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

PRESENTED JAN. 7th THROUGH JAN. 9th, 1987

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

INTRODUCTION

by

Sheldon C. Deal, D.C., N.D.

Past Chairman

This twenty second collection of papers of the members of the International College of Applied Kinesiology represents 16 papers written by 11 authors.

These papers will be presented by their authors to the general membership at the winter meeting to be held in Key West, Florida on January 7, 8, 9, 1987. The authors welcome comments and further ideas on their findings either in Key West or you may write them directly as their addresses are included in the Table of Contents.

These manuscripts appearing in this collection of the International College of Applied Kinesiology papers have been initially screened. However, neither the International College of Applied Kinesiology nor the Executive Board nor the Examining Board necessarily endorses or approves or vouches for the originality or authenticity of any statements of fact or opinion. The opinions and positions stated in these papers are those of the authors and not by fact of publication necessarily those of the International College of Applied Kinesiology or the Executive Board or the Examining Board.

The papers are being mailed out to the members well in advance of the Key West meeting. This will allow the membership at large to read the papers in advance which will save time at the winter 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.

*Diplomates

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Instructions to Authors of Collected Papers

The *Collected Papers of the Members of the ICAK* are published twice annually, at the summer and winter meetings. Manuscripts are reviewed for format, originality, and quality for reproduction. There is no review for authenticity of material. The ICAK recognizes that the usual procedure for selection of scientific papers is a blind review. The purpose of the *Collected Papers of the Members of the ICAK* is to stimulate creative thinking among its members. These papers are distributed only to the members of the ICAK for general evaluation and for the members to place into perspective the validity of the described approach. The purpose is to place before the membership primary observations which may lead to scientific investigations, new areas of research, and in-depth study, inspiring progress in the field of applied kinesiology.

Statements and opinions expressed in the articles and communications in the *Collected Papers of the Members of the ICAK* are those of the author(s); the editor(s) and the ICAK disclaim any responsibility or liability for such material.

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

Following are the current requirements which will be applied to the Winter 1986 papers which are due August 1, 1986:

1. The paper must be an original work and deal specifically with applied kinesiology examination and/or treatment techniques. Various techniques may be discussed if they are correlated with applied kinesiology manual muscle testing examination.

2. The paper must begin with the title, author's name, and an abstract. The abstract should be a brief description of the content of the article.

3. The body of the article is to follow the abstract and include an introduction, discussion, research procedure, and discussion of findings. Any or all of these topics may need to be addressed, depending upon each paper.

4. The paper is to end with a short summary of the author's conclusions.

5. Maximum total pages printed by one author per volume may be limited to 50 pages. Greater lengths will be considered on a space available, first-submitted first-served basis.

6. Quotes should be short, usually no longer than three lines, and should be referenced, giving credit to the original author. All referenced articles, books, or persons other than the author must be properly referenced at the end of the paper; for example, David S. Walther, *Applied Kinesiology, Volume I — Basic Procedures and Muscle Testing* (Pueblo, CO: Systems DC, 1981). If an article in a journal is referenced, the notation should read as follows: Walter Schmitt, Jr., "Fundamentals of Fatty Acid Metabolism — Part II," *The Digest of Chiropractic Economics*, Vol. 28, No. 2 (Sept/Oct 1985).

7. Any quotation of copyrighted material that is longer than that noted above must be accompanied by permission to print from the author and/or copyright holder. The permission must specifically note that the material is to be printed in the *Collected Papers of the Members of the International College of Applied Kinesiology*, copyrighted by the International College of Applied Kinesiology.

8. All art work must be original, or permission to print must be obtained from the author or artist, referenced in the article, and a copy of the authorization sent along with the article at the time of submission for printing in the *Collected Papers*.

9. Terminology or procedures that might be unfamiliar to some readers should be referenced at the end of the paper.

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

11. The body of the article should be double-spaced on plain paper. No papers typed on office letterhead will be accepted. The manuscript must be clear copy with dark print to ensure adequate reproduction in the *Collected Papers*. The margin on both sides of the paper must be a minimum of ¾", and the top and bottom margins ¾" when relating to 8½" x 11" letter-size paper. European authors should make note of the copy height of the American standard 11" paper size, which relates to approximately 28 cm.

12. Manuscripts that do not meet the above qualifications will be returned to the author, with recommendations for bringing the paper into ICAK guidelines for possible future publication.

13. Currently the articles to be published should be sent to the Education Committee Chairman in triplicate. The Education Committee Chairman is David S. Walther, D.C., 275 West Abriendo Avenue, Pueblo, CO 81004.

It is planned to establish a Publications Committee in the near future to review all ICAK publications.

INTER-EXAMINER AGREEMENT IN APPLIED KINESIOLOGY MANUAL MUSCLE TESTING

Katharine M. Conable, D.C.
Bert T. Hanicke, D.C.

ABSTRACT: In a study of inter-examiner agreement in manual muscle testing involving 110 muscles tested on 11 subjects, overall agreement was 78.2 %, with 7 subjects showing agreement of 90% or better. Respiratory faults appear to account for the discrepancies between examiners (agreement < 90%) for 3 subjects, and ocular lock apparently did so in the 4th. Retesting after these factors were eliminated resulted in 100% inter-examiner agreement in the first 3 subjects and 90% in the 4th.

Previous investigators have found considerable variability in the results obtained with manual muscle testing among different examiners testing the same subject and between machine and manual muscle testing. In 1979 Markham[16] compared muscle testing done manually using the methods described by Kendall, Kendall and Wadsworth[15] with the hand-held dynamometer and the Cybex dynamometer and found differences with each system. Blaich [2] reported that manual muscle testing and Cybex muscle testing did not show a useful degree of correlation. Goubel and Meldener[18] recently demonstrated that to achieve a reliable recording which matches the manual muscle test evaluation, inputs from both the examiner and the subject must be evaluated. Engel [8] found poor inter - examiner agreement

Blaich [1] found that respiratory phase of the patient could influence both manual and Cybex muscle testing. Other factors which have been reported to influence muscle tests and could be a source of discrepancies between examiners are: examiner phase of respiration, examiner or patient expectation, [21, 23] patient or doctor hand position ("hand modes"),[3, 5, 20] sex of examiner, [4] examiner neurologic disorganization or major bodily distortions on a surrogate basis, [6,7,8,11,19] changing environmental influences such as electrical or magnetic fields, and spectrum of light.[6,13,14]

In this study we addressed agreement of muscle testing results between two experienced examiners while controlling for some of the above variables and leaving others uncontrolled. Specifically, environmental factors were somewhat controlled for by having the subject remain in one room for all tests. This would leave most such factors constant with the possible exception of transient variations in magnetic or electrical fields. It also simulates an actual treatment environment in which most muscle testing takes place in practice. Gravity and motion effects were eliminated by having the patients rest supine for 5 minutes before being tested and by having the patient remain supine between examinations. Testing procedures simulated what actually might occur in a patient's visit where special precautions are not routinely taken.

The first examiner then came into the room and tested the following muscles bilaterally in this order: pectoralis major, sternal division, middle deltoid, upper trapezius, rectus femoris, tensor fascia lata. The subject was instructed to look straight at the ceiling and take no breath in or out during the actual tests - holding the breath neutral. The examiner ensured that this was done, as well as watching that the patient did not touch his body with his hands during the testing and that the head remained centered. Any test which was questionable as to eye position, head position, phase of respiration, or accidental therapy localization (patient touching own body) was redone until the examiner was satisfied he knew the state of the muscle. Results were recorded as strong or weak (facilitated or inhibited) for each muscle tested. The first examiner then left the room after requesting the subject to stay supine on the table and to refrain from discussing the examination with the other examiner.

The second examiner then came into the room and repeated the above instructions and muscle tests. Conable was examiner #1 for 6 subjects and Hanicke was examiner #1 for 5 subjects.

The examiners tested the muscles as they usually do, with no prior review of the procedures. No attempt was made to distinguish doctor initiated from patient initiated testing. [22] No attempt was made to control for examiner respiration, eye position or location of stabilizing hand. Although

Data Sheet Inter Examiner Agreement Study

Subject	Age	Sex	MT exper.	P.M.S.	P.M.S.	Delt.	Delt.	U. Trap.	U. Trap.	T.F.L.	T.F.L.	Quad	Quad	Tester	Agree	Retest	Retest
			tri/hrs AK	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.			Finding	Agree
1	37	F	DC2yr/200	s	s	s	s	s	s	s	s	s	s				
			SA 5	w	s	s	s	s	s	s	s	s	s	1st K	90 %		
2	40	F	tri 7/ 70	s	s	s	s	s	s	s	s	s	s	2nd B			
			SA 5	w	s	s	s	s	s	s	s	s	s	1st K	90%		
3	21	F	CA/ TFH	w	w	s	s	s	s	s	s	s	s	2nd B			
				s	w	w	w	s	s	w	w	s	s	1st B	60 %	Aug. insp.	100 %
4	32	F	DC 2mo/100	w	w	s	s	w	w	w	w	s	s	2nd K		assist	
			SA 6	s	s	s	s	s	s	s	s	w	s	1st K	30 %	Ocular	90 %
5	36	M	DC2yr/50	s	s	s	s	s	s	s	s	s	s	2nd B		lock	
				s	s	s	s	w	s	s	s	s	s	1st B	90 %		
6	35	F	DC2mo/200	s	s	s	s	w	w	s	s	s	s	2nd K			
			SA10,TFHins	s	s	s	s	w	w	s	s	s	s	1st K	100 %		
7	24	M	tri9/90	s	s	s	s	s	s	s	s	s	s	2nd B			
			SA 6	s	s	w	s	s	s	s	s	s	s	1st K	90 %		
8	35	F	tri 10/ 90	w	w	s	s	s	s	s	s	w	w	2nd B			
			SA 3	s	s	s	s	s	s	w	w	s	s	1st B	40 %	Temporal	100 %
9	28	M	tri 6/ 40	s	s	s	s	s	s	s	s	s	s	2nd K		bulge	
			SA 3	s	s	s	s	s	s	s	s	s	s	1st B	100%		
10	23	M	tri 9/130	s	s	s	s	s	s	s	s	s	s	2nd K			
			SA 5	s	s	s	s	s	s	s	s	s	s	1st K	100 %		
11	36	M	tri 7/ 80	s	s	s	w	s	s	s	s	s	s	2nd B			
			SA 4	s	s	s	s	s	s	s	w	s	w	1st B	70 %	Aug. insp.	100 %
														2nd K		assist	
Different # / %				7	32 %	4	18 %	3	14 %	6	27 %	4	18 %				

KEY:
- tri = trimester in chiropractic college
- SA = # trimesters worked as Student Assistant at Kinesiology Center
- hrs AK = # of hours of A.K. seminars attended to date
- TFH = one 12-hour Touch For Health Class
- TFH ins = Touch For Health Instructor

The greatest discrepancies between examiners were found with the pectoralis major, sternal division muscle (32 % disagreement) and the tensor fascia lata muscle (27 % disagreement). This may have resulted from slight variations in testing technique between examiners as noted by Walther.[23] However, all but one of these discrepancies disappeared when respiratory, or in one case ocular lock effects were eliminated. It may also be that these muscles are particularly sensitive to such effects.

The unexpected result was the apparent prevalence of "invisible" respiratory effects. When days intervened between the original test and retesting it is possible that other factors in life had already corrected some cause of poor inter-examiner agreement. However it seems very likely to us that the respiratory factor was in fact responsible for the observed discrepancies, in that correcting it did appear to result in a high level of inter-examiner agreement. This does agree with Blaich's findings on respiratory changes in muscle tone. [1] Future studies could control for phase of respiration by examining for and correcting cranial and sacral respiratory faults before or as part of the blind testing.

These results suggest that it is not sufficient to ask a patient to hold his breathing neutral and for the examiner to watch closely to eliminate the effects of respiration of muscle testing results. Although we may catch some patients "cheating" with respiration by observation, there are

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Application of Induced Neurovascular Response to "Filum Terminale Coccygeal Lift"

ABSTRACT: Induction of neurovascular response as described by Woodson can be used to determine the precise line of drive for Goodheart's filum terminale coccygeal lift.

In 1985 Goodheart presented an adaptation of Lowell Ward's coccygeal technique which he calls "Filum Terminale Coccygeal Lift"[1]. This involves a cephal and posterior skin traction on the coccyx, with the exact line of drive determined by marked reduction in musculoskeletal pain at the site of chief complaint or in the paraspinal muscles. At the Summer meeting of ICAK in 1986, Alan Woodson [2] presented the concept of using general spinal and paraspinal pressures to release spinal tension, determining line of drive by monitoring at a convenient neurovascular reflex point, such as the lambda. The correct vector and degree of pressure was observed to produce a strong pulsation at the neurovascular point which did not vary with the patient's respiration. Dr. Woodson observed that using this technique many patients experienced great relief of spinal tension, changes in breathing, improved muscular response, and symptomatic relief. Since reading this paper, I have used this technique with similar good outcomes.

coccygeal lift, with its accompanying meningeal release, as a major and fundamental therapeutic tool. Frequently the patient's breathing changes, with a big sigh accompanying the release of spinal tension, paraspinal and other pain is markedly reduced, Category I pelvic lesions recur much less frequently, and several times I have seen patients go through rapid emotional releases, with episodes of giggling or very brief shaking and crying. Of course when this passes - usually within less than a minute, the patient feels a great sense of change and relief. As Goodheart has observed, the upper cervical and cranial dural attachments often must be attended to as well, with usual A.K. procedures.

I would like to thank Dr. Woodson for his simple and very powerful observation, and recommend its application to procedures such as coccygeal lift and Logan Basic Technique which depend on a precise line of drive for their effectiveness.

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(2) Elmer J. Cousineau, "Chiropractic Meridian Adjusting" page 55, Collected Papers of ICAK, Summer 1982

(4) Same as (2)

(5) Elmer J. Cousineau, "Surrogate Testing by the Doctor"
(6) page 63, Collected Papers of ICAK, Summer 1982

CANDIDA ALBICANS---DIETARY VARIATIONS

by A.P. Karpowicz, D.C.

ABSTRACT:

The purpose of this paper is to seek out alternative, acceptable substitutes for the Candida Albicans patient who is on a very restricted yeast free diet.

INTRODUCTION:

As we have been made aware of the work of Dr. Orian Trust, Birmingham, Alabama and in our field Dr. George Goodheart, Candida Albicans is a ubiquitous enemy in our bodies. Similar to the Tuberculosis Bacillius, a germ which is in all our bodies however not dangerous when held in check by our natural defenses, Candida Albicans reminds us that this yeast proliferation is primarily caused by an excessive use of antibodics, prolonged Birth control usage and Steroids of the Cortisone type.

The yeast is fed by Carbohydrates especially of the type such as containing refined sugar in our famous junk foods, and those less obvious such as beer, wine, cheese, mushrooms, vinegar, etc. Because of the extensive use of yeast in everthing from literally bread to nuts, it is difficult for the patient diet-wise to avoid the foods that feed the Candida Albicans problem.

night, to quitting smoking, etc. is difficult to break. I think a complete change of diet is a large undertaking for the average person. So in conclusion, I feel that it is our job Doctors, to help smooth the transition and in essence truly treat the total person.

Thank you and your suggestions and subsequent findings would be of personal interest to myself. Feel free to contact my office. Thank you.

OBSERVATIONS OF NORMAL ENLARGEMENT OF THE FEET AND THE FIRST METATARSAL JAM

by Dr. Philip B. Maffetone D.C., P.C.

ABSTRACT: Millions of people routinely buy the same size shoes year after year, and some even wait for decades before checking their shoe size. Many adults are unaware that their feet can become larger with time. It has been observed by the author that foot size can increase for two reasons. 1. The foot may be compensating for the results of changes such as weight gain, postural imbalances, pregnancy, or exercise. 2. When the individual is properly treated, and proper balance is achieved, weight is then evenly distributed between the feet; the muscles and ligaments then relax, and the bones of the foot spread out.

The overall problem is one of patient education, as most people do not realize that their shoes are too small. In a two year period, from May 1984 to May 1986, size increases of up to 2 1/2 sizes were observed. No abnormal findings were found in the feet of these patients. These changes occurred in two types of patients: 1. New patients who, when comparing the size of their foot with the shoe size presently worn, were found to have shoes that were too small. 2. Competitive athletes, whose feet enlarged in the normal course of efficient training and racing. A random sampling of 45 competitive athletes showed that 24, or 53%, had improper shoe size.

The local problem is a jamming of the first metatarsal due to the tightness of the shoe. This jamming can then cause recurrent structural (or even chemical) problems in many areas, including the foot, knee, hip, pelvis, and spine. Using standard Applied Kinesiology procedures, the first metatarsal jamming can be diagnosed

are compounded, however, when the individual is unaware of the changes taking place. After a certain age, people no longer have their feet measured when buying new shoes, as they do not realize that their feet have changed in size. As a result, the same shoe size is worn for years, or even decades. This leads to a slow jamming of the toes, especially the first metatarsal. The calcaneal bone is often involved as a tarsal tunnel syndrome. Soon the toes become like springs. When the shoe is slipped on, the toes spring in, and the tightness of the shoe is never realized. This may result in classic foot related problems such as hammer toes, bunions, etc. The first metatarsal, however, is not as lax as the other digits. Therefore, the first metatarsal takes most of the abuse.

Any problem in the structural, chemical, or mental triad of the body may relate directly or indirectly to that jammed metatarsal. It is interesting to note that because of the slow onset of the problem, the first metatarsal is often asymptomatic - people usually don't complain of pain in that area. More often the stress is transmitted upward, and the symptom, whether pain or dysfunction, is felt in the ankle, knee, thigh, hip, low back, or even up as high as the neck or head.

ANALYSIS

Although the first metatarsal usually doesn't hurt, the patient's problem is very simple to diagnose. Proper observation of the feet will reveal trauma, or micro trauma, to the front of the foot, including discoloration of the nail bed (the all too common black toenail seen in runners and cyclists), blistering or callousing of the toes, first metatarsal swelling, enlargement, or pain upon

There is, however, a big drawback to measureing the foot with the standard devices used in shoestores: The shoe manufacturers' sizes are not consistent. Thus, their sizes do not always match up with these measuring devices. Yesterday's size 9 may be today's size 8 and tomorrow's size 8 1/2. For example, running shoes from some companies, like Tiger and Adidas, tend to run small, and many running shoe companies, with the exception of New Balance, make shoes in only one width. (Recently, Brooks, Nike, and Converse have begun to make certain models in different widths.)

Since most people's feet are at least slightly different in size and often do not fit into what the manufacturers have to offer, the ideal thing to do would be to get custom made shoes. Unfortunately, this quite inconvienient and expensive.

Most people do not have their shoes custom made. Therefore, it is important to educate patients so that they can find the proper shoes. Many people insist that their feet are a certain size and will absolutely refuse to wear a larger shoe, only to find that their metatarsal problem and any secondary symptoms soon return. Fortunately, there are a number of ways to choose the right shoes from those that are available in the shoe stores:

1. Spend adequate time trying on shoes in the shoe store. Find a harder surface rather than the thick carpet found in many shoe stores, where almost any shoe will feel good. If there is no sturdy floor to walk on, ask the salesperson if you can walk out of the store. If you can't, go shopping somewhere else. Try on the size you normally wear. Then try on a half size larger. If that feels the same, or better, try on another half size larger. Continue

frequently. So if you finally find the shoe that is perfect for you, buy several pairs. Be sure to try them all on; the same shoe may also vary in size, as some shoe companies do not have good quality control.

APPLIED KINESIOLOGY ANALYSIS

This common, asymptomatic problem takes only a few seconds to test and should be part of every new patient exam. The following Applied Kinesiology procedures should be used:

1. Find a strong indicator muscle, such as a strong psoas major.
2. Gently grasp the first digit with the thumb and index finger, gently forcing it posterior (into the foot), jamming the first metatarsal.
3. Retest the strong indicator, and observe for either strength or weakness.
4. Occasionally, either all of the toes need to be gently jammed, or the direction of the first metatarsal test needs to be more medial, as seen in a bunion.
5. Test for zinc, by having the patient ensalivate the tablet. Then retest using the above procedures.

CORRECTION

The following procedures should be used to correct the problem:

1. If the challange was positive, correct the first metatarsal in the opposite direction from the one that created a weakness. This will often also eliminate the need to correct other foot or ankle problems.
2. Correct any other foot or ankle problems that remain.

THE AEROBIC DEFICIENCY SYNDROME

by Dr. Philip B. Maffetone, D.C., P.C.

ABSTRACT: A common symptom complex seen in clinical practice may include complaints such as physical fatigue, low blood sugar patterns, pre-menstrual stress, mental fatigue, recurrent injuries in athletic individuals, overfatness, and circulation problems. A common complex of associated findings may include hormonal imbalance, deficiency in factors related to essential fatty acid metabolism, improper iron metabolism, and poor exercise habits. Any combination of these findings often results in a decrease in the functioning of the aerobic muscle fibers of the body. This decrease in function is seen as a deficiency. The common features of both symptoms and signs are so prevalent that the whole complex can be termed the Aerobic Deficiency Syndrome.

This paper is a synopsis of previosly discussed factors as well as other information not previously found to be related to this complex. This includes the use of carnitine to aid in fatty acid metabolism, the inhibition of iron by fluoride, the use of fluid replacement drinks with exercise, the effect of fructose on copper absorbtion, and the relationship of blood lactate levels to psyclatric symptoms.

These problems are found not only in the so called "athletes", but also in the general population since Everyone is an Athlete.*

INTRODUCTION

A syndrome is defined as "a symptom complex", or "a set of symptoms that occurs together"(1). Although it has recently been

* The title of a forthcoming book by the author.

applications.

2. Schmitt (4,10) also discusses aerobic and anaerobic relationships, especially relating to iron and retrograde lymphatic blockage.
3. Mowles (5,6) shows the relationships between iron, folic acid, and aerobic function, as well as the inhibitory effect of copper on iron (6).
4. Schmitt (7) also addresses the topic of copper and body quadrants.
5. A paper by the author (8) discusses the relationship between lack of aerobic activity and an excess of anaerobic activity as well as the importance of blood flow (9) during exercise.
6. The importance of co-enzymes and minerals and factors that inhibit fatty acid metabolism are dicussed by several authors (3-9).

These articles are available for those wishing to review the relationships between aerobic and anaerobic function and patient care. This is certainly not a complete list, as other excellent articles, workshops, and tapes are also available.

OTHER ASSOCIATED FACTORS

CARNITINE

Carnitine, a betaine, has been shown to play a major role in intracellular fatty acid transport (1,11,12). Fatty acid oxidation takes place in the mitochondria of the aerobic muscle fiber. The importance of proper dietary fat intake, digestion, absorption, liver function, and transport cannot be overemphasized. However, if all of these factors are working properly, low carnitine may be the rate limiting factor in the entire spectrum of fatty acid metabolism. Carnitine acts as a substrate of the acyl transferase enzyme complex and transfers the activated fatty acid from the cytoplasm into the

2.2 mg.). Nineteen of the individuals also weakened to NaF. In a second group of fifteen individuals who did not weaken to copper, only two weakened to NaF. None of the subjects weakened to NaCl. The author concludes: 1) that further investigation of the possible inhibition by fluoride on the aerobic system is needed, and 2) that fluoride has an effect on body chemistry similar to that of excess copper, which has an inhibitory relationship with aerobic function.

A certain amount of copper is needed by the aerobic muscles, and sometimes there is a need to use it nutritionally. It has been shown (24) that fructose in the small intestine will inhibit the absorption of copper. This may be significant in both the patient who requires more copper, as well as the copper toxic patient.

When necessary, it is possible to test the patient with both copper and copper containing substances, as well as fluoride containing substances using standard Applied Kinesiology. These substances are best tested against a previously weak muscle related to some aspect of aerobic metabolism.

BLOOD LACTATE LEVELS

High blood lactate levels have been associated with such "psychiatric problems" as panic attack, axiety, and agorophobia (13,14). These high lactate levels can be the result of excess anaerobic activity, a deficiency of aerobic activity, or any combination whose end result will mimic such a problem. If the aerobic muscles are not programmed (trained) to do what they should do (oxidize fatty acids), energy supplies will come from more predominant anaerobic metabolism - glucose - whose metabolic end product is lactate. Blood lactate is metabolized in the liver to

suicide by jumping off of bridge. Although she lived, she may never walk again.

These patterns sometimes resemble adrenal stress states, where anxiety and panic attacks are accompanied by high epinephrine and normal norepinephrine levels (16). These studies showed that the response to the stress of public speaking, for example, was high epinephrine, normal norepinephrine, but the response to aerobic exercise was opposite - normal epinephrine and high norepinephrine.

Using standard Applied Kinesiology procedures, it becomes easy to analyze a patient with problems possibly related to high lactate levels such as: faulty liver function, metabolic alkalosis, calcium metabolism, thyroid and adrenal funtion, the need for fatty acids - or substances to aid in fat metabolism - and other nutrients such as niacin, thiamine, riboflavin, and HCl.

FLUID REPLACEMENT TYPE DRINKS

Fluid loss during exercise and competition and the need to replace it is an important consideration for any athlete. Most of the fluid loss is water in the form of sweat through the skin and expired air through the lungs. Sweating is an important mechanism used for the control of body temperature. Some researchers (17,18) have shown that there is no change in plasma volume after prolonged exercise and suggest that this loss is intracellular. The most common replacement need in both the non-competitive as well as the competitive athlete is water. Competitive events of up to two hours in duration will only require water for replacement. (See Appendix 2, Race Recommendations).

Most fluid replacement drinks sold on the market will inhibit

contains high amounts of phosphorous which can lower tissue calcium even more.) The sweetness of four grams of sucrose (one teaspoon of table sugar) in 7 oz. of water is what a 2.5% glucose solution tastes like (20). The author has had a good response in athletes using a product called Recharge (Knudsen and Sons, Chico, CA 95973). It is made from diluted fruit juices and sea salt and is available in most health food storres.

Coffee has become a popular drink before exercise and competition. Caffeine will raise fatty acid levels in the blood (21,22), which is needed for aerobic efficiency. It is, however, most efficient when you teach (train) your body to do it on its own, rather than relying on a drug for function (see Appendix 2, Race Recommendations).

Lowered ascorbic acid levels have also been shown to affect fatty acid metabolism (23). These studies show that one gram per day of vitamin C for two weeks increased mobilization of fatty acids during exercise, with concomitant improved performance.

Using Applied Kinesiology procedures, it is an easy to test a patient, using any fluid drink on the market, to determine if the substance will actually improve function, thereby acting as a nutrient. They can be tested "in the clear", using a strong indicator muscle, or against a weak muscle. It is sometimes necessary to test these substances after an exercise session, or even competition, to determine the worth of such drinks for a particular patient.

CONCLUSION

The aerobic system of the body - the red muscle fibers and all

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APPENDIX 1

WHAT IS AN AEROBIC BASE?

The concept of base goes back to the Greeks. A bit more recently, Arthur Lydiard spent years advocating the benefits, which he said would improve race performance and increase the number of competitive years.

So what is a base? It's a period of time where there is no hard training and little or no racing. In other words, it is a period of all "aerobic" running, without "anaerobic" activity. What do "aerobic" and "anaerobic" really mean? Classically, the definitions center around utilizing oxygen, but for our use these definitions are not very practical. The average person just doesn't know or even care about oxygen uptake, and most people who do know a bit about it really have no way to apply it. It's like knowing that imported car horns sound in the musical key of G, and American made cars sound in the musical key of F. That's nice - good party conversation.

In the body, there are two types of skeletel muscle that we use for running, and everyday activity: aerobic and anaerobic. (There is actually, for those who go to very sophisticated parties, a third type called "B fibers" that are "fast twitch aerobic" in nature.) The aerobic muscles burn fat (fatty acids) for energy and are used in an individual's slower running. They are endurance muscles, providing energy for longer periods of time. The anaerobic muscles are very different. They burn sugar, not fat, for energy. They are used during relatively fast running, especially sprinting. However, they can only work for a very short period of time. In some animals, like the chicken, the aerobic muscles, which are red (the dark meat)

PR's after a period of base. Neil Wood ran a 5K PR of 14:43 following his base. Marianne Dickerson ran a 10K PR of 33:18 off her base. At least 80% of the people I work with will run their best races following base. The winter months are a good time to build base.

This base period is then followed by anaerobic track workouts in the early spring along with racing. I often use the month of July, to rebuid a short base of 4-6 weeks. Don't think that base running is always easy. You can really build to a very fast pace and still be aerobic. Many runners often complain that it gets to be too hard. Matt Centrowitz, Mike Starr, and Neil Wood have all approached the 5 minute per mile pace, aerobically. That's quite a feat.

When you take your pulse, take it for 6 seconds and multiply by 10. Perhaps you've seen (or heard) people running with heart monitors. Rob DeCastella uses one, but so does the guy next door who's just trying to break 40 minutes for 10K, as well as the woman who never competes but wants to get trim. The heart monitor has an alarm that sounds when you exceed your maximum aerobic pace.

The aerobic system doesn't peak until you are 35 to 40 years of age. Properly developing your aerobic system will not only make you run faster and for more race seasons. It will burn off that extra fat from the hips, clear it out of the arteries, improve your immune system, give you more energy, keep you relatively injury free, etc. In other words it is a way to build your running as well as your total health in a permanent way.

decreased slightly. This decrease would be later in the week as you approach race day and would vary with the individual. If intervals are being done, restrict this to one interval session, at least four days before the race. These intervals should be short (100 or 200 meter repeats).

3. Avoid getting a haircut during the week before the race. There is not much physiology to back this up. However, clinical information shows that a haircut will create a mild weakness in the body, so why take a mild chance. If you are racing every week and can not go more than a couple of months without a haircut, you should begin by reconsidering your race schedule.

4. Think about the race in a positive, realistic way. You have control over your conscious mind, and even though your subconscious may have some negative input that you can't always control at the moment (old tapes of fear, lack of self confidence, etc.), positive thoughts will make a difference. Positive mental input can make a difference of many seconds, if not minutes in your race.

III. THE DAY BEFORE:

1. Everyone should take off the day before the race. Some people should take off the two days before. Do not be afraid to experiment, and do not worry about not getting in your miles. Think quality, not quantity. If you are really tense the day before, and you feel like you want to go for a run, go for a walk and think about why you are so uptight. Tension can be negative, or it can be positive.

2. From a dietary standpoint, there are several considerations the day before the race:

(such as sodium or calcium) in the later stages of the race. This becomes a very specific thing, and must be dealt with individually. (The key is to determine the nutrients in which that person will tend to deplete, and then to increase those substances in the diet. At times it may be necessary to give a particular nutrient during a long race. Sodium is the nutrient most commonly depleted first.)

4. On warm days, it is important to get water on you as well as in you. However, make sure you are not going to get chilled along the way because of cooler temperatures. This is why the pores may close. If it is hot and you see a hose, run under it.

5. Most runners deprive themselves of a better time because they run the first mile too fast. You can run the first mile a little faster, assuming it is not a hard uphill. If you average an 8 minute pace for a 10K, you can go out at 7:45. If you average 5:30, you can go out at 5:20. If, however, you run that first mile at 7:15 or 5:05, respectively, you will go into debt and never recover from it. If you want to maintain efficiency, your mile splits should not vary too much. This pattern holds true for almost all athletic competitions.

VI. AFTER THE RACE:

1. As soon as the race is over, get more water, without ice. If it is a warm day, put water on you as well. Then dry off. It is a good habit to have a bag of dry clothes, shoes, and a clean, dry towel near the finish.

2. Once you have ingested some water and come to your senses, you can either jog or walk a warmdown or get some nourishment.

a. If you are going to warm down, run very easy or even walk

a. Do not eat any shellfish. This includes shrimp, lobster, clams, mussels, scallops, or any other similar creatures of the sea. Many people have an adverse reaction to shellfish, and this is amplified during any stress such as pre-race tension. Shellfish are also very toxic animals, and you will be producing enough toxins of your own the next day, in the form of lactic acid.

b. Do not eat any raw foods, nuts, seeds, black pepper, or corn (including popcorn). Raw foods would include raw fruits and raw vegetables. (Avocado is an exception, as it is a good pre-race food.) Again, you are in a mild (sometimes major) state of stress, and in that state your intestinal tract is not too efficient. So avoid things that could potentially create intestinal stress.

c. If you do not normally drink alcohol, do not get caught up in the pre-race festivities that sometimes exist. If you enjoy small amounts of beer or wine with your meal, then a glass or two is okay. That small amount of alcohol will help your fatty acid metabolism, which is endurance related. The carbonation in beer is too much for many people, so if beer bloats you, switch to wine. Do not drink bottle after bottle of beer thinking that it is a good way to hydrate yourself. Moderate amounts of alcohol will dehydrate you.

d. Eating whole grains should be part of your daily diet, so there is no need to discuss that. A good quality carbohydrate meal such as a whole grain pasta or brown rice cooked with some vegetables would make a good pre-race dinner. If you have a small piece of something for dessert, that would be okay, but do not eat large amounts of refined carbohydrates (sugar) thinking that you need all that sugar to run the race. You don't. (More about sugar will be discussed below.)

must then rely on glucose. It is very important to spare glucose for as long as possible for use later in the race.

c. A good meal may be something as small as a piece or two of dry whole wheat toast or as large as two eggs (without the whites) with two pieces of toast. Oatmeal with a small amount of butter would also be good. Avoid milk because of its potential for stomach upset.

d. Many people like to drink coffee before a race. Coffee will raise the level of fatty acids in the blood (a good thing), but so will a proper warmup. Let your body do the work rather than relying on a drug, like caffeine. If you are used to coffee before a race, consider the following:

(1). Drinking coffee will begin having a greater negative effect after about two hours. Therefore, if you drink it, consume it at least half an hour before the race. If you are running more than 15 kilometers, coffee should be avoided.

(2). Coffee has a dehydrating effect, so if you drink coffee, you will need more water before, during, and after the race.

(3). As mentioned earlier, do not use sugar or milk.

(4). Coffee has many psychological as well as physical effects. Among them is a feeling of self confidence. If you have to rely on a drug for that, something besides coffee is missing.

4. Warming up is the real key to competing at your potential. The following would apply to an individual who has a good aerobic base, and who is racing at distances up to and including 15K. If you do not have a good base, it is difficult to spend this much time running without a loss of energy. You should not even be racing if

eating in restaurants all present certain considerations.

1. During air travel, you are in a pressurized cabin, void of much humidity. As a result, you tend to lose moisture and mildly dehydrate. With flights lasting an hour or so, this is not a big problem. But on longer trips, it is significant. The remedy is simple: Drink small amounts of water throughout the flight. Having a water bottle on hand is a very convenient way to handle this problem.

2. On longer flights, you will also want to get up from your three foot by two foot seat and move around.

3. There are also a number of considerations regarding dietary habits on airplanes:

a. If possible, avoid eating at all on the plane. This may not be easy if it is a long trip from home to your final destination.

b. When you fly, your sympathetic nervous system is turned on more than normally, almost as if it was your first flight. As a result, your intestinal tract is not as capable of digesting properly. If you must eat, it would be best to avoid such foods as: raw fruits and vegetables, nuts, seeds, popcorn, and black pepper. These foods are difficult to digest normally. During the stress of flying, give your intestines a break.

c. If you generally hate airline food, or if you do not eat meat, you may be better off if you order (24 hours ahead) a special meal such as seafood or vegetarian, although these may include shellfish or lots of raw food. The ideal solution would be to pack your own food.

d. If you drink coffee, keep that to a minimum. This, too, will inhibit intestinal function (after the initial stimulation of

symptoms were almost instantly eliminated and the boy went on to complete recovery.[7]

Fletcher, an author of nutrition-oriented literature, advocated chewing food one hundred times. Research has identified a pathway between the mouth and the brain that may imply the existence of a nervous system mediator of nutrient distribution and utilization.[8] Correlating these and other observations and wondering whether the body was able to identify vitamins or nutritional supplements, Goodheart instructed a particular patient with a problem thyroid to chew a vitamin product known to enhance thyroid function. The patient, upon chewing the product, promptly fainted, a response that was totally unexpected. When revived she felt almost euphoric, better than she had in a long, long time. For the first time since Goodheart began treatment of this patient the muscle related to thyroid function tested "strong", indicating a positive response to nutritional supplementation.[9] Soon thereafter, Goodheart began a clinical study determined to isolate nutrient response. It was observed that some vitamin products would weaken and some would strengthen particular muscles. After much trial and error, specific muscles were found to be responsive to specific nutrients.

Another of the systems profoundly significant to proper body function finds its roots in the postulate of rhythmic movement of the bones of the skull. Cerebro-spinal fluid flow is directly related to nervous system health and is dependent

were followed by immediate return of muscle strength and often miraculous manifestations of restored organic function. This indicated a clear connection between structure and viscera (internal organs), a long-held theoretical postulate, that now had a visible, easily demonstrated representation in clinical practice.

As Goodheart's research continued, he became aware of the work of a chiropractic physician (Bennett) who in the early 1930's had discovered what was hypothesized to be remnants of embryological pulse centers that exist prior to the development of the fetal heart.[6] When these "vascular reflexes" or "pulse centers" were activated, a stimulation of blood supply to specific areas of the body was achieved and organ function enhanced. Goodheart correlated these reflexes with muscle testing and found that these "vascular receptors" acted like circuit breakers which, when overloaded, interrupted the circuit and must be manually reset. This is accomplished by a light tugging touch over the "receptor" site, most of which are located on the skull, activating a palpable pulsation (approximately 72 beats per minute) felt under the fingertips and indicating active stimulation of blood supply to the target organ or site in the body. The accomplishment of adequate stimulation was evidenced by an immediate restoration of muscle function specifically related to the "receptor site" which tested weak prior to "vascular receptor" stimulation.

In 1966 Goodheart published an article about acupuncture

eventually discovered that fixation or restricted movement of vertebral segments in relation to each other was the cause of many observations of bilateral muscle weakness. As far back as 1954, an osteopath, W. W. Martindale, found that he would occasionally produce some problems while correcting others with manipulation. He was hard-pressed to identify why some patients responded well and some did not. He observed that vertebrae were often lesioned in units of three. That is, vertebral areas were often fixed so that normal motion essential to spinal health was inhibited. The concept of fixation was also proposed by a doctor of chiropractic in the 1940's, Ted Blotok. He tried to move these fixations but was not able to accomplish consistently predictable results. Goodheart discovered that in order for normal motion to be restored these fixations must be unlocked by using two hands in the manipulative procedure contacting, simultaneously, two of the segments presumed to be fixed.[12] The analysis of the clinical usefulness ness of the chiropractic adjustment was now beginning to be observed. First vertebral fixation, then subluxation (misalignment) was able to be evaluated with the language of muscle testing.

In 1972 Goodheart published an article in a chiropractic journal entitled, "Cervical Challenge", which recounted the story of a young lady scheduled for surgery because of repeated incidents of spontaneous dislocation of her right shoulder while sleeping. She had come to Goodheart as a last resort hoping to avoid a seemingly inevitable surgery. After testing

of the needs of a body not satisfied with anything short of optimum function.

Goodheart had initially discovered that muscles can be injured at the point of origin or insertion. This was later to be classified under the major heading of neuro-muscular dysfunction. What of the "five factors" or systems of the body? How were they evaluated and incorporated into this system of analysis?

In the 1940's an osteopath (Chapman) extensively palpated the bodies of patients hospitalized for various ailments and recorded his findings according to hospital diagnoses.[5] He observed nodules in specific areas of the body that seemed to relate to each specific diagnosis. When vigorous massage was applied to these areas of nodulation improvement was often observed by both doctor and patient. He had discovered a specific receptor site for each internal organ hypothesized to act like a switch that operates the sump pump that drains that organ. This sump pump, the lymphatic system, is often described as a sewer system and often ignored, though it is the largest blood carrying network in the body. On the basis of these observations, Goodheart, having found a correlation between muscle weakness and organ dysfunction, discovered that particular muscles related to each of these receptors. His findings confirmed that these "lymphatic drainage reflexes" do exist and further that every weakened organ reflects itself as a weak muscle. The vigorous stimulation of these "reflexes",

spasm. A real case of primary muscle spasm is seldom seen. It is generally a secondary condition.[2]

Applied kinesiology allows the doctor to diagnose, through muscle testing, the need for the application of a variety of treatment methods resulting in the restoration of normal muscle function. This "new system of diagnosis" is able to demonstrate that when the need is diagnosed and supplied results are immediate.

The first patient diagnosed and treated with the principle fundamental to the premise of applied kinesiology was a man who was uanble to push anything and as a result consistently failed pre-employment physical examinations necessary to evaluate his ability to perform the tasks for which he was qualified. This condition, which was posturally observed as an elevated scapula (shoulder blade), had been present for fifteen years. Goodheart began his evaluation by having the patient put his hands on the wall as he proceeded to test the function of the muscle which holds the shoulder blade against the rib cage (i.e., anterior serratus) by pushing between the shoulder blades against the spine. The muscle failed to function. The weakness was obvious as Goodheart observed the inability of the anterior serratus muscle to hold the shoulder blade in position. Atrophy of disuse (loss of muscle size), a common concomitant to severe muscle dysfunction was not present. He proceeded in his evaluation as he palpated nodulations in the muscle insertion and intuitively began to apply hard, heavy pressure to "reduce"

causes muscle weakness. This rebound phenomenon, first described by Truscott, occurs because vertebral musculature reacts with equal force in the direction opposite the clinician's pressure causing, when released, a reaction exactly opposite the directional pressure.

In the 1940's osteopathic research had analyzed spinal subluxations with electrodes attached to the intrinsic musculature of the spinal segment. This research registered the degree of response to various electrical stimuli. It was noted that there was a reduced threshold of response if the vertebra was subluxated. The "facilitated" segments were hyper-irritable or "hot".[15] Goodheart had observed that the rebound of a normal, unsubluxated vertebra brought it back to its neutral position and produced no alteration in muscle strength. However, when the subluxated vertebra was challenged in the appropriate manner and aggravation of the subluxation occurred, resultant muscle weakness was an immediate and exact response. The vertebral challenge allows the doctor to be certain of the need to make a corrective adjustment of vertebral position or demonstrate the absence of the need for structural correction. It provides three useful things: precise analysis, immediate response and a reliability factor, that is, an ability to know with relative certainty that you have corrected the subluxation. This technique doesn't exclude other means of analysis such as x-rays, etc., but provides another measurement of function (or dysfunction) and a system of identifying whether a

therapy in the area touched by the patient.

In an attempt to explain the fundamental nature of "therapy localization", a number of hypotheses have been considered. Among these is the concept of electromagnetic energy suggested by the work of Karagulla[16] and Davis and Rawls[17] giving credence to the idea of adding energy to, or taking energy away from, an area in need of therapeutic attention. As time passed, Goodheart became familiar with the hypothesis of neurological function as described by Melzack and Wall[18] and suggested a neurologic explanation of the phenomena of "therapy localization". According to this explanation, it is as if, through the nerve fibers associated with touch, the body is being asked, "Is there a problem in the area I touch?" The body's answer is registered by a change in muscle strength. When you touch an area of the body with your hand, neurologically you are not touching once but twice. Your hand is touching your body and your body is touching your hand. The brain, the master control center, in the area associated with the sense of touch or sensation, has a disproportionate amount of neurons (nerve cells) assigned to the hand. It is this area that is primarily accessed, supported by the sensory fibers associated with the body area that is touched by the hand, focusing the attention of a large mass of neurons in the brain on the area presumed to be in need of therapeutic attention. The nervous system responds, as it responds to any important sensory input -- quickly and efficiently. When we are struck with an object or injured in any way, one of

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STUDY OF SPECIFIC MUSCLES TO DETERMINE DISC LESION AND CORRECTION

by

H. Louis Obersteadt, D.C.

ABSTRACT

After attending the "A-K Extravaganza" in Steam Boat Springs, Colorado, I decided to do a study of Dr. John Bandy's presentation of using specific muscles to determine lumbar disc lesion and direction of correction.

INTRODUCTION

A method of using specific muscles to determine intervertebral disc lesion and its level of involvement was presented by John Bandy, D.C. at the Nutri-West "A-K Extravaganza" in Steam Boat Springs, Colorado, March 1986.[1] George Goodheart, D.C., presented Category III in 1975 Applied Kinesiology Research and Workshop Procedure Manual[2] which is double hand therapy localization to the spinus processes of adjacent vertebra to determine the disc lesion and its level of involvement.

Having used Category III since 1977 to diagnose and treat disc lesions, I wanted to determine if there was a relationship between the different techniques. After testing fifty patients that showed one or more of the three specific muscles, and/or a Category III involvement, there appears to be a definite relationship between the two techniques. Category III is determined by having the patient therapy localize to the spinus processes of adjacent vertebra, for example, the spinus process of S1 with one hand, and the spinus process of L5 with the other hand. A weak muscle is tested for strengthening, or a strong muscle is tested for weakening. If there is a change in the muscle strength, then the transverse processes are challenged apart or together to determine direction of disc protrusion and direction of correction. In John Bandy's presentation, he demonstrated that the gastrocnemius, anterior tibial, and the rectus femorus (straight head) were

The anterior tibial was tested using the testing procedures in Applied Kinesiology Volume I.[3] The rectus femorus (straight head) and the gastrocnemius were tested using the procedures in Clinical Kinesiology Volume II[4] and Volume V[5] respectively. However, the two heads of the gastrocnemius were tested together by keeping the foot plantar flexed without rotation. If the test was positive (tested weak) for a specific muscle, the lumbar spine would be laterally flexed to check for strengthening of that muscle. Then the patient would be placed in a prone position and checked for a Category III. For example, if the gastrocnemius tested weak, the patient was tested to determine if it was a medial or lateral disc by laterally flexing the lumbar spine toward or away from the side of pain. If lateral flexion negated the weakness then the patient was placed in the prone position and checked for Category III. If the therapy localization to the two adjacent vertebrae spinus processes caused a weak muscle to strengthen, or a strong muscle to weaken, a challenge was made to the transverse processes to determine if the correction would be to open the disc space on the side of the challenge, or to close the disc space on the side of the challenge. If the lumbar spine is flexed away from the side of pain and the challenge showed the same, i.e. spreading the transverse processes apart on the side of pain, this would be the direction for correction and would be classified as a lateral disc protrusion to the nerve root. If the Category III was found first, then the procedure was reversed. The patient was asked to therapy localize to the spinus processes of the suspected vertebra, then a challenge was made to the transverse processes. If positive, the patient was placed in a supine position and checked for the specific muscle weakness for that nerve root level. Then the spine was laterally flexed to determine lateral or medial disc protrusion.

The rectus femorus (straight head) showed a 100% correlation to Category III, but of the twenty-four found weak, only six were negated with lateral flexion of the lumbar spine. Eighteen showed facet imbrications of L3 and this was determined by the Holmes Test.[6] The patient

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The Craniosacral System

Further Clarification of Principles, Mechanisms, and Techniques

Marc S. Rosen, D.C.

Abstract: The paper outlines the anatomy and theoretical physiology of the craniosacral system (cranial sacral primary respiratory mechanism) in terms of recent advances in cranial osteopathy. The etiology of "faults" within this system, and their clinical implications, are presented as a preface to a discussion of the application of manual muscle testing (coincident with phases of diaphragmatic respiration) as a primary means of cranial diagnosis. The mechanism of correction is perhaps in greatest need of clarification, with "exageration of the lesion" and "rebound correction" remaining, in the authors opinion, a poorly understood aspect of applied kinesiology.

Introduction: (cont.)

Why then are we faced with an enigma. On one hand cranial "faults" can be rapidly assessed, corrected, then reassessed. In that same clinical setting, dental, gait, chemical, or other potentially adverse factors can be evaluated for and appropriate therapeutic action taken to insure a more effective correction.

On the other hand cranial bone motion is oftentimes misunderstood. The nature of "faults" and how they give rise to their characteristic respiratory patterns and direction of "challenge" remains a source of academic confusion amoung both students and practitioners of applied kinesiology.

Fortunately there are authoritative sources that offer the perspective that is essential to this area of study.

Two remarkable texts in particular are quite comprehensive in their discussion of craniosacral principles and techniques. Namely, Applied Kinesiology Volume II Head , Neck , and Jaw Pain and Dysfunction - the Stomatognathic System by David S. Walther, D.C. (Systems DC: 1983) and Craniosacral Therapy by John E. Upledger, D.O. and Jon D. Vredevoogd M.F.A. (Eastland Press: 1983).

From these texts and other sources as will be referenced , the discussion will focus on:

- the anatomy , physiology , and physiologic motion of the craniosacral system
- the etiology and clinical implications of "faults" or "lesions" that interfere with craniosacral function
- the respiratory (diaphragmatic) patterns that identify specific "faults"
- the mechanism of challenge
- the mechanism of correction

Full comprehension of each item should easily resolve both questions of theory and of technique.

To study any aspect of applied kinesiology is to assume the responsibility for a wealth of information. The classroom demands are a long introduction to the committment of time and funds that runs a practice. The most efficient educational step would be to study the "concept". It is the concept that alerts us to the application of technique. The paper is intended for those who wish to review the "concepts" behind craniosacral therapy.

Discussion: (cont.)

fibers were made visible by modified staining technics. In addition, Upledger reported (8) that he had disected and traced a single nerve fiber from the sagital suture to the third ventricle in a monkey.

The "pressurestat" mechanism is then: a "feedback" or "homeostatic" mechanism that regulates the production of cerebral spinal fluid.

Presuming that Upledger is correct and that the choroid plexuses produce CSF at a rate that is faster than it can be resorbed, then the intracranial pressure could be expected to gradually rise toit's maximum. At that point, the neural elements within the sutures signal the ventricles so as to "shut off" the production of CSF. The arachnoid bodies in the venous sinus system are given the opportunity to "catch up" in their resorptive capacity. The CSF pressure should then drop, with the neural sutural reflex signaling the ventricle to resume the cycle.

In terms of this model, there is a rhythymic rise and fall in CSF pressure that has been found to exhibit a wave like pattern during 6-12 cycles per minute (9). It is productive of craniosacral motion and is mediated by a stretch type reflex that eminates from the cranial sutures.

The author has elected to bypass the issue of sutural ossification. The reference material should be consulted as this issue is one of conflict in modern anatomy. The discussion of the mechanics of cranio sacral motion will proceed upon the asumption that the cranial sutures do permit mobility so as to be sensitive to the described rise and fall in CSF pressure.

The reference material should also be reviewed for the research studies that have sought to document craniosacral motion and function (10).

The motion within the cranium is best understood by considering the mid-line (sphenoid , occipit) and paired lateral bones (temporals , parietals , frontals) separately.

The mid-line bones or the sphenoid and occiput, articulate at the sphenobasilar symphysis *. This articulation elevates and descends coincident with the rise and fall in CSF pressure. Sphenobasilar elevation is described as flexion , extension as it's depression or movement towards neutral.

* Upledger (page 10) feels that the sphenobasilar joint is more correctly named a synchondrosis. Most other cranial writings use the terms symphysis.

Discussion: (cont.)

The dural sheets or the falxes cerebri and cerebelli and the tentorium cerebelli became known as the "reciprocal tension membranes" (16). The dura is in a sense continuous with the connective tissue (especially the fascia) of the body. It's inelastic nature and it's firm union with the cranial bones allows a considerable potential for abnormal torsion or tensions to be transmitted into the skull.

Beginning with a firm attachment at the foramen magnum (and upper three cervicals) the dura envelops the spinal cord (and gives rise to the dural root sleves at the IVF) and eventually attaches at the anterior sacrum at it's second segment. Note also that the sacral and coccygeal attachments of the cauda equina and filum terminal.

The inelastic nature of this membrane causes it to lift the sacrum into a base posterior apex anterior position during the flexion/external rotation phase. Dural pull or tension is released upon extension/internal rotation. The result being a base anterior apex posterior sacral position (17) (18) (19).

Because of the articular facing between the sacrum and ilium (wider at it's anterior aspect) sacral flexion induces an external flare of the innominate (ASIS moves apart). Sacral extension will, as can be expected, move the innominates into an internal flare (ASIS move together) (20).

The sacrum is then the pelvic analogue of cranial midline bones. The innominates behave like paired lateral or temporal bones.

The reader is once again reminded that in terms of Upledger's pressurestat model, the described movements of the craniosacral system i.e. flexion/external rotation , extension/internal rotation, are in response to a rythymic rise and fall in CSF pressure. With the neural and connective tissue components of the mobile sutures mediating the production of CSF by the choroid plexuses, and the resorption by the arachnoid villi. The rise in CSF pressure to it's upper threshold encourages flexion of the midline bones and sacrum and external rotation of the paired lateral bones and innominates. The fall in CSF pressure to it's lower threshold results in extension of the midline bones and sacrum and internal rotation of the paired lateral bones and innominates.

Flexion and extension complete one cycle. Upledger describes craniosacral motion as proceeding through 6-12 cycles per minute (pg 6-9) and various functional and organic conditions that alter this rate.

Discussion: (cont.)

The etiology of cranial faults and craniosacral dysfunction:

Trauma

- birth
- dental
- cervical ("whiplash")
- blows to the head

Habits

- sitting or prone / chin resting in hand
- side posture / head rests against hand

Structural Imbalance

- unequal pull of muscles of mastication (malocculsion)
- unequal pull of SCM and upper trapezius

Remote Factors

- foot , gait dysfunction
- pelvic categories
- upper cervical subluxation/fixation
- dural torque
- T.M.J. dysfunction

Chemical and/or Allergic

The above was extracted from Walther"s Volume II (pages 24-27). While several of these items may require some elaboration, the discussion will not pretend to be comprehensive. Consult the reference for a more detailed study.

The passage of a newborn cranium through the birth canal is by nature precarious. Unfortunately, obstetrical intervention is by it's nature traumatic. Consequently, until "natural" childbirth procedures become the rule rather than the exception, neonatal craniosacral and upper cervical dysfunction may also be a rule (and not an exception).

Walther makes note of the fact that the human nipple is firm and requires a strong sucking effort during nursing. This is natures cranial adjustment, as ...

Discussion: (cont.)

The applied kinesiology principle of structural imbalance calls our attention to the relationship between sartorius/gracilis "weakness" and a posterior subluxation of the ilium (a category two). Students and practitioners of applied kinesiology are also well aware of how often this particular muscle imbalance is secondary to a functional hypoadrenia.

A cranial etiology then progresses as - hypoadrenia - sartorius/gracilis weakness - category two/posterior ilium - T.M.J. dysfunction - imbalance of muscles of mastication/malocclusion - torsion into reciprocal tension membranes - production of cranial faults.

A category three may involve a subluxation of the lower three lumbar vertebrae, or the lovetts of the upper cervical dural attachments. Of interest is the possibility that a cranial fault can torque the dura in such a way that the upper cervicals are in turn torqued. The lovett relation to the lower lumbars could give rise to a category three and it's characteristic subjective complaints of low back pain with or without sciatic neuralgia/neuritis.

An outline of the clinical implications of craniosacral dysfunction would read as follows: (after Walther's Volume II pg. 20-23)

Cranial Nerve Function

Neurologic Disorganization

- aberent afferents from sutures
- adverse effect upon visual righting and labrinthine reflexes

CSF Circulation

- theories of flow along axoplasm
- theories of flow through connective tissue
- possibly assists in hormonal transport

Blood Pressure

Endocrine

Visual / Auditory

- visual acuity , reading , tinnitis , hearing loss

Discussion: (cont.)

abdominal relaxation the inspiration phase of diaphragmatic respiration tends to favor craniosacral flexion. In this example, the system is fixed in flexion. A forced inspiration would force an already flexed sphenobasilar into more flexion. A situation that is referred to as "exaggerating the lesion or fault". It is reasonable to assume that the bodies response to such a stimuli would not be favorable.

When using a manual muscle test to monitor an adverse stimuli (in the craniosacral system) an "intact" or "strong in the clear" "indicator" muscle is tested after the patient has taken and held a forced inspiration or expiration. A change in the indicator confirms that the forced respiration has exaggerated an existing craniosacral fault. The nature of the fault is identified according to which phase of respiration resulted in a positive test (strong indicator became weak). Forced inspiration reveals a flexion fault and forced expiration reveals an extension fault.

There is a second diagnostic use of manual muscle testing. One that evaluates the effect of diaphragmatic respiration (and it's induction of a phase of craniosacral motion) upon a muscle that happens to be "weak in the clear".

In this instance, postural analysis, the T.S. line, an aspect of the history or physical exam, a laboratory test etc. leeds to a suspected muscular dysfunction. The muscle is tested and found to be "weak". An effort would be made to determine which factor was responsible. To evaluate the cerebral spinal fluid factor can be quickly evaluated by inducing a phase of craniosacral motion via diaphragmatic respiration. The "weak" muscle is then tested after the patient has taken and held a full or forced inspiration and then expiration. If inspiration should prove of some assistance and abolished the "weakness", then flexing the sphenobasilar joint also assisted the "weakness". Therefore, if flexing the sphenobasilar joint made something better, then flexion is what is needed as therapy. If the sphenobasilar joint needs to be flexed to "fix it", then it must be an extension fault. Since inspiration was what assisted in resolving a cerebral spinal fluid problem, the fault is named a "simple inspiration assist". If expiration assisted in abolishing the muscle "weakness", then the term "expiration assist" is used.

"Simple" refers to the fact that a full rather than a forced inspiration or expiration was held. When a forced respiration abolishes an existing "weakness" ..

Discussion: (cont.)

The force is further transmitted through the chain and into the sphenobasilar where the flexion fault is ultimately exaggerated as well. A strong indicator muscle would weaken under these circumstances and identify a flexion fault.

Now, a flexion fault was found to be made worse by inspiration (as inspiration serves to flex an already flexxed joint), it is then an expiration assist. When the mastoid is "challenged" in a posteriomedial direction, and the response is positive, then the "challenge" identified an expiration assist fault.

In an extension fault, the extension has internally rotated the temporal and brought it's mastoid process into a anteriolateral position. When a vector is applied to the posterior aspect of the mastoid so as to move it to eh anterior and lateral, then force is transmitted through the kinematic chain in such a way the sphenobasilar joint is brought more into extension. Holding the expiration phase of diaphragmatic respiration would accomplish the same. Therefore, this particular challenge would identify an inspiration assist.

It is the authors interpretation that the "challenge" of cranial bones does not involve the "rebound" phenomenon that occurs in the spine. Instead, the challenge pressure exaggerates a lesion by pushing it more "out of place", so to speak. Rather than have it rebound "out of place" as occurs in the spine. The difference lies in how the challenge determines the direction of correction when applied to the spine.

To consider the mechanism by which a cranial fault can be corrected, perhaps it is best to limit the discussion of a simple inspiration and expiration assist.

For simple inspiration assisted faults, the posterior aspect of the mastoid is contacted and pressure is directed to the anterior (and lateral) during consecutive inspirations. The pressure is relaxed during each expiration phase.

An analysis of this cranial correction or "adjustment" reveals that the contact on the mastoid actually extended the sphenobasilar (this is an extension fault whereby the mastoid remains "stuck" anterolateral). But isn't it interesting that the inspiration tended to flex the sphenobasilar joint and pull the mastoid posteriomedial. On the surface it appears as if the respiratory phase and the mastoid movement are in conflict.

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UPDATE ON GOLGI TENDON-SPINDLE CELLS

Julius L. Sanna, M.S., D.C.

ABSTRACT

This article is to associate current information involving golgi tendon-spindle; differentiation from trigger points and method of application of therapy.

Introduction

The mamlian muscle spindle is a highly specialized receptor structure that is fusiform in shape and is distributed throughout the fleshy parts of skeletal muscle. These spindle or intrafusal fibers are sensory in function, not contractile as are the extrafusal muscle fibers. They respond indirectly to stretch and interconnection to the extrafusal fibers through a shared, or common endomysium. The intrafusal fibers possess two basic configurations based upon the geometry of the nuclei on their equatorial regions. The nuclear chain is a single-file, linear collection of intrafusal nuclei and the nuclear bag is a sac-like cluster of intrafusal nuclei. Both types of fibers are innervated by afferent neurons.

A second form of receptor organ, the golgi tendon bodies or organs lie within muscle tendons immediately beyond their attachment to the muscle fibers. An average of ten - fifteen muscle fibers are usually connected in a

Inhibitory Nature of the Tendon Reflex

"If while still stretched the muscle is made to contract actively, the tendon organ will further increase its discharge, but the spindle discharge will decrease all together." (Kandel and Schwartz p. 289). Signals from the golgi tendon body are transmitted thru IB fibers both into local areas of the cord and thru spinocerebellar tracts to the cerebral cortex. The local signal is believed to excite a single inhibitory interneuron that in turn inhibits the motor neuron originally excited by the spindle response. When tension on the nuscle and therefore on the tendon becomes sufficient, the inhibitory effect from the tendon organ can be great enough to cause sudden relaxation of the entire muscle. This effect is called the lengthening reaction.

It is important to note that to cause the golgi tendon organs to reach threshold and fire initiating their inhibitory mechanism, tension within a muscle must be increased. To increase the tension within a muscle the muscle must be stimulated to contract. A muscle is stimulated to contract via the application of a stretch or a load which activiates the muscle spindle mechanism. By increasing the load upon a muscle, the tension within the muscle is increased and the golgi tendon organ mechanism is activated. An important consideration is the amount of force necessary to activate the tendon organs.

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KININ MEDIATED ALLERGIES AND PANCREATIC ACTIVITY

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

ABSTRACT: A diagnostic screening procedure and treatment procedures for kinin mediated allergy patterns are discussed. In kinin mediated allergy patients, CCK placed on the tongue will create universal muscle weakness which may be neutralized by the pancreas neurolymphatic reflex, pancreatic tissues, pancreatic enzymes and/or zinc. Correction of the appropriate factor(s) usually neutralizes the allergy pattern and neuromuscular food sensitivity testing is rarely necessary.

There are at least two types of general allergic reactions which occur in our patients. The most well known are immune system mediated allergies which result in the release of immunoglobulins as past of a cascade of events which results in the release of histamine and the classic localized tissue effects of vasodilation, swelling, itching, and so on. The less commonly recognized pattern of allergic reactions is non-immunologic in origin and involves the release of a family of protein molecules which are known as kinins. Bradykinin and cholecystokinin are two of these chemicals which are commonly dealt with in physiology books, but there are many different types of kinins which are polypeptide molecules and which have effects on tissues which are identical to histamine; that is, vasodilation, swelling, itching and so on.

sensitivities. Four of these difficult patients were coincidentally scheduled for food testing on the same day, and by the time the fourth patient was tested, it became quite apparent that there was a pattern that these patients had in common which had not as yet been identified.

We were aware that there are kinin mediated allergies that are different from histamine mediated allergies from reading articles by Bell[3] and Philpott,[4] but we knew of no way to test for these chemical patterns. Since it was thought that bradykinin was a common mediator in this type of non-immunologic allergic reaction, and since we had no source of bradykinin, we had felt at a loss to develop a muscle testing procedure to screen for kinin mediated reactions.

The fourth patient scheduled on the day mentioned above was the last patient of the day, and so after identifying a pattern of universal muscle weakness induced by several food substances, time was taken to try to identify a new pattern associated with this reaction. In the office was a testing bottle of a substance called "CCK" by Cardiovascular Research, Ltd., which was "A specially prepared gastrointestinal polypeptide, cholecystokinin (derived from bovine duodenum)." To our knowledge this product is no longer available. Recognizing that CCK was a member of the kinin family, it was placed on the patient's tongue with the immediate weakening of virtually every muscle in her body.

The next day, and since that time, nearly every patient in our office has been tested with this CCK substance to observe

food allergy has been proposed by Bell[3] who implicates a kinin initiated cascade of events which involves the blood clotting (fibrinogen) system and the complement cascade.

Bell[3] proposes that kinins released during kinin mediated allergic reactions may be the source of many cerebral symptoms which are so common with food allergy. She relates work which demonstrates the effects of bradykinin on the hypothalamus.

Our observations on kinin mediated allergic patients show that a kinin mediated allergy will induce muscle weakness of the gamma 2 type which reflects a supraspinal source of weakness.[7] This observation tends to support Bell's ideas regarding the hypothalamic effect of kinin mediated allergies.

As a consequence of our investigations, we now routinely test patients against oral insalivation of a CCK containing product whenever we observe a gamma 2 (patient-started test) weakness anywhere in the body. When weakness is induced by CCK, we find that it is a generalized weakness throughout the body on both gamma 2 and gamma 1 levels. This pattern was initially used to schedule the patient for neuromuscular hypersensitivity testing procedures wherein offending foods were identified by creating the same weakening pattern as induced by CCK when they were tasted. These patients were then placed on a restricted diet. Some of these patients, however, reacted to so many foods that an effort was made to discover a procedure which would aid these patients in desensitizing themselves to the foods.

Based on Philpott's ideas, we had patients who weakened with CCK hold the CCK in the mouth while other procedures were

FIGURE 2

$$H_2CO_3 \xrightarrow[Zn^{++}]{\text{carbonic anhydrase}} H^+ + HCO_3^-$$

Carbonic anhydrase, a zinc dependent enzyme, is necessary for the production of bicarbonate ions in the pancreas.

Since zinc is essential for the release of bicarbonate in the cells of the pancreas via its effect on catalyzing the carbonic anhydrase reaction (see Figure 2), and since zinc is an essential nutrient and the pancreatic glandular preparations are not essential nutrients as such, we supplement the patient with zinc if it tests positive.

Our second choice of a supplement if the patients responds to all of the listed substances would be pancreatic tissue. The differences in the various types of glandular preparations are discussed in Common Glandular Dysfunctions in the General Practice by this author.[8] These pancreatic substances apparently aid the pancreas in improving its function based on our clinical observations.

Our last choice for supplementation is pancreatic enzymes. Philosophically, it is preferred to get the pancreas working more efficiently by the use of manipulative and/or other nutritional therapies rather than to place a crutch under it by supplying its enzymes exogenously. In fact, in a number of patients, manipulation of the pancreas NL and attention to other pancreatic reflexes and subluxations in the mid-thoracic area clears the body of any reaction to CCK as well as to foods which previously caused weakness.

less likelihood for polypeptide fragments to initiate kinin reactions, or immunologic reactions for that matter as well.

CONCLUSIONS

There are a number of sources of gamma 2, supraspinal type muscle weakness, including histamine mediated allergies.[7] To this list should be added kinin mediated allergic patterns. Any gamma 2 muscle weakness may be due to the effects of kinins at supraspinal (e.g., hypothalamic) levels. The source of kinin production appears to be due to inadequate pancreatic activity both in the digestive system and systemically, usually with no apparent latissimus dorsi involvement.

When foods or their incompletely digested by-products initiate a kinin reaction systemically, there may be any number of symptoms created or aggravated, so symptoms are not necessarily a clue in identifying a kinin mediated problem. The oral challenge with CCK substance appears to be an extremely effective screening tool for these reactions. Manipulative and nutritional therapy directed at normalizing pancreatic exocrine activity appear equally effective in returning these patients to normal and aiding in the recovery of their symptoms, whatever they may be.

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BUT WHAT IF THERE'S NO WATER IN THE HOSE?

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

ABSTRACT: The rationale for the necessity of nutritional testing and nutritional supplementation in the chiropractic practice is presented in relation to the role of neurotransmitters in carrying messages from one nerve to another. The nutritional requirements for the production of three neurotransmitters, norepinephrine, serotonin, and acetylcholine are outlined. The use of nutrients as alternative to drug therapy in depression and other mental problems, especially those associated with stress, is discussed. The recurrence of emotional recall pattern and the need to repeatedly treat the emotional neurovascular reflexes is related to a need for folic acid, vitamin B-6, or pantothenic acid. The failure of other chiropractic treatments is related to a need for nutrients as neurotransmitter precursors to carry the healing message from one nerve to another.

Early chiropractic theory was explained in a simplified metaphor which is easy for anyone to understand. If you have a hose (nerve) which runs to a flower (organ or muscle) and someone steps on the hose (vertebral misalignment which presses on a nerve), then the flower will wither and die (disease),

to fire off the same chain of events. Eventually, these nerves end in msucles or organs and the release of a chemical at this site causes the muscle to contract, the organ to perform its function, and so on.

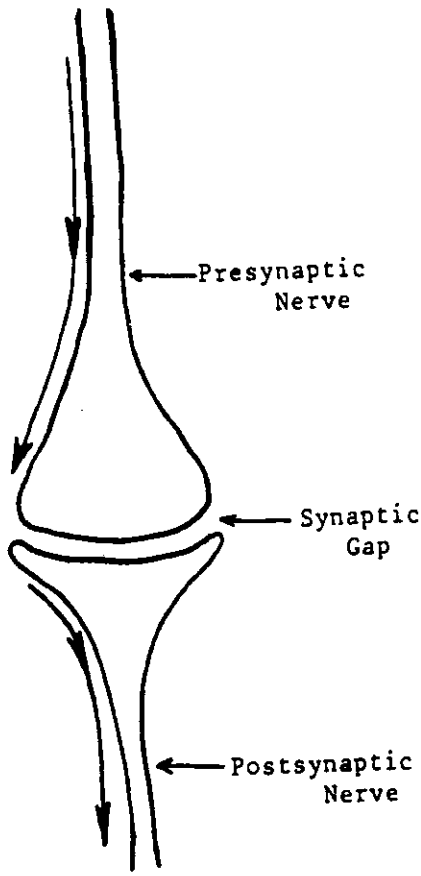


FIGURE 1a

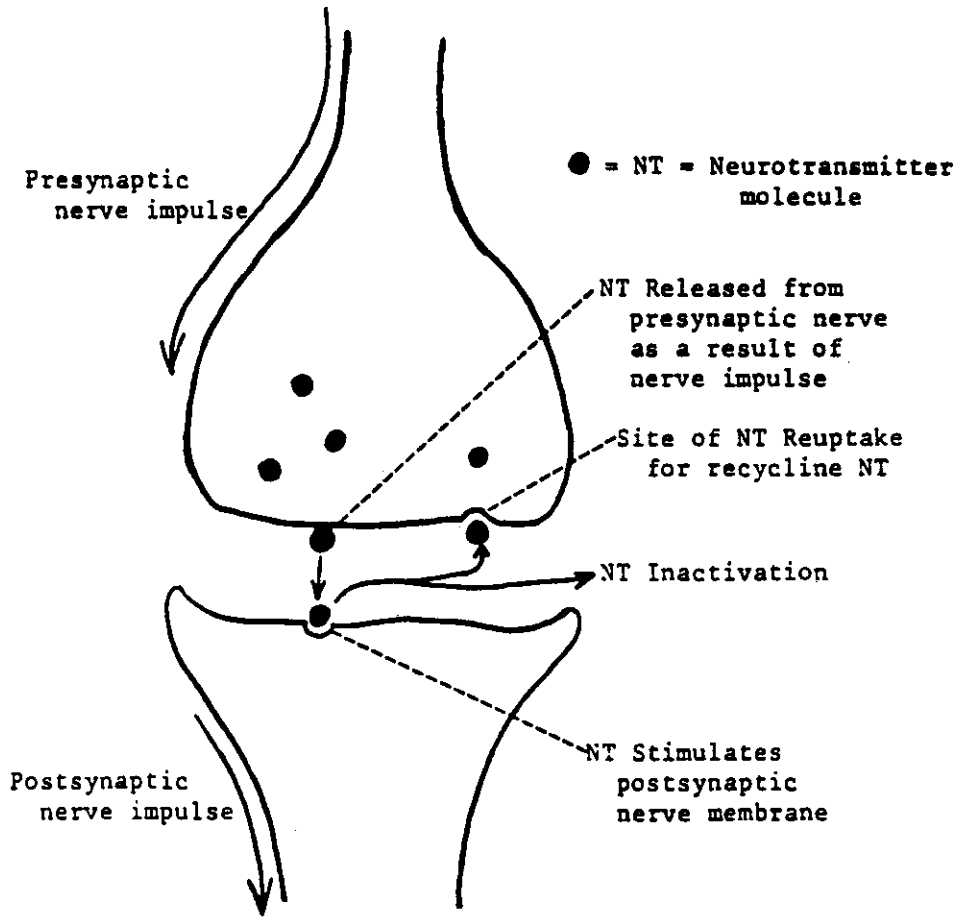


FIGURE 1b

In the brain, the way we feel is perceived by nerves which depend on an adequate supply of these neurotransmitters. How we think, how we feel, our sleepiness and our wakefulness, our ability to sense our surroundings, and how we react to various

entire lives constantly fighting against them. In applied kinesiology, we call this phenomonen "switching" and relate it to interference with normal nerve pathways carrying their messages. This is especially common in times when stress becomes distress and overwhelms us. And yet we observe that some people seem to handle stress better than others and even thrive on it.

Why do some people "go all to pieces," others experience depression, and still others stoically accept fate? There are obviously many, many reasons why people react differently, but one of the reasons is the chemical makeup of the person, particularly the availability of neurotransmitters.

A common treatment for depression is the use of chemicals known as tricyclic antidepressants and monoamine oxidase (MAO) inhibitors which affect norepinephrine and serotonin activity. Common tricyclic antidepressants include Elavil (amitriptyline), Aventyl (nortriptyline), Tofranil (impramine), Norparmin (desipramine), and Sinequan. Other common antidepressants which have similar actions on norepinephrine and serotonin are Ludiomil and Desyrel. MAO inhibitors include Nardil, Parnate, and Marplan. Both of these classes of drugs are widely used for both short term and long term care of depressed patients. Tranquilizers such as benzodiazepines (e.g., Valium, Ativan, Tranxene, Xanax,Librium, and others) which affect GABA activity are also extensively prescribed. The reason these medications work is that they affect the activity of neurotransmitters. They either increase or decrease the message carrying capacity

Many times, one treatment is adequate to drastically reduce symptoms. But more often repeated treatments have been found necessary. Sometimes, the original problem is alleviated but other problems crop up almost as if we weren't really fixing the problem, but just pushing it around the body. As more and more information about the nutritional basis for neurotransmitters has become available, it has become apparent that it is necessary in many people to reinforce their chiropractic adjustments with nutritional support to reduce the need for recurrent treatment. This is often because when we stimulate nerve pathways with our adjustments, there are not enough neurotransmitter substances in the nerves to keep up with the increased level of activity we have stimulated in the nerves by our treatment. It would be like turning on a closed faucet and no water coming out because there is no water in the hose or pipes.

It becomes the responsibility of the modern chiropractor to consider the nutritional status of his or her patients, if for no other reason than because the very nerves which are carrying the healing messages to our bodies are directly dependent on the nutrients we eat for the chemicals they need to carry these messages. In addition, the entire sense of well-being of our patients and ourselves is affected by the balance of chemicals in our brains, which is dependent on the balance of nutrients we ingest.

In this author's seminars and papers on "The Links Between the Nervous System and Body Chemistry" and "Muscle Testing as Functional Neurology," the effects of imbalances in body chemistry,

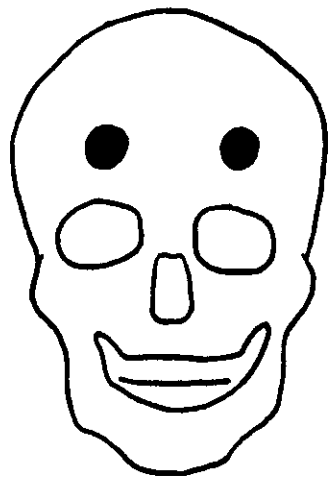
Since depression is associated with neurotransmitter imbalances, it seems reasonable that this type of imbalance is likely present in a large enough percentage of our patients to make it worthwhile of our consideration. And it is possible that the patient who was once depressed, still has the same neurotransmitter imbalance, but it is now showing other (e.g., musculoskeletal) manifestations. In other words, the problem may not have been fixed, but just pushed around the body.

The two neurotransmitters which are usually associated with depression are norepinephrine and serotonin. Antidepressant medications have their effect by modifying the utilization of one or both of these substances at the nerve endings where they are released. (See Figure 1b.)

Antidepressants act by two different methods to prolong the activity of the various neurotransmitters. (See Figure 1b.) MAO inhibitors block the inactivation of the neurotransmitters. Tricyclic antidepressants inhibit the re-uptake of the neurotransmitters at the presynaptic membrane.[2] (See Figure 2.) (Normally, these neurons release neurotransmitters and then, after they have stimulated the postsynaptic neuron, they are reabsorbed by the presynaptic neuron for recycling purposes.) Both types of drugs prolong the time the neurotransmitter is in the synaptic gap and make it more readily available to stimulate the postsynaptic neuron and/or be reabsorbed for recycling.

A patient with low norepinephrine activity is associated with a depression of a "I feel lower than a snake's belly on the ground" type. The person doesn't want to do anything but sit, alone, and feel bad, or possibly sleep all the time. In other words, norepinephrine is a sort of an "upper."

applied kinesiology understanding of the short-circuiting of the stomach activity associated with the emotional neurovascular circuits (See Figure 3), it becomes easy to understand how a less than optimal production of hydrochloric acid and pepsin by the stomach can aggravate the emotional stress by interfering with the digestion of the precursors of norepinephrine and serotonin.



Emotional NV's are the same as the stomach's NV's. Folic acid, vitamin B-6, and pantothenic acid are useful in neutralizing the recurrence of the need to treat these points.

FIGURE 3

Similarly, if a person has been under or is under considerable stress, the increased need for nutrients created by the stress can interfere with the conversion of the amino acids to neurotransmitters, due to the restriction of one or more of the vitamin and mineral cofactors which are necessary for this conversion. This can be due to either over-utilization of the nutrients or under-digestion and decreased absorption due to stress-induced gastrointestinal irritation. Interestingly, both norepinephrine and serotonin depend for their activation

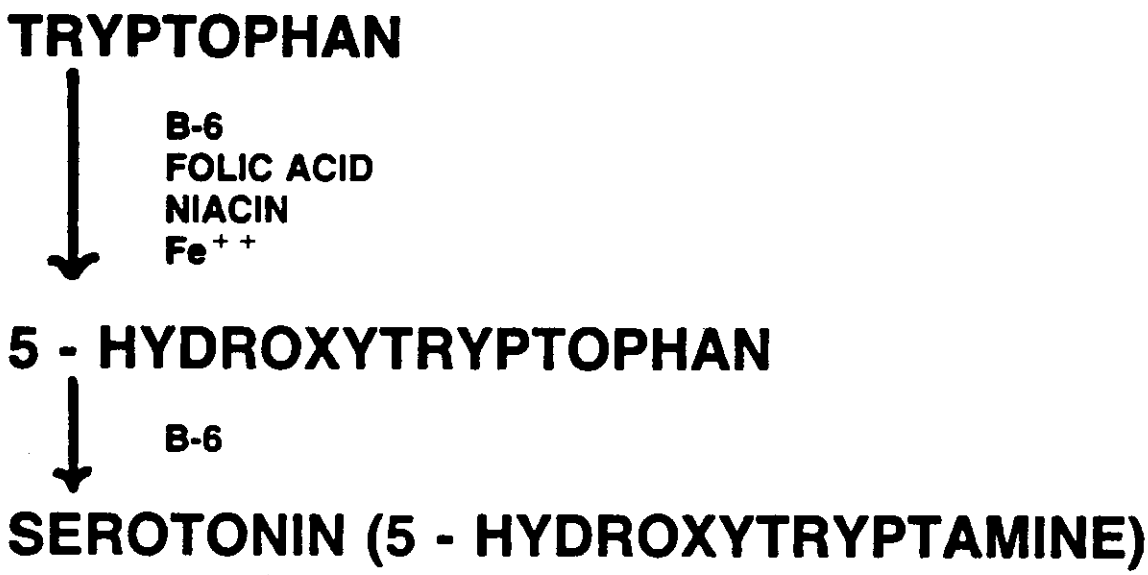


FIGURE 5a

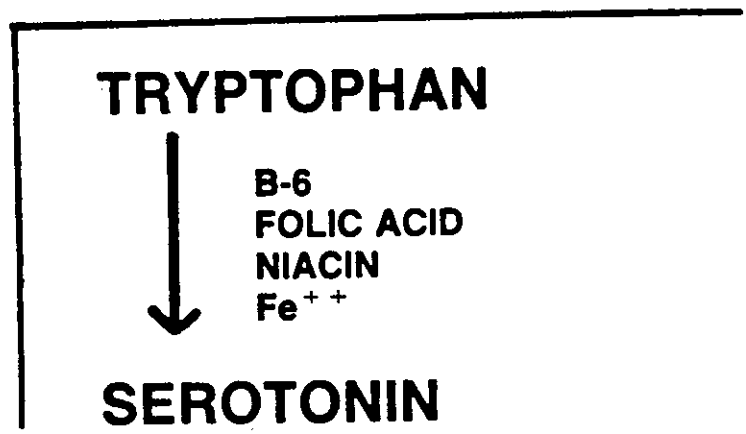


FIGURE 5b

The most important of these are folic acid and vitamin B-6 (pyridoxine). Since 1982, a year and a half prior to our learning about their neurotransmitter connections, we knew from empirical observations that folic acid and vitamin B-6 were important in neutralizing the recurrence of many emotional recall and emotional neurovascular patterns. In many patients, placing one of these nutrients in the mouth would block both the emotional recall pattern and therapy localization to the emotional neurovascular reflex points. (Figure 3.)

Goodheart[3] has shown choline to be useful in many patients by using it to strengthen weak muscles when tasting whole adrenal tissue induces general muscle weakness. We also find choline useful in this regard, and interpret the situation where whole adrenal weakens as an overly sympathetic dominant patient who needs parasympathetic (choline-acetylcholine) support. We also see patients who weaken on choline and subsequently strengthen on whole adrenal tissue. We interpret this pattern as an overly parasympathetic dominant patient who needs sympathetic support.

We have found pantothenic acid to be in the same category as folic acid and vitamin B-6 in blocking the emotional recall reaction. These substances break the viscious cycle precipitated by the stress and make coping so much easier, as well as block the recurrence of associated neuromusculoskeletal problems. Only in difficult cases do we resort to the amino acids themselves. As long as the patient is eating and digesting adequate amounts of protein, we are more concerned with manipulating the general diet or supplementing the appropriate vitamins or minerals.

One can see how the admonition to the stressed patient of, "You must eat at times like this to keep up your strength" takes on an entirely different meaning from the perspective of neurotransmitters and our recent understanding of how they affect how we feel. But just eating alone is inadequate. One must eat the appropriate types of foods to affect neurotransmitter activity.[4] It has been shown that eating carbohydrates (sugars and starches) increases serotonin activity

carbohydrate diet will increase serotonin ("calmer down") levels in the brain while lowering norepinephrine levels and aggravate the low depressive symptoms.

One might say that a good balanced diet is the best answer, but this is a "cop out" if one is armed with the knowledge regarding neurotransmitters and how diet affects their balance. We are not confronted with people in perfect balance, therefore, we must treat an imbalance with an imbalance. Using muscle testing as functional neurological evaluation gives us the tools to determine each specific person's needs. The following case history demonstrates the importance of this fact in the case of a patient with acetylcholine problems.

Case History: A 32-year old woman with a long history of low back pain had been stable for over one year when she presented on a Monday with severe neck pain and limitation of cervical motion. Two months previously, she had been restricted from eating eggs (it turns out unwisely) by her medical physician, due to a mildly elevated serum cholesterol (303mg/dl). Eggs are a good source of choline, and as it turned out, the only major source of choline in this patient's diet. She was treated for the neck pain which was primarily a right levator scapula muscle weakness with compensatory "spasm" on the left. She was 60% improved in the office, and gradually improved with no further treatment each day until Saturday morning, when she awakened fully recovered from the neck problem.

or painful problem is dependent on a clearly functioning nervous system, which is dependent on nutrient intake, which directly affects the ability of the brain to make the chemicals necessary to communicate with itself and other parts of the body.

The basis for chiropractic is to restore our patients to as close to optimal neurological function as is possible. This means that we must remove obstacles to normal nerve function, whether they be subluxations of the spine and extremities, other structural blockages and reflex points, emotional recall patterns, or whatever. But all of our procedures to remove the interference with normal nerve function have been based on the assumption that there is enough "water in the hose," that is, adequate neurotransmitter activity.

The number of our patients who are or have been depressed (not to mention other symptoms attributable to neurotransmitter imbalances or deficiencies) testifies to the need for our awareness of nutritional factors relating to the transmission of the nerve impulses. To achieve optimal health for our patients, we must not only correct the structural faults which are at the foundation of our profession, but we must evaluate and correct those factors which affect the transmission of the nerve impulses. This means we must evaluate our patients' blood and urine and employ other tests as indicators of nutritional imbalance which can effect neurotransmitters, hence the very nerve function we are working with on a daily basis. And based on these evaluations we must supply nutritional supplementation and counselling for our patients to enhance our effectiveness.

MOLYBDENUM FOR CANDIDA ALBICANS PATIENTS AND OTHER PROBLEMS

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

ABSTRACT: The essential trace element, molybdenum, is discussed in relation to its various metabolic pathways. Diagnostic approaches for molybdenum include applied kinesiological procedures based on strong muscles weakening when a patient sniffs aldehydes, ammonia, or Clorox, or tastes sulfur-containing amino acids. Other patterns indicating a need for molybdenum are the same as would be seen in a need for iron and/or an excess of copper. Each of these metabolic pathways are shown to be important in the problems of the Candida albicans patient, as well as other patients. Protocols for supplementation and natural sources of molybdenum are given.

INTRODUCTION

Molybdenum is an essential trace element in human nutrition which is understood about as well as it is pronounced.* In fact, there is no laboratory testing which has been standardized for the evaluation of molybdenum. Although it has been measured in both blood and hair, the normal values for these tests have yet to be established, and although it is accepted as an essential nutrient for humans, there has yet to be a recommended daily allowance or minimum daily requirement

*pronunciation of molybdenum: mo-lib'dĕ-num

MOLYBDENUM AND ALDEHYDES

Chemical aldehydes are best known as fragrances. The body also produces various aldehydes as part of its normal metabolic pathways. One pathway in the metabolism of the essential amino acid, threonine, is its conversion into acetaldehyde and then on into acetic acid for eventual production of acetyl coenzyme A. (See Figure 1.) Ethanol, or drinking alcohol, is also processed to acetaldehyde. The build-up of aldehydes can be very toxic to the body's tissues. Therefore, the body has an enzyme which breaks down the aldehydes to less toxic substances. This enzyme is called aldehyde oxidase, or sometimes, aldehyde dehydrogenase. Aldehydes encountered dietarily or environmentally or produced in the body must be handled by aldehyde oxidase metabolic pathways.

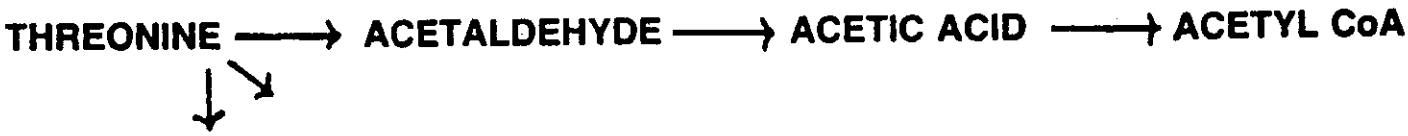


FIGURE 1

Acetaldehyde is a particularly toxic substance which, in addition to being produced from threonine and ethanol, is a product of the metabolism (i.e., fermentation) of carbohydrate in yeast - hence, the Candida connection. Acetaldehyde is thought to be the major source of tissue damage in alcoholics rather than ethanol itself. The conversion of acetaldehyde

enzyme is vitamin B-1 (thiamine) dependent. We had performed the functional blood test for red blood cell transketolase on a number of patients and found some of them to show a need for vitamin B-1. Knowing that nail polish remover is primarily acetone, we attempted olfactory challenging to observe the results. Eventually, we switched to using pure acetone for olfactory challenging because of the sporadic results we observed from using the nail polish remover.

A close look at the contents of the Cutex bottle revealed that besides acetone, a fragrance and a food color had been added. It is useful here to mention that when testing with sniffing acetone, a strengthening of weak muscles or a weakening of strong muscles has been demonstrated to be associated with a need for vitamin B-1. The proper B-1 tablet, either high synthetic dose or low natural source, or occasionally both, when insalivated, will block the weakening response to sniffing acetone in those patients who show it, and will likewise mimic the strengthening response in that group of patients.

Although we still occasionally use the nail polish remover as a screening test for acetone and B-1 involvement, we now know that many of our original sporadic observations were due to the presence of a fragrance (i.e., an aldehyde) in the product. We obtained a source of benzylaldehyde, which is the smell of almonds and quite pleasant. In the meantime, we had communicated with Mowles and found out about his results with formaldehyde. In an effort to find a less offensive odor,

A positive test is one in which the weakening response to aldehyde sniffing is negated by the nutrient substance in the mouth. Sometimes, one of the nutrients will strengthen a weak muscle but will not negate the aldehyde sniff response. We only supplement the nutrient which both strengthens the weak muscle and negates the weakening response of the aldehyde sniff.

Patients with aldehyde sensitivity will demonstrate a number of symptoms. The most severe cases we have observed are those patients with systemic Candida albicans allergy syndrome. Many of these patients are incredibly sensitive to any type of fragrance. This becomes easily understood in light of the idea that the Candida in the G.I. tract, vagina, or elsewhere in the body is giving off acetaldehyde as part of its normal metabolism. The excess stress which this must put on the aldehyde oxidase enzyme systems in the body's tissues leaves them unable to keep up with the extra demand. Supplementation with molybdenum and/or niacinamide, riboflavin, and iron will improve the patient's ability to handle the Candida generated aldehydes, as well as those encoutered in the environment.

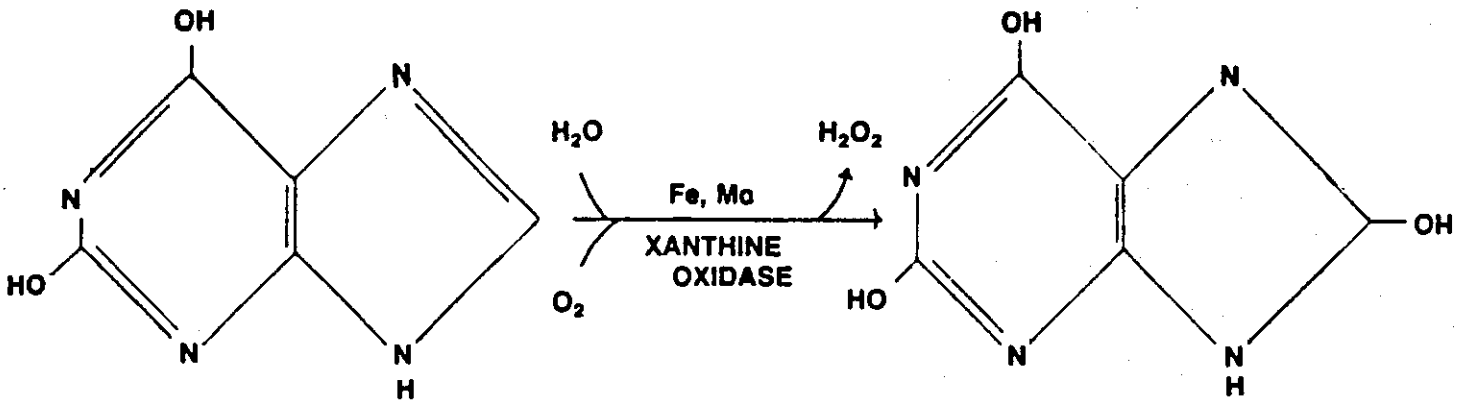
When an aldehyde sensitivity exists, there is considerable tissue irritation due to the buildup of these substances. It appears from our clinical observations that some patients fall into a viscious cycle of aldehyde sensitivity where there is a depletion of one or more of the aldehyde oxidase related nutrients whih leads to an increased tissue irritation from Candida produced acetaldehyde. This tissue irritation lowers tissue resistance which sets up vulnerability to future

related in most patients, but the most dramatic responses from controlling aldehyde sensitivity have been in our Candida albicans patients, on whom everything else had been tried. The addition of molybdenum, based on aldehyde olfactory sensitivity muscle testing has resulted in turnarounds in our most difficult Candida albicans patients. The most noticeable changes are seen in the sore, achy, sluggish, "flu-type" symptoms of which many Candida patients complain. The energy returns, the generalized musculoskeletal achiness improves, and mental sluggishness disappear, and sinus and nasal congestion clears up. Based on these observations on difficult patients, we now screen every Candida albicans patient with the aldehyde sniffing test and take appropriate measures sooner rather than later in these patients. The addition of molybdenum, in particular, has been a great boon to us in handling the Candida patient, and getting them out of whatever rut or viscious cycle the aldehyde sensitivity has put them into. Molybdenum is also important in caring for a number of other metabolic problems associated with the Candida patient, as well as other patients.

MOLYBDENUM AND AMMONIA METABOLISM

Mowles' study included olfactory challenging with ammonia in addition to formaldehyde.[4] There are many facets to olfactory challenging with ammonia which have been discussed by this author in his seminars and are the source of future papers.[7] Mowles chose to focus his study on the relationship of ammonia weakening response to the strengthening response from molybdenum. In twelve patient trails where the patient

the observation that sniffing ammonia causes a weakening of some of these patient's muscles. In a number of patients weakening upon sniffing ammonia, the weakening will be neutralized by the insalization of molybdenum, presumably due to its relationship to xanthine oxidase. (Occasionally iron will also neutralize the ammonia weakening effect, and there are many other factors which must be considered in this olfactory ammonia challenge.)



XANTHINE (ENOL FORM)

URIC ACID (ENOL FORM)

FIGURE 4

Because there are so many other factors related to the ammonia olfactory challenge, it is convenient in ammonia sensitive patients to use a muscle which is weak in the clear for screening for the appropriate nutrient(s) If molybdenum strengthens the muscle which is weak in the clear, it is held in the mouth while the ammonia is sniffed. Supplementation of molybdenum (or any other substance) is based on the nutrient

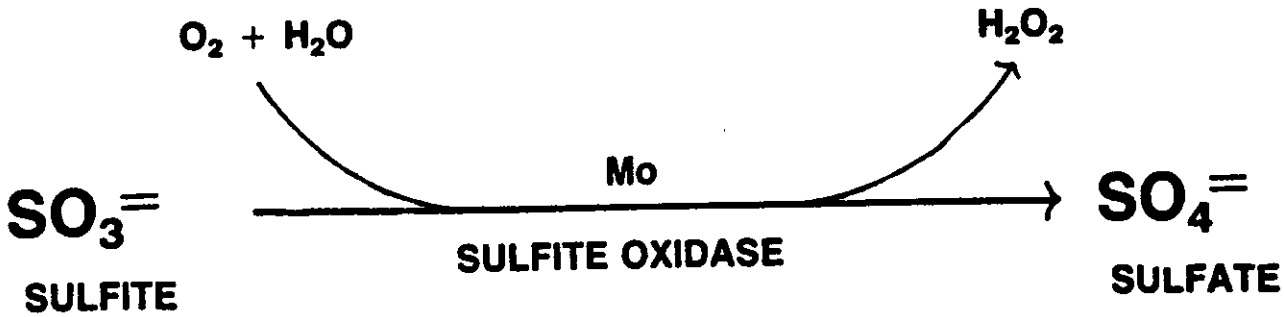


FIGURE 5

Sulfites are used by the food industry as preservatives. Metabisulfite is used to keep fruits and vegetables looking fresh in grocery stores and restaurant salad bars. This substance has created severe reactions in patients who are sensitive to it, and has even resulted in a few deaths. Asthmatics must be especially wary of this substance since it will trigger an almost immediate severe asthma attack. One of our sulfite-sensitive patients thought he had been poisoned and had such dyspnea that he felt like he was dying, collapsing on a restaurant table following eating of a salad from a salad bar in which a sulfite preservative was used. He eventually pulled out of the asthma attack, but now he asks first about the use of sulfites when eating out. Another patient in our practice simply avoids eating out altogether because of the fear of ingesting sulfites. Her reaction is extreme nasal and nasopharyngeal congestion, tachycardia and arrythmia, and complete exhaustion from sulfite contact. These patients will have weak muscles which respond to molybdenum, although their sensitivity to sulfites may or may not be affected by this supplementation, and avoidance of sulfite contact is usually recommended.

What we have observed clinically does not exactly fit what we would expect to see based on the metabolism pathways in Figure 6. A number of patients will demonstrate dramatic weakening of all of their muscles when ingesting methionine which will be negated by the simultaneous insalivation of molybdenum. In other words, it appears that in a molybdenum requirement, the body's ability to metabolize methionine is blocked. This may be seen in amino acid profiles by an elevated methionine.[1] This would make sense in the case of a magnesium deficiency as can be seen in Figure 6. But in the case of a need for molybdenum we would also expect to find a weakening from cysteine since the role of molybdenum is played beyond cysteine in the metabolic flow chart. We only occasionally see weakening from cysteine in these patients. We occasionally see strengthening from cysteine or no response from cysteine in these patients, but we are surprised at the infrequency with which we observe cysteine to cause weakness in these patients.

Some of these patients will show a strengthening response to taurine. Taurine has been discussed in an earlier paper by this author in its function as a free radical scavenger for the free radical OCl^- (hypochlorite ion or hypochlorous acid.)[6] In light of a molybdenum requirement, we have observed some patients who weaken on sniffing Clorox who are taurine responders and who also have this weakening response negated by molybdenum insalivation. Although this does not make sense based on the biochemistry presented, there must be some sort of negative

antioxidant depletion. Imagine the chemical stress a patient's tissues must be under when constantly exposed to free radicals, sulfites, acetaldehyde, and ammonia, all of which have accumulated in the tissues due to an unmet molybdenum requirement of the patient. Add to this the other factors involved in the Candida patient and it is easy to see why Candida can be difficult to treat, and why molybdenum can be such a great help in treating these patients.

MOLYBDENUM AND IRON METABOLISM

In 1981, this author reported the common observation of iron neutralizing retrograde weakening of patients' muscles.[12] Mowles observed that many retrograde type patients responded to molybdenum either in conjunction with or separately from iron.[4] Molybdenum plays a synergistic role with iron in the body. Clinically we are observing that any time one might use iron, one may also find a need for molybdenum.

Molybdenum and iron are related in a number of biological processes other than xanthine oxidase and aldehyde oxidase. In plants, there are iron-molybdenum dependent nitrogenase enzymes, for example, as well as several other areas which Mo and iron work together in important roles.[13] In humans, the highest concentrations of molybdenum are found in the liver, kidney (where xanthine oxidase is present) and the adrenal glands. High concentrations are also found in bone and skeletal muscle although, at present, Mo's functions in these tissues are unknown.[13]

We have found muscle testing indicators for Mo to be the same as for iron. This is especially true when a patient has

apparent functions paralleling those of iron may aid in producing a higher level of tissue oxygenation and thereby decrease the anaerobic environment in which Candida flourishes.

MOLYBDENUM AND COPPER

Molybdenum and copper are antagonists. Just as iron and molybdenum are synergists, and iron and copper are antagonists if they are not in balance with each other, so are molybdenum and copper antagonists. In patients who demonsrtate copper toxicity patterns, we have traditionally employed zinc and manganese to chelate the excess copper out of the tissues. Molybdenum is also useful for this purpose. Molybdenum antagonizes copper absorption and in a number of experiments in animals, copper and molybdenum have been shown to be directly antagonistic to each other.[2]

Mowles and I have observed an extremely high correlation in our practices of copper toxicity in patients with Candida albicans[5] as well as in women with menstrual and premenstrual disorders from functional hormonal imbalances.[14] It is possible that our geographical location (R.M. - Roanoke, Virginia, and W.S. - Chapel Hill, North Carolina) is responsible for this correlation. However, we find such a consistent pattern of low iron - high copper that we suspect copper's antagonism of iron and the resultant tendency toward an anaerobic environment for the yeast to grow as a major factor in these patients, as previously mentioned.

Occasionally we see a patient who has Candida and is low in copper. This seems contradictory to our other findings

The length of time to supplement a nutrient can often be based on clinical judgment and symptom response. In the case of molybdenum (and a number of other nutrients, especially those which have a relationship to red blood cells), however, we always maintain supplementation for a period of at least four months. This is based on the suggestion of Dr. George Miroff[15] who recommends that any nutrient which is associated with hematopoiesis be taken for a long enough period of time that each RBC in the body gets its full share. In other words, since the life of a RBC is 120 days, it is necessary to take these nutrients for at least 120 days to insure that the entire blood supply has had the advantage of this nutrient.

We have observed a number of patients who took molybdenum (or other nutrients which aid in RBC production and/or are taken up by the RBCs only during hematopoiesis) for one or two months and became asymptomatic, only to have the symptoms return one or two months later after stopping the supplement. Starting at 300 mcg and gradually reducing the dosage over four months time period has proven successful in our practice. In some difficult patients, we continue supplementation as long as they are symptomatic at a 100 mcg to 200 mcg level.

It is important to note that in cattle, molybdenum excess has been shown to decrease fetal growth.[1] Although no studies have indicated this in humans, we usually stop molybdenum supplementation in our pregnant patients, just to be on the safe side. If the obvious need for molybdenum returns, we will again supplement this nutrient, but at 100 mcg or less as a

SUMMARY OF MAJOR CLINICAL INDICATORS FOR MOLYBDENUM

1. muscles weaken on sniffing an aldehyde (e.g., acetaldehyde, formaldehyde, benzylaldehyde, etc.) and insalivation of Mo neutralizes this weakness.
2. muscles weaken on sniffing Clorox (hypochlorite) and/or tasting methionine and/or cysteine and insalivation of Mo neutralizes this weakness
3. muscles weaken on sniffing ammonia and insalivation of Mo neutralizes this weakness
4. whenever iron is indicated, Mo might also be indicated, such as in: aerobic testing muscle weakness patterns
   retrograde position weakness patterns
   low hemoglobin, RBC count, hematocrit or MCH
5. whenever copper causes muscle weakness, Mo may be necessary to chelate out excess copper.

Also discussed: If strong muscles weaken or weak muscles strengthen on sniffing acetones, this indicates a need for B-1.

[13]Rajagopalan, K.V. Molybdenum.in Biochemistry of the Essential Ultratrace Elements. ed. by Earl Frieden. New York: Plenum Press, 1984.

[14]Mowles, Richard, A Clinical Investigation of Pre-menstrual Syndrome and the Correlation with Copper, I.C.A.K. Collected Papers, Summer, 1983.

[15]Schmitt, Walter H., Jr., Muscle Testing as Functional Neurology. I.C.A.K. Collected Papers, January, 1986.

[16]Miroff, George, director Monroe Medical Research Laboratories, Southfield, N.Y. personal communication, 1984.

HOLOGRAPHIC MEMORY AS A REASON FOR THE DESIGN OF THE NERVOUS SYSTEM

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

Abstract: The use of the holographic memory theory as a possible explaination for the layout of the nervous system. The right side of the brain controls the left side of the body, and the left side of the brain controls the right side of the body.

To prepare for the task of teaching for the third year, I was reading the teachers manual. One of Dr. Goodheart's statements stuck in my mind. It was a very simple statement, "WHY IS THAT"?

This thought of "Why is that" kept coming back when I thought of the brain and nervous system. Why is that, our nervous system is laid out in the manner that it is? By this I mean why is it that the right side of the brain runs the left side of the body and visa versa. Could this set up happen strictly by chance and if so why? Or perhaps was there a greater design for this intermixing of neural information.

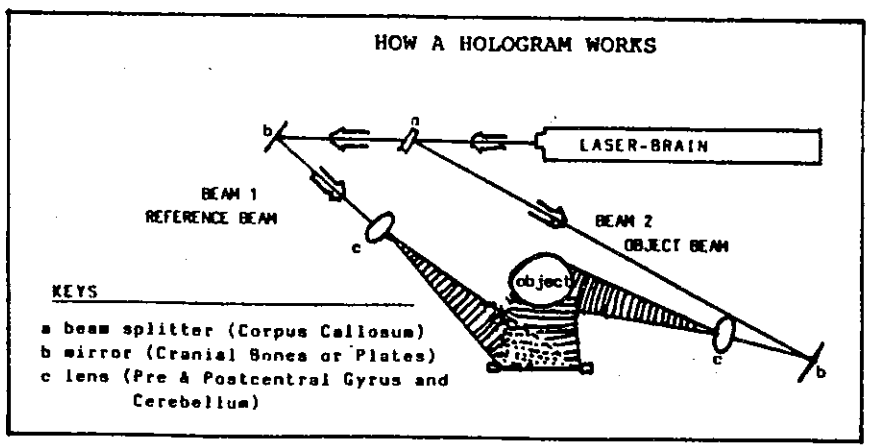
If we were to design the nervous system with what knowledge we have today would we have cross wired the system so that the right would run the left and the left would run the right? It seems to me a great waste to do it this way if it only happened by chance and not by design. Would it not have been easier, more conservative of energy, materials, and time just to have the right run the right and the left run the left? Or as I stated before could it have a design. And if it has this design "why is that" it is designed in this manner? Could it be there just to make it more difficult to learn about, more mysterious? I think not!

This interference pattern is one of the main bases for the formation of a hologram. Obviously this can be done by any force that resonates or vibrates or travels in a wave form, that means a hologram can be formed by light waves, sound waves etc.

In my last paper I used the concept of the resonant or vibratory pattern to explain surrogate testing by means of holographic memory.[6]

Now let us return to the concept of light holography and our nervous system. I will use the diagram showing the light source (corherent light) a laser beam will create our light source, then we take a splitter lens and create two beams of light which are inturn directed by mirrors from two different directions. One beam falls on the object called aptly the "object beam". The other goes directly to the photographic plate called the "reference beam".[7]

FIGURE 2



Since light travels in a wave pattern and since the angles of these two light sources meet at different angles an interference pattern is set up which allows the photographic plate to record the hologramic three dimentional image. The image has both height, width, and most-important depth which in the case of holography is a parallex quality.

As you can "see" the visual field from both eyes are split, this could be likened to the splitter lens that forms a hologram. Remember now that the lateral visual field from both eyes go directly back to its own side of the brain. This is one mirror to form the "reference beam". The medial, visual field goes back to the visual center by way of the optic chisma (the second mirror) forming the "object beam" so to speak. The fibers from the medial field cross over to the opposite side of the brain and travel back to the occipital region to join with the lateral visual field from the same side. This would gives us the interference pattern we need to form the hologram.[9] Refere back to Figure 3.

This would give the visual appratus the ability to record images similar to that of the photographic holograms with height, width, depth and the (parallex) view from two different points.

The auditory system is wired in a similar fashion in this instance the sound wave will be used to form the holoramic image.

Sound waves stimulate the tympanic membrane of the right ear, which are converted into vibratory pattern which in turn are converted to a hydrodynamic wave form in the cochlea of the ear. This in turn is converted to neural signals that travel from the cochlea by way of the spiral ganglion to the cochlea nerve. These tracts some of which synapse in the ventral chochlear nucleus others in the dorsal cochlear nucleus cross over to the opposite side and others travel to the same side of the brain in the superior olivary complex and synapse again sending branches across to the opposite side of the brain. The fiber synapse again in the medial geniculate body and finally arrive at the cochlea and acoustic area of the cortex.

Sound has a number of different qualities of pitch, timber, intensity (loudness). Quality or frequency is pitch, purity is timber, and intensity is loudness.

As you can note the three qualities of sound are similar to those of light formed hologram, with the three dimentional qualities of hight-intensity, width-frequency, and depth-pitch. Again giving us the ability to store information in the holographic record (memory).

This concept that the nervous system is laid out to facilitate holographic recording seems to follow through for all our senses and also for all our motor function since the tracts cross over and travel to the opposite side of the body, with some tracts going to the same side.

In chiropractic one of our tenents is that structure and function go hand and hand; that the way the nervous system is laid out will influence the way it will function. The function of the nervous system has created the need for the way it it structured (wired).

This could be carried back to my original idea that our nervous system cross over was facilitated because of the need of the nervous system to store it information in holographic engrams or encodings. Could explain the sturcture of the nervous system left side of the brain controlling the right side of the body and the right side of the brain controlling the left side of the body.

To have a hologram we must create an interference pattern to record this information and to do this we must have two different sources of information a "reference beam" and a "object beam". This would be similar to the brain sending or receiving signals some from the same side and others from the opposite side of the body.

POSITIVE ZINC TALLY AND THE FREQUENCY OF CRANIAL FAULTS AND TEMPORAL MANDIBULAR JOINT INVOLVEMENT

by: Allan Zatkin, D.C.

ABSTRACT: This paper is a retrospective study comparing patient's response to Zinc Tally to cranial fault and temporal mandibular joint involvement.

Over the past few months, we have been chronicling patients' responses to the Zinc Tally test. The Zinc Tally, by Metagenics, [1] is a saturated solution of zinc sulfate-hydrated as septahydrate which produces a varied response in its perception when insalivated. This response is governed by the balance of zinc in the patient's system. The more zinc available in the patient, the stronger the taste of the Zinc Tally solution. The suggested system of scoring the test is on a one through four basis. A grade of one is noted when the patient notices no specific taste or other sensation even after the solution has been kept in the mouth for about ten seconds. A grade of two is recorded when no immediate taste is noted but after a few seconds a slight taste variously described as "dry, mineral, furry or sweet" develops. A grade of three is when a definite though not strongly unpleaseant taste is noted almost immediately and tends to intensify with time, and a grade of four is when a strong and unpleasant taste is noted immediately.[2]

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1. Metagenics, Laguna Hills, California
2. Metagenics, Laguna Hills, California. Recommended evaluation scale for testing Zinc Tally.

Of 16 patients scoring a grade of one, six positive cranial faults and two TMJ involvements were noted. Of 32 patients scoring a grade of two, eleven showed positive cranial faults and seven TMJ involvements were noted. of 31 patients scoring a grade of three, five positive cranial faults and five positive TMJ involvements were noted. Of 23 patients scoring a grade of four, there were no cranial faults and four positive TMJ involvements.

CONCLUSIONS:

The results of the study generally speak for themselves. TMJ involvement was noted on all scores while other cranial involvement was noted only on scores 1-3. The frequency of these faults was not as high as I originally expected on those patients scoring a one. An interesting future observation would be to examine patients scoring a one in their body distortion posture to see if a higher frequency is actually present.

References:

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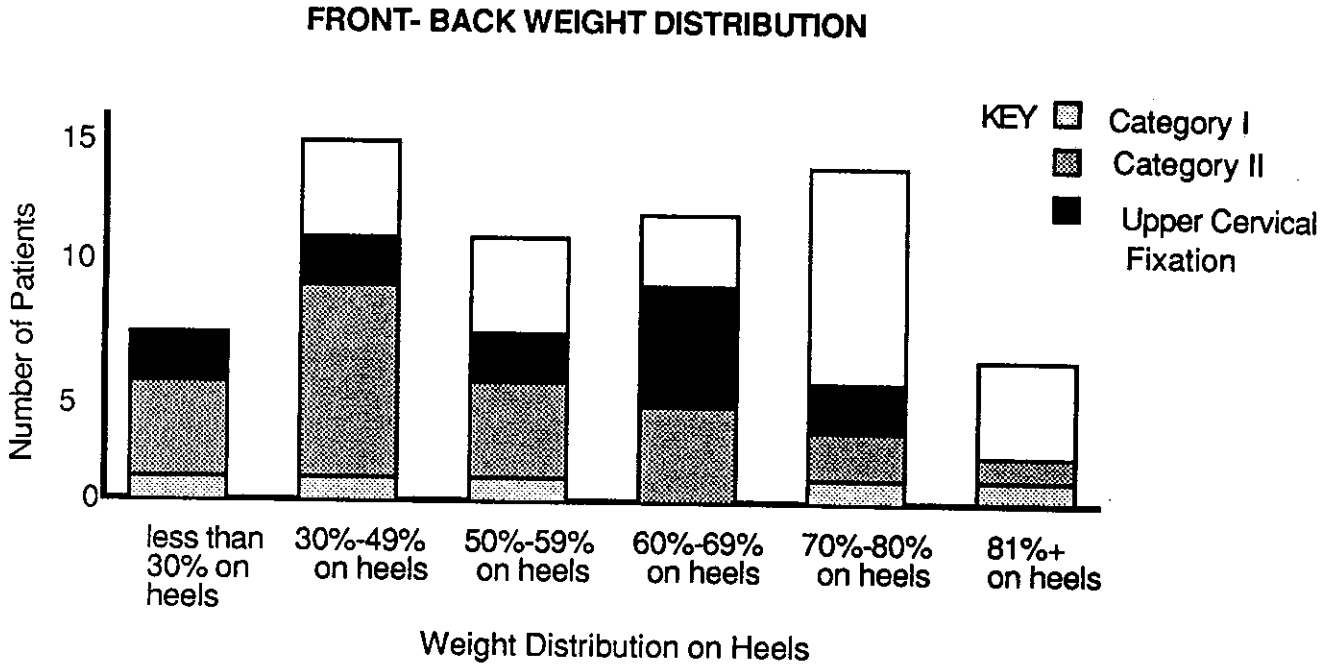
WEIGHT BALANCE AND STRUCTURAL INVOLVEMENT

by: Allan Zatkin, D.C.

ABSTRACT: This study compares variances in weight balance and the evidence of pelvic categories one and two as well as upper cervical fixations.

As a part of our initial work-up done on all new patients in our office, we measure height, weight and weight balance. We use a standard doctor's scale for height and weight and we use a computerized weight balance scale manufactured by Chirotron, Inc., Seattle, Washington, to measure weight balance from left to rightt and from front to back. For determining normals, we say that if a person is 61 inches tall, he or she should weigh 110 lbs. To this base we add five pounds for every inch increase. We also take into account a 10-15% variance for muscular development, age and other factors. Experience has shown that normal values for left/right and front/back are as follows: a right handed person should carry 5-10 lbs. more on the right foot. This imbalance is due to the fact that the liver being a solid organ is on the right side of the body and the stomach being a hollow organ is on the left side. Also included in this imbalance is general muscular development on the dominant side. A left handed person should weigh 0-15 lbs. more on the right foot for the same reasons, dominant side muscular development and stomach/liver relationship. Experience has also shown that regardless of handedness, every person's weight should be distributed such that 75% of the total body weight is on the back of the feet.



CONCLUSIONS:

The most interesting conclusion that I see is that while these structural involvements were preset in most weight categories, the percentage was lower in what we considered normal weight balance. Those who carried 1-10 lbs. heavier on the left showed a 60% frequency while the 0-10 lbs. on the right (normal) showed a 47% frequency and the 11-12 lbs. on the right category showed a 90% frequency of involvement. The front/back graph shows that the more the patient leans forward, the more frequent are his or her major structural involvements.

References:
Goldwait, Brown, Swain and Kuhns, Essentials of Body Mechanics, 4th Edition, J.N. Lippincott, 1945.

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