknown laws relating to particular facts. It is when we utilize the words scientific research and/or just research without defining why we are doing the research, that there is a lack of communication and understanding between the parties and some frustration occurs. By the above definitions we all agree that medicine and applied kinesiology are both applied science, as practiced by clinicians.

Goodheart aptly named his observations related to the testing of the movements of the body, applied kinesiology, because it was utilized by him and those that have followed his original discoveries in practical everyday practice of the healing arts practitioner in matters pertaining to daily living of themselves and their patients.

What are the "laws" of the science of applied kinesiology? I am utilizing the word "law" to mean the rules of conduct, recognized by the International College of Applied Kinesiology by custom and authorization of the governing bodies that is the Certified Teachers of Applied Kinesiology (CTAK), the International Board of Applied Kinesiology (IBAK), and the executive board of the International Collge of Applied Kinesiology (ICAK), by which people utilizing manual muscle testing can be said to be doing "applied kinesiology". What is the system or body of knowledge that is considered "applied kinesiology?"

In order to consider the answer to the question, I believe that further definition of term meanings must be considered. Another definition of science is any department of knowledge in which the results of investigation have been logically arranged and systematized in the form of hypotheses and general laws subject to verification and another definition is knowledge of facts, phenomena, laws and proxiomate causes, gained and verified by exact observations, organized experiment and ordered thinking. These definitions are easier understood in relation to the scientific approach of applied kinesiology than the earlier definitions, but both are satisfactory when we talk, if all are considered.

To consider the scientific nature of applied kinesiology we need to know what I mean by a "hypothesis." I would define a hypothesis as "an unproven scientific conclusion drawn from known facts and used as a basis for further investigation or experimentation" and further, "an assumption or set of assumptions provisionally accepted as a basis for reasoning."

Further, we need to differentiate between hypothesis and theory. The definition I am using for theory is "a plan or scheme existing in the mind only, that is a speculative or conjectural view of something as an integrated group of the fundamental principles underlying a science or its practical applications" and "the abstract knowledge of any art as opposed to the practice and a closely reasoned set of propositions derived from and supported by established evidence and intended to serve as an explanation for a group of phenomena."

In order to differentiate between hypothesis and theory we need to know what I mean by "fact". A fact is "something that I have actually observed and know to be true by my personal experience or something that I accept as actually existing or that has happened". A fact then is a personal experience for me for a particular time. My acceptance of the fact is subject to my greater knowledge on a personal basis. We have courts of laws and scientific journals that are arguing over what are the facts that various people can agree.

Groups are constantly forming because of the failure of individuals to agree on the "facts". As in law, facts are defined as an alleged circumstance or event presented to the jury as part of a case, considered apart from its legal interpretation by the judge.

When George Goodheart first presented the facts of applied kinesiology, he presented them as a hypothesis, that is, an unproven scientific conclusion drawn from known facts and used as a basis for further investigation and experimentation. Goodheart continued to systematically observe facts and receive reports (facts) from other applied scientific observers of their experiences (their personal facts). When the hypothesis had been observed and put into logical arrangements, a set of systematized general laws of applied kinesiology developed. These general laws were not spoken of as general laws of applied kinesiology, but they have been utilized as such. The International Board of Applied Kinesiology gives examinations related to the theories, hypotheses, laws and applications of applied kinesiology.

The problems that have arisen in applied kinesiology circles because we have not differentiated between what are the laws, hypotheses, theories and facts of applied kinesiology.

Further problems have arisen outside the circles of applied kinesiology because the facts (being personal acceptance of something existing or having happened, or something that I have personally observed or experienced), are not accepted as facts. Therefore, the hypotheses (the scientific conclusion drawn from the known facts), are not accepted and thus no applied kinesiology laws could be considered. If the laws of applied kinesiology cannot be considered, then no applications would be utilized. We have not defined our problems related to scientific acceptance of applied kinesiology.

The problem as I see it is that we are not diferentiating between the parts of applied kinesiology in words that are understood by other disciplines of science. I have already defined science and I believe that ICAK by that definition can be considered a scientific body. We must, therefore, utilize words in ways that are understood outside our scientific group to describe what parts of scientific methods we are functioning. When people in other disciplines say that we are "unscientific", utilizing the word in a tone that is derogatory, we need, in my opinion, to realize that they do not understand our methods and we are not making what we are doing clear to them. If we can taken responsibility for making our communication clear and understood, I believe, that we will be accepted as one of the most scientific of the applied scientific organizations in existance.

I propose that as part of the papers that are presented, that the authors decide if what they are presenting is a theory, a hypothesis, a technique based on applied kinesiology laws, a report of facts, observations of phenomena, philosophy, editorial, or comment. In this way, we will be able to determine through the policy set up by the members of this organization through the IBAK, CTAK and the executive board of the ICAK, what are the theories, hypotheses, laws of the science of applied kinesiology. The importance of our work in clinical applied kinesiology cannot be over emphasized. The applications of the scientific discoveries of the members made in clinical observation have already changed thousands, if not millions, of lives for the better. They have saved millions of dollars and have prevented much suffering in humanity. I urge all of you as members to realize that you are or can be part of the scientific observation of facts, phenomena and prominant causes, which when reported can be verified by exact observation of other members and be subject to ordered thinking and discussion. This is how science is done, we are doing it.

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REPORT ON I.C.A.K. VIDEOTAPES LIBRARY

by

Dr. C. Lance West, D.C., D.I.C.A.K.

During the past five years, I have had the pleasure of attending and videotaping numerous seminars which were taught by Dr. George Goodheart and Dr. Walter Schmitt. Many of these classes have been in Detroit, Michigan but, occasionally, I have traveled to classes in other cities.

My interest in doing the videotapes was first, to improve my own understanding of Applied Kinesiology; and second, to record the classes so those who were not able to attend could rent or buy copies so they could also keep up with the continuing development of Applied Kinesiology.

To date, I have numerous tapes and I felt it would be good to catalog them so those who might be interested would know what is available.

My video equipment is simple home video--not professional, and the quality of the videotapes has improved each year as I update my camera and video recorders. To date, I am on my fifth camera and video recorder, and they are continually improving the equipment so I expect to keep up with the latest as it is developed.

I have numerous video recorders for duplicating the tapes, since reproducing each tape is like making an original in that it is recorded from the video player direct to another video player and takes just as long to duplicate as the original did to film.

At present, all available tapes are VHS-6 hour speed (super long play). To rent or purchase any of the tapes listed, please contact:

Dr. George Goodheart
542 Michigan Building
Detroit, Michigan 48226

or

Dr. Walter Schmitt
1926 Overland Drive
Chapel Hill, North Carolina 27514

As they receive your orders, they will notify my office and I will send the tapes via United Parcel Service. They are to be returned to me after the two-week rental, if that is what you order.

All orders should include specific titles or numbers and a check to pay for the tape--either $75.00 rental per session or $150.00 purchase price. The profits from the videotapes are used for the development and research of Applied Kinesiology.

If there are special sessions such as Dr. Walter Schmitt's "Links Between the Nervous System and the Body Chemistry" which included a large notebook with each of the sessions, these tapes are only sold for the normal seminar fee of $200.00 and are not available as rentals.

Hopefully, as the years go by we will have a very complete video library.

Dr. Goodheart and Dr. Schmitt have authorized gifts of the ten sessions to the Palmer College of Chiropractic Applied Kinesiology Club, the Los Angeles College of Chiropractic Applied Kinesiology Club, and the National College Applied Kinesiology Club.

We are all indebted to Dr. Goodheart and Dr. Schmitt for their continuous efforts in Research and Development of the new areas of Applied Kinesiology, and also for their excellent presentations and sharing of their findings with us.

If there are any questions about the content or quality of the videotapes listed, please feel free to call me and I will do my best to help you with any information I have.

My telephone number is 419-475-4323. Call on Monday, Wednesday, or Friday from 8:00 a.m. to 6:00 p.m.

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OZM-101-ST-1	Stomatognathic System - 6/84 Session #1 Drs. Goodheart & Schmitt
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NBL-239-CBS-6	Links Between Nervous System & Body Chemistry Dr. Walter Schmitt Session #2 Chicago, Illinois
NBL-240-CBS-7	Links Between Nervous System & Body Chemistry Dr. Walter Schmitt Session #3 Chicago, Illinois

****INDEX****