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PAST CHAIRMAN I.C.A.K.

INTRODUCTION

By

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Past Chairman

This sixteenth collection of papers by the members of the International College of Applied Kinesiology represents 35 papers written by 18 authors.

These papers will be presented by their authors to the general membership at the Winter meeting to be held in San Diego on Nov. 30th, Dec. 1st and 2nd, 1983. The authors welcome comments and further ideas on their findings either in San Diego or you may write them directly as their addresses are included in the Table of Contents.

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

The papers are being mailed out to the members well in advance of the San Diego 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.

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* - Diplomate

MANUAL MUSCLE TESTING AND CYBEX MACHINE MUSCLE TESTING, A SEARCH FOR A CORRELATION

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I. Abstract

A comparison of manual muscle testing and Cybex machine muscle testing is presented. Various jaw positions were tested for their ability to produce muscle weakening on manual muscle testing. The same set of jaw positions and muscles were evaluated using the Cybex machine. The degree of correlation between the results of the two types of testing was evaluated. A control group (subjects who showed no manual muscle test weakening with the various jaw positions) performed the same battery of manual and Cybex tests. This data was used to establish confidence levels for the correlations found in the experimental group. The results of the two types of muscle testing were found to be statistically independent. The implications of this finding are discussed. A neurologic model for "strong" and "weak" muscle tests is presented.

II. Introduction

Since about 1975 several investigators[1,2,3,4] have tried to duplicate and objectively document the frequently observed changes in strength of the manual muscle test[5] through the use of the Cybex II Dynamometer.[6,7] These experiments have led to frustration and argument, because it has been found repeatedly that these two types of testing produce conflicting data. The closest correlation found to date is 40 percent[1].

Blaich[1] found a 40 percent correlation between Cybex and manual muscle tests of the hamstring muscle group in evaluating 12 patients while they held full inspiration and then full expiration. No control group was used. Nicholas, et al.[2,3] reports extensive use of manual muscle tests in his clinic to evaluate athletic injuries. But he found a poor correlation between Cybex and manual muscle test results when evaluating muscle inhibition after dermatomal skin stimulation. Smith[4] claims that a "very good strength correlation is shown" between Cybex and manual muscle tests of professional football players' deltoid muscles when evaluating the relative benefits of various mouth guards. But Smith's published data is incomplete, does not support the suggested 90 percent correlation, and reflects a very small sample size (nine people). Again, no control group was used.

In a related study, Triano[8] suggests that there might be a relationship between the timing of the EMG activity of a muscle and whether that muscle would be evaluated as strong or weak by a manual muscle test. The current investigation approached this theory only indirectly, by measuring EMG activity during the Cybex muscle tests. These measurements showed no correlation with the manual muscle test results, but the concept of a "weak" muscle actually being a "late" muscle merits further research. In another related study, Williams, et al.[9] documented the correlation between changes in jaw position and changes in Cybex muscle test results. Although this study did not investigate the manual muscle test, it is supportive of the experimental design of the current experiment.

During a manual muscle test, the operator judges whether the muscle is "strong" or "weak". This determination is based on several factors[5,10](the magnitude of the test force, the duration of the force, the presence of locking[11], body language of weakness).[12] During a Cybex II machine muscle test, the torque which the subject produces through a range of motion is recorded. The "strength" of the Cybex test has been evaluated by measuring the maximum torque value produced during each contraction effort. The purpose of the current study was to investigate the possibility of there being a useful degree of correlation between additional parameters of the Cybex muscle test and the results of manual muscle tests.

III. Materials and Methods

Prospective subjects for this study were first evaluated by manual muscle testing for the effects of various jaw positions on the "strength" of three muscles: left deltoid, right biceps brachii, right rectus femoris. (The choice of left or right was determined by positioning limitations of the Cybex equipment). Each muscle was tested while the subject held his jaw in each of four positions: relaxed, closed lightly, mandible to the left and closed lightly, mandible to the right and closed lightly. Each muscle and jaw position combination was tested three times. A prospective subject and a particular muscle on that subject were assigned to the control group if all the tests of that muscle were found to be "strong" and unaffected by jaw position. A prospective subject and a particular muscle on that subject were assigned to the experimental group if a particular jaw position consistently showed a weakening of that muscle relative to the other jaw positions. Twenty subjects were selected, ten for the control group and ten for the experimental group.

These same muscle and jaw position combinations were then evaluated using the Cybex Dynamometer, and the results were recorded on a Physiograph[13] six-channel strip chart recorder. The torque produced by the subject was recorded on channel #1. The height of this curve was measured at two points: (1) at the maximum value, which occurred near the

beginning of the range of motion, and (2) after 90 degrees of rotation through the range of motion. (Some subjects were unable to produce 90 degrees of range of motion. In these cases this measurement was made at 45 or 60 degrees.) The torque produced during each test was electronically integrated and recorded on the Physiograph channel #2. This torque integral curve, which represents the sum of the torque produced during the contraction effort (or, the total of the accumulated effort during that test), was measured at two points: (1) after 90 degrees of range of motion, and (2) at the end of the contraction effort (which was defined as the point at which the torque curve bottomed out after returning to the "O" baseline).

The electrical activity in the muscle being tested by the Cybex machine was recorded from surface electrodes placed on the skin over the belly of the muscle in question. This raw EMG signal was filterd and recorded on the Physiograph channel #3. The raw EMG signal was electronically manipulated to produce an EMG envelope. The height of this curve represents the quantity of EMG activity occurring at any moment during the test. This EMG envelope curve was recorded on the Physiograph channel #4 and was measured at three points: (1) at the moment of maximum torque, (2) after 90 degrees of range of motion, and (3) at the end of the contraction effort. The raw EMG signal was also electronically integrated. The height of this curve represents the cumulative summation of

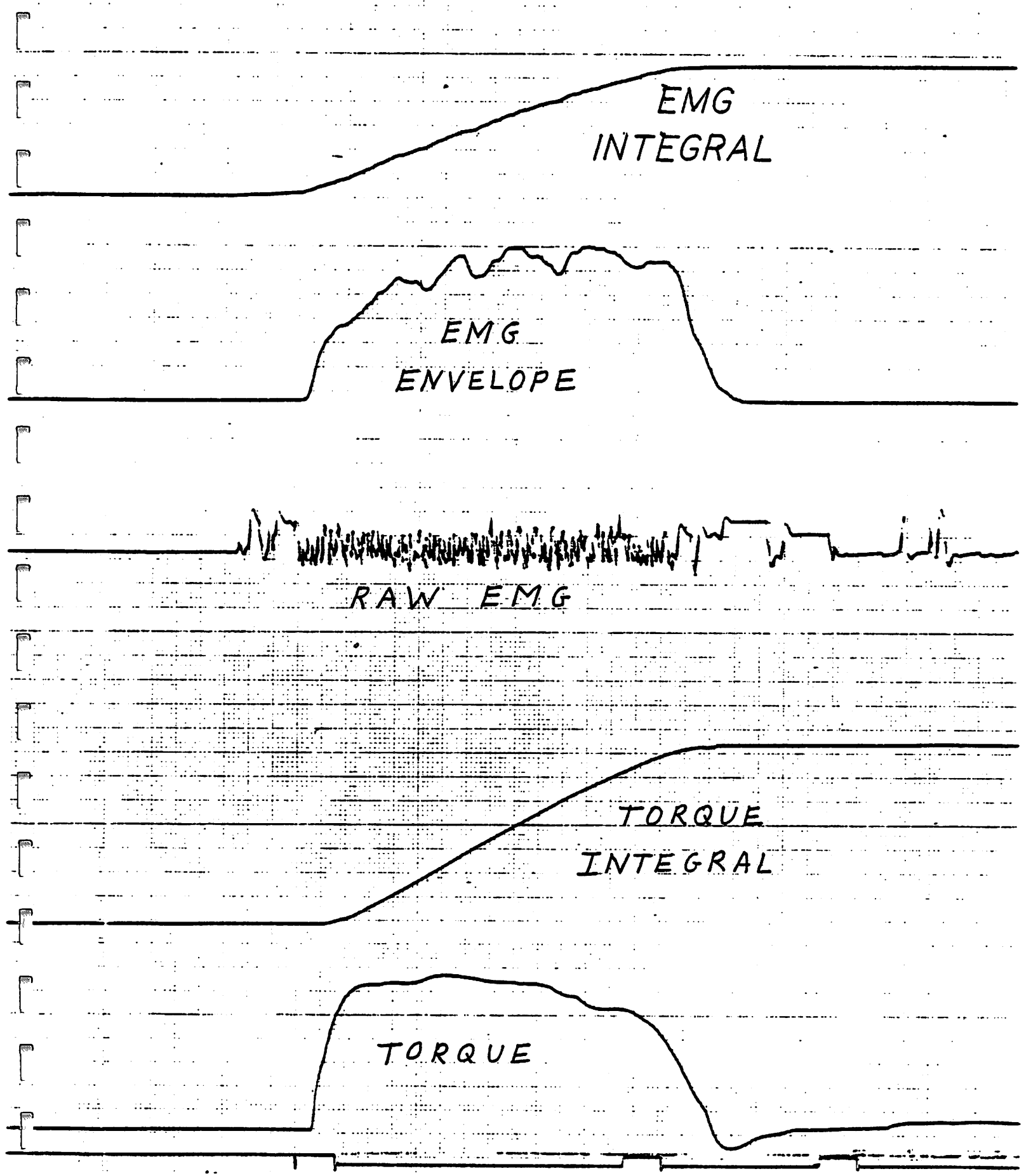
the EMG activity occurring during a test. This EMG integral curve was recorded on the Physiograph channel #5 and was measured at three points: (1) at the moment of maximum torque, (2) after 90 degrees of range of motion, and (3) at the end of the contraction effort.

Also evaluated during each Cybex test was the duration of the contraction effort. This was inferred by measuring the duration of the torque curve from the beginning of motion (which was recorded on the Physiograph by an event marker) to the end of the contraction effort.

Figure 1 shows a typical recording made on the Physiograph during a single Cybex muscle test. These graphs and their measured parameters were evaluated for correlation with the results of the manual muscle tests.

During the Cybex muscle testing phase of the experiment, each of the 20 subjects performed their appropriate muscle test with their jaw in each of the four positions. This sequence of tests was repeated five times. The graph of each of the contraction efforts was recorded and measured for the 11 parameters. For each parameter, the measured value was averaged over the five trials which had the jaw in the same position. This averaging helped to reduce the effects of variations in subject motivation and fatigue. Thus, a data sheet was produced for each subject which showed a performance

FIGURE 1



value for each of the 11 parameters for each of the four jaw positions.

The data sheet for each of the ten experimental subjects was scanned to determine which jaw position produced the lowest performance value for each parameter. It was then determined whether this "weakest" jaw position correlated with the "weak" jaw position found with manual muscle testing. Each of the 11 parameters was evaluated for its correlation with the manual muscle test results in the ten experimental subjects. Each parameter was given a degree of correlation rating as a fraction of ten, which was converted to a percentage.

The control group's data was used to determine which data from the experimental group could be considered significant. First, each control subject's Cybex graphs were measured, and the performance values were averaged and tabulated as for the experimental subjects. On the data sheet for each of the control subjects, the four performance values found under each parameter (one value for each jaw position) were averaged. The difference between this average and the lowest of the four performance values was expressed as a percentage of the average. Thus, a "percent variation" was calculated for each of the 11 parameters for each of the ten control subjects. Then, for each parameter, the "percent variation" values from the ten control subjects were averaged.

Thus, an "average percent variation" was calculated for each of the 11 parameters from the control group's Cybex test data. This normalization procedure permits comparison of the variation from the average for different groups of data without concern for the absolute values of the data.

The "average percent variation" values were then used to judge the significance of experimental group data. The data sheet for each of the experimental subjects was reviewed. For each of the 11 parameters, a "percent variation" was calculated as for the control subjects (i.e., the performance values for the four jaw positions were averaged; then the difference between this average and the lowest value was expresed as a percentage of the average). The "percent variation" for each parameter was then compared to the "average percent variation" found for that parameter from the control group data. Three categories of confidence were established that the "percent variation" found in a group of data for a particular parameter on the data sheet of an experimental subject can be considered significant relative to the control group:

Category I: Probably not significant -- if the "percent variation" for the experimental subject's data is <u>smaller</u> than the "average percent variation" for that parameter found in the control group.

Category II: Possibly significant -- if the "percent variation" for the experimental subject's data is larger than the "average percent variation" for that parameter found in the control group.

Category III: Probably significant -- if the "percent variation" for the experimental subject's data is larger than the "average percent variation" + (2 x the Standard Deviation) of the "percent variations" for that parameter found in the control group. This screening provides approximately a 98 percent confidence level that the data is significant. This calculation produces cut-off values which are two to three times the magnitude of the "average percent variation."

IV. Results

Table 1 lists the 11 parameters of the Cybex muscle tests that were evaluted for their degree of correlation with the manual muscle test results. The data represents the entire experimental group. If the control group data is not used to screen for probable significance or insignificance, the degrees of correlation shown in Column A are found.

Columns B and C represent two different levels of confidence that the degrees of correlation are based on data that is significant relative to the control group. Column B is based only on data that was judged "possibly significant" (see

above for explanation). Column C shows the degrees of correlation based only on data that was judged "probably signficant."

Table 1

	A	B	C
1. Torque, maximum value	40%	10%	0%
2. Torque, at 90° range of motion (ROM)	10%	0%	0%
3. Torque Integral, at 90° ROM	50%	0%	0%
4. Torque Integral, at end of contraction effort	50%	0%	0%
5. EMG Envelope, at moment of maximum torque	50%	30%	10%
6. EMG Envelope, at 90° ROM	10%	10%	10%
7. EMG Envelope, at end of contraction effort	40%	20%	0%
8. EMG Integral, at moment of maximum torque	50%	30%	10%
9. EMG Integral, at 90° ROM	20%	10%	0%
10. EMG Integral, at end of contraction effort	10%	10%	0%
11. Duration of contraction effort	10%	10%	10%

Among the ten experimental group subjects there was a total of 110 data groups (ten subjects, each with data for 11 parameters) which had the potential of showing a correlation with the manual muscle test results. Of these 110 data groups, 29 (26.4 percent) did show the correlation. This is very close to what would be expected to occur by chance alone (chance alone would predict a correlation in 25 percent of the data groups, or 27.5/110). Eliminating the data groups that were judged "probably insignificant" leaves only 35 data groups for evaluation for correlation. Of these 35 data groups, 13 (37.1 percent) did show positive correlation with the manual muscle test results. Chance alone would predict a correlation

in 8.75/35 data groups. Among the original 110 data groups, only eight were judged to be "probably significant". Four of these (50 percent) showed positive correlation with the manual muscle test results.

V. Conclusions

The results do not show a useful degree of correlation between any of the parameters evaluted during the Cybex machine muscle tests and the results of the manual muscle tests. The degrees of correlation found without regard to the control group are in line with the results of previous experiments. But it was found that use of the control group's data to establish levels of confidence that data being used from the experimental group was significant produced large reductions in the apparent degrees of correlation.

The conclusion was drawn that the manual muscle test and the Cybex machine muscle test are probably independent phenomena, at least as their results are currently being measured. Therefore, the Cybex Dynamometer is probably not a useful tool for future experiments designed to investigate and/or duplicate the manual muscle test.

VI. Discussion

The results of this experiment will probably produce one of two opposing impressions in the minds of the readers: (1) the manual muscle test has no objectively measurable basis, or (2) the objective bases of the manual muscle test simply do not show up in the Cybex muscle test. This dilemma can be confronted both philosophically (through discussion of ideas) and scientifically (through experimentation to challenge a hypothesis). Both approaches are necessary in any process aimed at establishing truth.

Future experiments designed to investigate and/or duplicate the manual muscle test need to be based on research into the physiology and biomechanics of the many types of muscle testing. The question must be addressed: What is the difference between the Cybex muscle test and the manual muscle test that does not allow their results to correlate? In the following discussion we will attempt to present some physiology, some biomechanics, some theories, and some hypotheses regarding muscle testing. It is important for the reader to realize that any statement in the following paragraphs which is not referenced to a source is a theory or a hypothesis. It is important that such statements be challenged! We offer this discussion with humility, and we emphatically request rebuttals, comments, questions, additions, and suggestions for other directions of investigation.

Before discussing the differences between manual and Cybex muscle testing, it is necessary to review the definition of the manual muscle test. For some testers, it is the determination of the maximum amount of force that a patient can successfully resist before he is moved out of the test starting position. This type of test ends as soon as the body part begins to move into an eccentric contraction. Kendall, Kendall and Wadsworth's muscle grading system[14] is based on this parameter. A second definition involves the concept of locking.[15] Here the tester is judging the steadiness of the isometric contraction, or the patient's ability to lock himself in the test starting position. A firm locking will be interpreted as a "strong" muscle test. But the tester will judge the muscle test "weak" if he senses mushiness, fading, pulsations or eccentric motion in the patient's response.

These two definitions of the manual muscle test seem to be in conflict, but actually they are expressions of different levels of skill and sensitivity in muscle testing. Recall the process of learning to muscle test. A beginner's ability to identify a "weak" muscle depends on his seeing the patient move out of the test starting position. Novice testers tend to overpower (and possibly injure) muscles in their desire to determine how much force the patient can resist. A more experienced tester knows that the muscle is "strong" as soon as he feels the locking sensation. He can

test a muscle with less force than a novice, and he can detect more subtle signs of neuromuscular dysfunction. An experienced muscle tester senses locking by feeling for subtle motion during the test. To understand the significance of this motion, let us return to the discussion of the differences between the manual and Cybex muscle tests.

The differences between manual and Cybex muscle testing are inherent in the instructions given to the patient at the beginning of each test. With a Cybex test, the patient is told, "Push as hard as you can through the entire range of motion." But with a manual muscle test, the patient is told, "Push only as hard as is necessary to keep me from moving you out of the test starting position." This difference in instructions produces a difference in the amount of force used in the two types of tests. The manual muscle test involves a much lower force (a lower percentage of maximum voluntary contraction) and therefore is testing the functioning of different muscle fibers and different motor neurons[16,17] than the Cybex test. The difference in speed of muscle action offers a second difference between the two types of tests. Fast motions, such as in the Cybex test, are primarily generated by the cerebellum; whereas slow motions, such as in the manual muscle test, are primarily generated in the basal ganglia.[18]

A third difference between the two types of testing lies in the fact that in a manual muscle test the patient must

monitor the doctor's test force and respond appropriately.[19] A manual muscle test evalutes the functioning of the patient's proprioceptive system, his ability to use that proprioceptive information in the central nervous sytem to quickly and smoothly modify his contraction force, and the integrity of the entire motor system (pyramidal and extrapyramidal systems, as well as the muscle itself). During a Cybex test, the patient's ability to gather proprioceptive information and to use that information is insignificant to the result of the test because there is no need for modification of the motor program during the test. This fact is well documented in the literature. "... [T]he greater the ability of the central nervous system (CNS) to 'predictively determine' a motor response, the less the need for peripheral sensory feedback...."[20]

> ... [W]hen the animal is in a predictive mode of performance, the movement pattern generated shows evidence of a substantial central programming component and an apparent independence of peripheral feedback.... In contrast, in the exploratory performance mode, the opposite pattern of results occurs; movements are clearly updated by feedback for successful execution."[21]

During a Cybex test, the patient determines the amount of force exerted, but in a manual muscle test the tester determines the amount of force involved. In the latter, the patient's job is to establish and maintain the delicate balance of forces between his effort and the tester's challenge which will result in an isometric contraction. This fine

balancing between eccentric and concentric contraction requires "complicated processes of neuromuscular integration to properly adjust the number and activity of motor units to the task."[22] After considering these significant differences in the physiology occurring during manual and Cybex muscle tests, it is not so surprising that their experimental data do not correlate well.

This brings us back to the two definitions of the manual muscle test. If a tester detects a low force capability in a muscle, he is probably finding a problem in the motor system. Physical therapists and orthopedists use this type of testing to evaluate both peripheral neuropathologies and myopathologies.[23] This also can include problems with the pyramidal and extrapyramidal systems in the central nervous system. Using the second definition, if a tester detects poor locking ability in a muscle, the problem could be anywhere in the proprioceptive system, the CNS integrative systems that make use of that proprioceptive information to modify the motor program in response to a changing load, or the motor system. These three systems: proprioceptive, integrative, and motor, form a neurologic loop from the muscle being tested (and the adjacent joints and soft tissues) to the CNS and back to the muscle. The goal of this neurologic loop during a manual muscle test is to maintain an isometric contraction in the test starting position. If there is dysfunction in this control loop, the tester will detect poor locking during the test. The faster the tester increases

the force at the beginning of the test, and the greater the force he applies, the more difficult it will be for the patient's control loop to avoid any droop from the test starting position. This is why the tester's technique must be consistent in the speed and the magnitude of force application; this is also why a tester can "get ahead" of a muscle if he does not allow the patient time to realize that the test is beginning. In summary, when a muscle tester judges the locking ability of a muscle, he is evaluating the integrity of this controlling neurologic loop. Good locking could be defined as the presence of a "zero droop control loop". In applied kinesiology, the manual muscle test is performed to evaluate both the force capability of a muscle and the integrity of the controlling neurologic circuits. In the absence of gross muscle or peripheral nerve pathologies, the manual muscle test as used in applied kinesiology is probably detecting changes in the efficiency of the patient's neurologic organization.

This brings us to a hypothesis as to why a "strong" muscle test can change to a "weak" one when therapy localizing a problem on the patient's body; or why a "weak" muscle can change to a "strong" one after appropriate applied kinesiology therapies are performed. This model hinges on the concept of selective attention.[24] It is well accepted that the human brain can only pay attention to one sensory stimulus at a time.[24,25] Our brain has the ability to select and pay

attention to one out of a group of concurrent stimuli and to screen out what it judges unimportant information at that time. For example, a young mother can sleep through the roar of an airliner or train passing near the house but will be awakened by a whimper from her baby. We often have conscious control over this selective attention phenomenon. For example, in the crowded room of a cocktail party we have the ability to select one person's voice to pay attention to. It has been found that mistakes that happen in an air traffic control tower are frequently due to the human operator selecting an unimportant bit of information to pay attention to, so that an important bit of information gets ignored.[26] This points out several important characteristics of the selection process. First, it is a continuous process with both conscious and unconscious priority systems. Second, it is fallible. Fatigue and stress increase the rate of errors and decrease our ability to control what we will pay attention to. The ability to concentrate and the ability to stay calm in difficult situations are expressions of effective selective attention. Stress of any sort (e.g. food additives leading to hyperactivity in children) diminishes the efficiency of our selective attention mechanisms and leads to neurologic disorganization.[27]

There is substantial evidence of a selective attention phenomenon at the interface between the proprioceptive system and the motor control system. The cells of the motor cortex

receive the proprioceptive information which is relayed via the thalamus.[28,29] These motor cortex cells receive information as to muscle length, muscle tension, joint movement, and position.[29] But the cortex also controls which incoming proprioceptive information it will receive or ignore.[29] Tsumoto, Nakamura, and Iwama[30] discuss how pyramidal tract activity can modify the transmission of tactile and passive joint information from the thalamus to the cortex. It has been documented that one of the functions of the descending fibers of the corticospinal (motor) tract is to synapse in the dorsal horn of the spinal cord to modulate sensory transmission.[31] Also, "information undergoing processing extraneous to the dominant cognitive activity under consideration is inhibited at the collosum, and ipsilateral pathways are also suppressed."[32]

Remember that the goal of the neurologic circuits controlling our motor functions (such as during a manual muscle test) is to provide rapid and smooth modifications of the motor program as required by changing loads. In order to achieve the ideal of a "zero droop control loop", all of the neurologic circuits involved must be intact. The presence of a selective attention phenomenon at the interface between the proprioceptive system and the motor system suggests that these circuits are <u>not</u> always intact. The CNS can choose to facilitate (make the circuits intact) or inhibit (make the circuits not intact) transmission of information along these motor control circuits as it deems appropriate at any moment.

It seems possible then that a "strong" muscle is one in which the motor control circuits are currently being facilitated and are therefore intact, producing a "zero droop control loop". It is possible also that a "weak" muscle is one in which the motor control circuits are currently being inhibited and are therefore not intact, producing less than a "zero droop control loop". In this latter condition, a muscle will display poor adaptation to changing loads. A muscle tester will feel a lack of locking, and he will label the muscle "weak".

The model for "strong" and "weak" muscles which is being proposed here offers a possible explanation for why a muscle's "strength" can change significantly within moments, which is a common clinical observation when therapy localization,[33] challenging,[34] or therapeutic techniques are employed. It seems that only a neurologic phenomenon could be effective so quickly. This model also offers a possible explanation for why therapy localizing a problem spot on a patient's body produces a weakening of a previously strong muscle. Perhaps when we have a patient therapy localize a spot, we are actually asking his body if there is a problem there that is more important than paying attention to the proprioceptive information coming from the muscle that we are testing. Clinical experience shows that a patient has to be paying attention to a muscle test in order for that test to be "strong". Perhaps therapy localizing a problem forces the patient's

attention selector to facilitate transmission of sensory information from the area of the localized pathology and to inhibit transmission of proprioceptive information from the muscle being tested.

This model also suggests that a "weak" muscle, with the muscle in the clear, might be indicative of a neurologic error by the patient's attention selector. For some reason the proprioceptive information from the muscle being tested is ignored. Various applied kinesiology therapies seem to be successful at "retraining" the neurologic decision-making mechanism. Thus, a state of neurologic disorganization,[27] or dysponesis,[35] can be detected by the manual muscle test and can be corrected by applying the appropriate therapeutic techniques.

VII. Summary

The Cybex test does not produce data that correlates well with the results of manual muscle tests. Consideration of the differences in the neuromuscular physiology between the two types of tests and recognition that the results of the manual muscle test depend on evaluation of a "locking" phenomenon make this lack of correlation more understandable. The conclusion is drawn that the Cybex equipment is probably

not a useful tool for future experiments designed to investigate and/or duplicate the manual muscle test.

Locking is a function of subtle motion that a muscle tester feels during a test. Good locking probably depends on the intact functioning of a neurologic control loop, which has proprioceptive, integrative, and motor components. The presence of a selective attention phenomenon at the interface between the proprioceptive system and the motor system offers a possible explanation for the clinical observation of rapid appearance and disappearance of good locking upon application of various applied kinesiology techniques. Perhaps a "strong" muscle is one with an intact neurologic control circuit, and a "weak" muscle is one whose neurologic control circuit is not intact. Future experiments should be designed to challenge this model.

We would like to close with some wisdom from Niels Bohr, the great physicist who first proposed the solar model of the atom. He said that a model for a phenomenon does not necessarily reflect the final reality of the situation. Instead, a model provides understanding of empirical observations, it provides a stimulus for further contemplation, and it provides a basis for forming new hypotheses that can be tested through experimentation. We offer the foregoing model of the manual muscle test in this same spirit. We welcome constructive criticism.

VIII. Acknowledgments

First, we would like to acknowledge George J. Goodheart, Jr., D.C.[36] who developed the original principles and practices of applied kinesiology. His work has stimulated our professional and scientific interests.

We thank David S. Walther, D.C. for providing research equipment and laboratory space, for his numerous consultations, and for his sincere interest in research which has helped to motivate our investigations.

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BIOCOMPUTER 'MODING' AS A TOOL FOR LEARNING MUSCLE TESTING

by

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Manual muscle testing consists of 'group' and isolated tests that are related to specific circuits and surface reflex areas of the body. Since accurate muscle testing forms the foundation of all A.K. procedures it seems critical that we should have a methodology by which to review and critique it. Such a methodology could be utilized both in evaluating and validating our muscle tests, as well as in providing a method of teaching muscle testing procedures to students of Applied Kinesiology. I believe that within the framework of the biocomputer model we are provided with just such a methodology.

The conceptual framework within which Applied Kinesiology operates requires at least three factors in order to exist in the realm of credibility. These three factors are an appropriate philosophical model, an impersonal feedback system, and a precise methodology with which to duplicate results. The biological computer model[1] with its concepts of modes and moding completes our current requirements for these three factors.

Using the analogy of the computer, the functioning of the body and the procedures of A.K. are easily described and readily accepted by both the general public and other health professionals. The widespread exposure to video recorders and home computers provides a readily accessible construct to which almost any individual can relate.

The introduction of 'moding' provides us with an opportunity to construct procedures and methodologies that can literally 'program' the body for feedback on specific information. By altering the variables we are

looking for it is possible to create procedures that cross check and test our current concepts. Once accepted such procedures could be valuable teaching aides with which to demonstrate the steps that anyone could utilize to verify our findings.

Presently there are over 260 muscle circuits and tests that form the basis of Applied Kinesiology. Of these, 77 are considered 'standard' A.K. testing[2] and as such are taught in the A.K. Basic 100 Hour Course. Approximately 180 other tests have been developed by various chiropractors and are taught and practiced at their discretion. The principle developer of this other muscle testing information is Alan G. Beardall, D.C..

Muscle testing, as the basis of Applied Kinesiology, is a highly complex skill that does not come easily to the typical beginning student of A.K.. Since being introduced to Applied Kinesiology in 1977 I have experienced the difficulties, frustrations, limitations and successes of trying to master muscle testing procedures. The relatively high attrition rate of practitioners in the basic 100 hour program speaks of the difficulty in teaching and learning the basic procedures. In looking back, I'm sure that I would not have mastered the ability to test individual muscle circuits had it not been for the careful and patient tutorship I have received.

The typical method of learning to muscle test is by observing an instructor who demonstrates a test and then critiques the student's effort at duplicating it. In the early stages of training the students results are usually uncertain, tentative, and frequently incorrect and the instructor is expected to point out the differences between right and wrong. Whenever the testing is incorrect, the instructor has to decide between two unsatisfactory alternatives. He can tell the student he is wrong, and again demonstrate the method, a procedure which in a class room often does nothing to build the student's nebulous confidence; he

can miss detecting the error, or worse yet, wrongly encourage an incorrect procedure so that the student inevitably is confused when on his own. In class many students seem intimidated by their colleagues or instructor's ability and do not fully engage in the learning process. Essentially, what most students are left with is a trial and error method of learning on their own.

What is ideally required by the student, is an entirely impersonal information feedback system which will tell him whether he is right or wrong with an impartial certainty. The "biological computer"[3] provides such an impersonal feedback system with which we can learn to test muscles.

The living computer (Biocomputer) model likens the body to an electronic computer with its logic and processing capabilities and the ability to store and display information. The human biological computer is more accurately a computer system analagous to a rocket in which four different computers interact to coordinate and process the functions. Each computer has both distinct and overlapping functions with every other computer so there are many backup systems for a particular function. The four computers in the body are termed local (kinetic), spinal (neurological), glandular (endocrine) and primary (cerebral). Each level has distinctive display and processing areas. The functional unit of the local or kinetic computer is the muscle. Muscles can be the display unit for all four levels of the body function. Within this context, muscle testing becomes a means of analyzing and monitoring any level of computer function.

Muscle testing along with a phenomenon known as therapy localization (T.L.) have formed the foundation of Applied Kinesiology diagnostic procedures. The introduction of the biocomputer model allows for the recognition of a third basic concept to the procedures of A.K. . This is the concept of 'moding'.

Various types of moding procedures have been utilized in A.K. without being recognized as such. These procedures are usually classified either as ways of finding so called 'hidden problems' or as ways of identifying specific conditions. Examples of moding are seen in our use of E.I.D., lateral tongue protrusion for occiput laterality, and twopoint therapy localization. Within I.C.A.K. the first direct reference to moding that I am aware of was made by Dr. Beardall during his presentation at the summer meeting 1981. He referred to the fact that therapy localization tells us that there is a problem but doesn't identify what it is. Moding was presented as a diagnostic tool to more directly let the body (computer) identify what is being TL'ed.

Dr. Beardall defines mode as "a form or manner of expression of the hand."[4] Essentially, I would describe a mode as a method of 'locking' the computer in to a particular function or program. A practical example of this aspect of moding is seen every day in the functioning of digital watches. A digital watch may have multiple functions such as seconds, lap timing, date, alarm, etc. all of which 'read out' or display on the face of the watch. Typically, by pushing the control buttons in a particular manner or sequence we can mode the watch for a specific function which it is capable of performing. Once moded correctly the watch will display only that function, even though it has the potential or capacity to operate in many other functions. It is locked in to that mode of operation. By learning which buttons to press and the correct sequence for a particular function we can predictably control the functioning of the watch. Moding is an extremely practical and useful concept in A.K.. We have muscle testing and therapy localization to tell us where to locate a problem and we have moding to tell us what we are therapy localizing.

Since moding is also a method of locking the computer into a particular function I feel that it can be used as a method of cross checking the validity

of muscle reflexes and muscle testing procedures and can thus provide a valuable teaching tool.

In 1981 I was testing a diplomate of I.C.A.K. for a persisting lower back and hip problem. This individual had been treated by extremely well qualified practitioners of A.K. and yet his hip problem persisted.

Since I had learned muscle testing from Dr. Alan Beardall, I tested muscles pertaining to the hip function in the manner I had been trained. Upon completing the testing what I observed was a startling fact. This individual was 'strong' ie. had intact muscle tone, in all the test positions that pertained to the recognized A.K. tests, but had gross muscle weakness in every test that deviated from these standard positions. It was as if only those circuits pertaining to known tests had been treated and were functioning. This experience drove home the necessity of knowing how to isolate muscle tests. If I didn't know how to test a particular muscle, I could easily overlook or miss a problem.

Given this experience I wanted to determine a method by which I could learn to accurately test a muscle that pertained to a certain set of reflexes. (ie. I wanted to know that when I tested an upper trapezius muscle, that is what I was really testing.) What I lacked was a method with which to double check myself. The concept of the body as a biocomputer with its capability for moding provided a paradigm upon which I could test not only the validity of the model but also the validity of the isolated muscle tests. What I proposed was that if a specific pre-determined set of reflexes truly reflected a certain muscle relationship, it should be possible to mode the computer so that it would 'read-out' only the muscle or muscles associated with those reflexes. I further reasoned that I should be able to find the involved muscle or muscles by therapy localization and specific muscle testing.

I therefore set up the following procedure:

1. The subject should be free of basic structural faults in the pelvis-lumbar, and upper cervical regions.
2. The subject should be clear to process information with no obvious switching present. ie. a) No change of muscle strength on therapy localization to acupuncture points K27.

   b) No change of indicator muscle strength when the finger tips of either hand touch the skin around the four levels of communication in Biocomputer model.[4] a) umbillicus b) xiphoid c) lips d) nose. If the subject is not clear on step 1 and 2, clear them in the appropriate manner or select another subject.
3. Test the muscles in the area to be tested. Record whether strong or weak.
4. Locate and mark vertebral level, [illegible] reflex (#1) and reflex [illegible] (#2) for the circuit being tested.
5. Have the subject touch tip of thumb and pad of index finger of one hand together.*
6. Have patient purse lips as if to kiss.** Maintain these 'modes' (lips and hands) throughout testing procedure.
7. First tap vertebral level, then reflex #1, then reflex #2, while patient maintains finger contacts.
8. Accepting only an extreme weakness as positive, test each of the positions as previously.

N.B. Alter positions of the test if necessary. Record which test if any was weak.

Using this procedure over the past two years, I have tested approx-

imately fifty muscle circuits and have observed the following phenomenon:

1. The body seems capable of manipulating its energy so that muscles that previously test weak will test strong when the biocomputer is moded for a specific circuit. ie. An apparent situation can be created in which every muscle in the body except one will test strong. This is a temporary situation that alters when the mode is released. I believe this is an example of how the body can adapt to a specific need and 'mask' an underlying problem.
2. The ability to isolate energy as in step #1 appears to be limited by:
   a) the specificity of the input that is programmed into the computer.
   b) the computer's ability to accept the input. ie. it appears that at times it will not or cannot overide an existing condition.
   c) gross switching in the patient - this causes a gross disorientation of input and display.
3. The toftness radiation detector will pick up the display or the 'read-out' of the muscle circuit that is being displayed.
4. The involved muscle will therapy localize. Sometimes the T.L. is very specific in location and direction of finger placement.
5. The involved muscle will test spongy in certain positions but will be extremely weak 'like butter' in certain specific test positions.
6. If insufficient input data is 'punched' in to the computer or if the sequence of input is incomplete, the computer will 'jam' or 'go on hold.' This jamming is evident as gross widespread muscle weakness and toftness radiation detector readings over large areas of the body. It is as if the body is saying, "I can't compute", and is on hold in its processing until a relevant piece of data can be put into the computer. This type of pattern occurs if I tap reflexes (NV, NL, etc.) that relate to organs

instead of muscles. I believe the reason is that the input is not specific enough for the biocomputer to fully process. If an appropriate area of the body (ie. related to the circuit being moded) is tapped the whole phenomenon reverses so that the weak muscles become strong and only the involved muscle is weak, therapy localizes, and displays to the radiation detector.

The above findings lead me to believe that we are in fact dealing with phenomena that at present are best described in terms of the biocomputer model. I am certain this information can be utilized to correlate and better organize the tremendous amount of data we have accumulated in Applied Kinesiology.

* * * *

Footnotes:

* A procedure for moding muscle circuits using the spinal computer as learned from A.G. Beardall, D.C.

** A mode for isolating muscle using the glandular computer as learned from A.G. Beardall, D.C.

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4. Ibid page 54

5. Ibid

A PROPOSED STUDY OF MANUAL MUSCLE TESTING PROCEDURES

by

Timothy W. Brown, D.C.

The following procedure is an example of what it is possible to test using moding and other procedures basic to A.K.. This procedure is designed to identify a muscle test related to a predetermined set of reflexes. By compiling data from procedures such as this it may be possible to upgrade and to resolve inconsistencies in our present knowledge.

Procedure

1. The subject should be free of basic structural faults in the pelvis-lumbar, and upper cervical regions.
2. The subject should be clear to process information with no obvious switching present. ie.
   a) No change of muscle strength on therapy localization to acupuncture points K27.
   b) No change of indicator muscle strength when the finger tips of either hand touch the skin around the four levels of communication in Bio-computer model. i) umbillicus ii) xiphoid iii) lips iv) nose. If the subject is not clear on step 1 and 2, clear them in the appropriate manner or select another subject.[1]
3. Test the muscles in the area to be tested. Record whether strong or weak.
4. Locate and mark vertebral level, reflex (#1) and (#2) for the circuit being tested.
5. Have the subject touch tip of thumb and pad of index finger of one hand together.*

6. Have patient purse lips as if to kiss.** Maintain these 'modes' (lips and hands) throughout testing procedure.
7. First tap vertebral level, then reflex #1, then reflex #2, while patient maintains finger contacts.
8. Accepting only an extreme weakness as positive, test each of the positions as previously.

   N.B. Alter positions of the test if necessary. Record which test if any was weak.

Alternate Procedure

An alternate procedure can be utilized if desired. After step #3 tape the north pole of a small ceramic magnet over a specific muscle acupuncture point, have patient touch thumb and index finger of one hand together (step 5), then tap reflex #1 for the circuit being tested. Test as per step #8 and record results.

Test Sequence A.

Vertebral level: Lamina process. Dorsal Ten Left Side (D10L)

Reflex #1: Temporal-sphenoid suture just superior to zygomatic process.

Reflex #2: 1st intercostal space -1" lateral to transverse process.

Acupuncture point: Stomach 10

Test Sequence B.

Vertebral level: Lumbar 4 - left side.

Reflex #1: Frontal bone - just superior to supra-orbital margin on superior temporal line.

Reflex #2: 6th intercostal space - 3" lateral to spine.

Acupuncture point: Pericardium 9

Test Sequence C.

Vertebral level: Lumbar 2 - left side.

Reflex #1: Occiput - 1" medial to occ. mastoid suture.

$1\frac{1}{2}$" superior to base of skull.

Reflex #2: Right - 5th intercostal space - 2" lateral to spine.

Acupuncture point: Bladder 58

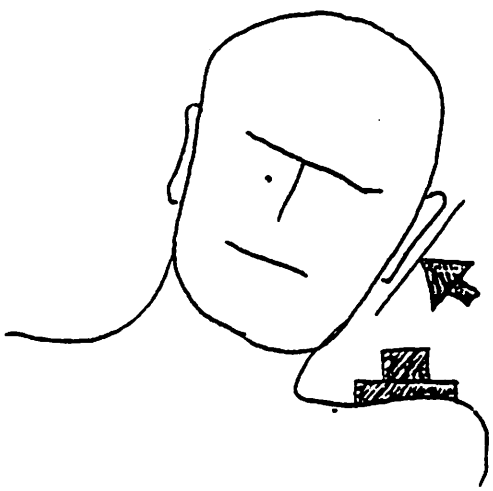
For each muscle test record whether strong or weak for each procedure.

Note any alterations from tests as outlined.

I invite you to test this procedure and I welcome any feedback regarding its use. Any data received will be compiled as a research project.

PRETEST	PROCEDURE #1	PROCEDURE #2	PROCEDURE #3
MUSCLE TEST #1			
#2			
#3			
#4			
#5			

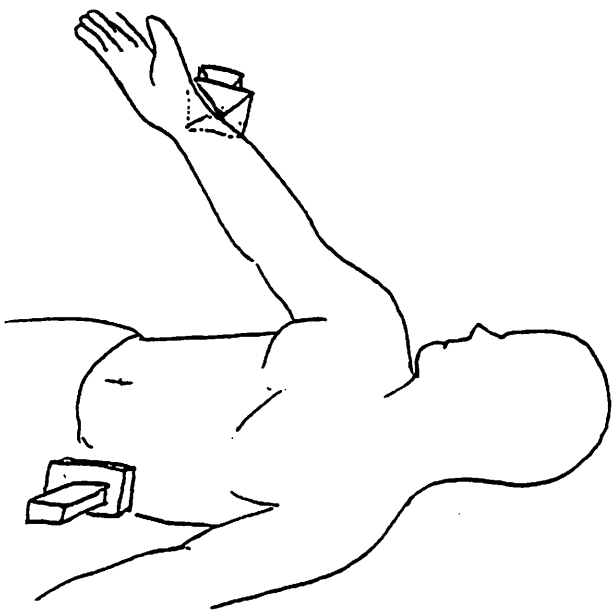
#1



Patient: Supine: Keeping face in coronal plane laterally. Flex neck on trunk to approximate shoulder.

Doctor: Brace shoulder and with palmar contact along temporal and parietal bones attempt to laterally extend neck on shoulder.

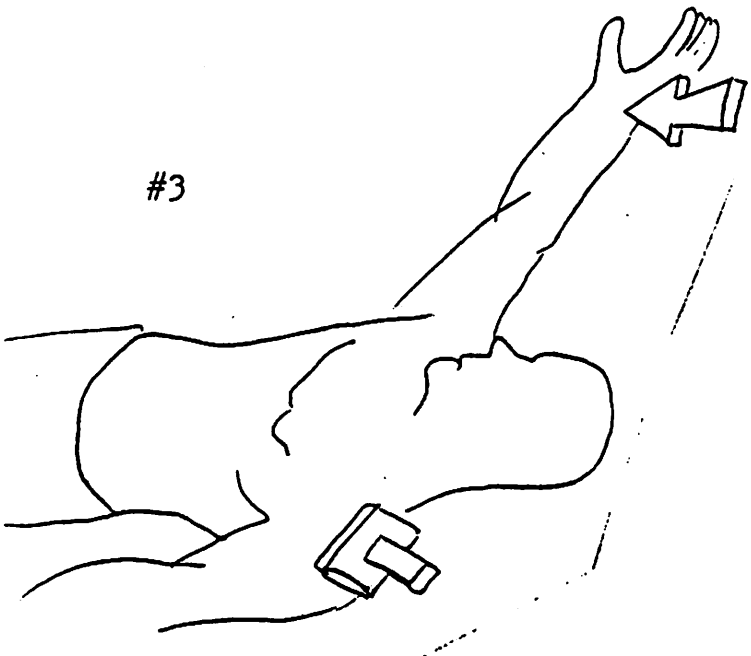
#2



Patient: Supine: Shoulder flexed 45 degrees and abducted 45 degrees. Thumb cephalad.

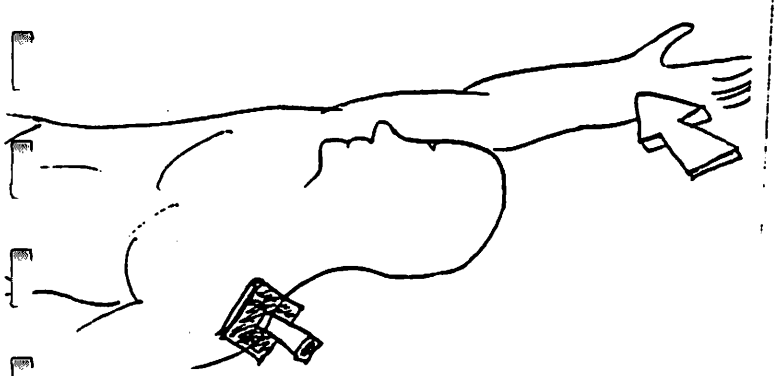
Doctor: Brace waist on opposite side. Contact distal forearm and press to adduct arm across body.

#3



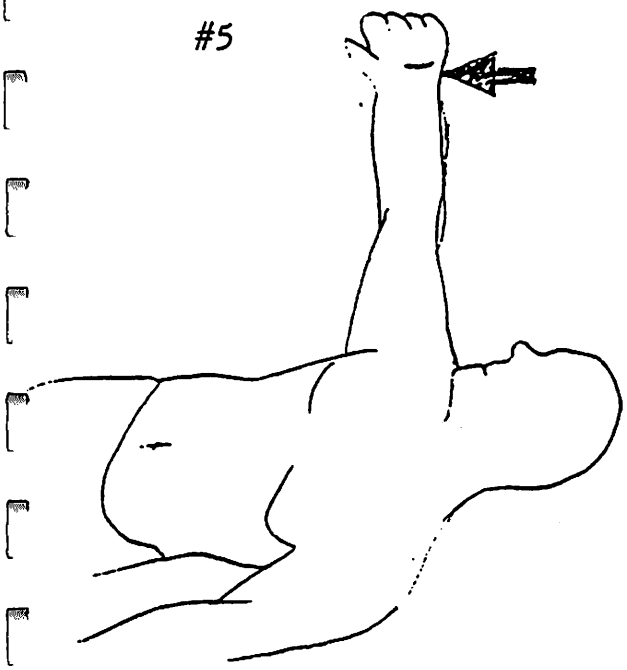
Patient: Supine: Abduct arm 120 degrees. Internally rotate arm until thumb points caudally.
Doctor: Brace contralateral shoulder. Contact forearm and adduct arm through coronel plane towards feet.

#4



Patient: Supine: Abduct arm 180 degrees to place deltoid muscle to ear. Fully internally rotate forearm.
Doctor: Brace contralateral shoulder. Contact forearm distal to elbow to abduct arm through coronal plane.

#5



Patient: Supine: Flex shoulder 90 degrees. Fully internally rotate forearm so thumb points caudally.
Doctor: Adduct shoulders so thumb is midline and scapula is pulled forward. Brace opposite shoulder contact distal forearm to extend shoulder through sagittal plane.

THE INTERNAL ISCHIUM

by

Timothy W. Brown, D.C.

The internal ischium subluxation pattern is a medial displacement of the ischial tuberosity relative to the femoral bone. A common pelvic fault, it is primarily associated with a dysfunction of the obturator internus muscle and manifests independant of the Category I, II, III,[1] IV[2] or PRYT patterns.[3]

I am grateful to my good friend Alan Beardall, D.C. for introducing me to the concept of the internal ischium. Since my initial awareness of its existence I have found it to be a common and significant pelvic fault that correlates with a specific pattern of muscular imbalance.

The obturator internus muscle has its origin on the margins of the obturator foramen, obturator membrane, obturator fascia, and the pelvic surface of the hip bone behind and above the obturator foramen and inserts into the medial surface of the greater trochanter.[4] An extremely thick powerful muscle its function is to rotate the thigh laterally, and abduct the thigh when the limb is flexed.[5] A weakness of the obturator internus muscle commonly results from a fall on the side of the buttock that leads to a medial displacement of the greater tuberosity of the ischium away from the respective femur.

The internal ischium subluxation pattern correlates with low back instability, leg symptoms such as heavy, 'dead' legs, poor circulation and varicosities. It is associated with a pubic bone inferior - superior torque pattern, weak medial thigh muscles particularly adductor brevis, gracilus and pectineus and dysfunction of the cloacal reflexes. The pattern is most easily screened for by testing the adductor brevis muscles for weakness.

TEST PATTERN

A) Pubic symphysis torque pattern - Indicators: Patient supine.

1. Test for adductor brevis muscle weakness[6]
2. Test for weak gracilus muscle[7]
3. Test for weak pectineus muscle[8]

The above weaknesses are negated by a proper challenge or TL to the pubic symphysis.

B) Internal ischium

1. Challenge by pressing on the ischium with medial pressure. Strong indicator muscle will weaken if the internal ischium is present.
2. Correlate with weak obturator internus muscle.[9]

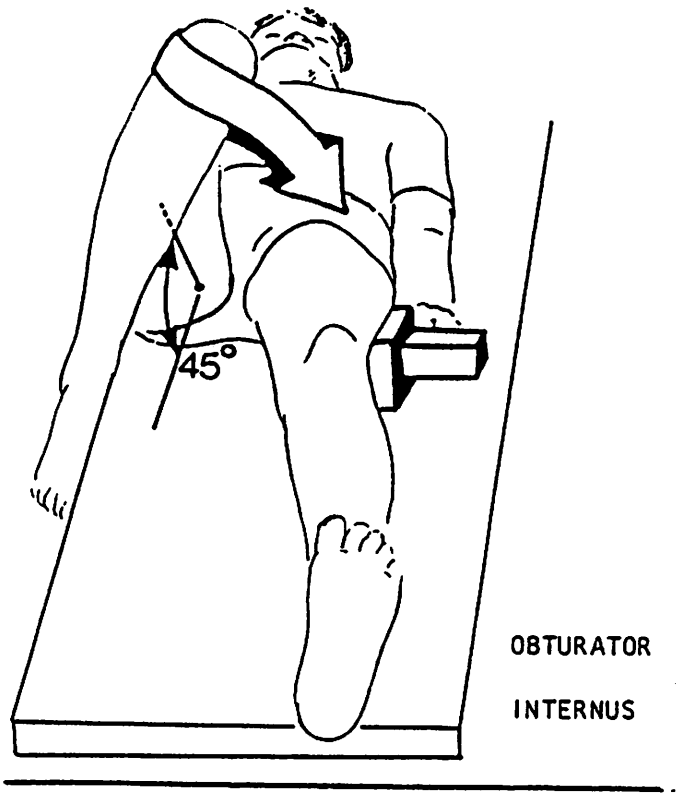
CORRECTION: The correction of the internal ischium pattern involves four steps.

1. Correct pubic torque pattern with patient supine.
2. Correct internal ischium subluxation with either
   a) Prone pumping technique: place a Dejarnette S.O.T. Block under the ipsilateral ASIS and contact the medial surface of the ischial tuberosity to pump it towards the femur head.

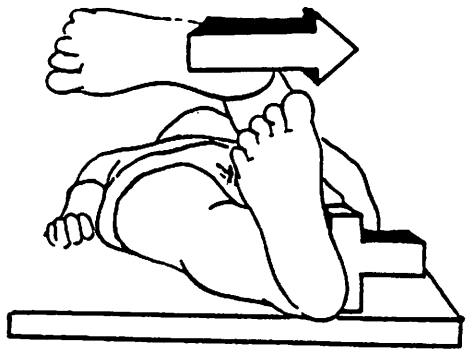
   or

   b) Side posture manipulation: with the involved side up, adjust like a typical illium except keep elbow close to table so thrusting vector is medial to lateral.
3. Diagnose and correct individual cloacals usually posterior and anterior involvements. These can be therapy localized when active and are treated by manual pressure in a circular motion.
4. Diagnose and correct any specific muscles still found to be weak.

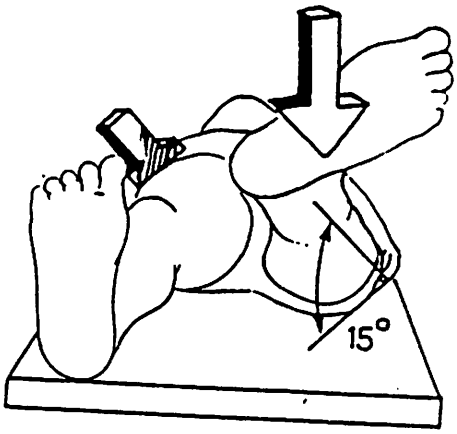
MUSCLE TESTS



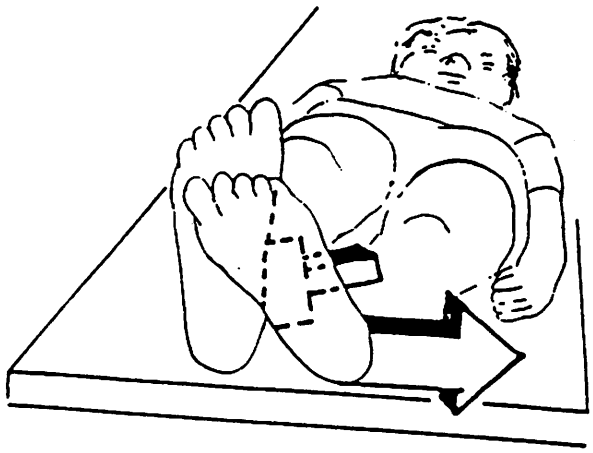
OBTURATOR INTERNUS



ADDUCTOR BREVIS



PECTINEUS



GRACILIS

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" The Calcaneal Subluxation and Recurrent Low Back Pain."

Dr. Elmer J. Cousineau, D.C.

Abstract:

A posterior medial subluxation of the right calcaneous bone of the foot is accompanied by a left psoas major and a right adductor weakness. Its correction eliminates these muscle weaknesses, but it is stabilized by adjusting the Fourth Lumbar.

Introduction:

The serendipitous discovery of the effect of vertebral and extremity subluxations upon the strength of the muscles of the body is a constant, consistant and repetitious theme of the research papers submitted by members of the I.C.A.K..

When another member of this group tests the application of these discoveries and finds them to be true, advantageous and efficacious, it in turn leads to the discovery of other applications of the same discovery. As Dr. George Goodheart so aptly expressed it: "Answers are found, for which there has not as yet been posed a question."

Such is the case for the discovery of subluxations of the lower extremity, the foot and the ankle. The use of the Gait Reflexes is one such discovery by an I.C.A.K. member.

The discovery by this author that the medial subluxation of the right calcaneus was always accompanied by a weakness of the left psoas major and of the right adductor muscle group led to correction of a recurring subluxation of the Fourth Lumbar vertebra and recurring low back pain.

The correction of the Lumbar Four subluxation without the correction of the right calcaneus would allow the return of the Lumbar Four subluxation upon the use of the lower extremity, as in walking.

The correction of the calcaneus subluxation without the correction of Lumbar Four would permit the return of the calcaneal subluxation.

Along with the calcaneus correction and befor the correction of the Lumbar Four it was found that there was a "switching" of weakness of a right abdominal muscle to the left abdominal, either rectus or obliquus. Also if the left quadratus lumborum was weak prior to the calcaneal correction, its weakness would also "switch" to the right quadratus lumborum.

The Calcaneal Correction

Patient: Supine, legs extended together, hands along body.

Adjustor : At foot of table facing patient headward.

Adjustor's Left Hand: Grasp patient's right foot at heel, cupping in the hand with the thumb extended along the distal end of the fibula, thenar eminence

of thumb on lateral portion of the heel at the calcaneus. The extended and joined fingers of the left hand wrap around the heel on medial side of the calcaneus.

Adjustor's Right Hand: wraps around the dorsum of the foot just inferior to the patient's talus bone. The thumb is placed upon the plantar surface of the patient's foot.

The Corrective Thrust: The foot is slightly dorsiflexed upon the lower leg. The entire relaxed leg is then slightly abducted sideways from the other leg and traction is applied to the foot and leg to take up any slack in the knee and hip joint. At full tension a quick tug is given to the foot while the grip upon the heel is tightened. This causes a shearing or torqueing motion to be applied to the medial surface of the calcaneus to move it laterally upon the talus and fibula.

Post Check of Muscle Strength will show a return of strength to both the left psoas major and the right adductor muscles, without the correction of the Lumbar Four. Correction of it will stabilize the calcaneal correction.

- 0 -

" Muscle Weaknesses Corrected by Cervical Adjusting "

Dr. Elmer J. Cousineau, D.C.

Abstract:

All pelvic muscle weaknesses may be corrected by upper cervical adjustments of the occiput, atlas, axis and third cervical vertebrae.

Weakness of muscles affecting the arms may be corrected by adjusting the third through the seventh cervical vertebrae.

Introduction:

For the information relating to the muscle weaknesses corrected by adjustment of the cervical vertebrae I wish to acknowledge the source as being that discovered by Dr. Robert Ridler, D.C. of Seattle, Washington during the 1970s when he was reading XRays for chiropractors of that state while he was a Diplomate Roentgenologist.

Your author was heavy into Applied Kinesiology and muscle testing, and demonstrated such for Dr. Ridler, especially the correction of muscle weaknesses by Neurolymphatic and Neurovascular Reflexes.

Dr. Ridler prided himself upon his ability as an adjustor and so found upon his own research those segments of the spine that would perform the correction. He found it by a simple method of pushing upon the spinous process of each vertebra of the spine until he found the one that would tightn the muscle in question at that moment. Then upon adjusting it he rechecked for weakness.

Muscle Weakness Corrected by Cervical Adjusting (Contd) page 2

Your author recorded this data and reproduces it here at this time for benefit of members of the I.C.A.K. for such use as they wish to make of it.

Upper Cervical and Pelvic Muscle Weakness

The Occiput corrects leg adductor weakness. Adjust for posteriority of occiput on side of m. weakness.

The Atlas corrects for lateral leg movement as abduction. Posteriority corrects gluteus medius and psoas m. Anteriority correct fascia lata of opposite side.

The Axis corrects for forward or backward leg motion. Posteriority of axis body on side of m. weakness corrects gluteus maximus and quadriceps.

The Third Cervical corrects piriformis m. weakness. Posteriority of body on side of m. weakness.

Lower Cervicals and Arm Muscle Weakness

The Fourth Cervical corrects Deltoideus muscle weakness.

The Fifth Cervical corrects for Latissimus Dorsi weakness.

The Sixth Cervical corrects for Pectoralis Major Clavicular.

The Seventh Cervical corrects for Pectoralis Major Sternal.

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312. E. Pioneer Avenue, Puyallup, Washington 98371

LIGHT PUPILLARY CHALLENGE OF ADRENAL INSUFFICIENCY

by

Frederick J.Dieterle,D.C.

ABSTRACT: In clinical observations it has become evident to us that shining a bright penlight into the patient's pupil, that the patient with postural hypotension or adrenal insufficiency will weaken a strong indicator muscle as well as a sartorius. There appeared to be some individiauls that both were Hypoadrenic and yet not show up in blood pressure measurements, Rogoff sign, and sartorius adrenal syndrome testing. This particular pupillary light challenge allowed us to monitor the adrenal circuit and obtain the necessary information. This is a simple and most useful challenge and can be used in conjunction with all the usual kinesiological procedures.

TESTING CATEGORY:

Patient initially is posturally evaluated. After the usual blood sugar questionaire and case history. Blood pressure measurements supine and standing, as you know should show an elevation of 10 to 12 points going from supine to standing as well. The usual therapy localization procedures auditing the alarm points, pulse points vertebral challenge. Rib tenderness, pupillary response and the usual kinesiological testing procedures are employed as well.

One then do the pupillary light challenge. In order not to evoke a pineal gland response we do not darken the room entirely, but adjust the room to a subdued lighting condition and patient is in a supine position to test any strong indicator muscle before and after the light challenge. This evokes a positive response- ie: muscle weakness and you may then correlate this with all the other indicators as well as using a double contact and temporal tap to audit the nutritional circuit, this is performed with the light challenge and the nutritional supplement sublingual simutaneously.

We have tested both in standding, sitting and supine positions to see if this alters in anyway the pupillary response. To date, the one individual has shown greater sensitivity to the testing procedure in a sitting position as opposed to a supine position.

This is not to be confused with challenging the effect of flourescent, incadescent and full spectrum lighting or use of color therapy. I found this test to be quite useful and sometinges more sensitive than others. I hereby submit this for your appraisal and evaluation and possible use if you so desire.

CONCLUSION:

The Pupillary light challenge is effective in the case management of hypoadrenia and can be used as a adjunct to and in correlation with other kinesiological procedures, It is not recommended that it be used solely or in lieu of anyother form of already established testing.

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PECTINEUS MUSCLE AND THE MANAGEMENT OF INGUINAL HERNIA

by

Frederick J.Dieterle,D.C.

CLINICAL OBSERVATION: We have determined that there is a positive relationship between the pectineus muscle and the existance and symptomatology of an inguinal type hernia. Usual methods of alleviating this condition such as a slant board and organ lift to retract the abdominal contents has been useful buth there has been persistant and reoccuring patterns of continuous muscle spasm or shortening of the muscle sheath. Very good response has been noted allowing the utilization of well known kinesiological procedures that are performed on the pectineus muscle.

GENERAL INFORMATION: In order to evaluate the relationship between the pectineus muscle and the inguinal hernia we took those patients that were diagnosied with inguinal hernias and those that had the symptomatology that amounted to an inguinal hernia and tested the pectineus msucel as well as all other pelvic muscles. We found it to be involved in the single case of inguinal hernia without exception. A sample of only my patients isstill significant. The pectineus muscle was therapy localized, because of the therapy localization was usually positive both to the spindle or the belly of the muscle and the origin insertion of the muscle as well. By challenging the muscle it was found to be hypertonic. By applying pressure directly on the belly of the muscle, as in a Nimmo procedure, it then most certainly would cause a radiating pain and pressure sensation just above the inguinal ring deep in the abdomen.

CLINICAL PROCEDURE: The most effective procedure was to utilize a fascia release technique as well as a spray and stretch technique as per Jeanette Travell's procedures. Patient was also instructed to fully stretch the abductor muscle group and the pectineus, simutaneously doing golge tendon technique and spreading origin insertion of the pectineus apart combined with lifting techniques such as in opeing the ileocecal valve, but lower. We feel that the key to success of this procedure lies in the inteorelationship between the structures of the attachments and the anatomy of the inguinal ligament and inguinal canal in relationship to the attachment of the pectineus muscle. We are also investigating a possible reactive muscle link. Attach please note Figure A- excerpts from Gray's Anatomy, 35th British Edition.

CONCLUSION: This procedure should be useful in the management of inguinal hernias. A study is underway as time permits, and we will provide an update, if at all possible, sometime in the future.

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contains non-striated muscular fibres, forming the *dartos* muscle, which is supplied by the genitofemoral nerve. From the scrotum it may be traced backwards into continuity with the superficial fascia of the perineum. In the female it is similarly continued from the abdomen into the labia majora and perineum.

The deep layer of the fascia, thinner and more membranous than the superficial, contains a considerable quantity of elastic fibres. It is loosely connected by areolar tissue to the aponeurosis of the external oblique; but in the median plane it is more intimately adherent to the linea alba and to the symphysis pubis, and is prolonged on to the dorsum of the penis, forming the *fundiform ligament* (5.45). Above, it is continuous with the superficial fascia over the rest of the trunk; below and laterally, it blends with the fascia lata of the thigh a little distal to and parallel with the inguinal ligament (5.45); below and medially, it is continued over the penis and spermatic cord to the scrotum, and from there may be traced backwards into continuity with the membranous layer of the superficial fascia of the perineum (p. 529). In the female it is continued into the labia majora and thence to the fascia of the perineum.

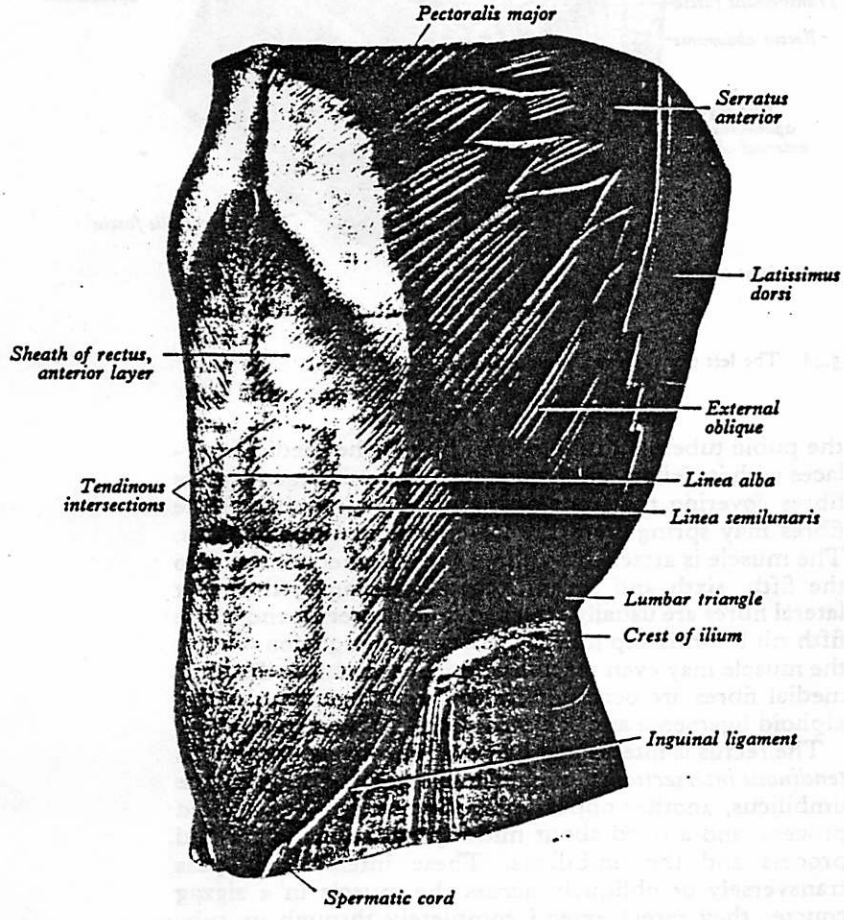
In the child the testis can frequently be retracted out of the scrotum into the interval occupied by loose areolar tissue between the external oblique and the deep layer of superficial fascia over the inguinal canal. This interval is sometimes called the *superficial inguinal pouch*.[88]

The obliquus externus abdominis (5.44), curved round the lateral and anterior parts of the abdomen, is the largest and the most superficial of the three flat muscles in this region. It arises, by eight fleshy slips, from the external surfaces and inferior borders of the lower eight ribs; these slips interdigitate with the digitations of serratus anterior and latissimus dorsi, and are arranged in an oblique line which runs downwards and backwards, the upper ones being attached close to the cartilages of the corresponding ribs, the lowest to the apex of the cartilage of the last rib, the middle ones to the ribs at some distance from their cartilages. From these attachments the fibres diverge as they pass to their insertions. Those from the lower two ribs pass nearly vertically downwards, and are attached to the anterior half or more of the outer lip of the ventral segment of the iliac crest (p. 346); the middle and upper fibres, directed downwards and forwards, end in an aponeurosis, opposite a line drawn vertically from the ninth costal cartilage to a little below the level of the umbilicus, and then inclining laterally to the anterior superior iliac spine. The muscle fibres rarely descend beyond a line from the anterior superior iliac spine to the umbilicus. The posterior border of the muscle is free (5.58).

The *aponeurosis* of the external oblique is a strong membranous structure, the fibres of which are directed downwards and medially. In the median plane its fibres end in the *linea alba* (5.44), a tendinous raphe which stretches from the xiphoid process to the symphysis pubis. At the raphe it is continuous with the aponeurosis of the opposite muscle and the two together cover the front of the abdomen. Below and medially the aponeurosis is attached to the upper border of the pubic symphysis and the pubic crest as far as the pubic tubercle. The margin of the part of the aponeurosis between the anterior superior iliac spine and the pubic tubercle is a thick band, folded internally upon itself to present a grooved upper surface; this is the *inguinal ligament*. A small expansion from the medial end of the inguinal ligament is attached to the pecten pubis—the *lacunar ligament*. From this end of the inguinal ligament fibres also pass upwards and medially to join the rectus sheath and the linea alba; these constitute the *reflected part* of the inguinal ligament (5.50).

The muscular and aponeurotic parts of the external oblique are invested by external and internal layers of fascia of which the former is better developed. The upper and lower digitations may be absent; digitations or even the whole muscle may be reduplicated. Digitations may also be continuous with pectoralis major or serratus anterior.

Nerve supply. The ventral rami of the lower six thoracic spinal nerves.



520 5.44 The left obliquus externus abdominis.

The inguinal ligament (5.46), the lower border of the aponeurosis of the external oblique, stretches from the anterior superior iliac spine to the pubic tubercle. It is convex downwards towards the thigh and continuous with the fascia lata. Its lateral half is rounded and oblique; its medial half gradually widens towards its attachment to the pubis, is more horizontal and supports the spermatic cord.

The lacunar ligament (pectineal part of the inguinal ligament) (4.58) is the extension of the aponeurosis of the external oblique which passes backwards and laterally from the medial part of the inguinal ligament to the medial end of the pecten pubis. It is triangular, and almost horizontal when the body is upright; it is a little larger in the male, and measures about 2 cm from base to apex. Its base, directed laterally, is concave and thin, and forms the medial boundary of the femoral ring; its apex is attached to the pubic tubercle. Its posterior margin is attached to the pecten pubis, and is continuous with the pectineal fascia; its anterior margin is continued into the inguinal ligament. It has superior and inferior surfaces. A strong fibrous band, the *pectineal ligament* extends laterally from the base of the lacunar ligament (4.58) along

[88] D. Browne, *Br. med. J.*, 2, 1938.

uscle may be described as forming continuous loops om the middle of the inguinal ligament as far as the nica vaginalis and then returning to be attached to the ubic tubercle. In the female a few fibres descending on ie round ligament of the uterus represent the lateral part cremaster.

Nerve supply. The genital branch of the genitofemoral erve, derived from the first and second lumbar spinal erves.

Action. The cremaster pulls up the testis towards the perficial inguinal ring. Although its fibres are striated, is not usually under voluntary control. Stroking of the edial side of the thigh evokes a reflex contraction of the uscle and this *cremasteric reflex* is much more active in ildren. The precise value of this action, whether pro-ctive, concerned with temperature regulation, or other nctions, remains uncertain.

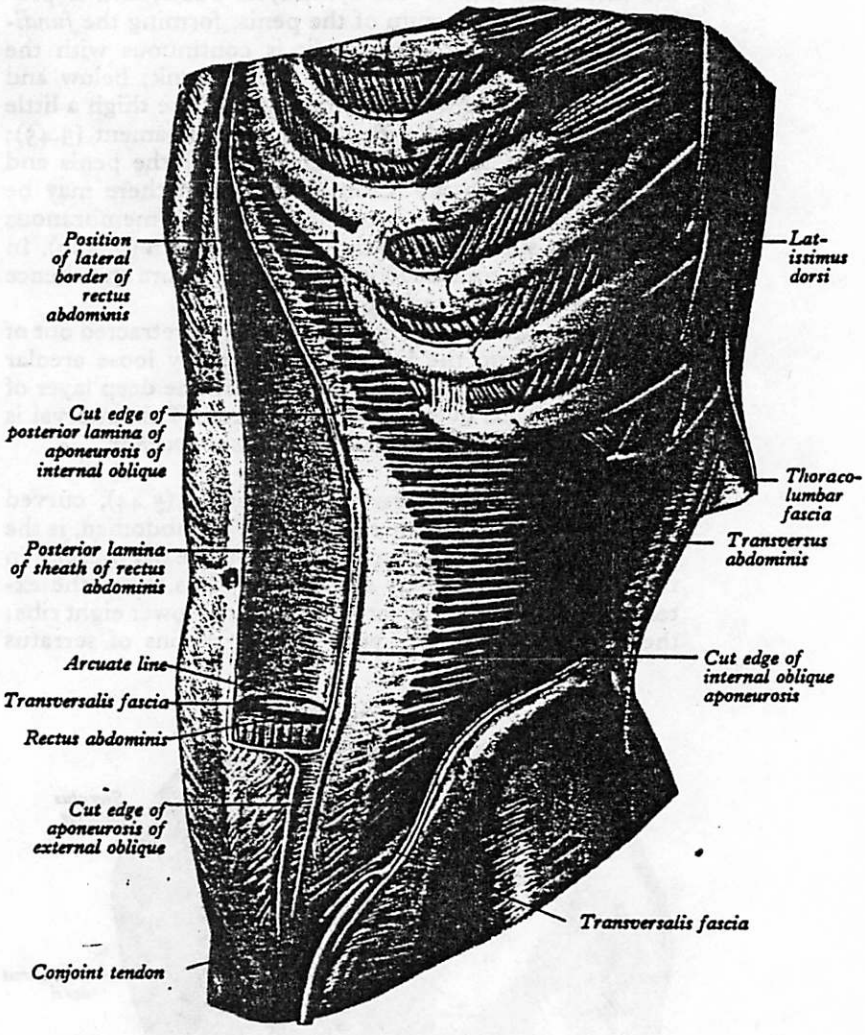
The transversus abdominis (5.48), so-called from ie direction of its fibres, is the innermost of the flat uscles of the abdominal wall, being internal to the ternal oblique. Its muscle fibres arise from the lateral ird of the inguinal ligament, the anterior two-thirds of ie inner lip of the ventral segment of the iliac crest, the oracolumbar fascia between the iliac crest and the velfth rib, and the internal aspects of the lower costal rtilages (usually six), where it interdigitates with the aphragm (5.41). The precise origin of the fibres, hether from the inguinal ligament direct, or from ljacent iliac fascia has been the subject of dispute.[30] The uscle ends in an aponeurosis of variable extent, the wer fibres of which curve downwards and medially to-ther with those of the aponeurosis of the internal olique to the crest and pecten of the pubis, forming the *njoint tendon.* The rest of the aponeurosis passes rizontally to the median plane, and blends with the nea alba; its upper three-fourths lie behind the rectus bdominis and blend with the posterior lamina of the oneurosis of the internal oblique; its lower fourth is in ont of the rectus. The upper muscular fibres of the ansversus abdominis are continued medially behind the ctus (5.48) and the posterior lamina of the aponeurosis the internal oblique, sometimes being continuous ross the midline with the opposite transversus. Near ie xiphoid process they reach to within 2 or 3 cm of the nea alba. The muscular fibres of the transversus run into ie aponeurosis along a line which is concave medially .48), the aponeurosis being widest opposite the origin the muscle from the thoracolumbar fascia.

Fusiform defects filled with fascia occur in the lower uscular and the aponeurotic parts of both the internal olique and the transversus abdominis. The two muscles e sometimes fused, or the transversus may be absent.

The conjoint tendon (falx inguinalis) of the internal olique and transversus (5.50, 51) is mainly formed by ie lower part of the aponeurosis of the transversus, and inserted into the crest and pecten of the pubis; it escends behind the superficial inguinal ring, thus serv-g to protect from behind what would otherwise be a eak point in the abdominal wall. The attachment to the ecten pubis is frequently absent. Medially the conjoint ndon is directly continuous with the anterior wall of the heath of the rectus abdominis. Laterally, it may be ntinuous with an inconstant ligamentous band, named ie *interfoveolar ligament* (5.51), which sometimes con-cts the lower margin of the transversus to the superior mus of the pubis; it occasionally contains a few muscu-r fibres. Muscular fasciculi, attached to the pecten ubis behind the conjoint tendon, may reach the trans-rsalis fascia, the aponeurosis of the muscle, or even the

Nerve supply. The ventral rami of the lower six thoracic and the first lumbar spinal nerves.

The rectus abdominis (5.52) is a long strap muscle, broader above, which extends along the whole length of the front of the abdomen, separated from its fellow by the linea alba. It arises by two tendons; the lateral and larger is attached to the crest of the pubis and may extend beyond



5.48 The left transversus abdominis.

the pubic tubercle to the pecten pubis; the medial interlaces with its fellow and is connected with the ligamentous fibres covering the front of the symphysis pubis. Some fibres may spring from the lower part of the linea alba. The muscle is attached by three slips of unequal size into the fifth, sixth and seventh costal cartilages; the most lateral fibres are usually attached to the anterior end of the fifth rib but this slip may be absent, although conversely, the muscle may even reach the fourth and third; the most medial fibres are occasionally connected with the costo-xiphoid ligaments and the side of the xiphoid process.

The rectus is intersected by three fibrous bands, named *tendinous intersections*; one is usually situated opposite the umbilicus, another opposite the free end of the xiphoid process, and a third about midway between the xiphoid process and the umbilicus. These intersections pass transversely or obliquely across the muscle in a zigzag course; they rarely extend completely through its substance and may pass only halfway across it; they are intimately adherent to the anterior lamina of the sheath of the

COMMON EXERCISES
THAT INTERFERE WITH PATIENT RECOVERY

By Daniel H. Duffy, D.C.

ABSTRACT: Swimming is contraindicated during the recovery of hypoadrenic patients due to the decrease in body temperature produced by the usually lower than body temperature water encountered in both indoor and outdoor swimming. Any slight lowering of body temperature initiates a hypothalamic response resulting in glandular stimulation over and above normal requirements. Bicycling is contraindicated especially in gait related problems due to the overactivity of the lower limbs in proportion to upper limb activity. Repetitive exercises not in conformity with normal gait mechanisms are potential trouble makers. The bicycle seat also abuses the integrity of the levator ani muscle and the pelvic diaphragm.

Four difficult patients with recurrent hypoadrenic signs and symptoms were finally found to have one thing in common—regular trips to the "Y" for swimming exercises.

Elimination of the swimming resulted in lasting corrections of the signs and symptoms. The constant stimulation of an already overtaxed glandular system by prolonged swimming prohibits recovery in the difficult patient. This observation emphasizes the need for lasting correction of body temperature in order to give the glandular system a "rest" so to speak. Pituitary drive technique is quite effective in producing a normal temperature but almost always requires other intervention to maintain it. Many factors other than direct thyroid involvement are in need of correction and when all physical factors are corrected a simple nutritional need left unfulfilled will not allow basal temperature to reach and maintain normal.

Many patients have been seen with recurrent pelvic problems which seem to be related to bicycle riding. One acute patient was seen following a night in an intensive care unit due to left chest and arm pain. Cardiovascular examination at the hospital was negative and the patient was discharged with a prescription for tranquilizers and analgesics. Careful questioning by this writer revealed that the pain had started in the left first rib head area following a day of bicycling and gradually spread to envelope the entire left shoulder, arm and chest with angina like symptoms. When first seen the patient was in a position of acute antalgia, hugging the left upper limb (ear to shoulder, arm clasped to chest). No pain was present in the lower back however correction of a sacroiliac subluxation on the affected side immediately relieved the upper limb and chest pain. The subluxation of the sacroiliac joint involved the hamstring and levator ani muscles on that side.

Repetitive exercise not in confotmity with normal gait mechanisms will eventually produce problems in most patients. The only repetitive exercise recommended by this writer is walking with a free arm swing.

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AN UNCOMMON FINDING IN GAIT TESTING

By Daniel H. Duffy, D.C.

ABSTRACT: Occasionally while testing for anterior gait responses a weakness is observed in the lower limb only regardless of the testing mode. Four of these instances have been found to respond to pumping of the sacrum and ischium on the phase of respiration that abolishes the weakness.

A patient was examined and found to have a weakness of the lower left limb when tested in the anterior gait fashion.[1] Every known technique was used to abolish the weakness to no avail. Finally, the pelvis was examined for a sacroischial respiratory correction need and this correction abolished the condition of the "one half gait" response. Since this first occasion approximately a year ago I have seen four occasions of this. No effort was made to correlate sacroischial respiratory correction need with the existence of the "one half gait" response. The need for sacroischial respiratory correction is fairly frequent and should especially be checked when there is persistent rib head pain following good category correction of the pelvis. Following correction of the pelvis, dorsal spine and first rib head via the "limbic Technique" any further rib head pain is usually abolished by sacroischial technique. TL of the sacrum and ischium abolished by inspiration calls for pushing the sacrum and ischium together on inspiration with the reverse for expiration.

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THE ASYMMETRICAL SAGITTAL FACET SYNDROME

By Daniel H. Duffy, D.C.

ABSTRACT: An asymmetrical sagittal facet (ASF) in the lumbar or lumbosacral joint produces a tendency for a unilateral forward slip on the ipsilateral side which responds to a variety of approaches for short periods of time. Sole lift on the affected side eliminates the recidivism.

A large overweight diesel mechanic was treated for low back pain, worse in the erect position, aggravated by standing for even short periods of time. This pain would be alleviated temporarily by the patient by flexing the affected limb and placing the foot on a chair or other elevation. The pain was of such intensity that the patient was seriously considering changing his vocation.

When initially seen, pelvic corrections and spinal adjustment effected excellent but short lived relief varying from one day to one week. Further attention to the gluteus maximus and abdominals also gave some relief, complete but shortlived, on several other occasions, corrections to the muscles of the lower leg and foot on the affected side gave similar results.

Final and lasting correction of this difficult case came from the observation of an ASF on the affected side and the suggestion of a sole lift to be placed on that side [1]

I have since that time had the occasion to use the sole lift on several occasions in order to effect a lasting correction. Of six cases treated, two were gradually taken off the sole lift and continue to function for over a year without resorting to further need of the lift. Permanent lifts should be built into the footwear once the permanent need is established. Since many patients with ASF do not have symptoms attributable to the deformity it is logically assumed by this writer that if we properly balance the mechanism we should be able to do away with the lift. Proper balancing is not always possible for obvious reasons, therefore the opinion of this writer is that most sole lifts will probably require life long use.

The symptomatic picture presented by the patient is acute pain, often of sciatic distribution, which responds to a seemingly infinite variety of corrections only to recur in full bloom so to speak.

The ASF may not appear in the classical manner on xray, i.e., the facet articulation may nor be classically perpendicular to the horizontal but may be angulated or as was the case with the patient mentioned herein, barely noticable as a very slight curving articulation encompassing the lower lateral portion of the lumbosacral articulation. Good film and careful inspection are required to detect the presence of this type.

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Walking Disc

by Kenneth S. Feder, D.C.

ABSTRACT:

Therapy localization in a weight bearing walking posture may be necessary to assist in the diagnosis of a Category III. Once the diagnosis has been established, it may also be necessary to correct the Category III with the patient in the proper walking posture.

There are many patients who exhibit discogenic related symptomalogy, but however, show a negative therapy localization and challenge while in a prone position. After utilizing and finding success with Dr. Goodheart's non-weight bearing gait technique, I attempt to employ a weight bearing gait technique on those patients who showed a negative TL in a non-weight bearing position.

The procedure and correction is outlined as follows:

1. The patient stands with feet together with hands in proper therapy localization position for determining disc involvement. In a previous paper concerning disc therapy localization, I referred to this as a "framing of the disc". The neck flexors having previously been tested clear and found strong should then be tested to determine if a positive therapy localization exists. If a positive therapy localization exists, then treat as follows: challenge and determine the side of involvement while the patient is in a weight bearing position. The side of involvement is the side that produces muscle weakness. The adjustment is made with the patient in a standing position with the correction administered to the side that produced muscle weakness while utilizing the appropriate phase of respiration. For further details on the correction of disc involvement, refer to my paper entitled "Flexion-Traction Technique".

2. If therapy localization is negative in a weight bearing posture, have the patient continue to TL the suspected disc lesion; however, have the patient advance either the right or the left leg. The TL may then reveal a hidden disc with one or the other leg advanced.

Having determined which advanced leg produces a positive TL, continue having the patient advance the same leg while you challenge to ascertain the side of involvement. (Note: the challenge will not reveal involvement if the non-involved leg is advanced.)

CORRECTION:

Once the side of involvement has been determined, have the patient advance the non-involved leg (the leg that did not produce a positive TL) and proceed to correct the disc in a weight bearing position.

This procedure has been extremely effective in treating difficult non-responsive disc related problems. It can also provide an extraordinary cost savings by eliminating the expense of an adjusting table.

Best Foot Forward Technique

by Kenneth S. Feder, D.C.

ABSTRACT:

This paper serves as an expansion to the walking disc procedure. Many faults may not surface unless the patient is placed in a weight bearing posture with the proper gait. Correction may not be attained if the patient is not placed in a weight bearing position in the proper gait.

After experiencing much success with the walking disc technique, I attempted to apply the same principles of diagnosis and treatment to general faults. The following approach may be utilized to diagnose and treat any previously learned AK procedure.

Having found a negative TL in both non-weight bearing and weight bearing, proceed as follows:

1. Advance either the right or the left leg while therapy localizing the suspected involvement.
2. Once determining that a positive TL exists, challenge the area with the leg advanced which produced the positive TL.

CORRECTION:

Advance the leg which did not produce the positive TL while you adjust the involved area.

A BRIEF ANATOMY OF THE COLON, RECTUM AND ANUS

By Bert T. Hanicke, D.C.

Diplomate I.B.A.K.

A brief description of the division of the colon, rectum and anus giving the major anatomical relationships and landmarks. Special attention is given to terms used in Applied Kinesiology.

This paper was prompted by a desire to refresh our members' memories concerning anatomical names of various parts of the colon. This desire was given strength when at several meetings I observed good Kinesiologists making errors in labeling the parts they were working on. As an example, at the Summer Meeting of I.C.A.K. 1978, Dan Gleeson, D.C. gave an excellent paper and demonstration involving the descending colon and the sigmoid colon.(1) His paper was anatomically accurate. Since that time I have seen this same procedure called the left I.C.V. Technique, and the Valves of Houston Technique, neither label being anatomically correct, hence this paper.

Colon (Large Intestine)

That part of the alimentary canal between the ileum and the anus and is approximately 140 cm in length in the male and 130 cm in length in the female.

Cecum

A cul-de-sac forming the first part of the colon and located below the level of the ileocecal valve. It lies in the right iliac fossa upon the iliopsoas muscle. The apex of the cecum is usually located near the middle of the inguinal ligament just behind the abdominal wall.

Ileocecal Valve

Located at the terminal part of the ileum in the colon and marks the upper boundry of the cecum. The valve usually lies near the middle of a line drawn from the A.S.I.S. to the umbilicus or just below it.

The valve appears as a narrow transverse slit about 1.2 cm in length.

Appendix (Vermiform process)

Attached to the apex of the cecum usually about 2.5 cm below the ileocecal valve. Only its attachment to the cecum is fairly constant. It lies approximately 2.5 cm below McBurney's point.

Ascending Colon

It has an average length of 20 cm and extends from the cecum to inferior surface of the liver where it forms the right colic flexure. It reaches the liver, lateral to the gall bladder.

The ascending colon lies anterior to the right kidney and iliacus and quadratus lumborum muscles. Some coils of the ileum usually separate it from the anterior abdominal wall. The hepatic flexure is at the level of the second or third lumbar vertebrae.

Transverse Colon

It has an average length of 40 to 50 cm and extends from the under surface of the liver to the spleen. It is smaller in caliber than the ascending colon. The liver, gall bladder and stomach are located above (cephalic) while the coils of the small intestine are located below.

In the standing posture the transverse colon may lie anywhere from above the iliac crests to level of the pubes.

Descending Colon

Extends from the splenic flexure to the pelvic brim and is covered anteriorly by some coils of the small intestine and rests posteriorly against the lower pole of the left kidney and quadratus lumborum muscle.

It terminates by crossing the psoas muscle and external iliac vessels to join the sigmoid colon.

Sigmoid Colon

Extends from the descending colon to the rectum. At its start it is at superior rim of the minor pelvis and its termination is anterior to the sacrum at the level of S2 or S3.

It moves from the left pelvic wall toward the right pelvic wall then bends back on itself to move toward the left until it reaches the midline and ends in the rectum. Its junction with the rectum forms a very acute angle and is also the narrowest part of the entire colon.

The sigmoid colon usually has more or less contact with the bladder, uterus, loops of the small intestine and at times may contact the cecum.

Rectum

This is the terminal portion of the colon and lies anterior to the lower portion of the sacrum. It can be divided into two sections, the rectum proper and the anal canal.

The Rectum Proper is approximately 10 to 12 cm long and internally presents three transverse folds, the valves of Houston. This section of the rectum is in contact with the coils of the ileum anteriorly and with the sacrum posteriorly. In the male it contacts the bladder, the vesiculae seminalis, ductus deferentes, and the prostate. In the female, it contacts the cervix of the uterus and the vagina.

The Anal Canal is approximately 2.5 cm to 3.5 cm long and passes through the pelvic floor and ends at the anus. The mucus membrane has a series of vertical folds known as the Rectal Columns of Morgagni.

Anus

This is the aperature by which the intestine opens externally and contains the sphincter muscles.

Areas of the colon free to move extensively because of the attachments of the peritoneum:

Cecum
Appendix
Transverse Colon (most movable)
Sigmoid Colon

Areas of the colon not free to move to any great degree because of the attachments of the peritoneum:

Ascending Colon
Descending Colon

Areas of the colon have less than free movement but not greatly restricted:

Rectum
Anal Canal

Movement or displacement of the rectum and anal canal is most likely caused by weakness in the muscles of the pelvic floor.

References

This paper was a composite of references from as early as Morris's Human Anatomy edited by J. Parsons Schaeffer, 1942 to the most recent Grays Anatomy. This was necessary because the terminology varied from year to year.

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SPHENOID ROTATION IN THE CORRECTION OF DURAL TORQUE PROBLEMS

BY
DARREL W. HESTDALEN, B.A.,D.C.

Abstract:

The correction of sphenoid rotation has been significant in achieving a lasting correction of the dural torque problem.

Introduction:

Stress on the dural membranes of the spinal cord and its extension into the cranium is an important consideration in human functioning. The torque on the dural membranes created by gait abnormalities is of primary importance in the correction of numerous problems.

Dr. George Goodheart has described the dural torque mechanism and its correction.[1] The correction of the femoral head angle into the acetabulum is necessary to alter the gait dysfunction. Review of the anatomy of the dural tube system makes it seem likely that a corresponding fixation or abnormal anatomical relationship could exist at the opposite end of the tube system.

The spinal dura mater is attached at the sacrum at the inferior aspect and the foramen magnum, second and third cervicals at the superior aspect. The dura is continuous with the cranial dura mater and extends in the form of the falx cerebri, falx cerebelli, and the tentorium cerebelli. The tentorium cerebelli is attached to the petrous portion of the temporal bone and to the anterior and posterior clinoid processes of the sphenoid.[2,3,]

Upledger and Vredevoogd have written that the dural tube system can exert and influence upon the tentorium cerebelli and that tension at either the inferior or superior ends of the dural tube may be transmitted to the other end.[4]

This transmission of tension is of prime concern with the function of the sphenoid as it can be altered by dural torque problems resulting from walking with an unequal stride.

Procedure:

18 female and 12 male patients,ranging in age from 11 to 72 years of age, were found to have a dural torque problem and were re-examined after various periods of time. The patients were tested for toe turn in, piriformis and psoas function in supine and supine gait position, contralteral blocking of the glenoid and acetabulum, and E.I.D.. In the prone position they were checked for first rib tenderness and categories 1,2,and 3. Category 1 was the most frequent finding and would therapy localize with one hand dorsal and the other hand palmar over the sacroiliacs. For a detailed description of the testing procedures see the 1983 Workshop Manual.[5]

After correction of the category findings and the femoral head the patient was returned to a supine position and tested for E.I.D.. All patients showing a positive E.I.D. would show a positive therapy localization and challenge to the sphenoid. The sphenoid was challenged by pushing cephalward on the cruciate suture and by rotating the sphenoid A-P on one side and P-A on the other side. Correction was done as indicated by challenge on the phase of respiration that abolished the positive challenge.(See figure 1) Occasionally the correction included a slight cephalward- caudalward distortion as well. Proper vectoring of the challenge will indicate the proper correction.

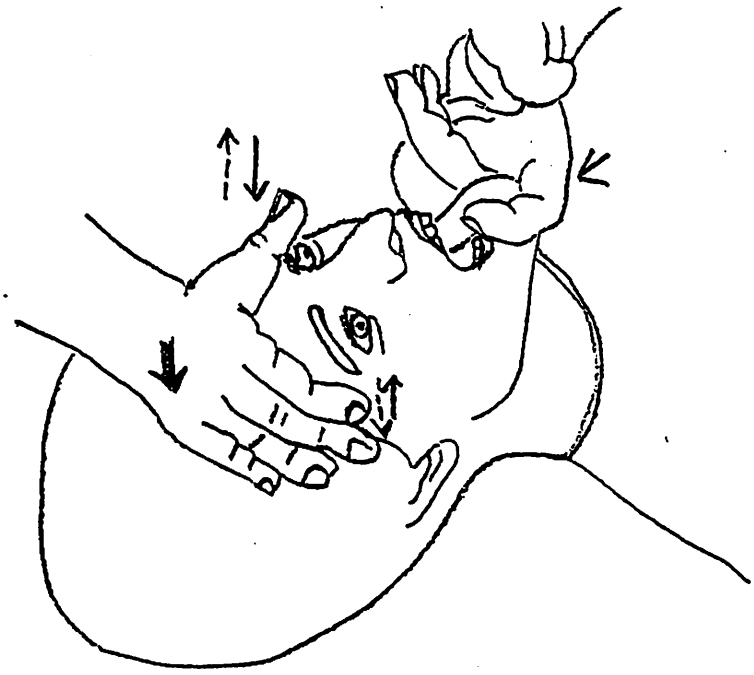
Of the 30 patients found to have a dural torque problem and were followed with a re-examination, only 2 were found to have the problem return. They were re-examined at the shortest 1 week and the longest was 7 weeks after the correction. One of the patients that had a reoccurence was a more difficult than average patient with several complicating problems. Both of these patients have retained a correction for at least 2 weeks after the second correction.

Discussion:

Review of the anatomy of the dural tube system and the opinions of Upledger and Vredevoogd correlate with the findings of this clinical research. The E.I.D. phenomenon also correlates with the sphenoid rotation as this would stress the attachments of the ocular

musculature with the sphenoid.[6] Correction of the dural torque mechanism as described by Goodheart[7] with the addition of the sphenoid correction,as described here, has shown to be of lasting quality in this study.

The clearing of the dural torque problem has shown to be a valuable aid in the correction of numerous problems in the patients in my practice.



-Figure 1-
Hand positioning for challenging and correction of sphenoid rotation.

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Computer Clearing and Positive Therapy Localization

by
Darrel W. Hestdalen, B.A., D.C.

Abstract:

A means to clear adaptations to elicit a positive therapy localization of an abvious problem is presented. Specific acupuncture points, pulse diagnosis points, and alarm points are used to determine areas of adaptation in the meridian system and the musculo-skeletal system.

Many times therapy localization(TL) to an obvious problem has not produced a positive test. (i.e. a previously strong indicator muscle shows weakness while touching a designated area of the body.) Several methods have been offered to achieve positive TL including dorsal hand contact, RNA, moistening the fingers, etc..

I feel that if the body is functioning normally, and a problem exists, TL to that area should produce a weakness in a previously strong indicator muscle.

Dr. Alan Beardall has presented a model for understanding the adaptive processes the body may utilize if a problem can not be resolved promptly.[1] Beardall has also presented a computer model for understanding the functioning of the nervous system.[2] Guyton's Medical Physiology also presents a computer model of the nervous system.[3] Beardall has determined specific acupuncture points to be key factors in the normal communication of the nervous system.[4]

Dr. Dan Gleeson presented a system evaluation to diagnose neurological interference and other faults within a meridian circuit. This is used to determine subluxations, cranial faults, adhesions, meridian imbalances, and nutritional needs.

The acupuncture points associated with the local, spinal, endocrine, and primary computer levels, as determined by Beardall,[4] are illustrated on the chart on page 3. If the patient TL the area of concern and the test is negative, have him/her continue to TL the area while the acupuncture points are TL. When a positive test is found, hold that point and the area of concern until a pulse is equally felt at both points. If the hyoid is the positive finding it needs to be challenged and corrected with golgi-tendon and spindle cell therapy. This has

shown to be faster and as effective as pulsing in S9. Usually only one point is found to be involved but several points may need to be treated.

If a positive TL to the area of concern is not achieved after the acupuncture points are cleared then go to the pulse diagnosis points. The patient should TL the area of concern and the Dr. can quickly screen the pulse points. Determine the meridian system involved and have the patient TL the alarm point and the area of concern. I have found the quickest way to screen for subluxations and adhesions is to use the pinch and scratch method. Clear the subluxations and adhesions that are found and check for a positive TL to the area of concern. If the TL is negative return to the alarm point and search for cranial faults, meridian imbalances, neurolymphatic, etc. Usually clearing the subluxation, adhesions, and cranial faults will produce a positive TL to the area of concern.

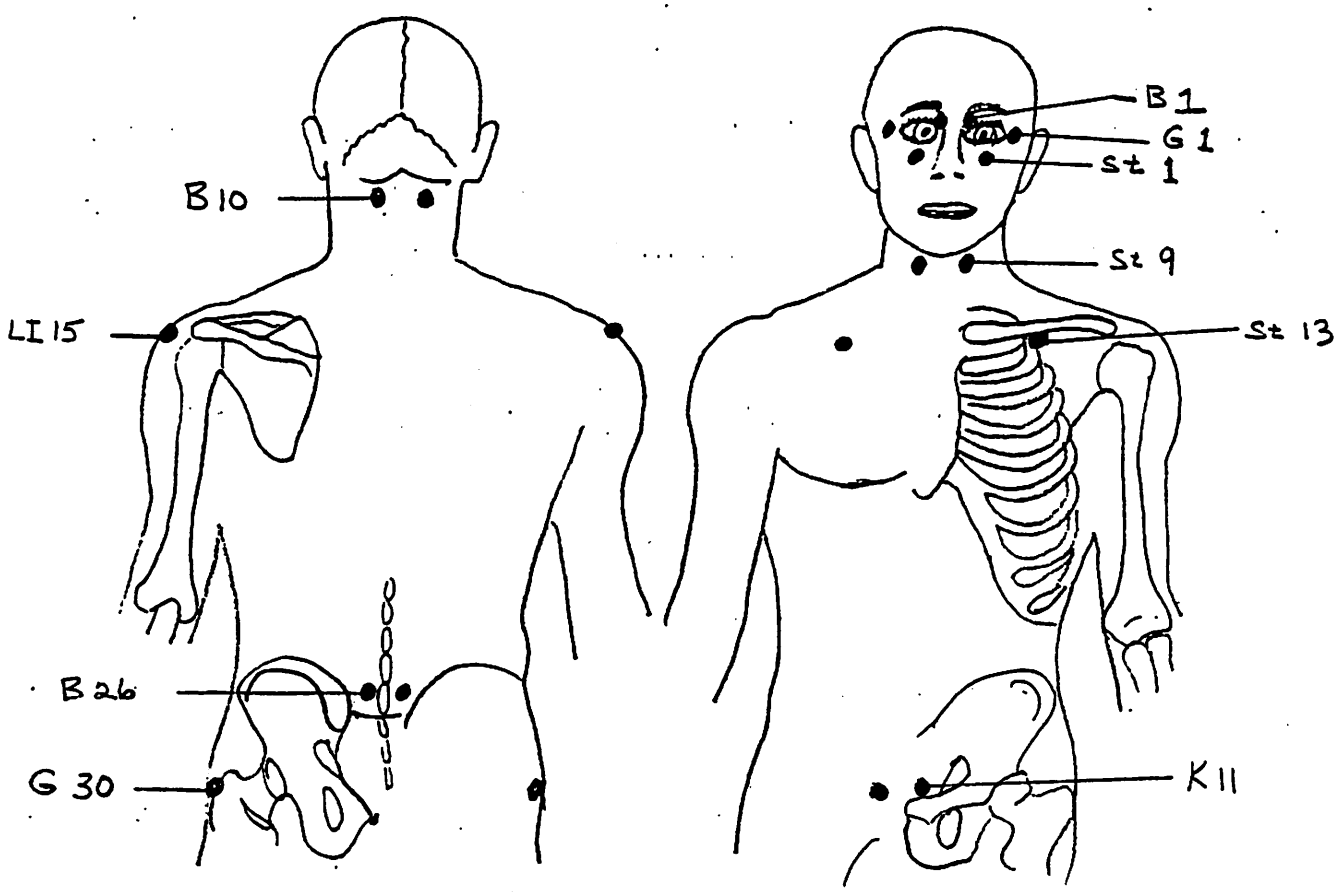
I have found this procedure often times will clear other areas to show a positive TL, if there are multiple areas of concern. But sometimes it will require repeating the procedure for another area.

It appears that the acupuncture circuits and the musculo-skeletal adaptations occur in order to allow the body to continue to function by covering up a problem. I refer you to Beardall's description of this process for a more complete illustration.[1] Unlocking the circuits allows the brain to focus in on the problem to direct the healing process and allows the applied kinesiologist to make a better evaluation of the problem.

Once the positive TL has been achieved, use your preferred means of determining the subluxation, ligament, muscle, nutritional need, cranial fault, emotional involement, etc..

I have found that this procedure can easily be accomplished in 5 minutes. It has helped me save time by removing adaptive problems that do not need to be treated and leading me to a problem the body can not resolve without outside help.

Chart of acupuncture points related to computer levels.



Local computer- St 13, K 11, B10, B26

Spinal computer- G30, LI 15

Endocrine computer- Hyoid or St 9

Primary computer- B 1. St 1, G 1

Those underlined have been found more often involved.

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Dr. Stephen J. Kaufman

Extensive Lymphatic Drainage Technique

Abstract: A simple technique is described for greatly enhancing general bodily functioning.

All kinesiologists are aware of the vital importance of the lymphatic system for general body functioning, and we all use a variety of lymphatic enhancement techniques for specific corrections. The usual approach is to identify the problem area or areas and apply as many varied and diverse kinesiological techniques, lymphatic and otherwise, to that specific area as practical.

Applied Kinesiology, in addition to its tremendous clinical usefulness in clearing up specific health problems, also has much value in raising a person's energy level and feeling of well-being, and stimulating the body to clean itself out. A technique we find quite useful is to strongly stimulate the lymphatic system by testing and correcting pectoralis minor stretch weakness and/or fascial involvement. Then TL and treat as many active neurolymphatic centers as you can, especially related to the thymus, liver, stomach, pancreas, small and large intestine. Treat these vigorously, and re-test and re-treat under all high gain

conditions; RNA, opposing thumb-little finger, scratching, right and left - brain, neurolymphatic junction technique (described elsewhere) EID, cerebellar make and break contacts, palm up/palm down and mixed, contralateral A-P lymphatic technique, breath holding, local and temporal tapping, etc. This may take quite some time, so you may want to train an assistant to do this, or have the patient schedule two visits the same day. (Our office always charges for whatever additional techniques or time the patient requires.) At any rate, really stimulating the lymphatic system strongly, especially through the stomach, thymus and liver, has been productive of excellent response. We will devote one or more sessions solely to this, and do no other adjustments or cranial procedures, etc. At the same time, very frequently, patients will feel the next day like they've "been hit by a truck", and of course the areas stimulated will be very sore, but following this there is usually a marked overall improvement in the patients condition, both generally and frequently symptomatically as well.

This concept is of course nothing new. The key here is to focus for the time being <u>solely</u> on neurolymphatic treatment, and to <u>thoroughly</u> and <u>completely</u> clear up especially the stomach, thymus, liver and intestinal NL's. The extra time involved is necessary but well worth the results.

Dr. Stephen J. Kaufman

Cranial/Gait Relationship

Abstract: A slight modification of testing in gait position is reported, with outstanding clinical usefulness.

At the summer meeting of ICAK, and on research tape #79, Dr. Goodheart presented the concept of testing a patient in gait position for uncovering acupuncture pulse therapy localization and hidden Category I's. He further suggested TL'ing associated point-spinal subluxations and other 5 finger IVF factors in gait position. Since then, this author has been challenging (not TL'ing) cranial faults with the patient in a prone gait position (the hypertonic psoas leg advanced over the side of the table.) After the cranial faults are negative to challenge or TL in a normal position, very frequently one will find, in gait position, when challenged, universal, sphenobasilar, glabella pituitary drive, and especially occipital counter torque faults, as well as a lesser occurring frequency of the other known faults. These are then corrected, if possible, with the patient remaining in gait position. Therapeutic results have been outstanding, with patients reporting a great improvement in head and neck tension and pressure, and brain clarity and function.

Reference

Dr. George Goodheart, Research Tape #79.

Dr. Stephen J. Kaufman

Neurolymphatic/Neurovascular Junction Technique

Abstract: This paper presents an unusual method of finding hidden neurovascular or neurolymphatic involvement.

This author has frequently wondered why different muscles have the same neurolymphatic and neurovascular turn-on centers, e.g. the gluteus medium and the piriformis have the same neurolymphatic above the pubic symphasis, the TFL and quadriceps have the same neurovascular point at the parietal eminence. The idea was conceived that perhaps, among other functions, these areas served as some sort of junction point or relays between different muscles, and if the relays were not working properly, muscle function would be interfered with.

A patient was tested with intact TFL and quadriceps. These were tested sequentially several times. No reactive muscle situation was found. The quadriceps/TFL NV was then therapy localized. No weakness was found.

However, when the patient continued to touch the NV point and the quadriceps was tested and then the TFL, the first muscle tested did not weaken, but the second muscle tested did! (The order of the muscles tested did not matter; in fact, after the second muscle weakened, the first muscle weakened when tested again, as long as the patient continued to hold the NV.) In other words, the neurovascular showed no neurologic abnormality or

weakness when it was therapy localized against one of its related muscles, but when several muscles related to it were tested, it was apparently incapable of acting as a shunt and it blew out.

Further investigation showed that this phenomenon held true for other muscle/reflex combinations. TL to the pubic symphasis was negative, and no reactive muscle combination pertained to the piriformis/gluteus medius tests, but when testing the piriformis and then the gluteus medius while the patient continued a contact on the pubic symphasis, a weakness occurred which then persisted on both muscles as long as the patient maintained the contact. Treatment of the indicated point, whether neurolymphatic or neurovascular, abolished the presence of this type of weakness.

Dr. Stephen J. Kaufman

Cervical Counterstrain Technique

Abstract: A clinically useful technique for the cervical column is presented.

One quite useful school of thought in osteopathy is called counterstrain therapy, developed by Lawrence Jones, D.O.. Methods of therapy localizing for this technique are in research, but one useful technique is herein presented.

Have the patient turn his/her head to one side, and test a strong indicator muscle. Generally it will stay strong, barring the presence of a cervical subluxation. Have the patient roll his eyes to the same side the head is turned. No weakness should occur, barring the presence of an ocular lock. Now have the patient roll his eyes to the side opposite the way the head is turned. (If the head is turned to the right, have the patient shift his eyes left.) If no weakness occurs, repeat on the opposite side. Frequently, the patient will weaken with the head turned one way and the eyes shifted to the opposite side. This is conceptually an indication of a torqueing in the dura mater, which attaches to the investing fascia of the eyes. Turning the head and shifting the eyes puts an undue further strain on the dura and causes a weakness.

Cervical Counterstrain
Technique
Dr. Stephen J. Kaufman
Denver, Colorado

To correct, have the patient relax his head and neck in your hands, and turn his head as far as it will go to one side while the patient shifts his eyes in the opposite direction. (It will be hard for him to relax and shift his eyes at the same time). Just hold his head in this position for 6 to 8 seconds. Now roll his head the opposite way, and have him again shift his eyes opposite the way his head turns, and hold for 6 or 8 seconds. Repeat this procedure 5 times for each side, and then turn the head with the patient's eyes rolling in the same direction as the head turns, and repeat this procedure for the same length and number of times. In other words, once a weakness is found, turn the patient's head to the right, and have him roll his eyes back to the left, and hold. The have him turn his head to the left and roll his eyes back to the right. Repeat this procedure five times. Then have the patient roll his head to the right and have him roll his eyes to the right, also. Then have him roll his head and eyes to the left and hold. Repeat.

Whether or not this technique "unkinks" the dura is hard to say, but it definitely relaxes the neck and reduces cervical symptomatology.

Reference

Strain and Counterstrain - 1981. Lawrence H. Jones, D.O. American Academy of Osteopathy, Colorado Springs, CO.

Dr. Stephen I. Kaufman

Precise Determination of a Category II

Abstract: A method is presented which reveals a far greater number of Category II sacroiliac problems than was previously thought.

According to Applied Kinesiology thought a one-handed palmer or volar therapy localization to the sacroiliac joint is generally indicative of a Category II osseous sacroiliac slip, whereas a two-handed TL indicates a Category I fault of the respiratory boot mechanism. The SI joint can also be challenged. Dr. Goodheart in the past has implied that a Category I problem is a frequent occurrence in the patients he sees, whereas most SOT practitioners feel that 85% of the first-time patients they see are category II's. These doctors, of course, usually define a Category II by the arm fossa test, as opposed to therapy localization or challenge.

In an effort to clarify this issue, the present author has exhaustively searched for Category II lesions on his patients for the past four years. Our experience is summed up as follows; when a patient's SI joint is TL'ed or challenged only (without regard to any other testing) a Category II problem will frequently fail to reveal itself. Category I's seem much more common.

However, if the arm fossa test is first performed on both sides, and the SI joint is then re-challenged, a Category II is

frequently present. In other words, performing the arm fossa test itself allowed the Category II to show up by challenge or TL.

Of course, the arm fossa test can be used alone to determine the presence of a Category II. In addition to merely testing the fossas in the clear, testing each fossa with the following additions will reveal many more Category II's:

1) Test each fossa during held inspiration.

2) Test during held expiration.

3) Test each fossa with the patient TL'ing the anterior fontanelle with the opposite hand. (This frequently reveals a weakness, and is considered a form of switching by Dr. David Denton.)

4) Test after patient chews RNA.

5) Test with patient TL'ing the Now point (be certain the now point does not TL in the clear, without fossa contact).

The complete testing for a Category II thus takes a minute longer than merely TL'ing or challenging. However, when tested thoroughly in this

fashion, we find Category II's almost ubiquitous on first patient visits. Correcting them usually has the patient returning the next visit with a Category I, which is then corrected, restoring normal pelvic mechanics.

To remind you, when a Category II is corrected, check and correct the following in the case of a reoccurring problem;

1) Sartorius and gracilis on the posterior illium/ short leg side.

2) Hamstring, adductor, and quadriceps on the anterior illium/long leg side.

3) TMJ (usually closed, usually ipsilateral to the involved SI.)

4) Cranial sutural release, as taught by DeJarnette.

5) Skin adjustment over the SI joint.

6) Neurolymphatic drainage three spinal segments above the SI joint, usually contralaterally. (TL simultaneously with the SI joint).

Precise Determination
of a Category II
Dr. Stephen J. Kaufman
Denver, Colorado

7) Tapping the SI joint for bone memory.

8) Ligament interlink with the contralateral sternal-costal joints.

9) Intraosseous lesions of the illium.

10) Extraspinal segments related by Beardall's bone sequence (atlas, cuboid, temporal bone, triquetrum, etc.)

PRELIMINARY OBSERVATIONS OF EXERCISE, FITNESS AND TRAINING

by

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

ABSTRACT: Exercise is an excellent means of building and maintain one's health. Proper procedures are necessary to attain a high efficiency of exercise, thereby avoiding the pain and frustration of injury and ill health. If and when a disability occurs, it is an indication of 1) improper function (whether structural, biochemical, and/or mental); 2) poor exercise habits which are often due to aerobic deficiency and anaerobic excess; and 3) poor exercise techniques, which include training for the competitor. The scope of this paper will include the "average" person who attempts regular exercise, some of whom compete on a local level for enjoyment, and the competitive athlete who has attained higher levels of fitness and competition. The principle objective is to show the problems of present thought regarding the economy of exercise and to offer solutions. These solutions result in improved health, and in the case of the competitor, improved performance. Discussions will center around defining and redefining aerobic and anaerobic exercise including the neurological and nutritional components of each activity. As one exercises, or trains, specific phases are encountered. These phases are cyclic, like the seasons, and include neuromuscular, hormonal and cardio-vascular. Factors that inhibit efficiency and progression relate to these phases and will be discussed. Running will be used as a standard; however, similar if not identical attitudes can be held for other types of activities and competition whether aerobic or anaerobic in nature. It will also be shown that increased and improved aerobic activity will result in an increase in anaerobic efficiency.

In the last 15 to 20 years, hundreds of articles and books have flooded our population resulting in what is now referred to as the "exercise boom". Many professionals have seen the unfortunate aspects of this trend in the form of injuries of various types. Some of the more popular books have been authored by professionals, coaches, and athletes themselves. (1, 2, 3, 4, 5)

The problem with exercise, as it is done today, is that most individuals are exercising at a low level of efficiency, often resulting in a high level of stress. Selye (6) had researched and written extensively about stress. Goodheart (7, 8) has clinically adopted diagnostic and therapeutic techniques related to structural, chemical and emotional stress. These procedures that are utilized in Applied Kinesiology can and should be adopted to the athletic individual. Goodheart and Schmitt (9) have discussed some of these procedures.

The author has had personal contact with various coaches, including Bill Dellinger, 1984 U.S. Olympic coach, and Arthur Lydiard. World class athletes such as Grete Waitz, Matt Centrewitz and British olympian, Geof Smith, have also been treated by the author, as well as other nationally ranked runners. He has also worked with various athletic organizations including Adidas and Athletics West (Nike). Formerly known as a nationally ranked runner, the author is still running and competing.

When asked why they exercise, many people will give a variety of answers, most of which are unfounded. The purpose of exercise is to improve one's health, both mentally and physically. All too often, however, the rules are broken with the result being a <u>loss</u> of health.

A, B, AND C FIBERS

In order to understand these rules, definitions are needed. The word "aerobic" has become a popular term. Skipping the academic definitions and relating the word aerobic clinically to exercise, it can be defined as "steady, uninterrupted, and daily". (1) It is an activity which should be monitored by the individual. Physiologically utilizing aerobic muscles will result in predominantly burning fat as an energy source, instead of glucose. Goodheart (10) has described some important relationships between aerobic and anaerobic muscle function. Montcastle (11) describes aerobic and anaerobic function from a neuromuscular standpoint. Baily (1) also evaluates aerobic and anaerobic activity, allowing the reader practical applications. Henneman and Olson (12) discuss the characteristics of A, B, and C fibers, relating to anaerobic, mixed, and aerobic function respectively. The blood and lymph supply is greatest around the purely aerobic or C fibers, less around the mixed or B fibers and least around the anaerobic or A fibers. It is thought that a single motor neuron innervates a specific type of muscle fiber.

In other words, there are three types of neurons that innervate the three types of muscle fibers. (See Figure 1) C, B, and A fiber activity becomes an important relationship regarding exercise, and especially "training". Aerobic muscle fibers are oxygen consuming. Their red appearance is due to the iron containing myoglobin. They function as slow twitch endurance fibers innervated by small diameter neurons and have mitochondrial enzyme systems predominantly for fat burning.

The anaerobic fibers are void of myoglobin and are termed "white". They are "fast twitch" and short duration in function, innervated by large diameter neurons, and predominantly burn glucose due to the need for a rapid energy source.

It is this author's belief that any exercising individual, whether competitive or non-competitive, should utilize aerobic muscle function 80-95% of the

NEUROMUSCULAR RELATIONS

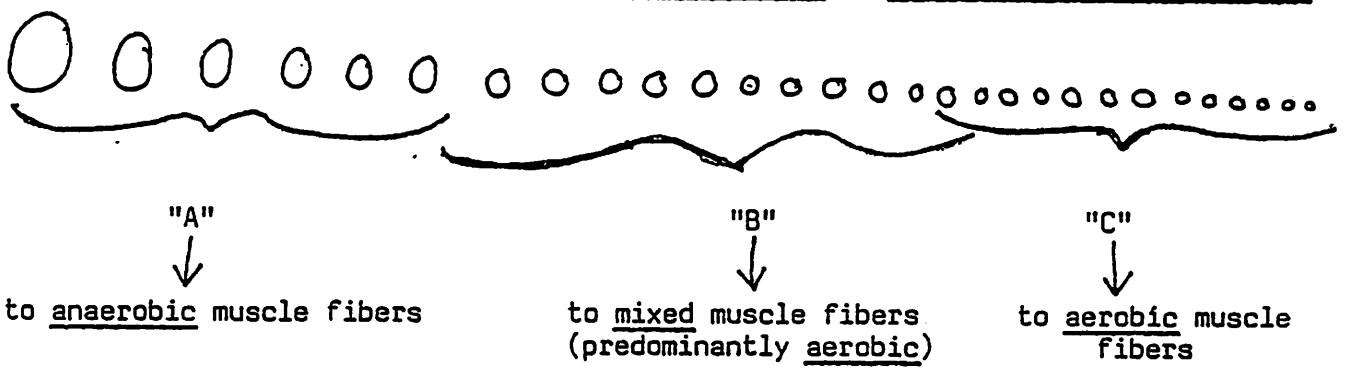


FIGURE I

STAGE	PHASE I	PHASE II	PHASE III
	1. Neuromuscular	1. Neuromuscular	1. Neuromuscular
	2. Hormonal	2. Hormonal	2. Hormonal
	3. Cardiovascular	3. Cardiovascular	3. Cardiovascular

FIGURE 2

time during exercise or training. From a competitive standpoint, this would translate into improved competition and a longer competitive life.

We should realize the "total person" ([13]) concept of health, viewed as an equilateral triangle. The structural, chemical and mental aspect of one's health is influenced by these factors. A certain balance or homeostasis is required to maintain good health.

MAXIMUM AEROBIC PACE (MAP)

As we monitor patient progress, so we must monitor exercise progress. As the body in general works harder, or walks and runs faster, we generally see an increase in the heart rate. This change in heart rate can be used as a monitoring system. Having the patient manually check the pulse during activity is often very subjective and difficult. Most runners who stop to check their pulse obtain inaccuracy within the six to ten seconds they are counting the pulse. A person in good condition will have a dropping of the pulse rate anywhere from one to one-and-a-half beats per second, if starting at 160 beats per minute. From a standpoint of heart recovery, this is good. But it becomes difficult to check the pulse accurately during running. The author has found that many runners, while running on a track or road are constantly perceiving their pulse rate to be lower than it actually is. I have used many devices for checking pulse rates and have settled on a monitoring device from Respironics Inc., 650 Seco Road, Monroeville, Pa. 15146. This picks up the heart rate directly from the chest wall rather than from a peripheral pulse. Monitoring the pulse <u>during</u> activity is essential, as this becomes the determining factor for an aerobic state. The first step is to determine what the <u>maximum</u> pulse should be to maintain an aerobic state.

Baily (1) describes several ways, giving formulas to determine a pulse range. I use several other factors including the Dynavite Bike, modified Masters-Two-Step, exercise history, pulse pressure, and the general health of the individual. Running with the heart rate in this range then, would indicate the person was exercising aerobically. This becomes a practical method for monitoring an exercise. It is not practical to draw blood samples or do other elaborate tests daily just to see what state (aerobic or anaerobic) a person is exercising in. This will also replace the old fallacy that runners have regarding aerobic running. Some say that if you can easily carry on a conversation while running, then you're not overtraining. I was a sprinter in high school and college and, as a result, can carry on a conversation at a very fast anaerobic pace! Many people have run themselves into the ground using this old idea. Once you find the maximum pulse rate, subtract 10 to get a "range". For example, if you determine that in order to run aerobically your heart range would be at maximum 140-150 beats per minute, you could run at that heart rate in general without regards to pace. The time of the activity and heart rate are the two most important factors. Present ideas dictate that pace and distance are most important. However, time and heart rate relate to quality where pace and distance relate to quantity. Exercise must be qualitative before it can be quantitative. The minimal time needed to benefit aerobically is 12 to 15 minutes. The fastest that one can run within their heart range is called the maximum aerobic pace (MAP). This MAP will increase as time goes on, as will be discussed later. There are many factors that will inhibit progression and efficiency of exercise. These will also be considered later on.

PHASES OF EXERCISE

If you have exercised before, then you are aware that there are certain

phases your body goes through to accomodate the increased stress of exercise.

The author contends that three phases exist with three specific aspects in each stage (see Fig. 2).

Phase I is the beginning phase of exercise. As a person starts on a walking or running program, demand is placed on the neuromuscular relationships which must compensate to accomodate (it is assumed that mechanical, biochemical and mental factors that inhibit progression do not exist, as will be discussed). The hormonal and cardio-vascular aspects of the individual obviously function as well, but the muscle and nervous systems lead the way. Overexercising in this stage results in excessive soreness or pain, sprains and strains. It is quite similar to local trauma to the muscle as one is going beyond the neuromuscular capacity of the muscle. Therapeutically, origin and insertion technique may be used when indicated. Calcium, Vitamin F and Vitamin B (<u>not</u> B-Complex) should be ruled out. Water is often needed by these individuals as dehydration is very common. Within a short period of time (a week to one month), the second stage begins playing a larger part. This stage is hormonal in nature; the adrenals should not be excessively stressed. However, due to human nature, we tend to make attempts at speeding up natural processes. If this happens, a "typical" hypoadrenia reaction will soon follow. Progression will stop, often accompanied by a secondary physical breakdown (ankle, knee or hip, for example, which relates to posterior tibial, or sartorious function). These people often become cranky and irritable due to a low blood sugar level and dietary habits should always be evaluated.

In evaluating the heart rate of these individuals, <u>during</u> running it is often well above their MAP, and they are functioning in an anaerobic state. The remedy is often as simple as slowing them down.

Properly monitored to insure an aerobic level, a person should progress through this stage within 2-4 months. With the neuromuscular components and the hormonal relationships still improving, the body slowly transfers its emphasis to the cardio-vascular system. The first sign of this stage is a significant decrease in the resting heart rate (best checked before getting out of bed in the morning). There is an increase in function and development of arteries, arterioles, capillaries and veins andthe myocardiam becomes stronger and more efficient. During this stage, the person is usually running farther, and if properly monitored, will be running faster with the same effort. With longer exercise times, there is an increased demand on capillary beds and they are encouraged to develop further. Rarely will a person break down in this stage, if over-exercised. Rather the body will usually force itself back to the hormonal stage as an attempt to "try again". Those individuals who break down in Stage 3 will have the "heart attack" while running, as is heard about all too often. This problem will not be picked up on EKG or stress test unless it is given while the person is in Stage 3. These test are often given during Stage 1, before the person embarks on a fitness program or soon after. This is just another reason why monitoring a person as well as giving follow-up care is important.

Phase II is an intermediate phase with the same stages-neuromuscular, hormonal and cardiovascular. Many people can enter Phase II within a years time, depending on their discipline, care and genetic make-up. There is a common indication of this Phase while running (especially with a heart monitor apparatus). You feel you need to run faster than you would like in order to keep the heart rate within the aerobic range. For example, while wearing a monitor, you may be quite comfortable at an eight minute per mile pace. Your heart rate is only 125, but your aerobic range is 140-150. You will still benefit at a heart rate

of 125 and you will maintain the benefits that have been developed thus far. However, if you want to lower your MAP, some running at that level (within a heart rate of 140-150) must be done. Between this first stage and the second stage of Phase II, are where most injuries occur. Screening procedures (as will be discussed) are important here. Protein metabolism seems to be extremely important in the first and second stages of Phase II. Rubel[14] and Schmitt[15] discuss the importance of protein needs. Checking the blood for globulin levels is a simple procedure. A globulin above 2.9 is an indication of poor protein utilization, in that the body is stealing protein from itself. Often times in this stage (Phase II, stage 1), a low globulin will be seen of which Chusid [16] shows myosin to be globulin X. Applied Kinesiology procedures should obviously be used to evaluate all aspects of metabolism, especially the digestive tract and the liver.

Stage 2 of Phase II as mentioned relates to hormonal influences. Thyroid hormone begins to play a more important role as the individual continues to exercise more and more. If thyroid function is good, then entering the third stage - cardio-vascular - will be easy and efficient. This correlates with Barnes [17] in his finding that patients susceptible to heart attacks usually have poor thyroid function. Stage 3 in Phase II is virtually the same as in Phase I; but the individual may now be referred to as an "athlete". They are often running longer runs (10 - 15 miles) whereby they are sometimes totaling 40 - 50 or more miles per week. Again I emphasize that this is still "aerobic" as it is defined. At this point in time, our athlete has often been tempted into competition (which is strictly anaerobic). This adds another possible means of overtraining as excessive competition can be excessive stress. However, as will be shown, if the regular day to day exercise, in this case running, is aerobic, the person is safe and still building their health. Certainly

by this phase and stage, seasonal competition, even once or twice a month is healthy. The athlete will still have 80-95% aerobic activity in the course of a week's time. From a training standpoint, anaerobic activities can begin.

A good next step would be to have the individual develop the medium sized neurons that innervate the B fibers. Again, these fibers are mixed, being predominantly aerobic with some anaerobic function. This is the aspect of exercising and training that is so often neglected. It is at this point in time that the person will start doing anaerobic exercises to excess in the form of interval workouts and harder daily runs. This is uneconomical, as the B fibers never fully develop. An excellent way to do this is to have the individual run at the MAP, perhaps twice a week, with a gradual increase in distance. This is a very high level of aerobic activity so the runner should experience no soreness or pain. This will gradually bring the MAP down even further, and for the competitor translates to faster race times. Phase III is usually reserved for the national or world class athlete, or for those individuals who can devote a good amount of time to training. It is a continuation of the cycle-neuromuscular, hormonal, and cardio-vascular. Further discussion of Phase III is beyond the scope of this paper.

The Exercise Triangle

Many people are content staying at the beginning of Phase II. As a matter of fact, running 2 miles per day will eventually get you there. It is important to completely go through Phase I to obtain the health benefits of exercise which includes lowering the total percent body fat.

The system of exercise and training today relies heavily on chance to properly and wholistically train the athlete. A runner needs to develop muscle fibers that are innervated by all different sized neurons, which means exercising

through various intensities. Without an efficient monitoring system of checks and balances, this is rarely done. When the runner "skips" steps, he or she is less effective a competitor and more vulnerable to injury or systematic breakdown (depending on the phase and stage of breakdown).

The exercise, or training triangle (see Figure 3) shows, from bottom to top the proper procedural program that will 1) build and maintain health, and 2) enable the athletic individual to attain a greater level of achievement over a longer period of time. The base of the triangle is just that, a base. The running during the forming of this base has often in the past been referred to as "long slow distance" or "LSD". All too often, the runner, after building not quite a large enough base, skips to a lot of anaerobic activity. They erroneously think that in order to run fast you must train fast. Lydiard (2, 18) agrees that anaerobic training, though necessary, is overemphasized and uneconomical. Building and maintaining an aerobic base is by far the most common area of neglect in athletes and non-competitive individuals that I have seen. The vast majority of people who have developed this problem have the need to regress and redevelop (or develop) an aerobic base. This takes time (3 months to a year) and some people don't have the discipline. As the individual goes through the various phases and stages, they begin using slightly larger diameter neurons for muscle function. This happens automatically, when properly monitored. Actually their pace is down between 8 and 7 minutes per mile, they begin to use more of the B fibers which are predominantly aerobic. In this part of the triangle, the individual begins to develop stamina and endurance as the runs are longer and naturally faster. Here is where the MAP becomes important. At this point, the average pace of the run will be slower than the MAP. As mentioned earlier, the MAP will be faster but the runner will not always want to run at that pace. For example, a runner may,

Preliminary Observations of Exercise Efficiency

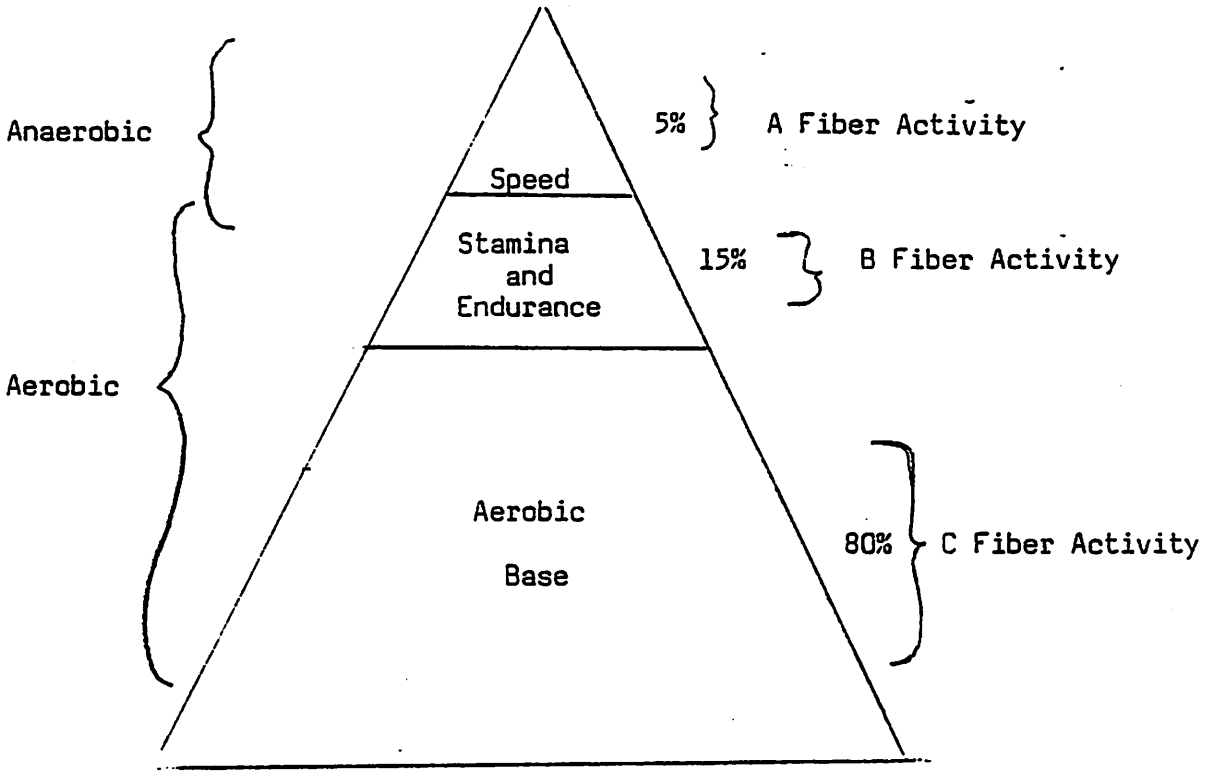


FIGURE 3

on an average day run 10 miles at a 7½ minute per mile pace, which is more comfortable. The MAP for that athlete may actually be 6½ minutes per mile. At this point in training, it is important to develop more of the MAP once or twice a week (not on consecutive days) in order to develop the B motor neuron/muscle fiber relationships. The final step before serious racing can take place, is to develop speed. This can effectively be done in 4-6 weeks at the minimum. Dellinger[19,20] has developed a system of interval training that is very efficient. Any type of speed work by the athlete should predominantly "train" the nervous system. This step continues to high level competition and like Phase III goes beyond the scope of this paper. It must be understood that as more non-aerobic activity is engaged, aerobic efficiency and progression will slow down. If a runner is attempting to build an aerobic base, but insists on racing once a week (highly anaerobic), proper aerobic progression will not occur.

When competition is entered, there exists a normal ratio between the MAP and the race pace (Fig.4).

If, for example, a runner's MAP is 8 minutes per mile, the race pace average per mile for a 5,000 meter (3.1 mi.) race should be 6½ minutes. A runner with a MAP of 6 minutes per mile would race this distance at 5 minutes per mile. This is again assuming that the factors that inhibit efficiency, as will be discussed shortly, are of little significance (i.e. iron deficiency, Category II, etc.). If the runner is not within the proper ratio, then a problem exists with training. If it is not corrected, improvement will be limited and the athletic potential of the individual will never be realized. In addition, the athlete becomes prone to injury. In the example last used (9 minutes per mile MAP/6 minutes per mile race pace), the race pace time is primary as this is actually what the runner ran. The 9 minutes per mile MAP then, is out of proportion; it should

5000 METER (3.1 Miles)

MAP (per mile)	RACE PACE (per mile)	RACE TIME
10:00	7:30	23:18
9:00	7:00	21:45
8:30	6:45	20:58
8:00	6:30	20:12
7:30	6:00	18:38
7:00	5:30	17:05
6:30	5:15	16:19
6:00	5:00	15:32
5:45	4:45	14:45
5:30	4:30	13:59
5:15	4:20	13:28
5:00	4:15	13:12
4:45	4:10	12:57

FIGURE 4

be 7½ minutes per mile. This indicates lack of aerobic efficiency and is by far the most common problem seen. This person then needs to develop an aerobic base.

Simply stated, as the MAP lowers, the race pace will also lower, having done nothing but aerobic activity. (See Figure 4) In other words, improving the aerobic efficiency (aerobic base) will improve anaerobic activity (racing). This author has utilized these relationships with runners, swimmers, cyclists, tennis players and weight lifters, many of whom were beyond local level competition.

FACTORS THAT INHIBIT EXERCISE PROGRESSION

It must be understood that any factor that inhibits normal posture and gait will inhibit exercise performance and progression. The factor(s) may be structural, biochemical, or mental in nature. Some of these inhibiting elements are discussed briefly.

We should be able to control our progression regarding exercise. In other words, if the average person wishes, they should be able to progress to the point of competition of at least national level. At that point, genetic factors and social factors (like having to work) dictate much of the progression. Too many people "plateau" much too soon. This is not normal. It means something has prevented normal progression and that something must be determined. As mentioned, lack of aerobic base is by far the most common training factor, but other factors that inhibit exercise progression also exist.

Many people become frustrated when after exercising for months, they do not progress or lose the inches or the weight, even while they are exercising properly (i.e., within their aerobic heart range). Goodheart (10) has shown that iron levels in the muscle can be low without concomitant low

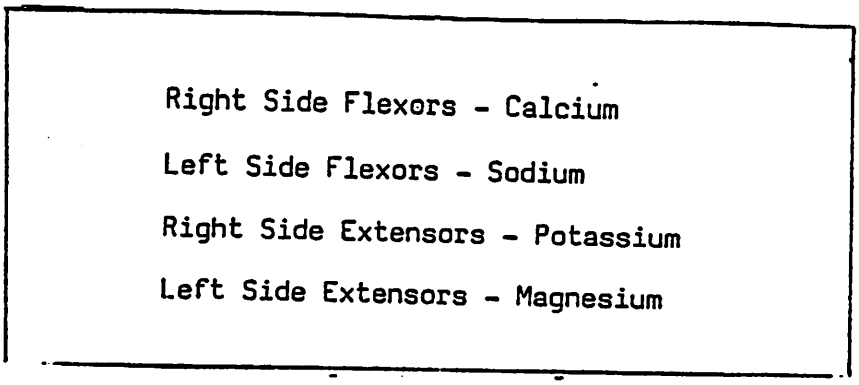
blood levels of iron. In order to find this problem, aerobic muscle testing is used. If a weakness is found, having a patient insalivate a source of iron such as Ferrofood (Standard Process Labs), will abolish the aerobic weakness. Other obvious relationships such as lymphatic drainage, dietary sources of iron, digestion and absorption, and utilization must also be considered. In order for iron to be efficiently useful, copper must be present. Mowels [21] and Schmitt [22] have shown that excessive copper will actually inhibit iron metabolism. Excess copper is stored in the liver and the brain. [23] Once absorbed, copper combines with albumin and is transported to the liver where it is stored as ceruloplasmin. Excess copper should be eliminated via the bile. This explains why when in some people with high copper levels, "pulling" out the excessive copper may mimic a gall bladder or other gastro-intestinal problem. High copper levels, which are common, can be determined by having the patient insalivate a nutritional source of copper. Schmitt [22] states that this will create a weakness in almost any muscle properly tested, being careful to allow ample time for the patient to taste the substance.

The author has found a high correlation between excess copper and, 1) people who are exercising properly and are unable to lower their fat content, and 2) athletes (mainly runners) who are unable to build and/or maintain an aerobic base. The reason again seems to be excess copper having an inhibitory effect on iron which then inhibits aerobic muscle function. Copper will also inhibit folic acid. [21] Folic acid (and B-12) may then cause defective D.N.A. synthesis.[24] Patients usually do not require iron forever. When there is a recurrent need for iron or folic acid, check for copper. Zinc and/or manganese will "pull" copper out of the system. Attempts should be made to first utilize as many factors as possible to accomplish this. In other words, fix what you find and save the nutritional aspects until the "end". All too often, nutritional products are used before any attempt is made to allow the body to do

it's own work. Fix what you find and retest for copper.

The electron poising system as described by Issacs [26] and elaborated on by Schmitt [26, 27] and Tolen relates heavily to the exercising individual (especially in Phase II) and most especially to the athlete. A deficiency of aerobic activity (and an excess of anaerobic activity) will cause a clockwise rotation of the electron poising curve, as these individuals will often show the need for pituitary drive technique.

Schmitt [21, 30] has discussed the four quadrants represented by superimposing the electron poising curve on the human body. As shown in Figure 5, specific nutrients relate to specific quadrants of muscle activity.



Observing an exercising individual will reveal specific muscle imbalances, as anyone knowledgeable in Applied Kinesiology could demonstrate. I have seen some very exciting changes while working with runners on the track and roads. Two typical examples are given:

Example I - Runner X was in the fifth mile of a run at MAP on a 400 meter track. At this point a general left sided flexor weakness was observed (this patient previously had a weak rectus abdom. and quadriceps femoris). This irregular gait was consistent for the next mile.

I then gave runner X a NaCl tablet which was kept in the mouth. Within

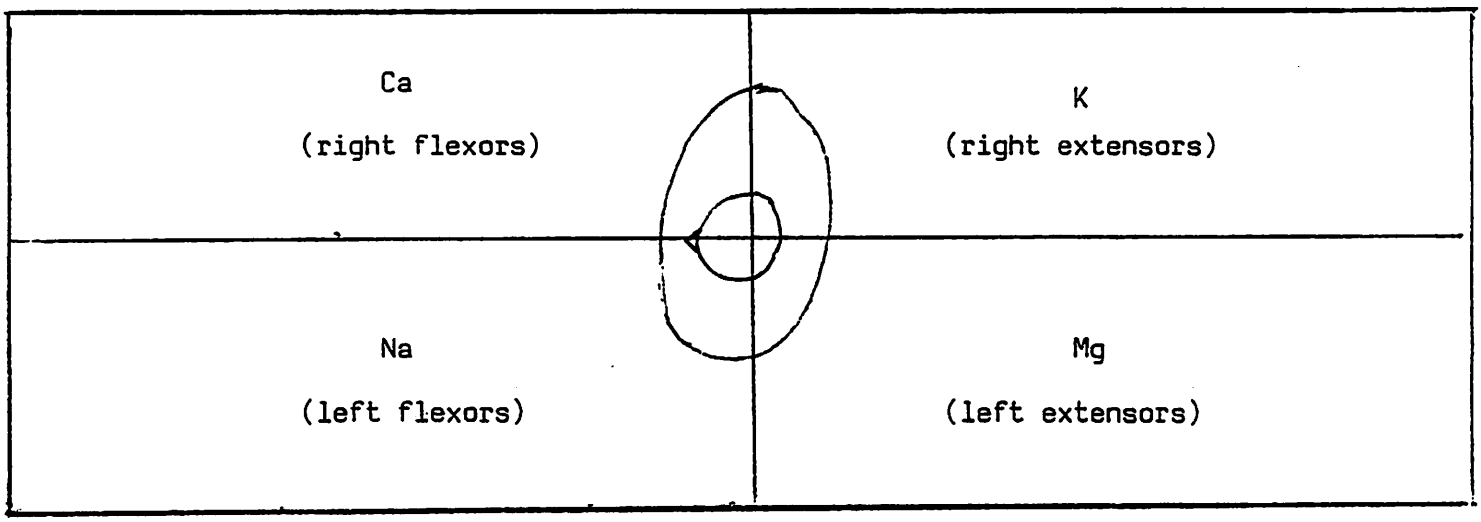

Ca
(right flexors)
K
(right extensors)
Na
(left flexors)
Mg
(left extensors)

FIGURE 5

¼ mile, the gait improved to the original pattern observed in miles one to four. It should be noted that the sound of the runner's shoes hitting the track changed dramatically.

Example II - Runner Y was observed to have an excessive right rotation with the left leg taking a longer step. This became noticeable after 3½ miles on a track, running at MAP. The runner was given calcium with no corresponding change after ½ mile. The runner was then given magnesium with little change after ½ mile. However, Calcium Lactate with magnesium (Standard Process Labs) was given and held in the mouth as mentioned above. Within 400 meters a distinct change was observed.

It should be noted that these and other runners (including most runners used for the statistics in Figure 4) had been under care; significant structural and biochemical imbalances did not exist.

Again, it should be emphasized that it is not the purpose in Applied Kinesiology to find nutrients that neutralize muscle imbalances, but to view the total person. Runner X previously had an adrenal problem, therefore, it makes sense that sodium loss under stress of running would make itself known.

CONCLUSION

These observations merely serve as an introduction to a field which has been misunderstood and abused: that of exercise fitness and training. Any individual wishing to gain from exercise in a healthy way must predominantly exercise aerobically. Up to 95% of regular activity should be this lower intensity, fat burning, efficient exercise. Without some system of monitoring, however, people will overexercise and overtrain. Using a heart monitor during activity is a highly efficient way to ensure safe, efficient and progressive exercise.

In the case of a runner, it will allow the pace to increase while still maintaining an aerobic state, which will also increase the anaerobic race pace. Other areas of performance such as swimming, cycling, weight lifting, baseball, tennis etc. will also be improved. Exercise must follow a certain progression, or order of events (physiologically) in the form of stages and phases. Trying to skip to higher levels without fully developing other levels neuromuscularly, will result in both injury and poor efficiency. For the individual trying to lose weight, inches, and get in shape, skipping steps means frustration, discomfort and failure. For the competitive athlete it means injury and failure to reach true potential.

There are two main factors that will prevent proper progression. The first is the lack of understanding of what "aerobic" is and the failure to monitor it as an exercise. The second are the structural, biochemical and mental stresses that the Applied Kinesiologist sees and should be able to properly manage in a daily practice.

This is an exciting area of endeavor, and it is hoped that further studies can be done in this field. My hopes are to add such methods as Stop Action Video as well as Blood Analysis during specific levels of activities. Rebuilding an understanding of efficient and healthy exercise habits, as well as training techniques, will be a great addition. This will add another small part to building the total health picture.

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CONTRALATERAL REACTIVITY

BY

Nancy L. McBride, D.C.

Abstract- This paper discusses the theory that most muscle weakness, especially following an injury, of chronic duration and even many recent injury patterns will eventualize in a pattern of reactivity to the same muscle on the opposite non-injured side. This occurs especially frequently with those patients involved in athletic endevors.

I realized one day in the office while testing a patient on a reevaluation visit, that what I at first believed was an error in testing was really a reactive muscle pattern being exhibited. I thought I had accidently tested the wrong side in my reevaluation of a muscle that had previously tested weak and had been treated. I tested the opposite muscle and then retested the original muscle marked on my chart and that muscle then showed weak. We all know that the pattern of muscle reactivity is to test a muscle, test another muscle, and then quickly retest the originally tested muscle. If there is a pattern of reactivity between the two muscles tested then the one originally tested will become weak after testing the second muscle. In the past I submitted a research paper with a chart which I use to keep tract of commonly occuring groups of reactive muscles such as the hamstring group being reactive to the quadriceps group and vice versa. What I did not realize was how often one side of the body is reactive to the opposite side.

A case in point was a patient who had been thrown out of a moving auto and landed on the left side. After clearing all the obvious muscle weaknesses and pelvic faults I began testing all of the weight bearing muscles on the left side against their exact counterpart on the right side in the outlined pattern of reactive testing. To my surprise, every weight bearing muscle above the knee on the left side was reactive to the right side same muscle. That is to say, the TFL on the left was reactive to the TFL on the right, and so on.

I especially find this pattern in patients who have been involved in car accidents or injuries where the pain has persisted for a duration of months or years. I find this pattern especially prevalent in patients who run, jog or participate in strenuous sports such as racquetball and tennis. I feel this pattern is also a great contributing factor in gait stress center locking mechanism which is exhibited when the patient is placed in the facilitation gait position and the muscles of inhibition are found to be strong. It seems that when the reactivity between muscles is cleared and the stress centers of the gait mechanism are cleared many other problems are eliminated in the process.

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RIGHT CONCEPTION VESSEL - LEFT CONCEPTION VESSEL
RIGHT GOVERNING VESSEL - LEFT GOVERNING VESSEL

by Ineon Moon, D.N., C.Ac.

ABSTRACT

The Conception Vessel and the Governing Vessel are known as two of the Eight Extra Meridians in traditional acupuncture. They have been accepted as running dead-center on a sagital plane of the torso. In the AK community, there has been some controversy as to the direction that the Conception Vessel (in particular) runs. If we postulate a right Conception Vessel and a left Conception Vessel, with different running directions in each, we will get consistent results. The concept of a right and a left Conception Vessel and a right and a left Governing Vessel is not new.[1] In this paper, the author examines the concept of right and left separation using the AK approach, and finds it to be true.

In early practice of Applied Kinesiology we therapy localize on the Alarm points (Front-Mu points) to determine excess or deficient energies in the internal organs or acupuncture meridians. We know that there are two lungs and two Lung alarm points, two Kidneys, and two Kidney alarm points, ascending and descending colon, and two Large Intestine alarm points. The single organs, however (heart, Pericardium-Circulation-Sex, stomach, triple warmer, small intestine and bladder) were known to have single alarm points along the Conception Vessel in the middle of the body. Liver and Gall Bladder are split so that one alarm point is on the right while the other is on the left, and we see the same for the spleen-pancreas.

Regardless of the number of organs, however, organ-meridians are paired right and left. The only exception, in most texts, has been the Conception Vessel and the Governing Vessel. We question why the Conception and Governing Vessels are accepted as single meridians, instead of paired right and left. Using the old AK concept, if we find an excess or deficiency on the Pericardium (Circulation-Sex) meridian by therapy localizing to its Alarm point, it is impossible to know if the imbalance is on the right or left meridian. Yet if we therapy localize slightly laterally to the point (CV 17), yet not so far as to TL the Kidney meridian, we will reveal the condition of the PC meridian that is to that same side. (Therapy localization to alarm points being done by single or double hand.[2])

The right side of CV 17 will reveal the condition of the right side Pericardium meridian, while the left side of CV 17 will reveal the condition of the left side Pericardium meridian. This can be verified by therapy localizing to the Connecting point of the PC meridian (PC 6) or its source point (PC 7). In practice, an excess or deficiency to a meridian will only show unilaterally in most cases, following the law of energy conservation.[3] Often, in fact, it is found that one side of a meridian is excessive while the other side of the same meridian is deficient, that is, that there is a right to left side imbalance. At any rate, it is important to determine which side of the body a meridian is imbalanced, and in which way, so that proper treatment may be given. Knowing the mid-line alarm points (for the pericardium, heart, stomach, triple warmer, small intestine, and bladder meridians) have both a right and left side componenet, makes diagnosis and treatment accurate.

According to our investigations of the direction of energy flow in a meridian via the "meridian-running" method,[4] for the male, the left sided Conception Vessel carries yin energy downwards while the right side carries yin energy upwards. The sides are reversed for the female. As for the Governing Vessel, the left side for the male carries yang energy upwards, while the right side carries yang energy downwards, and again, this is reversed for females. This should clarify the controversy over the direction that the enery runs in both the Conception and the Governing Vessels. This can be confirmed by challange and muscle response known as Applied Kinesiology. *The subtle differences can only be detected keeping in mind the Relativity Principle.[5]

CONCLUSION

Applying the universal laws of Energy Conservation and the Relativity Principle into Applied Kinesiology, we can prove and find new techniques precisely, especially, as we have shown in this paper, left and right meridian components for the Conception and Governing Vessels and its bearing on left and right Alarm points for the pericardium, heart, stomach, triple warmer, small intestine, and bladder. This technique is extremely useful in determining whether an imbalance is left or right meridian, preventing mistaken therapeutics applied bilaterally.

* The Governing Vessel can be tested using GV 14 both right and left, with the finger circling method or magnet rotation. Treatment can be done by demagnetizing method.[6]

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A WHOLE BODY LOOK AT THE CORRECTION OF TMJ DYSFUNCTION

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WALTER H. SCHMITT, SR., D.C.

The temporomandibular joint (TMJ) is the most important articulation in the body. The oral cavity including the TMJ is responsible for half of the neurons in the precentral and postcentral gyrii of the cerebral cortex, and due to the numerous interconnections with other sensory and motor neurons, the TMJ can have effects, both positive and negative, throughout the entire body. Likewise, the entire body must be analyzed in determining sources of TMJ dysfunction.

Correction of TMJ problems can have beneficial effects on both musculoskeletal and visceral disorders. But, by the same token, many factors can cause and/or aggravate TMJ-dysfunctions. One of the faults of clinical chiropractic in the past has been the tendency to look at the body with blinders on as if the spine were the only structure worth paying any attention. Similarly, many dentists have failed to look outside the oral cavity for answers to TMJ problems.

Through the investigations of applied kinesiology (AK) in the chiropractic profession, a considerable amount has been learned between

the relationships of the TMJ and the rest of the body. This paper outlines some of the major factors to be considered in a whole body approach to the correction of TMJ problems toward the end that the dentist may recognize factors in his TMJ patients which originate outside the oral cavity. These extra-oral factors can be the primary sources of the patients' symptoms. In these patients, therapies directed only at the TMJ may result in less than hoped for responses, since the TMJ problem may be caused or aggravated due to these other factors. Obviously, TMJ and occlusal problems are often the source of symptoms elsewhere, as is well described by many authors.

To put the correction of TMJ problems in proper perspective, we have employed the following priority system of evaluation. This procedure details a step-by-step approach to TMJ correction :

1. Level the head (occiput)

   A. Correct structural misalignments (subluxations)

   B. Balance postural muscles which affect head level

2. Correct cranial bone articular lesions (cranial faults) in order to align the condylar fossae.

3. Balance TMJ muscles

4. Dental care of the oral cavity

A. Use TMJ equilibrating appliance ("splint")

B. Correct occlusion problems with dental intervention

Employing this procedure, one can be assured that potentially unnecessary or overly extensive procedures will be undertaken only after ruling out faults which may be more fundamental. Further, the use of this step-by-step procedure in the care of TMJ patients will enhance the effectiveness of necessary dental procedures, often greatly shortening overall treatment time. We will briefly discuss the importance of points one, two, and three in the overall care of the TMJ.

1. Level the head (occiput).

As can be seen in Figures 1-A, -B, and -C, an unlevel head can cause stress into one or both TMJ's, due to the altered proprioceptive and/or gravitational effects of this unleveling. All patients should be examined in a standing position to determine the head level prior to commencing other procedures. Postural analysis is normally performed by comparing the levels of both mastoid processes -- a line drawn from one mastoid tip

to the other should be parallel to the horizonal place.

The TMJ's must serve two masters : 1. their anatomical relationships, and 2. gravity. When the head is level, the anatomy and the effects of gravity are in harmony. When either the head level or the functional anatmony of the TMJ are altered from optimal, both are affected. Correction of the TMJ by dental procedures may cause changes which result in a return to normal head level. However, if structural or muscular imbalances are present, they may create TMJ problems and therapy aimed at TMJ correction in these cases will be less than totally effective.

Spinal misalignments (chiropractic subluxations), especially those of the upper cervical and atlanto-occipital areas alter postural relationships of the head on the neck. Correction of these subluxations by chiropractic manipulation results in an immediate return to normal mechanical and postural patterns when such subluxations are the only problem present. Head unleveling, observed by one mastoid process which may be as much as one-half inch to one inch higher than the other, is a frequent consequence of an occipito-atlantal subluxation, and proper manipulation can normalize this imbalance immediately.

Muscle imbalances, especially of, but not confined to, the cervical muscles (e.g., sternocleidomastoids, upper trapezii, etc.) also contribute to head unleveling. These muscle imbalances may arise from many sources. Muscle imbalances have been shown to be due to primary muscle weakness and secondary muscle hypertonicity. The muscle weakness may be identified by standard muscle testing procedures. Applied kinesiology (AK) procedures are designed to strengthen weak muscles by manipulating various reflex points and other areas and by supplying specific nutritional support, when indicated by AK testing.

When the head is unlevel, the TMJ's are attempting to satisfy both their anatomical relationships in the condylar fossae as well as providing proprioceptive feedback which is in harmony with their normal certical plane which parallels that of gravity. When the head is unlevel, confusion becomes present in the nervous system since these two factors are out of synch. The result is stress in both structural and neurological arenas and symptoms are often those of TMJ involvement (Figure 1-C).

2. Correct cranial bone articular lesions.

The bones of the skull, including the mandible, have a regular, slight pattern of movement which is more or less parallel with respiration. The cranial bone movement is microscopic in nature, and takes place at the sutural articulations which are anatomically designed for such movement. This can be readily observed by studying a disarticulated skull or detailed drawings or photographs of the individual bones. The highly trained individual can palpate these movements, but it is more readily monitored by electronic sensor equipment.

The movements of the skull bones are quite complicated, but for this discussion, we will simplify these movements and discuss only one plane of motion for a given bone, even though most cranial bones move through two or three planes during their respiratory cycle. For example, the temporal bone, when viewed from the lateral (See Figures 2-A, -B, -C) rotates like the movement of a wheel, with the mastoid process moving posteriorly on inspiration (Figure 2-C) and anteriorly on expiration (Figure 2-A). This rotation causes a shift in the orientation of the condylar fossae during the respiratory cycle, therefore affecting the

TMJ function. The mandible's respiratory movement is such that the mouth tends to open during inspiration and close during expiration. Of course, since these movements are microscopic, they are rarely observed in patients.

Cranial bone respiratory articular movement may be compromised for a multitude of reasons. When part or all of the cranial respiratory mechanism malfunctions, we say there is a "cranial fault" present. Cranial faults are lesions where one or more cranial bones are "jammed" and not moving through their normal respiratory excursion. The most common cranial faults involve lesions of the temporal bones which become "stuck" in either their inspiration range of motion or their expiration position. Cranial faults may be present in such a way that either one or both temporal bones may be lesioned, in either the same or opposite phases of respiratory motion.

Cranial faults then, may alter the normal relationahips at one or both condylar fossae, resulting in abnormal proprioceptive feedback from one or both TMJ's. Most TMJ patients suffer from one or more cranial faults. The cranial faults in these patients may be the cause or the

result of TMJ dysfunction, but once they are present, they can become entities which require specific correction in order to insure complete and rapid resolution of TMJ problems.

Another common cranial bone fault is the tilting of the sphenoid bone when viewed from anterior to posterior. That is, the sphenoid often gets stuck in a tilted position, causing an elevation of the greater wing on one side and a lowering of the greater wing on the other, in relation to other cranial structures. These patients can be observed to have a slightly protruding eyeball on the side of the sphenoid elevation.

In addition to the effect of a sphenoid tilt on the temporal bones and the other cranial bones, the sphenoid tilt fault has major implications in TMJ dysfunction, due to the attachments of the external pterygoid muscles and the internal pterygoid muscles to the pterygoid processes of the sphenoid. When the sphenoid is stuck in this tilted position, there will be imbalances in the pterygoid muscles which manifest themselves when the mandible is in motion (which as mentioned previously, is the case with each respiration, not to mention talking, chewing, swallowing, etc.). Many cases of so-called "muscle spasm" of the external pterygoid are no

more than reactions caused by the tilting sphenoid, and it is common for such a so-called enternal pterygoid "muscle spasm" to return to normal tonicity in seconds when the appropriate sphenoid correction is made. Compare this with the three to six months of dental intervention usually necessary to achieve external pterygoid muscle balance! Again, if the proper priority of treatment is followed, correction of TMJ problems becomes much more rapid if not more complete.

3. Balance TMJ Muscles.

Applied kinesiology is the discipline based on identifying and correcting muscle imbalances throughout the body. AK research has shown that nearly all muscle imbalance, anywhere in the body, is due to a primary weakness of one muscle with the antagonistic muscle being merely a compensatory or secondary hypertonicity (often referred to as a "muscle spasm." Muscle spasm is not a primary entity as is generally accepted by traditional concepts. Since 1964, the work of George Goodheart, D.C., of Detroit, Michigan, has focused on identifying muscle weakness (i.e. muscle inhibition) by standard muscle testing procedures. The most frequent causes of muscle weakness may be likened to a "short-circuiting" within the nervous system which can readily be corrected by "resetting"

the appropriate circuit breakers. Correction of these weak muscles has been consistently shown to automatically achieve relaxation of tight or hypertonic (antagonistic) muscles by reciprocal innervation. No longer is therapy directed at tight or "spastic" muscles. Not only is such tight muscle therapy often incomplete and/or short-lived, it has no effect on strengthening the pirmary problem, the weak muscle. With muscle weakness present, permanent correction of any musculoskeletal problem, including the TMJ, is impossible.

AK investigation has developed numerous techniques for correcting the inhibition causing muscle weakness which include various reflexes, acupuncture points, nutritional supplementation, cranial bone fault correction (as discussed above), spinal manipulation, and many more techniques, depending on the source of the problem. The intricate interaction of the TMJ muscle requires that they must function without interference, even if all of the other above-mentioned factors are functioning properly. TMJ muscle imbalance must be corrected as the third factor in treatment priority in the course of treating TMJ problems.

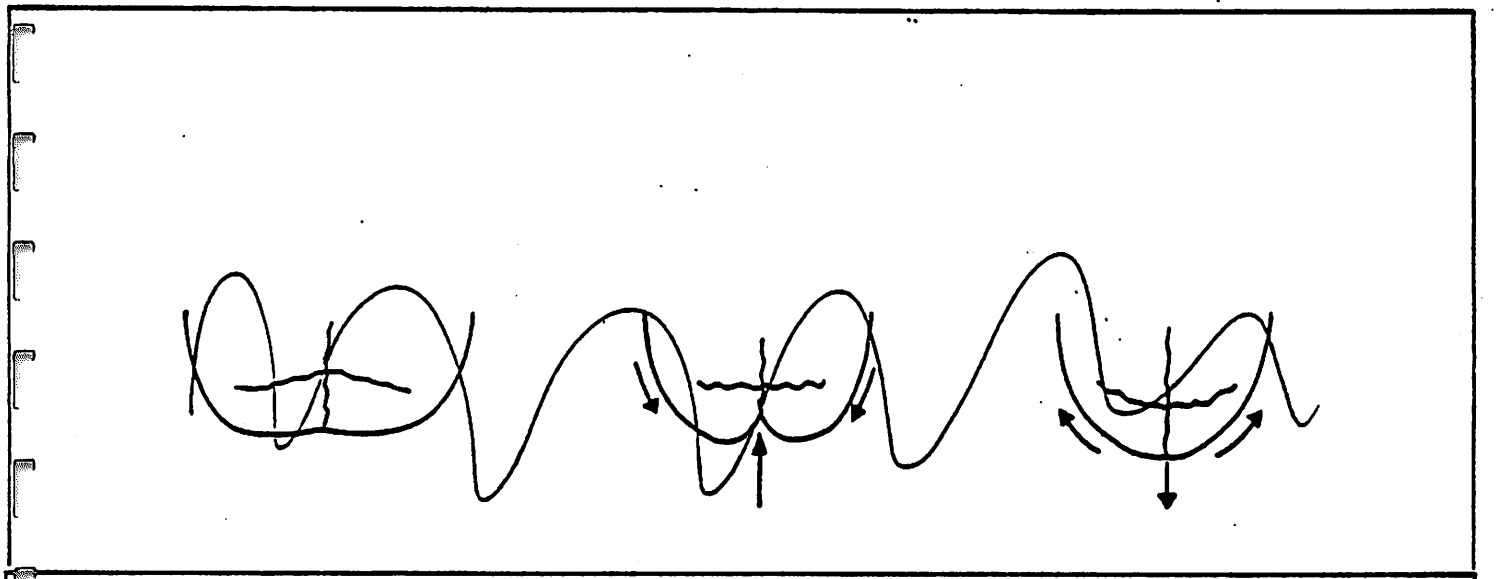
Correcting TMJ muscle imbalance follows correction of the other potential problem areas and may be performed by various techniques,

depending on the source of the problem. AK diagnostic techniques are designed to identify problems and isolate the sources of muscle imbalances, whether they be found in short-circuited reflex areas, nutritional faults, stress-related problems, or within the intramuscular receptors (e.g., muscle spindles, golgi tendon organs) themselves. Immediate changes in such parameters as point of first contact, width of opening, lateralization on opening and closing, and so on can be seen following TMJ muscle balancing techniques. These imbalances, along with head leveling and cranial correction, should be made prior to dental intervention (priority #4) in the care of TMJ problems.

CONCLUSIONS

Dentists often build appliances for their patients which are called bridges. It is hoped that this paper may serve as a chiropractic "bridge" to the dental profession. The cooperation and interaction of the two professions has served as the basis for outstanding health care opportunities for the patients of both professions in many areas of the country. The dentist may be taught to perform a number of the procedures listed in 1, 2, and 3 above. When difficulty is encountered, such as the necessity

for structural or spinal manipulation, the dentist needs to be aware of the role of the chiropractor in correcting these problems. Likewise, the D.C. must be educated as to the necessity and availability of dental consultation in those cases requiring procedures in the dental care of the oral cavity listed in point #4. More and more numbers of chiropractors and dentists are founding relationships toward these ends and for the benefit of all of their patients.



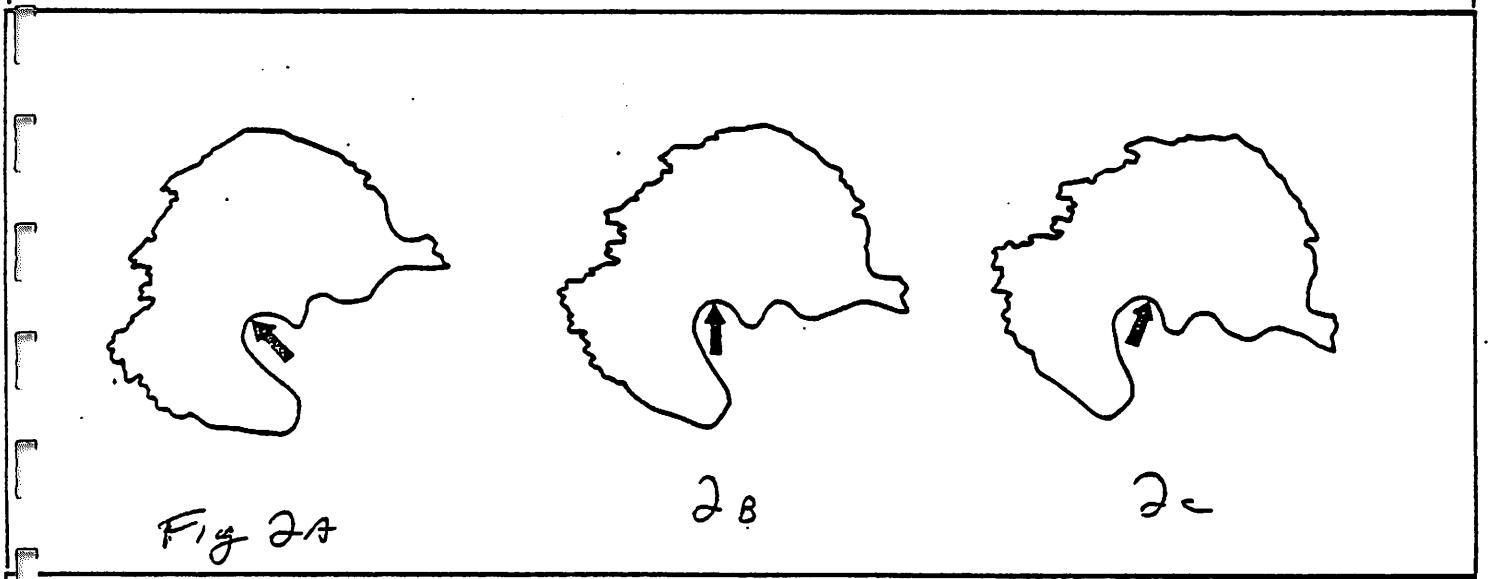
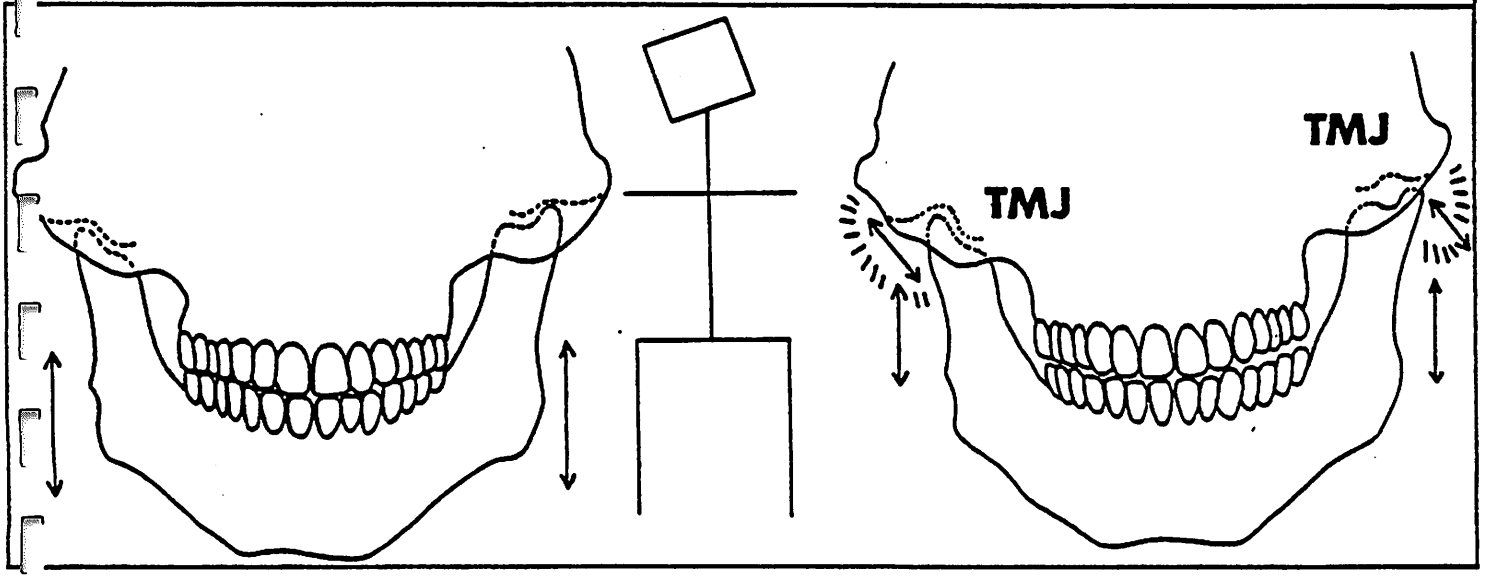


Fig 2A 2B 2C



12-2

Fig 1A 1B 1C

AN APPLIED KINESIOLOGICAL APPROACH TO CANDIDA ALBICANS ALLERGY

Richard A. Mowles, D.C.
Walter H. Schmitt, Jr., D.C.

ABSTRACT: This paper will examine the current approach to Candida albicans as a systemic allergen. These principles will be utilized to incorporate Applied Kinesiology diagnostic principles in order to help interpret neuromuscular patterns. This paper will also incorporate the ideas concerning Candida into our general understanding of body homeostasis.

INTRODUCTION

Candida albicans is known to most clinicians as an organism which is a normal inhabitant of the mucous membranes which cover the internal surface of your digestive, respiratory and reproductive organs. Most physicians associate Candidiasis with common vaginal yeast infections and the cause of oral thrush infections. Otherwise, Candida albicans has traditionally received little interest as a source of problems to patients and to be a complicating factor with a patient's clinical response to treatment.

In 1978, Dr. C. Orian Truss of Birmingham, Alabama, published the first of his series of articles describing Candida as a common source of allergic reactions. The fact that Candida lives in the gastrointestinal tract of all adult humans, and that everyone has a normal level of circulating antibody to Candida testifies to both its ubiquity and its generally being regarded as a benign symbiont of the human gastrointestinal tract.

In fact; however, Candida albicans has been implicated as one of the most frequently encountered severe allergens known with consequential symptomology ranging from neurological dysfunction, endocrine dysfunction, gastrointestinal dysfunction, to such bizarre symptoms as muscle and joint numbness and tingling. Along with these symptoms, many of these patients become increasingly sensitive to a variety of environmental substances such as tobacco, perfumes, petroleum products, exhaust fumes and many common foods including breads, sweets and milk. This can be well understood since Candida wreaks havoc with the immune system and in particular, the adrenal glands. When a person becomes sensitive to Candida,

there is no avoiding it since the parson carries it with him or her constantly. The patient, much to their distress upon seeking professional help are told, "Your evaluation shows no disease. Your symptoms must be stress." These people are commonly labeled "hypochondriacs." These individuals end up wandering from doctor to doctor seeking help.

Candida's effects on the immune system, in particular the thymus gland, are to literally stimulate its function with a constant allergic irritation to the point of unresponsiveness. One study showed abnormalities of the thymus - dependent immune system in the majority of patients with chronic mucocutaneous candidiasis, although the manifestations and the severity of the defects were not the same in all patients.[2] The effects of constant histamine release becomes a constant stressor to the adrenal glands leading to an exhaustion of the adrenal glands and any of the myriad of symptoms associated with severe chronic functional hypoadrenia. It is as if Candida acts like a rubber hose being constantly rapped across the wrist. Initially, it is no problem, but with continued and prolonged stimulation, the wrist (thymus/adrenal axis) becomes paralyzed and totally unable to function to any distressor stimuli.

Immune system suppression and adrenal exhaustion are further complicated by the local irritation Candida can cause in the gastrointestinal tract. When under an ever - present allergic barrage, the intestinal walls become inflamed as in any food allergy, thus disrupting the normal digestion of nutrients and impairing nutrient absorption. This usually leads to an open ileocecal valve syndrome which is recurrent and very difficult to stabilize. The potential for toxic reflux from the colon into the small intestine where absorption occurs leads to further compli-

cations in clinically understanding the patient, not the least of which are signs and symptoms of a persistently congested or toxic liver problem.

Candida proliferation is also seen dependent upon the proper sex steroid hormone balance. Low basal estradiol levels which is seen in the early follicular phase are involved in the maintenance of normal antibody titres to Candida. A study showed that the administration of low doses of estradiol to rats showed a higher antibody response in uterine secretions.[3] Another study observed low antibody titres to Candida albicans in pre - pubertal girls (lower estradiol production) with active vaginal candidiasis, suggesting that the maturation of humoral immunity to Candida albicans may be linked to sex steroid hormones, especially estradiol.[4] In contrast, in two normal subjects whose sera were collected every day of a complete cycle, the normally rising estradiol levels in the late follicular phase were associated with markedly reduced antibody titres. This observation suggests that estradiol at higher concentrations exerts a suppressive effect on antibody levels.[5] Truss puts emphasis on yeast being worst when progesterone levels are high as in pregnancy and the luteal phase of the menstrual cycle.[6] It appears that the proper relationship of the sex steroid hormones will give the most optimum antibody titres to Candida. Our experience indicates that the most severe Premenstrual Syndrome (PMS) patients have Candida albicans allergy. We also see definite hormone imbalance associated with PMS.

Candida has two forms: a rather quiet Y (yeast) form and pathogenic M (mycelial) form. A recent study has shown that, in vitro, the

absence of biotin causes Candida to differentiate to the pathogenic M form.[7] Under normal circumstances, Candida in the gastrointestinal tract is kept in check by the normal friendly flora which produce biotin. Any oral antibiotic which disrupts the normal gastrointestinal floral balance may allow a flare up of Candida as a consequence. All clinicians have had experience with patients who were never the same following some infection or other condition which was treated with oral antibiotics. Candida is often the culprit here, activated by the suppression of the normal gastrointestinal flora.

TREATMENT

The original treatment protocol recommended by Truss is the use of nystatin, a rather innocuous antifungal agent. Truss also suggested local nystatin treatment of the vaginal and oral cavity when there is any evidence of Candida present in either of these two cavities. Further clinical investigation has led to a number of adjunctive measures to combat Candida including dietary restrictions of yeast containing products and carbohydrates, the avoidance of steroids, the avoidance of oral antibiotics, the desensitization to Candida, alternative anti - fungal medications, various vitamin and lifestyle recommendations in the hopes of controlling yeast proliferation.

DIAGNOSIS

The problem with the Candida albicans problem is that there is no definitive diagnostic procedure as yet published. Since everyone has circulating antibodies to Candida, there is no blood tests which is specific for identifying the Candida - allergic patient. Reports on the value of serum antibody tests are often contradictory, and the situation

will remain controversial and inconclusive so long as the data from the various laboratories are not derived from standard test systems, standardized antigens, and comparable patient selections.[8, 9, 10, 11, 12, 13, 14, 15] One study concludes that the diagnosis of systemic Candidiasis should never rest on serologic findings alone, nor should the tests be regarded as useless because there are false - positives and false - negatives. The diagnosis should rest on all available diagnostic data.[16] These include predisposing illnesses, iatric factors,[17, 18] Candida mucocutaneous lesions,[19] Candida chorioretinitis,[20] positive blood cultures and Candida counts of 10,000 to 15,000 per millilitre or more in catherized urine.[21] Truss himself admits that it is unclear just what it is about Candida's presence that creates the overwhelming allergic response to immune system suppression. Diagnosis must presently be made by induction (eliminating other causes of the patient's symptoms) and by a therapeutic trial with nystatin (common brand names include Nilstat® and Mycostatin®) and/or related aforementioned procedures. Fortunately, Nystatin is one of the least toxic drugs known to man, because very little of the drug is absorbed into the body from the intestinal tract. However, occasional side effects may occur, including nausea and skin rash, but in general a misdirected therapeutic trial causes little trouble.

Candida albicans allergy may be suspected from a patient's history by one or more of the following:[22]

1. History of taking antibiotics, birth control pills, or cortisone - type medicines, plus a history of consuming foods with a high content of sugar or yeast. (Yeasts thrive on sugar and refined carbohydrates. Symptoms are also aggravated by yeast - containing foods, especially breads, cheeses, beer and fruit juices.)

2. History of depression, mental illness (and other nervous system symptoms) and symptoms involving the reproductive organs and digestive tract, often associated with symptoms in many other parts of the body.

3. Sensitivity to chemical fumes, odors and common foods.

4. Response to the administration of nystatin and other medicines which remove yeast from the body.

From our experience we have seen several other factors:

1. Depressed thymus and immune system function.

2. Clinical response to biotin therapy or other therapy at reinstating normal gastrointestinal flora.

3. Open ileocecal valve and/or similar gastrointestinal tract involvement.

There are many other factors which could be summarized about the Candida albicans allergy patient, but we have employed some of the above to develop both Applied Kinesiological diagnostic screening methods for Candida as well as naturally oriented treatment regimens for these patients.

APPLIED KINESIOLOGICAL EVALUATION OF CANDIDA

As chiropractic physicians, we are not licensed to prescribe medications, including nystatin. This fact has served as a blessing in that it has forced us to look at the Candida patient from a different perspective than medical physicians, and this perspective has yielded a great deal of useful diagnostic and therapeutic information which may be employed when encountering the potential Candida patient.

As Applied Kinesiologists, we use standard muscle testing procedures in our approach to diagnosis. Muscles which test weak can be seen to strengthen by a variety of procedures, one of the most important of which is oral nutrient testing. A substance which the patient requires will cause a weak muscle to become strong when the substance is placed in the

patient's mouth and insalivated. Similarly, a substance which is an allergen to the patient, or which is toxic to the patient will cause a weak muscle to strengthen when it is salivated.[23] Another tenet of Applied Kinesiology (AK) is that one possible cause of muscle weakness is a viscerosomatic reflex pathway from a specific visceral structure for each specific muscle - i.e. a muscle/organ connection.

AK testing has yielded considerable information about the Candida patient. The majority of Candida patients demonstrate the following patterns:

1. A weakness to testing of one pectoralis major, sternal division. This muscle is related to the liver. (Other muscle weakness frequently observed in these patients are the tensor fascia lata, related to the colon, and the iliacus, related to the function of the ileocecal valve).

2. Weak muscles strengthening on insalivation of Antronex. Antronex is the product name of a natural anti - histaminic product produced by Standard Process Laboratories.[24] It has been suggested as an excellent screening tool for allergies when used with AK testing.[25]

3. Weak muscles strengthen on insalivation of biotin. Biotin is usually necessary in the Candida patient as a natural anti - Candida substance. It is especially necessary when nystatin is unavailable.

4. Weak muscles strengthen on insalivation of glandular preparations containing thymus tissue. Since the constant irritation by the ever - present Candida proliferation suppresses the thymus and the immune system, thymus tissue gives a general strengthening response to weak muscles in addition or in exception to the presence and response of infraspinatus (thymus related) weakness.

5. Weak muscles become strong on insalivation of an herbal product named Imu-Stim.[26] This product is primarily a highly active source of Echinacea, a well known herbal immune system stimulant. Multiple muscle weaknesses strengthen in many Candida patients when Imu-Stim I is placed on the tongue, presumably due to the wide ranging adverse effects of immune system suppression.

6. Weak muscles strengthen when nystatin powder is placed on the tongue and insalivated. We use this as a confirmatory test that Candida is present and causing trouble. In general, our experience with testing various forms of antibiotics with AK muscle testing procedures dictates that they will weaken the patient unless they are quite necessary to the patient at that time. The strengthening response from nystatin is contrary to what is normally suspected. In fact, we have observed a weakening response or no change in muscle strength response in several patients who we believe were misdiagnosed as Candida allergy patients.

7. The patient will demonstrate a general weakening effect in most muscles of the body when insalivating one or more sources of yeast products. We tested bakers yeast, brewers yeast and lactic acid yeast. Some patients weakened on all sources of yeast and others on only one or two of the yeasts. This test is a good demonstration to the patient of the necessity of avoiding yeast and yeast containing products (mushrooms, aged cheeses, beer, fruit juices and dried fruit). This yeast catagory can also include supplements with a yeast base. Some patients' systems are so devastated by Candida that even supplements which their systems may need for optimum function will create an adverse effect on their metabolism due to the fact that a yeast base is utilized which facilitated Candida proliferation. The individual can be orally challenged to see if there is systemic weakness produced by that nutrient. Even though one particular muscle may strengthen to oral challenge, there may be systemic weakness produced due to the yeast base. Most of the time there will be a generalized weakness produced upon salivation of that nutrient.

8. There will usually be a general muscle weakness upon salivating a nutritional source of copper. Copper excess has been implicated as a problem in approximately 80% of Premenstrual Syndrome (PMS) patients.[27] We find a strong correlation between Candida allergy and PMS. The exact correlation between Candida and copper is not clear. We see in the clinical evaluation of a patient, through hair trace mineral analysis, that the deficiency or excess of a mineral can cause the same set of symptomatic presentations. The unavailability of copper or the excess of copper tends to decrease the availability of oxygen in the tissues. This has importance in the fact that Candida albicans, being an anaerobic organism,[28] could possibly be affected by the tissue oxygen content. Many enzymes such as cytochrome C oxidase are copper dependent. Cytochrome C oxidase is involved in the last step of the respiratory oxidation chain within the cell. As a result of a copper deficiency, anaerobic metabolism can occur, this is to say a growth of cells without the presence of oxygen similar to fermentation. Copper is necessary for aerobic metabolism, therefore any

deficiency of copper[29] is an indication of reduced aerobic metabolism.

Dr. Paul Eck, PH.D., has researched minerals and their interrelationships in biochemical metabolism[30] based on how that system oxidized glucose. Dr. Eck's research has shown that high copper slows down the body's oxidation physiology. Dr. Eck feels that copper slows down oxidation by its effect on the thyroid gland.[31]

Apparently copper has a very strong correlation to the enhancement of Candida.

9. We also see in these patients a need for zinc supplementation. These individuals show a cranial spinal torque pattern frequently, which is challenged and neutralized by zinc and/or manganese as reported by Schmitt.[32] It has been reported that micromolar concentrations of the divalent cation zinc suppress the formation of the mycelial form of Candida in vitro.[33, 34] In addidion, a depression of the in vivo level of zinc has been implicated in Candida pathogenesis in individuals suffering from acrodermatitis enteropathica,[34] a disease related to zinc deficiency. Therefore, the concentration of the divalent cation zinc in the body fluids of infected individuals may be directly related to the growth, phenotype, and pathogenicity of Candida.

10. There appears to be some correlation between Candida and selenium deficiency. The exact mechanism of how Candida is correlated with selenium is unknown. At present the only known function in man for selenium is a component of the antioxidant enzyme, glutathione peroxidase.[35] Glutathione peroxidase protects vital components of the cell against oxidative damage. Therefore selenium prevents the decay of cellular function. It is known that selenium improves the function of mitochondria (energy - producing units of cells)[36] by protecting them from the lack of oxygen. There could be some association with a need for selenium and the anaerobic environment which promotes Candida. Selenium also detoxifies cadmium, an environmental pollutant. Cadmium binds with zinc in the body and we have already discussed a correlation between zinc deficiency and Candida. It is possible that selenium's strong influence on the body's immune system suppresses Candida. Drs. John Martin and Julian Spallholz and their colleagues at Colorado State University found that dietary supplementation with selenium at levels above those recommended as nutritional requirements[37] enhanced the primary immune responses.

CONCLUSIONS

This paper has examined Candida albicans and its implications with physiological processes. The examination of individuals with the many tools available in Applied Kenisiology enable the physician to acknowledge the presence of this anaerobe and to obtain a more concise picture of why the hyopadrenia and/or hypothymic response is slow or why the ileocecal valve keeps reoccurring or why a patient may have an adverse reaction to a particular supplement.

A lot of emphasis has been put on Candida as the causative culprit in many clinical problems. We feel that Candida is not the cause but merely another complication, sometimes stubborn, but one that has to be considered in order to adequately understand the patient's response to Applied Kinesiology treatment. We know that Candida exists in a symbiotic relationship to bacteria within the body. When the body's homeostasis is disrupted either biochemically, structurally or phychologically then there is neurological interference to possibly allow Candida to become rampant. Unless the body's homeostasis is restored, then the Candida will continue its physiological course. The accepted treatment of Nystatin along with dietary alterations does not correct the reason of Candida's proliferation. We, as Applied Kinesiologists, have the approach to adequately view the body's homeostasis holistically. When this approach is followed, the body will set up proper system function and return to homeostasis. Only when this homeostasis is accomplished does Candida become suppressed and its many effects on body function eliminated.

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EXPOSING THE HIDDEN CATEGORY II

by
Dale K. Sandvall, D.C.

ABSTRACT: The author shows another method of exposing a category II.

When the patient is in the supine position, have them TL to the sacro-iliac joints with both hands. If both palmar and dorsal TL are negative repeat the same but with the patient doing the Fabere-Patrick test on one side at a time.[1] This position will often test weak when there is a hidden category II present. The diagnosis should be confirmed by challenge to the involved sacro-iliac joint.

CONCLUSION

The author has shown another method of exposing a hidden category II.

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EVALUATION OF THE CONTRACTED PECTINEUS

by
Dale K. Sandvall, D.C.

ABSTRACT: The pectineus is usually evaluated with the adductor group which includes the adductor brevis, longus and magnus. In this paper the author shows a method of exposing the contracted pectineus.

When the patient is in a supine position, have him externally rotate the foot and abduct the leg. Then have him perform a straight leg raise test. The leg will go higher on the side of the weak pectineus and will be lower on the side of contracted pectineus.(1)

CONCLUSION

The above simply shows how a correlation may be made between applied kinesiology and orthopedics. It is the author's opinion that more research should be done in an attempt to correlate the two examining procedures.

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APPLIED KINESIOLOGICAL MANAGEMENT OF INGUINAL HERNIAS

by

Dale K. Sandvall D.C.

ABSTRACT: Too often we accept the standard treatment for a particular condition. In this paper the author shows an alternative to the standard surgery for inguinal hernia.

According to Dr. George Goodheart, the key to the management of inguinal hernias is usually found in the correction of the very tight pectineus muscle on the side of herniation.[1] On the opposite side, often there is a weak adductor group, usually found weak in the clear or weak with TL to the NL points. The contraction of the tight pectineus soon becomes too much for the congenital weakness in the anterior division of the internal oblique abdominal and the abdominal soon gives way to the herniation. Thus there usually will be a weakness of the anterior division of the internal oblique abdominal on the side of herniation. The side of tight or contracted pectineus may be evaluated as explained in the paper by the author, "Evaluation of the Contracted Pectineus".[2]

Correction consists of first reducing the tightness of the pectineus on the side of hernia. This may require origin-insertion and muscle spindle therapy as if one were trying to weaken the muscle. If palpation of the pectineus reveals fibrous nodules present, it may require fascia stretching to the pectineus. Prolonged NL to the adductors opposite the side of herniation proves to be very beneficial. The weakness in the anterior division of the internal oblique abdominal usually responds well to NL activity and Standard Process Vitamin E. Also, it should go without saying that all factors of the IVF should be evaluated and treated . accordingly, for any muscle weakness

Other specific muscles to evaluate are the psoas, often in a reactive pattern or foot subluxation, and the gluteus medius. Presence of a category I or II should be corrected and the goal should be to get equal turn in and turn out of the feet.[3]

An elastic support should be fitted and worn to help keep the hernia reduced for approximately 4-6 weeks. The standard truss is not advisable since it has a tendency to push inward, thus aggravating the hernia.

CONCLUSION

The author has tried to outline an alternative to the standard surgical repair of inguinal hernia. According to Dr. George Goodheart[4], in his many years of experience in

treating this condition. only once was it necessary to refer a case out for surgery.

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THE USE OF ANTRONEX AND HISTIDINE AS SCREENING TOOLS FOR FOOD AND OTHER ALLERGIES

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

Abstract: Antronex, a natural anti-histamine, will strengthen weak muscles and histidine, the amino acid precursor to histamine, will weaken strong muscles in histamine-type allergy patients. Testing with Antronex and histidine on all patients is recommended during initial examination and for patients with recurrent muscle weakness pattern.

Allergic reactions are manifested in the body by histamine reactions and kinin reactions. The use of anti-histamine products for the histamine type allergic reaction is well established. One natural product which has anti-histamine effects and is available without a prescription is Standard Process Laboratories' Antronex.[1] This product has been shown to be useful through years of clinical experience of this author and colleagues for all sorts of allergies including hay fever, food allergies, and other allergic manifestations, such as asthma and histamine type headaches.

An earlier paper by this author discussed the use of Antronex as a useful tool for uncovering hidden muscle weakness in patients similar to the use of ribonucleic acid.[2]

We have also used Antronex therapeutically based on muscle testing (usually weakness of a pectoralis major, sternal (PMS)) in treating various types of patients with allergies. "Antronex contains 10 mg. of Yakriton from liver. A liver extract having a natural anti-histaminic nutritional effect." (Label information) Although there has never been a specific muscle related to Antronex or any other anti-histamine, the PMS has always seemed a logical choice based on the nature of Antronex as well as the clinical observation that it is usually weak in allergic patients (as well as many other patients).

The bane of any physician is the patient who doesn't respond, or possibly worse, the patient who responds temporarily but always slips back to his original pattern. This patient is particularly frustrating to the applied kinesiologist who is constantly monitoring for muscle weakness and attempting to correct it. During the past year, we have observed that recurrent muscle weakness patterns in patients, including the recurrent open ileocecal valve, have been due to either heavy metal and mineral intoxication or allergy, usually food allergy. A screening test for mineral imbalance of copper, manganese, and zinc was presented in an earlier paper by this author.[3] We now will present a screening test for allergic states based on the use of Antronex.

Traditional AK testing for food allergies and/or toxic substances has been performed by placing the substance in

the mouth and observing for strong muscles becoming weak. There are numerous articles on various allergy testing procedures and among these are recent contributions to I.C.A.K. Collected Papers by Sanna[4] and Klepper.[5] A further screening test for allergy or toxicity was presented by Goodheart in 1980 with the technique of identifying "blood allergy" reactions.[6]

If a patient experiences an allergic reaction which is demonstrated by weakening of multiple muscles, usually all of the patient's muscles, when a drop of the patient's blood is placed on his tongue, then it seems curious why the same substance circulating in the patient's bloodstream does not create a constant weakness pattern in the patient. Obviously, the patient with total muscle weakness could not function, so it is reasoned that the allergic patient shows a constant weakness of only those muscles which were the most involved with his allergic state.

Recurrent muscle weakness is often due to a systemic allergic reaction triggered by frequent exposure to an allergen, such as a food allergen, which is eaten several times a week. If this reaction is of a histamine type, whatever muscles are weak at the time of testing will strengthen when Antronex is placed in the mouth and insalivated. There may be any number of weak muscles and any pattern of muscle weakness, but if all weak muscles strengthen with Antronex, that patient is presumed to be an allergic patient and allergy screening is considered.

Screening for the histamine type allergy patient can be performed by testing with Antronex, but we have observed another pattern which seems to confirm the histaminic type allergy patient. The chemical precursor to histamine is the amino acid histidine. When patients show a strengthening muscle response with Antronex in the mouth, the vast majority of them (over 90%) also demonstrate total or near total muscle weakness upon placing of the amino acid histidine on the tongue. This makes sense based on the logic that supplying more histidine would tend to increase histamine production and the allergic patient already has more histamine than he knows what to do with, which is why Antronex, as an anti-histamine works in the first place. It is also important to note at this point that histamine is a putative neuro-transmitter substance and therefore may have a major impact on certain histamine-dependent neural pathways. Investigation into the muscle testing significance of this fact is presently ongoing and findings will be reported at a later date.

When we observe a patient who strengthens with Antronex and weakens with histidine, we then proceed to test for allergic reactions (muscles weakening) to various foods and other substances. These Antronex/histidine responsive patients who undergo thorough allergy testing by AK methods of placing the suspected food or substance in the mouth, or by inhaling airborne allergens, have always been found to be

highly sensitive to one or sometimes many substances. Removal of these substances from the diet (or environment) has resulted in rapid improvement in these patients and an end to the recurrence of muscle weakness patterns.

However, it is believed that many patients who have allergies or sensitivities to certain substances have acquired these sensitivities and that they may be returned to a state where they are no longer sensitive (or allergic) to the substance. This is especially true of the patient who all of a sudden during adolescent or adult years begins to react to a substance, and may even be true of the child or infant who exhibits allergy type symptoms. Anyone practicing applied kinesiology has observed this pattern in hay fever and other allergy patients when appropriate measures are performed.

We have observed that histidine testing is a useful screening device for the amelioration of allergic reactions. Just as a patient who recovers from a milk allergy is no longer sensitive to milk when tested by AK methods, so will a histamine type allergy patient no longer weaken when histidine is placed on the tongue when that patient's sensitivity is corrected. This often occurs during a single treatment session. That is, the patient originally strengthens on Antronex and weakens on histidine. Treatment is rendered and immediately following, the patient no longer weakens on histidine AK testing. We have correlated the

histidine reaction with other, specific reactions to foods such as wheat, milk, and yeast, and the histidine and food reactions parallel each other precisely. It is important in these patients to invoke a period of avoidance of known allergens in order to let the body recover. Histamine is one of the most potent adrenal (epinephrine) stimulating substances and even though the patient may show rapid improvement in the office, avoidance of allergens for several weeks or months is often necessary in order to allow recovery of over-stimulated adrenal glands and the sympathetic nervous system. Prudent re-entry into the diet or environment of the offending substance can then be undertaken without danger of re-initiating the same old vicious cycle. Specific treatment patterns which will neutralize the histidine (allergic) sensitivity are presently being investigated and appear to be directly related to the mechanics of the pelvis, especially the sacrum. These findings will also be reported at a future date.

The use of Antronex and histidine as screening tools for allergic and histamine type patients is an important tool in the diagnostic workup of our patients. It can be confirmed by a variety of laboratory allergy testing procedures, all of which are controversial or should be at the present time. Oftentimes, our patients are better with AK treatment even before the laboratory reports return. But the critical factor is the identification of the allergy pattern in the first place and to this end, testing with Antronex and histidine should be a part of every applied kinesiologist's initial examination.

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INDUCING RIGHT BRAIN/LEFT BRAIN ACTIVITY IN THE OFFICE SETTING

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

Abstract: Right brain and left brain activity can be induced in allergic-type patients who previously test negative to these patterns. These patients must show weak muscles strengthening upon insalivation of Antronex. Further testing with histidine in the mouth will demonstrate multiple muscle weaknesses and many of these muscles will respond to right brain or left brain activity.

One principle widely accepted in applied kinesiology is that if a therapy is to be of value, it must be able to be detrimental if inappropriately applied. An example of this is making patients worse by reversing cranial respiratory techniques. Since the introduction of our AK understanding of right brain/left brain patterns in muscle testing by Goodheart in 1979,[1] we have learned several methods of dealing with these problems. However, the exact nature of the right brain/left brain patient has yet to be determined. This paper hopes to shed some light on this matter.

In another paper[2] in this edition of the I.C.A.K. Collected Papers, this author discusses the use of Standard Process Laboratories product, Antronex,[3] a natural anti-histamine, as a screening tool for food and other allergies. Also discussed in this paper is the weakening effect created by insalivation of the amino acid, histidine, in allergic patients. If a patient has muscle weaknesses which strengthen

with Antronex, there is usually a general weakness of most, if not all, of the muscles in the body upon insalivation of histidine. Histidine is the amino acid which is the precursor to the substance histamine. Accompanying this weakness is the introduction of right brain and left brain factors into many of the muscle weaknesses induced by the histidine insalivation. Prior to testing with Antronex and histidine, screening of muscles weak or strong in the clear reveals no right brain or left brain activity.

Procedure:

1. Test several muscles weak in the clear.

2. Identify that the patient is an allergic-type patient by a strengthening of these weak muscles with Antronex placed in the mouth.

3. Place histidine in the mouth and observe multiple muscle weaknesses becoming present.

4. With histidine still in the mouth, test a variety of weak muscles with right brain and left brain activity. Note changes.

The pattern of right brain/left brain activity which has been observed has not been specific with regard either to the muscle tested, the side of the body involved, or the phase of brain activity which is produced. Some of the multiple weak muscles will not show any right brain/left brain activity. Others will show strengthening with both right and left brain activity. And any combination of

patterns has been observed. The important point is that it is possible in the allergic-type patient to induce various non-specific patterns of right brain/left brain activity by placing histidine in the mouth.

CONCLUSIONS

It is concluded from these observations that patients demonstrating right brain/left brain activity may fall into the category of allergic patients. Although this may be an erroneous assumption, the patient with recurrent right/left brain patterns certainly deserves a screening test for food or other allergies.

Since histamine is a putative neurotransmitter in addition to its effects on body chemistry at the cellular level, it is very possible that the activation of excess histamine in the body from allergic reactions has a spillover effect into the nervous system, in some way activating histamine-dependent pathways and creating right brain and/or left brain problems. Obviously, further investigation into this pehnomena on both the clinical level and literature search level is indicated.

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THE BODY QUADRANTS, POLARITIES, ELECTROLYTE LEVELS, ELECTRON POISING, AND BODY TORQUE

More on the Links Between the Nervous System and Body Chemistry

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

ABSTRACT: The body's needs for its four major cations are linked to the body structure via changes in the strength of trunk flexors and extensors in the following manner: right flexor weakness-calcium; left flexor weakness-sodium; right extensor weakness-potassium; and left extensor weakness-magnesium. These cations are related to metabolic and endocrine function according to mineral analysis principles set forth by Dr. Pauk Eck. Support for Eck's principles is found when examining the electron poising curve as it relates to body structure and adding the effects of the cations.

Cation levels and endocrine function are related to specific patterns of body torquing and gait, as seen by the muscle balances of major flexors and extensors. These patterns are further related to the electron poising curve as a model for correlating all of these factors. The relationship of amino acid neurotransmitter precursors (tyrosine and tryptophan) to these patterns is discussed as well as the correlation with nasal ionization patterns.

The relationship of thyroid and adrenal function to oxidation and reduction patterns of body chemistry is demonstrated. A theory for endocrine involvement without the presence of expected muscle weakness pattern(e.g., thyroid-teres minor weakness) is presented.

INTRODUCTION

Two previous papers by this author have dealt with specific patterns of body chemistry being reflected in specific muscle testing patterns.[1,2] In the second of these papers,[2] changes in body structure were postulated to parallel changes in body chemistry patterns demonstrated by the relationships of the electron poising curve. Each of the four quadrants of the body (as viewed from above) are represented by one quadrant of the electron poising curve graph. See Figure 1. The polarity of each of the body quadrants was discussed in this paper[2] based on the work of Davis and Rawls.[3] The positive and negative changes of each body quadrant are also represented in Figure 1. Likewise, the relationship to body structure

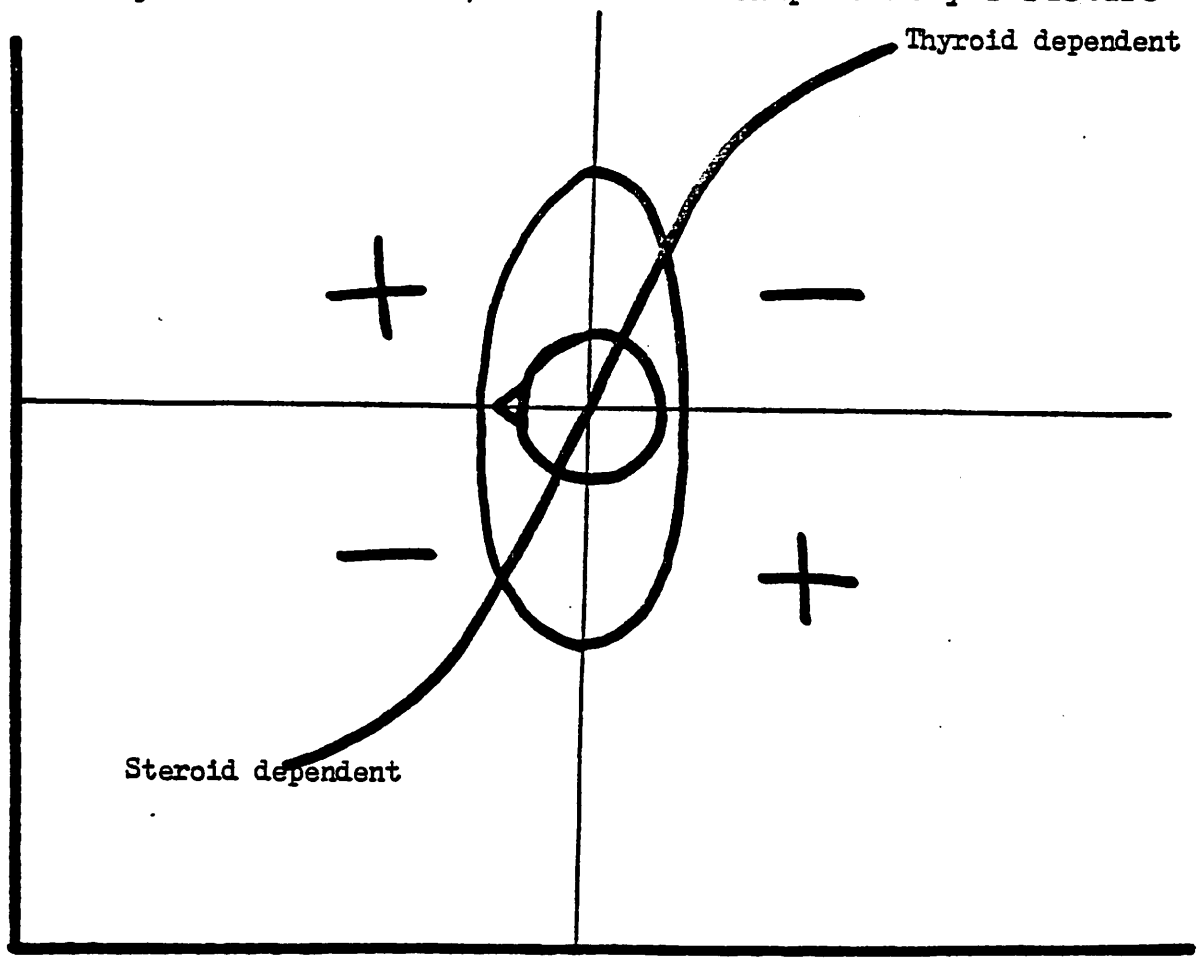


Figure 1

Electron Poising Curve - Body Quadrants and Polarities

of the trace elements copper, manganese, and zinc was discussed.[2]

NOTE: Any techniques described in this paper must be viewed and employed in the context of these two previous papers,[1,2] or their efficacy will not be apparent.

In evaluating mineral status of the body, whether via muscle testing, hair tissue mineral analysis, or blood levels of minerals, one must make a differentiation between the trace elements (e.g., copper, manganese, zinc, selenium, chromium, etc.) and the electrolytes (e.g., calcium, magnesium, sodium, and potassium). The trace elements are greatly dependent on dietary intake and intestinal absorption for maintenance of proper levels. The electrolytes are in considerably greater abundance and, although they are also dependent on dietary intake and absorption, the body has hormonal and other self-regulatory mechanisms for these substances. Without self-regulation, one could easily kill oneself by merely over-consuming or under-consuming one of these substances at one meal due to the critical nature of the body's ionic and electrolytic balance. Just as the body is postulated to demonstrate specific structural patterns reflecting the trace mineral status, so it is now postulated that there are specific structural patterns associated with the electrolyte imbalances of the four major cations: calcium, magnesium, sodium and potassium.

BODY QUADRANTS AND THE CATIONS

Each of the four quadrants of the body in Figure 1 are represented by groups of major flexors of the spine and pelvis on the anterior of the body, and by groups of major extensors on the posterior of the body. Major flexors in this regard would be, for example, the abdominals, the psoas, and the rectus femoris. Major extensors would be the gluteus maximus, hamstrings, latissimus dorsi, erector spinae, and middle and lower trapezius muscles. Each of these major flexors and extensors exerts significant influence on the flexion or extension of the spine and pelvis.

The valences of calcium and magnesium are both plus two; i.e., Ca^{++} and Mg^{++}. The valences of sodium and potassium are both plus one; i.e., Na^{+} and K^{+}. Calcium and magnesium, therefore, are more positive than sodium and potassium. Or stated in another way, sodium and potassium are negative in relation to calcium and magnesium. If we attempt to find a parallel between these cation charges, the body structure, and the electron poising curve, we find that we can place calcium in the right anterior quadrant (+), magnesium in the left posterior quadrant (+), sodium in the left anterior quadrant (-), and potassium in the right posterior quadrant (-). See Figure 2.

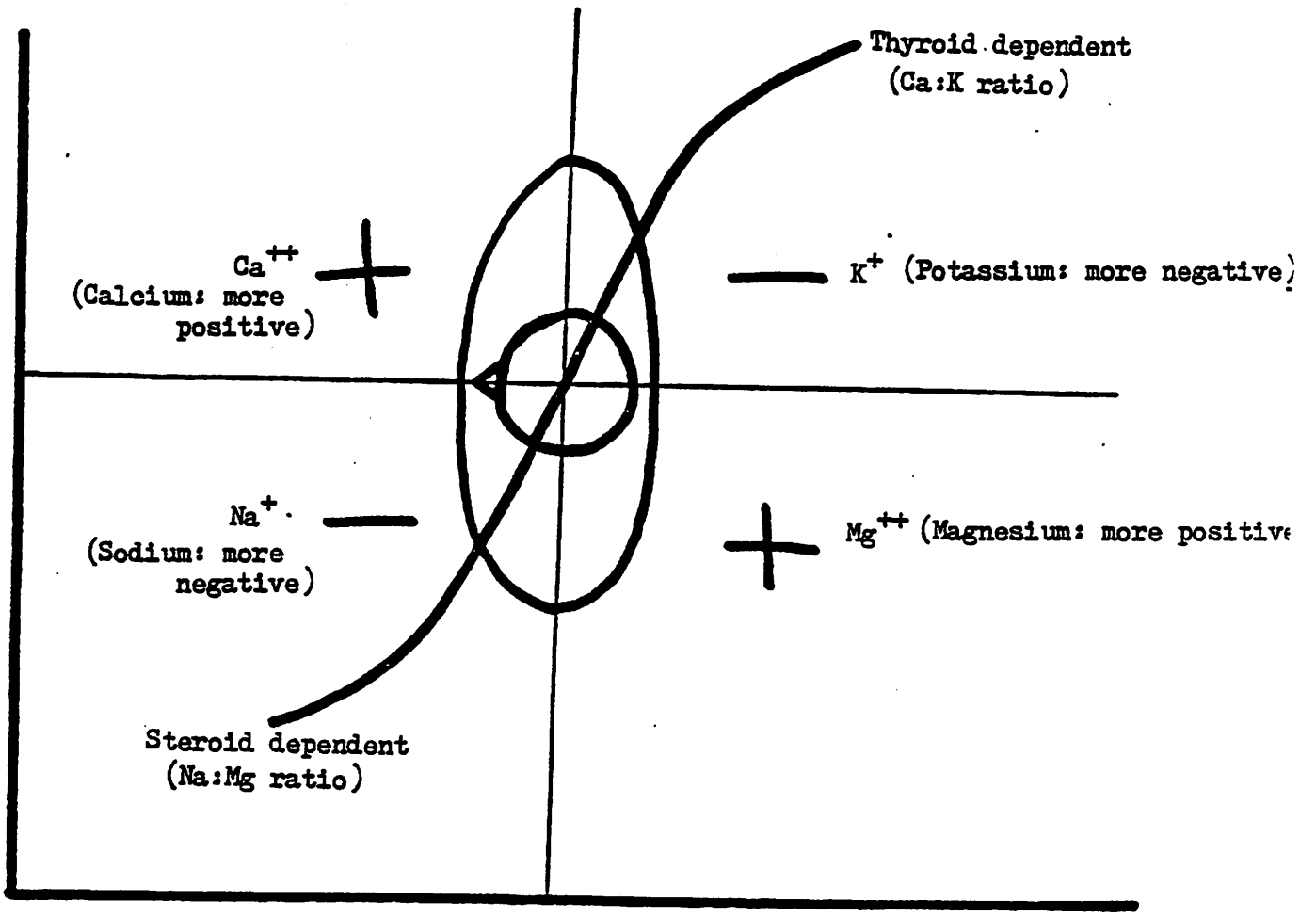


Figure 2

Body Quadrant Polarities and Related Cations

The work of Dr. Paul Eck in interpreting hair tissue mineral analysis tends to confirm these observations.[4] Eck states that the level of thyroid function can be determined by examining the calcium to potassium ratio (Ca:K). Both of these electrolytes are represented by the right side of the body in Figure 2, as is the thyroid-dependent part of the electron poising curve.[2] Similarly, Eck relates the level of adrenal (steroid) function to the sodium to magnesium ratio (Na:Mg). Both of these electrolytes are represented by the left side of the body in Figure 2, as is the steroid (adrenal/ovarian) dependent part of the electron poising curve.

THE CATIONS' EFFECTS ON THE ELECTRON POISING CURVE

If we examine the effects of these four cations on the electron poising curve, we can postulate that they each have an effect on the curve similar to the direction which their associated muscles act. That is, calcium and sodium exert influence similar to the right and left flexors, respectively. And potassium and magnesium exert an influence on the body and the electron poising curve similar to the right and left extensors, respectively. These effects are represented by the heavy, horizonal arrows in Figure 3.

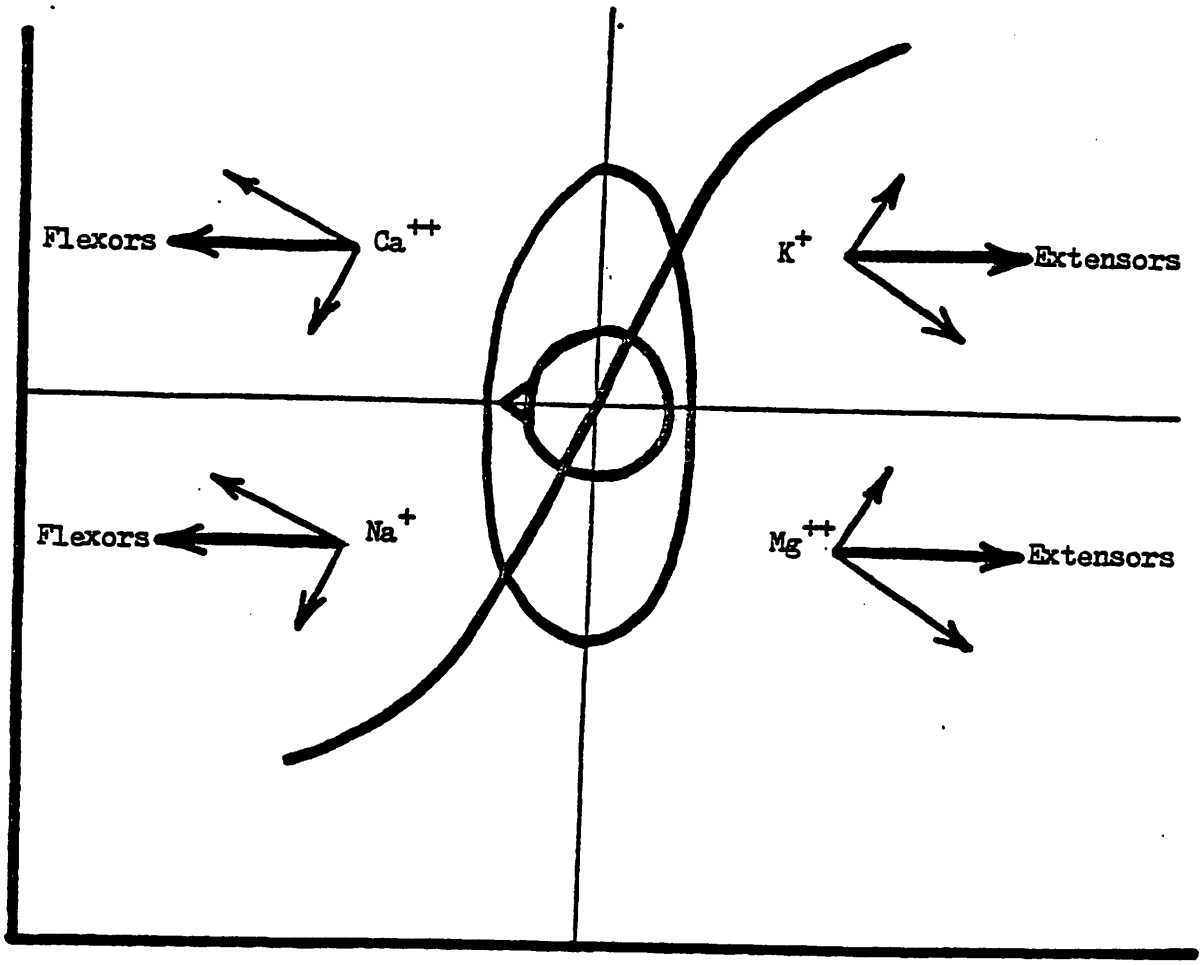


Figure 3
Effects of Cations and Their Associated Vectors Which Affect the Electron Poising Curve

To fully understand the effects of these cations, it is necessary to break down the directional factors of flexion and extension into vectors which can be related to changes observed in the electron poising curve. These vectors are seen as the thinner arrows in Figure 3. Note that these thinner vector arrows run either parallel to or perpendicular to the curve. Breaking the flexion and extension factors into these vectors makes sense since we know that the body does not flex or extend without some other factor of rotation or tilt. Further, it must be remembered that all movements take place in three planes, and the model in Figure 3 considers only two planes.

In Figure 4, we can see a summary of the effects of calcium, sodium, potassium, and magnesium which run parallel to the curve. We know that both calcium and sodium are primarily extracellular ions and that they are both necessary for cell wall depolarization and muscle contraction. In other words, the presence of calcium and/or sodium will tend to enhance the depolarization-contraction process. This pattern is consistent with what we know about the nature of the electron poising curve, as is shown in Figure 5.

The electron poising curve represents the physiological relationships which affect a cell, particularly a muscle cell, during depolarization (contraction) and repolarization (relaxation). Those substances which promote depolarization (contraction) help induce changes in the cell which cause

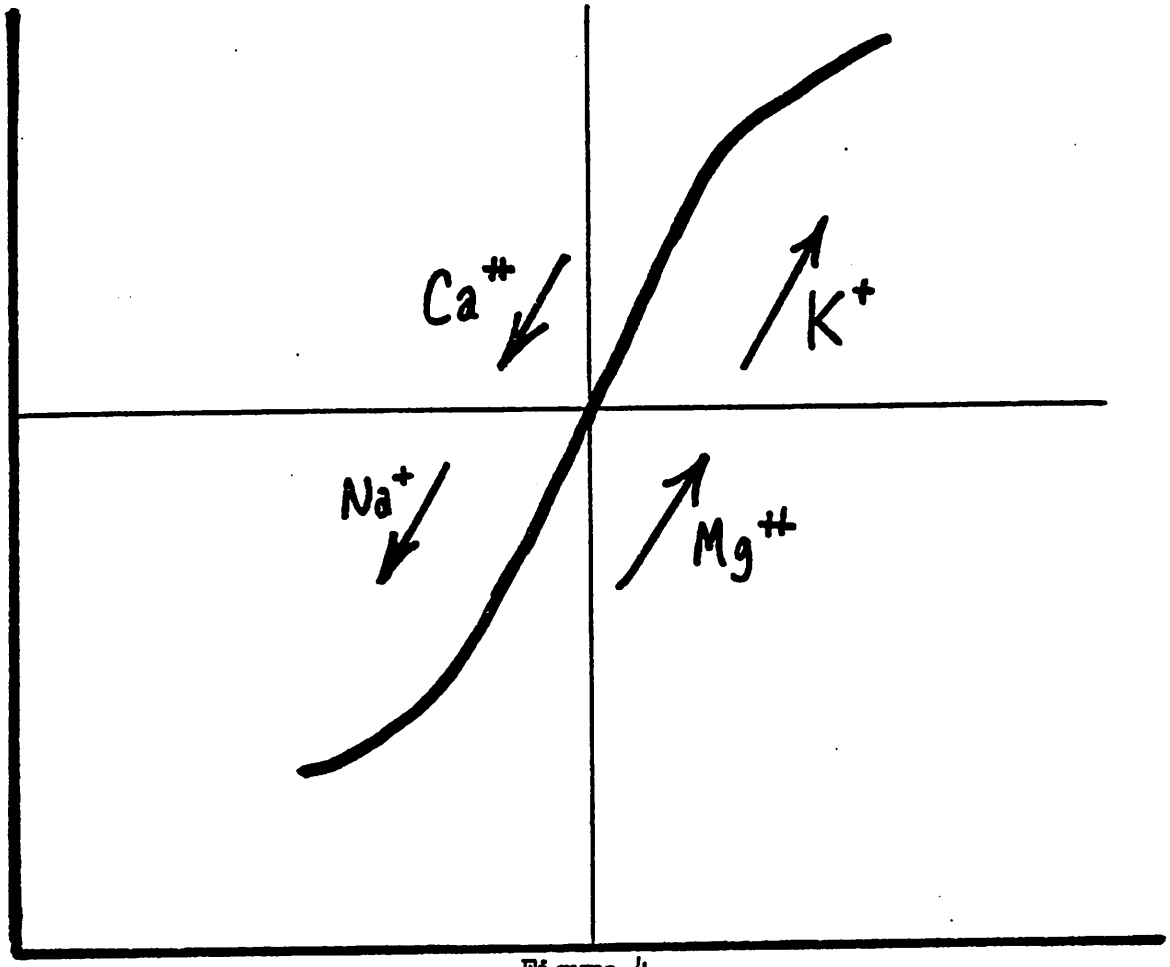
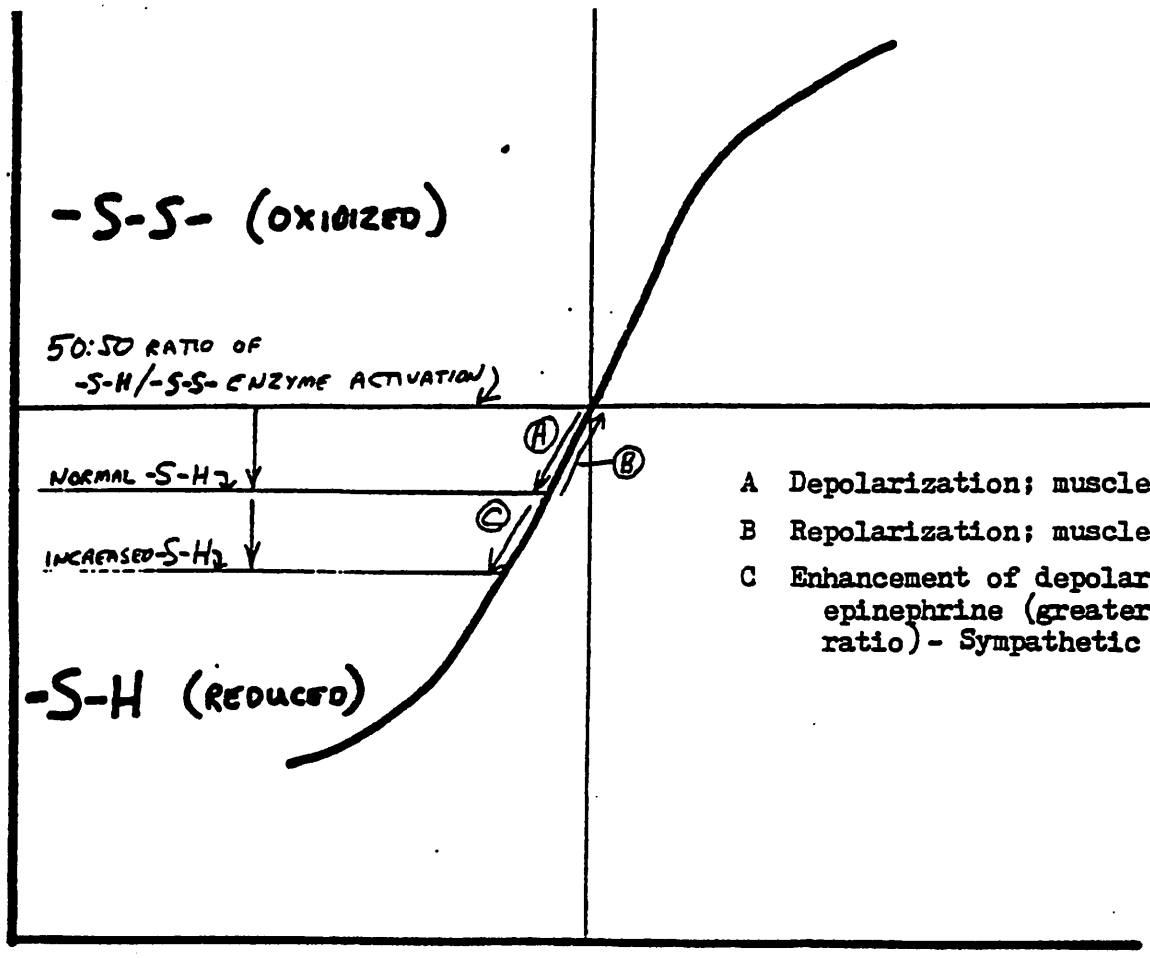


Figure 4



A Depolarization; muscle contraction

B Repolarization; muscle relaxation

C Enhancement of depolarization by epinephrine (greater S-H/-S-S- ratio) - Sympathetic Dominance

Figure 5

a move along the curve from its mid-point, down and to the left. Repolarization (relaxation) is represented by a return up and to the right to the mid-point. (See Figure 5.) Epinephrine enhances depolarization (muscle contraction) as shown in Figure 5 and this is the situation which accompanies a sympathetic dominant patient. Note that the effects of calcium and sodium in Figure 4 parallel the direction which enhances depolarization, that is, moving down and to the left along the curve, as described in Figure 5. Calcium and sodium work in parallel with depolarization and contraction, and also in the same direction as epinephrine and sympathetic dominance.

Potassium and magnesium are seen in Figure 4 to exert an influence along the electron poising curve in the direction up and to the right. This is the direction which corresponds to repolarization and muscle relaxation as represented by Figure 5. We know that potassium and magnesium are primarily intracellular ions and they are well known to be necessary for muscle relaxation. Further, potassium and magnesium are alkaline ash minerals, and as such, they support the parasympathetic nervous system. In other words, potassium and magnesium counteract the effect of the sympathetic nervous system and epinephrine. This can readily be seen by comparing the directions of the effects of potassium and magnesium in Figure 4 with the effect of epinephrine represented in Figure 5.

If we examine the cations' vectors which run perpendicular to the electron poising curve, we see the pattern described by Figure 6. Note that the influence of calcium and magnesium working together in this Figure tends to rotate the curve in a counterclockwise (CCW) direction. Similarly, the influence of sodium and potassium working together rotates the curve in a clockwise (CW) direction. More will be said of these rotation patterns later in this paper.

In a similar manner of looking at the effects of the various combinations of cations' vectors. we can observe in Figure 7 that the influence of the calcium vector from Figure 6, combined with the potassium vector from Figure 4, tends to pull the entire curve in an upward direction (toward the oxidation side of the graph). Likewise, combining the vector effect of sodium from Figure 4 with that of magnesium from Figure 6 demonstrates the tendency to pull the entire curve in a downward direction (toward the reduction side of the graph). These patterns will also be discussed later in this paper.

MUSCLE PATTERNS ASSOCIATED WITH CATION IMBALANCES

Following treatment, we always perform a post-check postural analysis on our patients. At this time, we frequently observe residual postural muscle imbalance which is then further investigated and corrected. This author's experience has been that the most common residual muscle weakness pattern has been one of a high shoulder and low

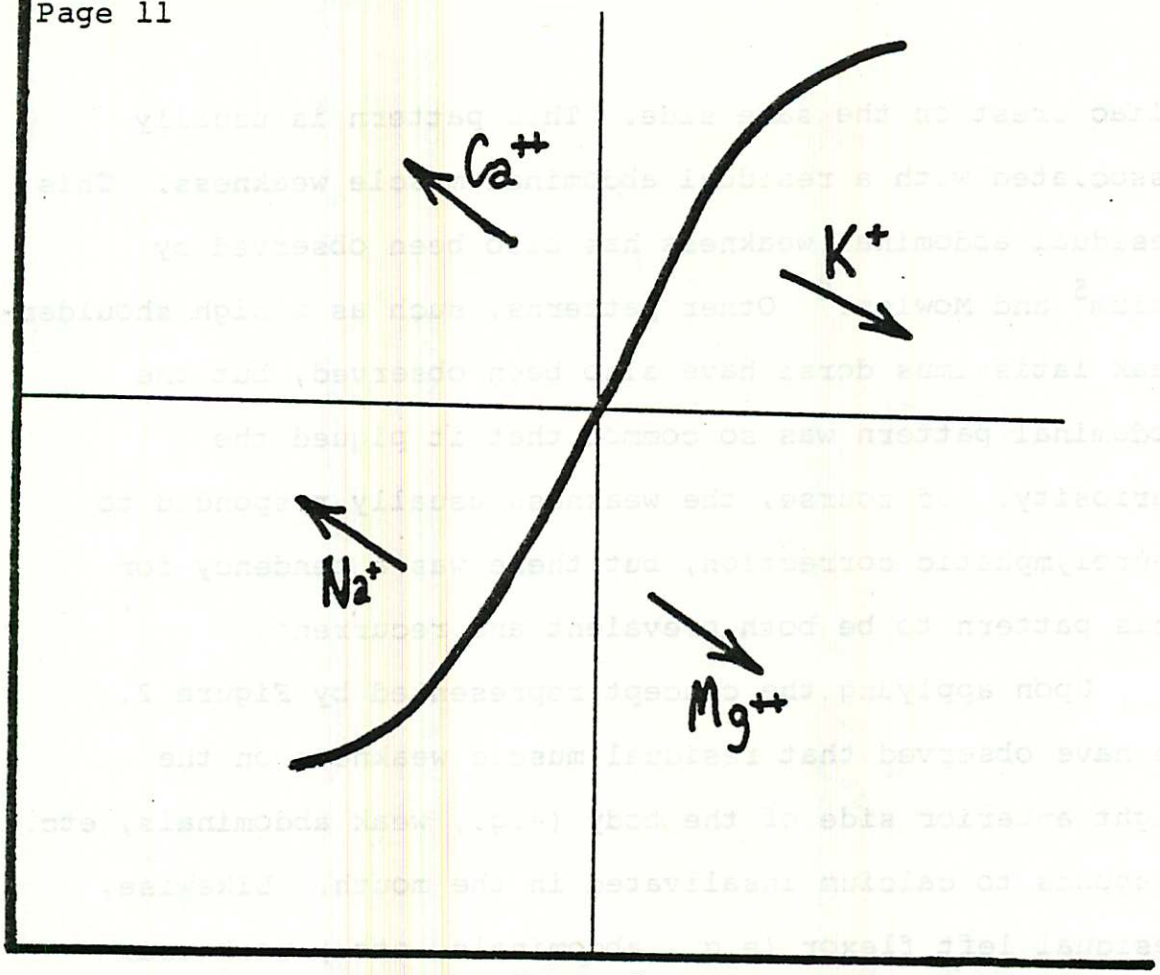


Figure 6

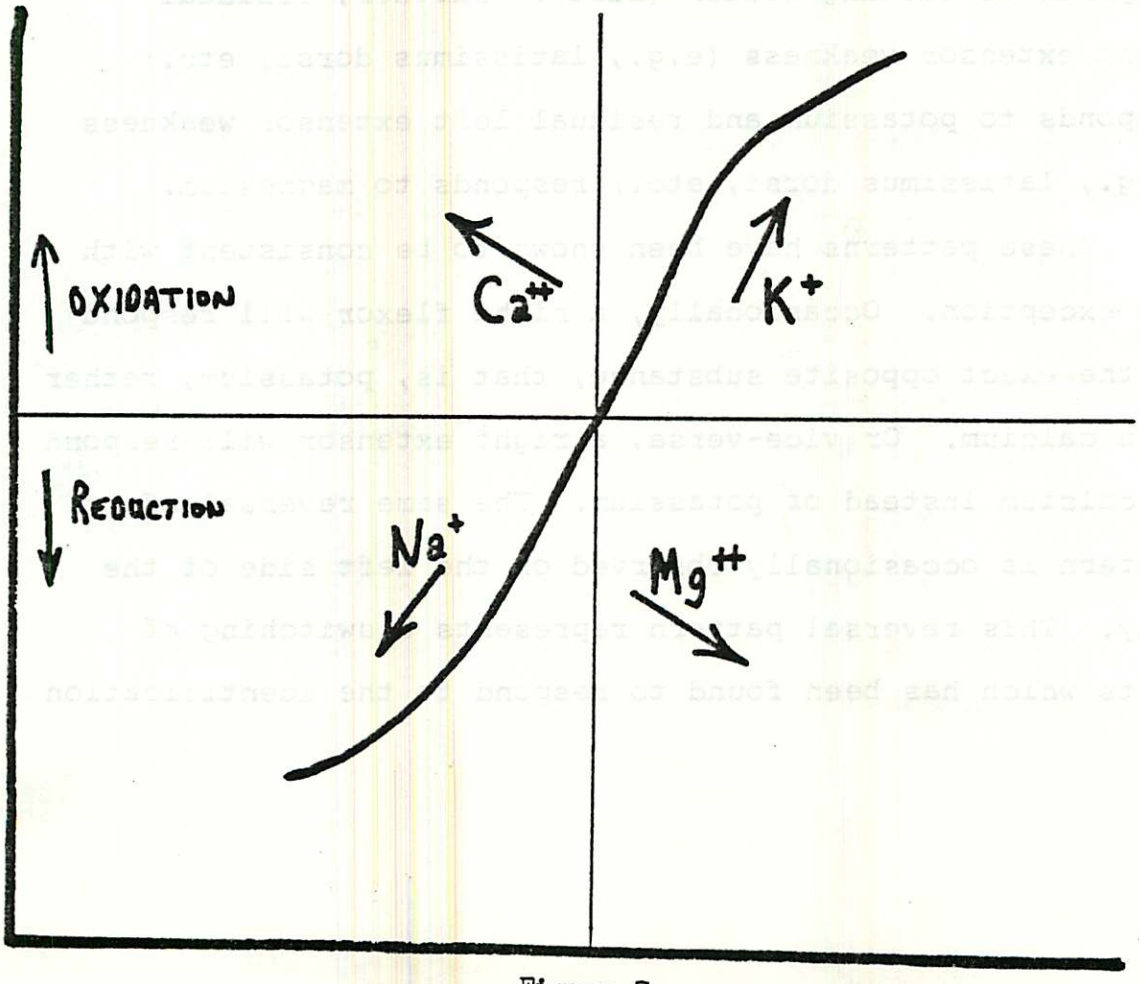


Figure 7

iliac crest on the same side. This pattern is usually associated with a residual abdominal muscle weakness. This residual abdominal weakness has also been observed by Colum[5] and Mowles.[6] Other patterns, such as a high shoulder-weak latissimus dorsi have also been observed, but the abdominal pattern was so common that it piqued the curiosity. Of course, the weakness usually responded to neurolymphatic correction, but there was a tendency for this pattern to be both prevalent and recurrent.

Upon applying the concept represented by Figure 2, we have observed that residual muscle weakness on the right anterior side of the body (e.g., weak abdominals, etc.) responds to calcium insalivated in the mouth. Likewise, residual left flexor (e.g., abdominals, etc.) weakness responds to tasting sodium (salt). Further, residual right extensor weakness (e.g., latissimus dorsi, etc.) responds to potassium and residual left extensor weakness (e.g., latissimus dorsi, etc.) responds to magnesium.

These patterns have been shown to be consistent with one exception. Occasionally, a right flexor will respond to the exact opposite substance, that is, potassium, rather than calcium. Or vice-versa, a right extensor will respond to calcium instead of potassium. The same reversal of pattern is occasionally observed on the left side of the body. This reversal pattern represents a switching of sorts which has been found to respond to the identification

and correction of the tilted sphenoid cranial fault by nasal-sphenoid technique. Upon correction of the tilted sphenoid, the expected pattern of Figure 2 is re-established.

A very interesting additional pattern was observed in most of these patients. Upon supplying the appropriate electrolyte and observing a muscle strengthening response, another major flexor or extensor muscle in an opposite quadrant, previously strong following treatment, spontaneously becomes weak. For example, placing calcium in the mouth strengthens a weak right abdominal muscle, but causes spontaneous weakness of the right latissimus dorsi. With the calcium in the mouth, the addition of potassium to the mouth causes strengthening of both muscles and no other identifiable weakness. (Note: This often does cause a positive therapy localization to occur at left Sp-21, but a discussion of this factor will be left for another paper.) Or the strengthening of the right abdominal with calcium may cause spontaneous weakness of the left gluteus maximus (for example) which then responds to magnesium.

The pattern of one electrolyte uncovering the apparent need for another is the rule rather than the exception. The pattern has always been observed between flexors and extensors. That is, correcting a flexor weakness (Ca or Na need) uncovers an extensor weakness (K or Mg need). Or, vice-versa, upon correcting an extensor uncovers a flexor weakness. It has not been seen between right

and left flexors. Nor has it been observed between right and left extensors. It has only been seen between a flexor and an extensor, or vice-versa. The reason for this is unknown.

A few patients were supplemented with these minerals based on the observed pattern, but it was reasoned that, for the most part, electrolyte levels were self-regulated by the body's homeostatic mechanisms, and supplementation of these dietarily abundant substances should usually be unnecessary. The next step, then, was to identify what mechanisms were at fault to allow an apparent need for these minerals to be present.

CATION REGULATION BY THE ENDOCRINE SYSTEM

Further investigation via therapy localization (T.L.) revealed at least a partial answer to this question. When a residual major flexor or extensor muscle weakness was found, its relationship to the appropriate mineral was established by oral testing. The substance was then removed from the mouth and the weakness was allowed to recur. Since hormonal control plays an important role in electrolyte self-regulation, T.L. to each of the endocrine organ neurolymphatic reflexes (NL) was performed until one (or more) NL was observed to neutralize the weakness of the residual weak muscle.

For example, a residual weak right abdominal responds to calcium. Calcium is removed from the mouth and T.L.

to all significant endocrine NL's reveals that T.L. to a parathyroid NL neutralizes the weakness. This NL is then treated with the subsequent strengthening of the abdominal. Usually a spontaneous weakening of one of the major extensor muscles is then observed. For example, the left latissimus dorsi. It responds to magnesium. The magnesium is removed from the mouth and a screening by T.L. of all of the potentially related endocrine NL's is similarly performed to identify the source of this muscle weakness/mineral imbalance. The latissimus strengthens with T.L. to the ovarian NL. It is also treated. Usually, the muscle related to the endocrine organ is not weak, nor has it been found weak during the treatment of the patient. However, there are often clinical signs of a problem in this organ, such as dumping blood pressure (postural hypotension) in an adrenal problem or low temperature in a thyroid problem. It is felt that this procedure may play an important role in identifying endocrine involvement which escapes the usual organ-muscle weakness pattern. We have observed several dramatic blood pressure changes using this procedure which were previously unresponsive to treatment.

CATION BALANCE, TORQUE PATTERNS, AND THE ENDOCRINE SYSTEM

Upon identifying weakness in two quadrants, one can extrapolate the observed muscle imbalances to parallel changes in the electron poising curve. Since the structural

relationships of the body parallel chemical relationships expressed by the electron poising curve, a patient who shows weakness in the left flexors quadrant (responsive to sodium) and the right extensors quadrant (responsive to potassium) will appear as in Figure 8, torqued in a counterclockwise (CCW) fashion. This is the case, regardless of which quadrant was found weak first or second.

Likewise, a patient who shows weakness in the right flexors quadrant (responsive to calcium) and in the left extensors quadrant(responsive to magnesium) will appear as in Figure 10. This represents a clockwise (CW) torquing when viewed from above. Note that the shift in body structure in these two patterns is accompanied by counterclockwise (CCW) or clockwise (CW) rotations of the electron poising curve.

NOTE : In this discussion and hereafter, when referring to torquing of the body, we assume that we are viewing the body from above. Sometimes, the pelvis can torque one way (e.g., CW) while the shoulders torque in the opposite way (e.g., CCW). Always look at the torque pattern in reference to what the pelvis is doing while sitting on top of the femur heads. The primary torque in these discussions is of the pelvis on the femurs, and any opposite torque of the upper body (or head) is viewed as compensatory and to be ignored when studying the figures and discussion in this paper.

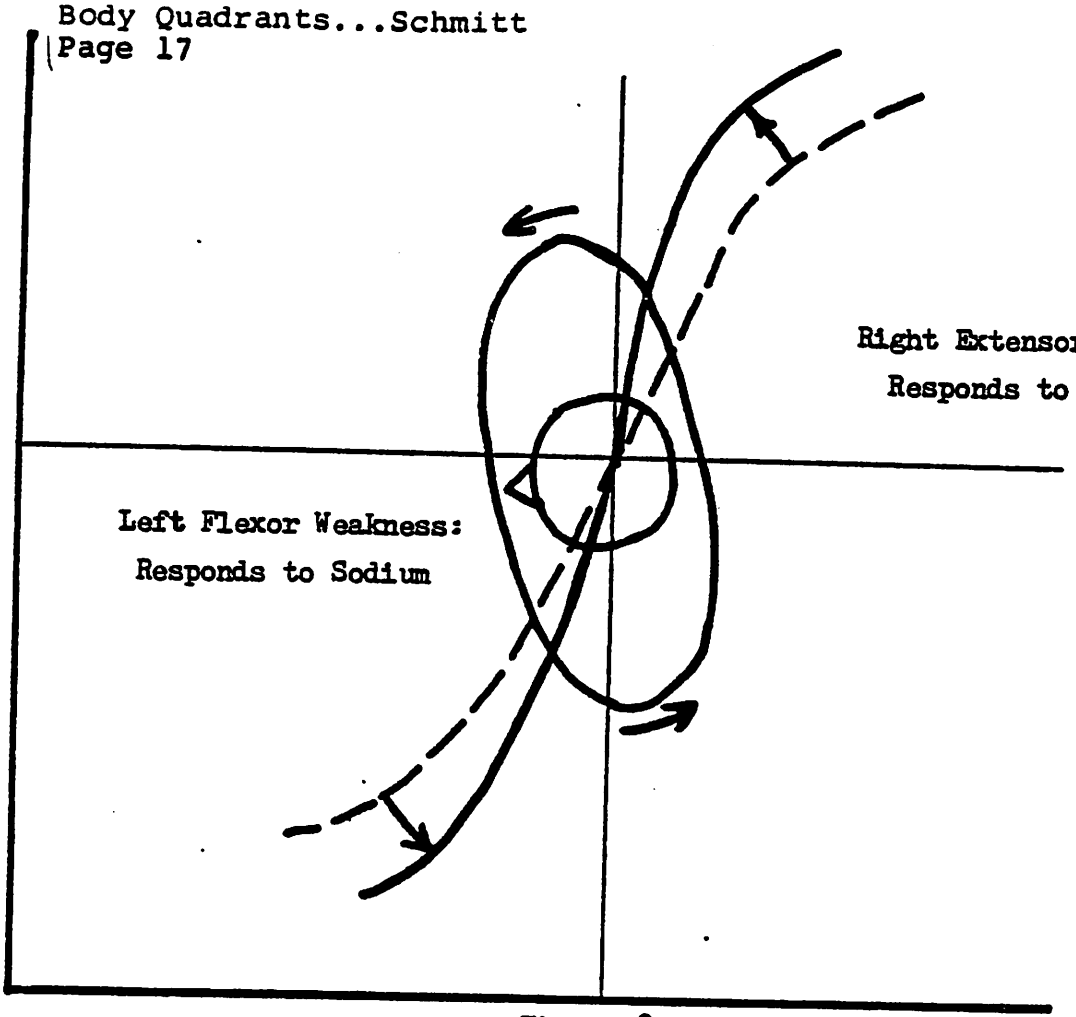
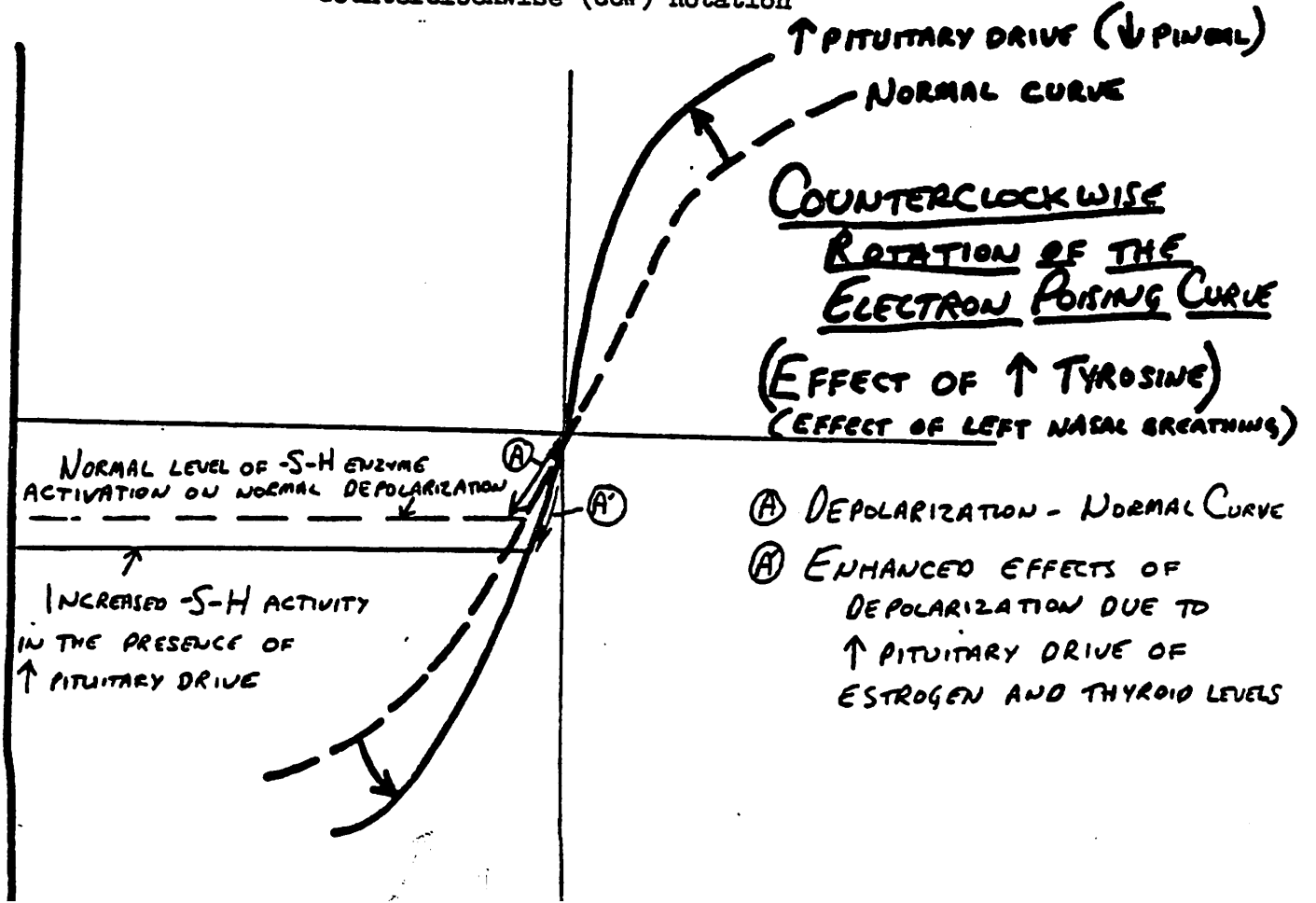


Figure 8
Counterclockwise (CCW) Rotation

Fig. 9



In a previous paper by this author,[1] CCW and CW rotations of the electron poising curve were related to enhancing and dampening of the general endocrine system, respectively. The CCW rotation of the curve in Figure 8 is associated with increased pituitary drive and heightened endocrine system activity, as is summarized in Figure 9. This pattern is also associated with increased tyrosine availability as a precursor to both hormonal (thyroid, epinephrine) and neurotransmitter (norepinephrine) substances, that is, the "uppers" on both hormonal and neurotransmitter levels. This pattern is also associated with decreased pineal gland function and decreased tryptophan availability in relationship to tyrosine.

Clockwise rotation of the electron poising curve, as seen in Figures 10 and 11, is associated with a dampening effect on the endocrine system. This occurs in increased pineal gland activity which has an inhibitory effect on pituitary drive. It is also associated with increased availability of tryptophan (in relationship to tyrosine). Tryptophan is the precursor to the pineal hormone melatonin, as well as the precursor to serotonin, a "downer" type of neurotransmitter (in a sense) as well as a dampener at the cellular level, based on its effects on the electron poising curve. (See previous paper.[1])

Eck[4] uses the relative hair levels of calcium, magnesium, sodium, and potassium to define what he calls "slow oxidizers," "fast oxidizers," and "mixed oxidizers."

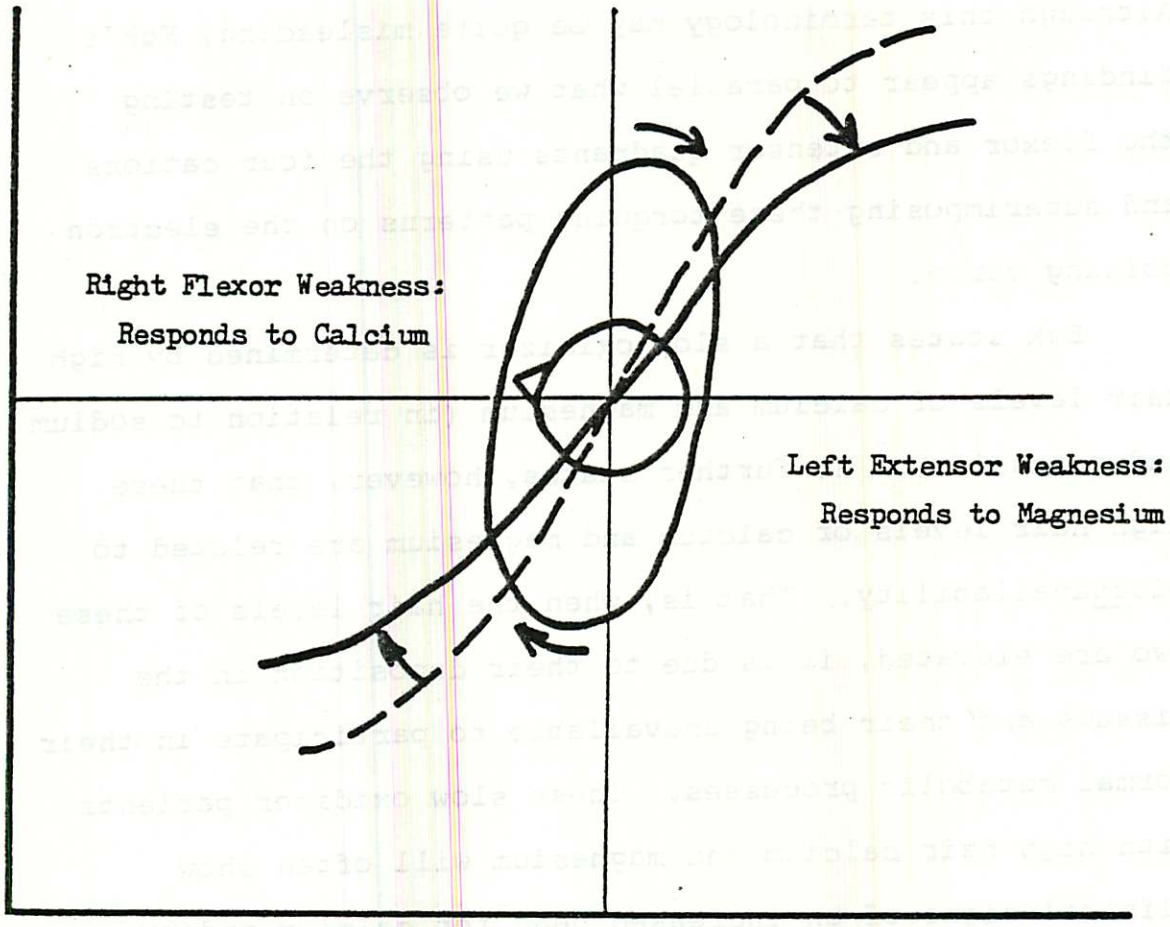
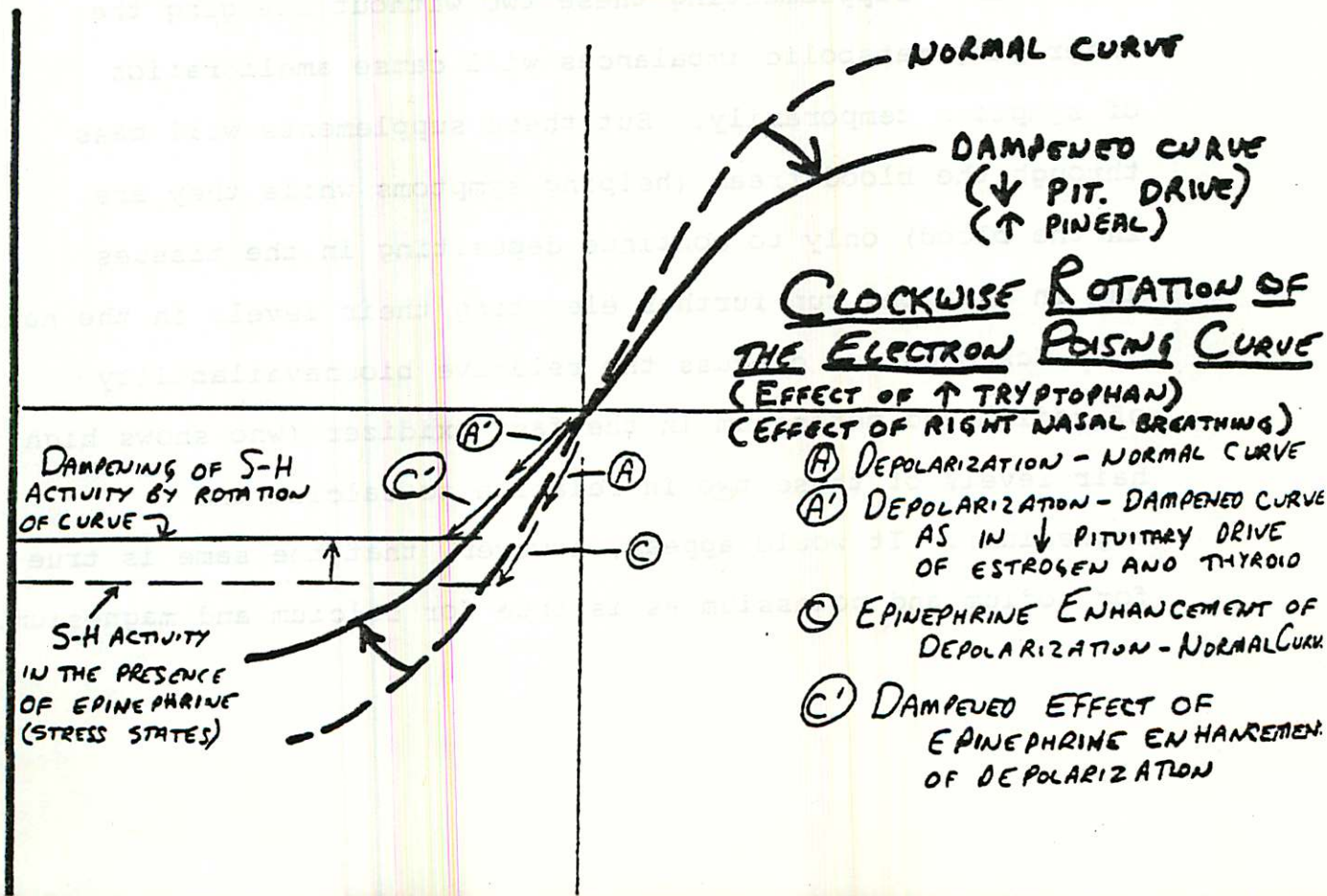


Figure 10
Clockwise(CW) Rotation

Fig. 11



Although this terminology may be quite misleading, Eck's findings appear to parallel what we observe on testing the flexor and extensor quadrants using the four cations and superimposing these torquing patterns on the electron poising curve.

Eck states that a slow oxidizer is determined by high hair levels of calcium and magnesium (in relation to sodium and potassium). He further states, however, that these high hair levels of calcium and magnesium are related to biounavailability. That is, when the hair levels of these two are elevated, it is due to their deposition in the tissues and their being unavailable to participate in their normal metabolic processes. These slow oxidizer patients with high hair calcium and magnesium will often show clinical signs of an increased need for calcium and/or magnesium. Supplementing these two without changing the underlying metabolic imbalances will cause amelioration of symptoms temporarily. But these supplements will pass through the bloodstream (helping symptoms while they are in the blood) only to continue depositing in the tissues and in the long run further elevating their levels in the hair.

Eck does not discuss the relative biounavailability of sodium and potassium in the fast oxidizer (who shows high hair levels of these two in relation to calcium and magnesium). It would appear, however, that the same is true for sodium and potassium as is true for calcium and magnesium.

That is, in a fast oxidizer, there is apparently a deposition of sodium and potassium in the tissues (including the hair) leading to their biounavailability. Increasing these two in the diet would give only a temporary response at best, with a long-term aggravation of symptoms and the hair mineral imbalance pattern.

In the slow oxidizer, then, there is an apparent need on the surface for calcium and magnesium, even though supplementation of these is not indicated. If we compare the flexor-extensor torque pattern associated with an apparent calcium-magnesium need (Figures 10 and 11), we observe that the electron poising curve is rotated in a CW fashion, also indicating a slowing down of metabolic processes (i.e., increased pineal activity/decreased pituitary drive and increased tryptophan/decreased tyrosine availability). Although Eck's terminology of "slow oxidizer" may be difficult to interpret, the general concept of a "slowing down" runs through both approaches.

Likewise, when sodium and potassium are elevated on hair analysis, if we postulate that this represents a similar (to calcium and magnesium) biounavailability, then the fast oxidizer patient will show an apparent need for these two cations. The need for sodium and potassium is represented by Figure 8 with a CCW torquing of the body and a parallel rotation of the electron poising curve. This CCW rotation of the curve is associated with increased

metabolic function by increased pituitary drive and decreased pineal function (and increased tyrosine/decreased tryptophan availability, as in Figure 9). Again, Eck's concept of the "fast oxidizer" and its associated sodium-potassium pattern parallel exactly what is observed in changes in body structure (torquing) when there is an apparent need for these minerals. And this pattern exactly parallels the relative rotation of the electron poising curve associated with "a speeding up" of the metabolism.

STRUCTURE, GAIT, AND BODY CHEMISTRY INTERACTIONS

Goodheart has recently been investigating body torquing patterns in relation to body mechanics, structural faults, and gait patterns.[7] We see direct parallels between this work and the electron poising-mineral balances discussed above and in a previous paper dealing with copper, manganese, and zinc patterns.[2] Our findings indicate that there is a complex pattern of interaction between hormones, vitamins, and minerals. Depending on the state of the patient when presenting to the office, we sometimes find it necessary to correct structural and gait patterns to balance the apparent chemical imbalances. Just as often, however, we find it necessary to correct chemical imbalances in order to correct structural and gait patterns. It is sort of like the chicken and the egg - one is not quite sure which came first, and therefore which to correct first, the body structure or the body chemistry. We have certainly noted rapid return to normal gait patterns in most patients when

we approach the body on a chemical level, and have also noted apparent chemical imbalances self-correct when appropriate structural and gait changes are made. It appears, however, that the patient who must continually walk with a longer stride with one foot, or the patient whose side of greater stride changes from side to side on subsequent visits is much better approached on a chemical level with appropriate supplementation employed to reinforce structural and gait correction. We have been able to eliminate the need of consciously walking with one step greater than the other in most patients when we have first employed the indicated supplementation and/or related treatment (i.e., endocrine NL's) to de-torque the patient.

Let us take, for example, a patient who is in a "slowed down" or dampened chemical patterns, as in Figures 10 and 11. Goodheart[7] has identified torquing patterns in patients by employing varying stride length as well as a torquing of the patient lying supine on the treatment table with orthopedic wedges (DeJarnette blocks) placed under one hip and the opposite shoulder. Gait differential and/or block placement to aggravate the patient's torquing pattern have resulted in muscles weakening. If we employ this block placement torquing technique to identify weakness to the patient in Figure 10, we will find weakness occurring when the blocks are placed under the left hip and the

right shoulder. Underlying chemical imbalances will recreate this torquing pattern of the electron poising curve transposed to the body structure and cause the patient a continuing need to walk with varying strides as well as a recurrent torquing pattern every time the patient returns to the office.

The weakness induced by the blocks in this patient may be neutralized by placing calcium and magnesium in the mouth simultaneously; or by placing tyrosine in the mouth. Tyrosine causes the curve (and the body) to rotate in a CCW torque direction, thereby neutralizing the weakness caused by the blocks. (See Figure 9.)

At this point, the tyrosine (or calcium and magnesium) placed in the mouth often brings out another hidden problem. This is usually a cranial-spinal torque pattern[8,2] or a category one pelvis. In the case of the category one patient, tyrosine (or calcium and magnesium) in the mouth negates the necessity of employing a gait pattern (i.e., one leg hanging off the table) or alternate dorsal and palmar T.L. patterns to identify the presence of the category one. With the appropriate substance(s) in the mouth, a simple two-handed palmar T.L. to the sacroiliacs of the prone patient causes the expected indicator muscle weakness. The category one is corrected in the usual fashion. Need for gait changes are rapidly eliminated when the structure is approached as secondary to the chemical

imbalances which create a torquing of both the body and the electron poising curve.

The fact that electrolyte imbalances, for example, can create major muscle imbalances which result in body torquing patterns is easily demonstrated. Take the patient in the above example, who has weakness of a right flexor and a left extensor which has responded to calcium and magnesium, respectively. (Figure 10) After correcting these weaknesses by using the appropriate minerals, remove them from the mouth and place the opposite minerals, that is, sodium and potassium, in the mouth. These will cause a weakening of every (or nearly every) muscle in the body. Tryptophan may likewise tend to cause a weakening response (although mild) in this patient by aggravating the CW torquing. Since the muscle quadrant weaknesses are found following correction of all other major structural corrections, it can readily be seen how these mineral imbalances and/or underlying endocrine imbalances severely limit the potential for correction by the patient's self-regulatory systems, even if the patient is instructed to walk with a varying stride pattern.

NASAL RESPIRATION PATTERNS AND BODY TORQUE

Right and left nasal ionization respiration patterns were first described as affecting muscle patterns by Goodheart in 1976.[9] Goodheart relates his findings that the right nostril positively ionizes air inhaled through it and the left nostril negatively ionizes air inhaled

through it. This right-positive and left-negative pattern correlates with the polarities on the anterior side of the body previously discussed.

More recently, Goodheart[10] has demonstrated the relationship of nasal respiration to body torque patterns. He has based these findings on the research of David Shannahoff-Khalsa as reported in the January 3, 1983 edition of the Brain-Mind Bulletin.[11] Shannahoff-Khalsa reports that there is definite increased electroencephalographic (EEG) activity in the right cerebral hemisphere during left nasal breathing. Likewise, there is increased EEG activity in the left cerebral hemisphere during right nasal breathing. Goodheart has related this cerebral hemispheric activity to body torque patterns based on the placement of orthopedic wedges (DeJarnette blocks) and variance in stride length. To summarize, if the patient weakens with a greater stride with the left foot, he will also weaken when, in the supine position, a block is placed under his left hip and another block is placed under his right shoulder. This weakening pattern will be neutralized by left nasal respiration.

Goodheart's reasoning behind this pattern is that the left nasal breathing increases right brain activity which in turn increases general activity in the left side of the body. Since left leg gait patterns weaken, it is reasoned that increasing right brain activity will enhance cerebral outflow to the left lower extremity and balance

out the weakening reaction. The fact that the right arm is torquing anteriorly (or swinging forward during gait) in these patients and that the right arm is controlled by the left brain seems to be contradictory to this reasoning. That is, increasing right brain function by left nasal respiration would seemingly only aggravate this right arm pattern and this has been a somewhat perplexing point. An extension of the concepts of nasal ionization helps to clear up this issue as well as put nasal ionization technique in perspective with the electron poising curve/ body torque framework.

In the book, The Ion Effect,[12] the author, Fred Soyka, relates the work of Dr. Albert P. Krueger on the effects of ions on physiological function. Krueger demonstrated that "an excess of positive ions caused the overproduction of serotonin in mammals...." We know that the effect of serotonin as a neurotransmitter, in the general sense, is as a "downer." The precursor for serotonin, tryptophan, is also the precursor for the pineal hormone, melatonin, which is a "downer" for the entire endocrine system. The effect of increased levels of the neurotransmitter serotonin from increasing tryptophan levels was discussed in a previous paper[1] and is summarized in Figure 11. Combining all of these facts, we can see that increasing right nasal breathing increases positive ionization of the air being breathed which increases serotonin production, which causes a general dampening

(or downer) effect in the nervous system which parallels the curve we see in Figure 11 and the body torque we see in Figure 10. Therefore, right nasal respiration increases the torque of the curve and the body in a CW direction. This means that right nasal respiration would also aggravate the weakness induced by the blocks placed under the left hip and right shoulder as well as aggravate the left-footed longer gait pattern. And therefore, in contrast, the left nasal breathing pattern would be expected to have the opposite pattern, that is, of neutralizing this torque weakness and the left gait weakness, which is exactly what Goodheart has found, but for expanded reason.

The left nasal breathing pattern will negatively ionize the air being breathed. If we can extrapolate what we know about right nasal breathing (positive ionization - increased serotonin - CW torquing of the curve - dampening) to the left nasal breathing, then we can postulate the following : left nasal breathing, which increases negative ionization of the air being breathed, will cause an increase in neurotransmitter activity of norepinephrine and a general enhancing of endocrine function (i.e., increased pituitary drive), which will ultimately cause a CCW rotation of the electron poising curve (Figure 9) and a similar torquing of the body (Figure 8). These patterns of nasal ionization fit perfectly into our previously reported concepts of the electron poising curve/body structure relationship,

as well as supporting what Goodheart has reported regarding the structural and gait patterns in relation to nasal respiration. When two things equal the same thing, they equal each other. It is felt that these findings help to neatly tie together the structural and chemical sides of the triangle as we know them at the present time. There are other patterns of activity which complement these findings. However, these will be presented in another, later paper.

OVER-OXIDATION AND OVER-REDUCTION

An alternate pattern of electrolyte imbalance from that described above involves flexor and extensor weakness on the same side of the body. For example, a patient has a weak left rectus femoris which responds to sodium. With sodium in the mouth, a weakness of the left latissimus dorsi becomes apparent. The left latissimus then responds to magnesium. This pattern is associated with what is observed in Figure 12. Note that sodium and magnesium are both associated with the left side of the body and the lower, steroid-dependent half of the electron poising curve. It is assumed that these patients have a deficient steroid (adrenal/ovarian) pattern which allows the curve and the body chemistry to readily become over-oxidized (i.e., move towards the upper side of the graph into the area of greater oxidation). The lower half of the graph is associated with reduction, therefore, a steroid deficient patient will tend to move up on the graph (to the right in the body) in this situation, as seen in Figure 12.

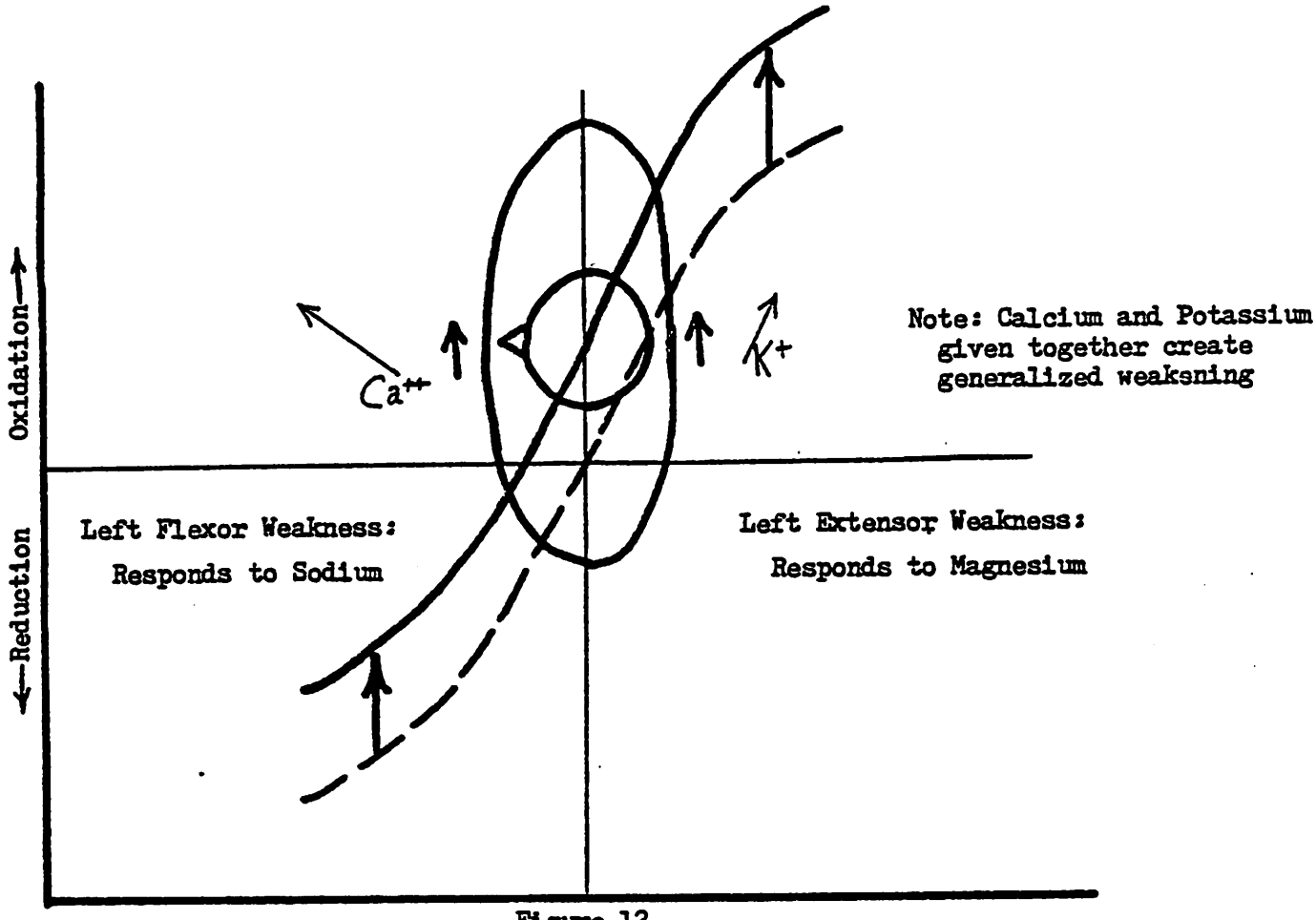


Figure 12

Shift Into Over-oxidation (Under-reduction)

This over-oxidized/under-reduced patient will usually weaken all over the body if the sodium and magnesium are removed from the mouth and replaced by calcium and potassium. Note that the calcium and potassium vectors present in Figure 7 tend to pull toward increased oxidation. (These patients will occasionally be weakened by tasting thyroid tissue and/or iodine, both of which tend to increase oxidation.) This is the classic hypoadrenic patient, although there may be no evidence of a sartorius, gracilis, or other adrenal related muscle weakness. Some patients will not weaken with the calcium and potassium

but will demonstrate the presence of a ligament stretch-adrenal stress syndrome pattern.

The weakness(or ligament stretch pattern) induced by calcium and potassium will be neutralized if either adrenal tissue or an anti-oxidant material is placed in the mouth along with the weakening minerals. Anti-oxidants which have been observed to neutralize the weakness caused by calcium and potassium in this patient include: vitamin E, selenium, and superoxide dismutase. A specific pattern of muscle imbalance other than just mentioned has not yet been identified to confirm the need for these anti-oxidant materials, but investigation into this phenomenon is ongoing.

By the same token, some patients will demonstrate a weakness of a right-sided flexor which responds to calcium and a right-sided extensor which responds to potassium. Removing the calcium and potassium from the mouth and placing the opposite substances, sodium and magnesium, together in the mouth creates weakness of most, if not all, muscles in the body. This pattern is demonstrated by Figure 13. The sodium and magnesium vectors from Figure 7 which tend to pull the curve toward reduction are also included in Figure 13. The weakness induced by sodium and magnesium in these patients is neutralized by thyroid tissue and/or other substances which tend to increase oxidation such as iodine. The use of anti-oxidants and/or adrenal tissue may have a weakening effect on this patient.

These patients are over-reduced (under-oxidized) and are very likely to have low body temperature which is difficult to raise. This is the classic hypothyroid patient. There may or may not be a weakness of the teres minor.

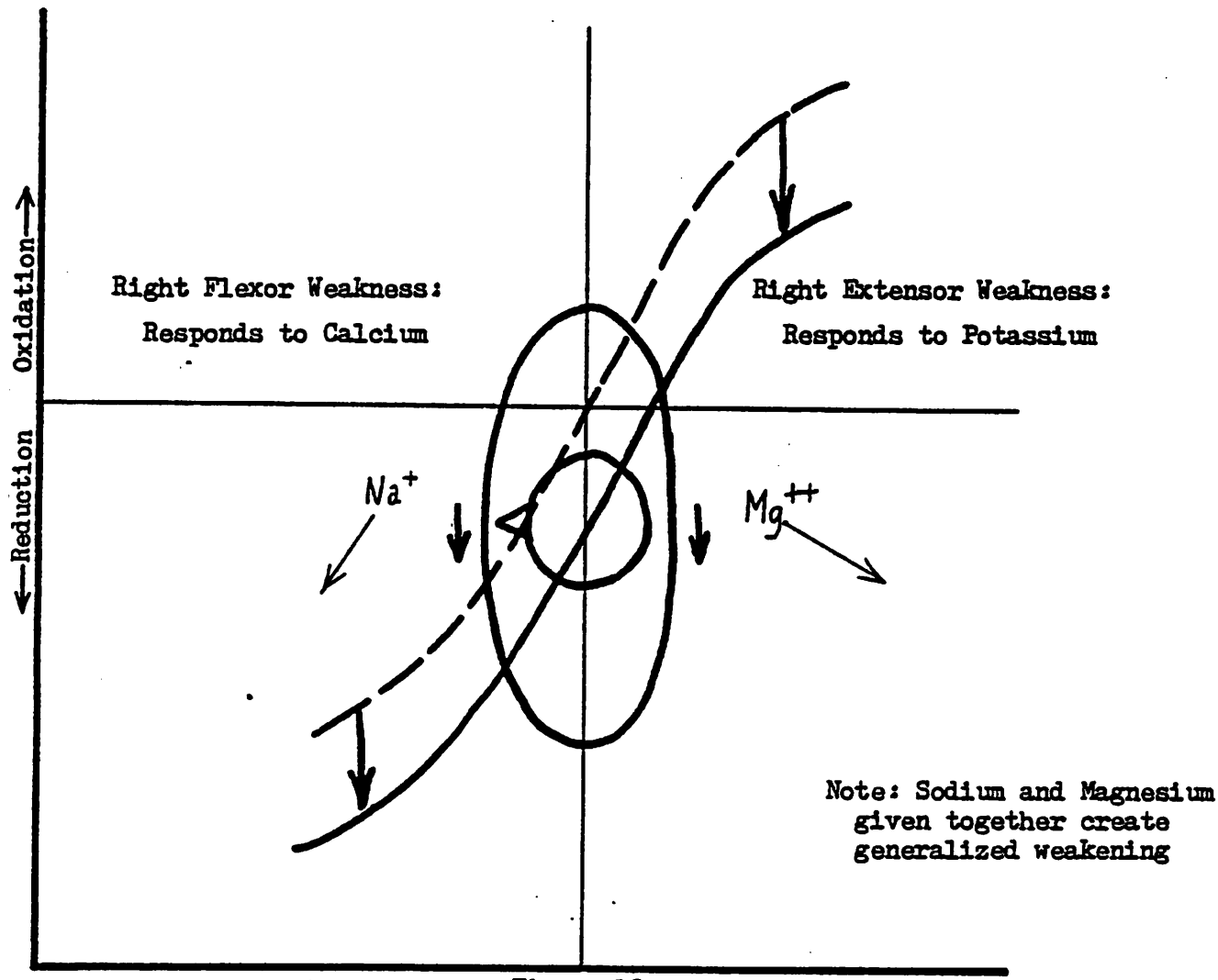


Figure 13
Shift Into Over-reduction (Under-oxidation)

ENDOCRINE INVOLVEMENT WITHOUT EXPECTED MUSCLE WEAKNESS

It is almost heresy to suggest to an applied kinesiologist that there can be an adrenal problem without any sign of sartorius, gracilis, etc. weakness. Likewise, one risks excommunication from I.C.A.K. by suggesting

lowered thyroid function with no evidence of teres minor involvement. And yet every applied kinesiologist has observed the patient with a positive Ragland sign (postural hypotension), paradoxical pupillary reflex, and/or increased second heart sound at the pulmonary area, typical patterns of hypoadrenia, but with no presence of adrenal muscle involvement. Similarly, we have all seen many patients with a low temperature and/or sluggish Achilles tendon reflex time (photomotogram), typical hypothyroid signs, with no evidence of teres minor involvement. It would seem that these patients are categorized as having "difficult or unusual" problems and thereby do not challenge the tenets upon which we base our AK practices.

The observation of the patterns in Figures 12 and 13 give us an alternate explanation to these "difficult or unusual" patients. Suppose that the adrenal gland, for example, is intact and able to function normally. We would not expect to find a sartorius, etc. weakness in this case. But suppose that due to a variety of mineral imbalances (or potentially vitamin imbalances or hormonal imbalances), the adrenals are required to function only at a very low level. For them to increase their function would be to create an imbalance in the system and/or stress some other organ or nutrient level. In other words, if the body was "stuck" in a pattern as in Figure 12, increasing adrenal function might create such an adaptation that the electron poising curve would have to rotate to compensate

and alter the levels of pituitary and pineal function causing even worse imbalances in the system, so we have signs and symptoms of hypoadrenia without the expected weakness of the sartorius, or gracilis, etc.

Since the adrenals are fully capable of responding, but for other reasons they are operating at low levels of activity, we would find clinical signs of a hypoadrenia without specific evidence of adrenal gland muscle involvement. These patients, at the same time, may be benefitted by stimulating the adrenal reflex points, especially the NL. The reflex stimulates the organ even when the muscle may not be involved, just as it stimulates the muscle when the organ is not a factor.

We have all seen those hypoadrenic patients who dramatically responded after one treatment and also those who require months or years to respond. Those who respond rapidly may have done so because their adrenals were intact and only showing signs of lowered function as a compensation to some other chemical or structural imbalance in the body. Those who respond slowly or fail to respond may well have actual adrenal gland functional disease and require a long time to heal themselves. This might also explain why patients show such varying responses to the use of adrenal tissue glandular preparations. Everything that has been said above regarding the adrenal glands may also be correspondingly true for the thyroid gland and teres minor involvement. We may, very simply, have to

rethink our approaches to many endocrine involvements.

CONCLUSIONS

The body chemistry is reflected in the body structure, as can be observed via manual muscle testing patterns. The relationships of the electron poising curve are based on sound physiological principles, and these relationships serve as the basis for interpreting changes of body structure as paralleling changes in body chemistry. The combination of the principles of this paper with those of the previous two by this author[1,2] affords the doctor the opportunity to further understand the status of the physiology and biochemistry of his patients.

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Negative Therapy Localization in the Glaucomatous Patients

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

Abstract: Clincial observations of the lack of therapy localization in patients with glaucoma.

This paper contains observations on glaucoma that I have found interesting. Over the years I have treated a good number of patients with this disorder.

As you know there are two types of glaucoma: open or wide angle and closed or narrow angle. The open angle usually develops slowly and is usually more easily controlled with medication.

Findings: My study contained 47 cases ranging from ages 13 to 75 years old. The group consisted of 32 males and 15 females.

I did my regular kinesiological examination and the one constant finding was the presence of the Heath cranial fault, which I corrected. All these patients remained stable but usually required medication to maintain normal ocular pressure.

The one other constant finding I came across was that none of the patients show any positive therapy localization to the effected eye. I wondered why this would happen. I tried our techniques to uncover the hidden problem such as E.I.D., Cerebellar Therapy Localization, RNA, Temporal Tapping, Right and Left Brain Activity, etc. None of these methods seem to be able to bring to the forefront the positive localization to the glaucomatous eye. I wondered how could this be, that therapy localization seemed to be a very useful tool in uncovering the presence of functional or pathological conditions. Why in these cases of glaucoma did it not show anything?

I did not find the answer till I took Dr. Alan Beardall-Clincial Kinesiology course. His theory is that the body has four major computer centers (Local, Spinal, Endocrine, C.P.U.).

He states that many times the body goes into an adaption rather then healing completely. The information that the patient has glaucoma is in the computer storage in the form of neurological memory, but I did not know how to program the computer to retrieve this information.

If the patients nervous system was not aware that hte patient has glaucoma how could it ever heal this condition?

Procedures: The way I used Dr. Beardall's approach I would have the patient localize the involved eye with (eye lid closed) with the finger tips. The patient was then requested to place the finger tips (all five) over each of the computer centers (umbilicus, xyphoid process, lips and nose). At this point one of the centers would usually produce a positive two point therapy localization.

Once I found the involved computer center I would have the patient contact that center with one finger at a time till the correct center was located.

I made whatever corrections were indicated by the computer network and tracing each center till I had a positive single (one point) therapy localization to the involved eye in the clear.

This should now mean that the body is aware of the patients' glaucoma. It seems reasonable that if the body is made aware of the pathology it might be able to heal it. If the healer within is unaware how could healing ever take place.

At the present time I do not have evidence as to whether or not the patients' will go to complete recovery. However, when this information becomes available I will report on it.

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THE MOST ANONYMOUS, PANDEMIC, MALTREATED SYNDROME IN THE WORLD - ILEOCECAL VALVE MALFUNCTION

By: Brice E. Vickery, D. C.

As Dr. Goodheart predicted, "the better you get, the worse your patients get", meaning, of course, that as you increase your skills the more serious, desperate cases you attract. As the severity of our cases increased, we noted the higher incidence of ICV and bowel problems was also increased.

I do not think the ICV gets the proper attention it deserves among even expert Kinesiologists, let alone the parttimers and the one, two tricksters. It is to the masters, the experts in A.K., that I primarily address this paper, for all the rest are followers.

There are only three (3) major ways ICV malfunction can be diagnosed:

1. Applied Kinesiology
2. Barium Radiograph of the Bowel
3. Electro-acupuncture According to Voll -- or possibly other types of electro-acupuncture.

Since, in my experience, the medical radiologist largely ignores the passage of barium into the ileum that excludes the entire medical profession and leaves the recognition of the syndrome to the many disciplines who are leaping into Electro-acupuncture According to Voll (E.A.V.) and the Chiropractic Applied Kinesiologist.

In evaluating how many supposed Applied Kinesiologists treat the ICV properly, I am appalled at the slap, dab, rub-em-and-get-on-with-it techniques that are used and they all say it's "fixed".

A case in point: At the 5th E.A.V. Congress in Chicago, Dr. Voll had made some astounding discoveries about a certain young lady on stage -- one of his diagnoses was Morbus Krohn -- ICV!?

I struck up a conversation and asked the young lady if possibly she could go to a chiropractor and have her ICV corrected and to get some colonic lavage. She replied: "she was under the care of a doctor who practiced A.K. and had eight (8) colonics". I told her that I thought she had fooled her doctor and could I please test her. Standing using neck flexors and two (2) handed contact therapy localization, she showed nothing -- with a sharp slap on her side, her head flew upward.

It was a great pleasure to verify what Dr. Voll had found 15 minutes earlier.

The point is, we can be often fooled and unless the doctor understands the pathological implications, he may pass off the ICV as "goat feathers", "they" say is important.

Before we get into classifying causes of ICV, let's look at the pathophysiology. The bowel has contained some 40 known deadly poisons -- to name a few: indole, skatole, phenole, creosol, putrescine, cadaverine, ethylamine, isoethylamine and other ptomaines. The bacteria, etc., will be described later.

The wastes from the blood arrive at the inner wall of the colon and cannot be excreted due to areas jammed with feces and irritated epithelium. They are reabsorbed into the body. This caused malfunction of the colon because it should excrete its products just as the kidney does urine. The ICV malfunctions, and via the portal vein, these poisons and bacteria are rapidly transported to the liver along with our food. Check the liver when an ICV is found and it will therapy localize. The liver cannot handle this amount or flood of toxins and these toxins bathe every cell of the body.

Next, check the kidneys, they are always positive and attempt to do the colons job, too. Smell the breath of your ICV patients because the lungs pitch in too! Therapy localize the entire bowel, it will show to the rectum! (See collected papers, Summer 1981). Hyperpsoas is usually concomitant and is the number one reason for facett jamming or imbrication -- about 25% - 30% of low back problems. In cases where this condition has prevailed chronically, the "itis" of the kidneys becomes "osis", and bilateral psoas weakness is the final result.

Allergies, asthma, arthritis, high blood pressure, herniated IV disc, sinusitis, hay fever, tonsilitis, hepatitis, and an endless list of diseases all relate to this syndrome. The reason for this can be better appreciated when the bacterial and viral nosodes from the Stauffen Pharma are used for testing either with A.K. or E.A.V.

with the honeycomb. Below is a list of these known by Dr. Voll or that we have personally found. Please note that bacteria like B Coli and others develop into toxic strains.

B Coli
Morgan
Proteus
Areobacterium C Coli
Clostridium Perfringens
Bacillus Cereus
Lambia Intestinalis
Toxoplasmosis
Kleysiella Sp.
Clostridium Difficile
Bac. Pyocyaneus
Pasturella Pestis
Versinia Enterocolitis
Camphylobacter Fetus
Herpes Zoster
Salmonella Typhosa
Entercoccinum
Peptococcus
Staphyococcus Auerus
Edwardsiella Tardia
Monilia Albicans
Parathphus A & B
Shigella Kruse
Shigella Flexneri
Clostridium Botulinum
Bac. Subtilis
Thermibacterium Intestinalis
Thermibacterium Bifidus
Vibrio Cholera
Herpes Simplex
Herpes Progenitalis

The foregoing named bad actors should shake the heart of the most courageous practitioner when he realizes that, that bastion of the sanctity of the rest of the body the ICV, has fallen!

This past year, we have classified ICV Syndrome thusly:

1. ICV caused by upper cervical subluxations - Secondary ICV.

2. ICV caused by diaphramatic hernia - Secondary ICV.
3. ICV caused by intestinal intoxication and dysbacteria (toxic bowel) - Primary ICV.

There are occasionally other causes, but these are the main categories. Another interesting thing - we do not recognize the "closed" ICV. Every case that ever demonstrated this mode has been successfully reversed into an "open" ICV, especially since the fine work by Dr. David Walther, D.C., et al. We are convinced that a closed ICV is a mirror image due to switching mechanisms.

TRAUMATIZED INTERVERTEBRAL DISC
APPLIED KINESOLOGICAL DIAGNOSIS TREATMENT
WITH
ROUTINE CAT SCAN EVALUATION

By: Brice E. Vickery, D.C.

ABSTRACT

We Chiropractic Physicians who practice A.K. have the finest, most sensitive tool for the diagnosis and treatment of herniated and bulging spinal discs yet developed. Discarding the invasive myelogram, we chose the later CAT scan (III & IV generation) for cross comparison and verification as routine in our treatment of disc injuries.

INTRODUCTION

We practice in rural West Redding, Ct. - a town of approximately 7000 people and 40 square miles. When people drive 20 to 30 miles from populated towns of 30,000 and upward, there is a reason for it. The answer is a final definitive answer to the varying degrees of pain they have been experiencing from weeks -- to years!

This is a common experience for all members of the College, I realize, but the added comparison of another frame of reference - CAT scan - is a learning, evaluating, confirming experience that will improve not only your treatment but the confidence you SHOULD have in the outcome of your treatment.

HISTORY

After becoming certified Applied Kinesiologists in 1977, my wife Marilyn and I were expanding our horizons at a meteoric rate. One of many conditions was, of course, the IV disc syndrome and I was not satisfied with the "pumping" taught at the time but limped along until an article by Dr. Dan Duffy (1), came to my attention. This revolutionized our treatment and made it the nearly certain

thing it is today.

My thanks, again, to Dan and, as he told me in Detroit, to Dr. George Goodheart who developed it.

After working with this a few years, we eventually dropped the therapy localization and utilized only the challenge part of the technique. This is when we caught on fire, and with later developments such as Cerebellar Tapping and E.I.D., we felt unconquerable.

A patient came in one day with his myelograms under his arm -- diagnosis: herniated L5-S1 Disc. He didn't want an operation, so we sought to help him. A.K. showed not only L5-S1 but L4-L5 and L3-L4. These were all successfully treated.

This was the beginning of our search for verification. I was tempted but decided against the myelogram as being too invasive -- harmful to the cranial sacral flow and probably having a fatality rate.

On September 10, 1982, a man sought our services for low back pain. We did not recognize his problem until the 3rd visit but not before he suffered another injury inbetween -- L5 disc was diagnosed. This was successfully treated -- however, my patient's agency, upon hearing of my diagnosis, ordered him for an orthopedic diagnosis, which included a CAT Scan. The diagnosis was verified in New York City and an operation was recommended.

My patient, who now had no pain, of course, refused. We had a repeat CAT Scan at the Danbury Hospital, which showed the following on 12/22/83.

COMPUTED TOMOGRAPHY CONSULTATION REQUES

221

HEAD CT | BODY CT

WHEEL CHAIR ☐ AMBULATOR
STRETCHER ☐ AMBULANCE

Dr. Vickery
5/6/43
Male
27 97 29

CAN PATIENT HOLD STILL FOR 30 MINUTES	YES	NO
ALLERGY TO CONTRAST MEDIA	Y	NO

REQUESTED INFORMATION IS MANDATED BY STATE REGULATORY AGENCIES

PERTINENT X-RAY	NO	NEG	POS	FINDINGS	PREV	NR	DEF	IS	FINDINGS
NUCLEAR MEDICINE	NO	NEG	POS	FINDINGS	DATE				

IS THERE A SPECIFIC QUESTION YOU WANT ANSWERED?

CLINICAL DATA IMPORTANT PLEASE COMPLETE — HISTORY PHYSICAL EXAM

LS SPINE: L4-5 Had disc ?? Healed.
Compare to prior CT. Patient Will bring CT.

ATTENTION: Dr. Roberts no Surg

The patient will bring films.

COMPUTED TOMOGRAPHY OF THE SPINE: 12-22-82

Axial computed tomography of the lumbar spine was performed with contiguous 1.5 mm. sections obtained through the L4-5 and L5-S1 disc and contiguous 5 mm. sections through the corresponding posterior elements. In addition, several contiguous 1.5 mm. sections were obtained through the L3-4 space.

There is no evidence of spinal stenosis. There is no evidence of facet arthropathy or hypertrophy. The L3 disc space is normal. There is no evidence of significant posterior protrusion.

Spondylitic ridging is noted at the L4-L5 space and an ill defined area of increased density is noted obliterating the left antero-lateral epidural fat. This is poorly marginated, but is probably distinct from the dural sac and is highly suggestive of herniated disc.

The L5-S1 level is unremarkable. There is no evidence of disc protrusion.

CONCLUSION: The findings are highly suggestive of left disc herniation at the L4-L5 level. Direct one to one comparison with the outside examination of 10-5-82, is not possible as relatively thick sections I.E: 5mm. axial sections were employed during that examination. but the findings are similar on the current examination.

However, if surgery is contemplated confirmation with myelography is advised.

CT REPORT

Michael W. Richter, M.D.
lag 22 22 82

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PHYSICIANS COPY

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SAMPLE CAT SCANS WITH CORRELATED FINDINGS

	DATE OF SCAN	A.K. DIAGNOSIS	CAT REPORT	DOCTOR REPORTING	HOSPITAL
1.	12/17/82	Bulge/herniated Disc L 4-5 L 5-S1	Negative - normal spine	Stuart L. Roberts, MD	Danbury, Ct.
	Comment:	This patient had pain for one year -- no pain after treatment.			
2.	12/22/82 Comparison scan to previous one of 10-5-82	Bulge/herniated Disc L 5-S1	Spondylitic ridging in L4-L5 space Disc Herniation L4-L5	Michael W. Richter	Danbury, Ct.
	Comment:	This was the case that started us using CAT Scan. The original CAT Scan was relatively crude using 5mm sections against 1.5 mm sections at Danbury Hospital. It is interesting to note that Dr. Richter's comment: "however, if surgery is contemplated, confirmation with myelography is advised", shows that it is possible with this evidence to consider an operation, while this patient, though antalgic when first seen, is functioning normally after routine treatment.			
3.	12/28/82	L5-S1 Disc B/H	Normal Exam	Michael W. Richter	Danbury, Ct.
	Comment:	This Disc was difficult to find. No improvement (9 visits) with many problems corrected made us search for a disc problem with severe stressing. When identified, there was normal recovery with routine treatment. It is very plain to me why CAT Scan was negative!			

SAMPLE CAT SCANS WITH CORRELATED FINDINGS (2)

	DATE OF SCAN	A.K. DIAGNOSIS	CAT REPORT	DOCTOR REPORTING	HOSPITAL
4.	1/8/83	Buldge/herniated disc L5-S1	Herniated L5-S1	Robert W. Rottenberg	Danbury, CT.
	Comment:	This person was seen at Danbury Hospital 12/9/82 - was given Percodan. We diagnosed and started treatment 12/15/82. Uneventful recovery.			
5.	2/9/83	L3-4 Disc B/H	Negative for Disc Mild facet osteo-arthritis	Robert W. Rottenberg	Danbury, CT.
	Comment:	This lady had a near fatal head-on-crash New Year's Eve '82. Expert surgical attention saved her. She had dislocated pelvis, smashed ribs and a crushed foot. Unfortunately, the CAT Scan could not verify our AK findings to help her legally. Uneventful recovery.			
6.	5/19/83	L5-S1 Disc B/H	L-4-5 central concen-tric bulge of disc with focal hernia-tion encroaching neural foramen left, L5-S1 marked loss of disc space w/proliferative degenerative changes	J. J. McSweeney	St. Joseph Stamford, CT.
	Comment:	This patient was being driven three times a week to a Chiropractor in Long Island, N.Y. by her sister who drove from N.Y. State to pick her up. What endurance - 6-7 hours of driving each trip. This doctor (?) has a reputation for sport s injuries. When we first saw her, she could not stand for more than 10 minutes at a time. The doctor had been giving her adjustments 3 times a week for 6 months (completely contra indicated)! Severe left sciatic pain was eliminated and longer recovery time was needed because of the severe neuritis and intraforaminal inflammation. Complete recovery was attained, however, of all dysfunctions including discs.			

SAMPLE CAT SCANS WITH CORRELATED FINDINGS (3)

	DATE OF SCAN	A.K. DIAGNOSIS	CAT REPORT	DOCTOR REPORTING	HOSPITAL
7.	3/24/83 comparison 6/6/83	L5 - S1 Disc Bulging/herniated left	L3-4 L4-5 Normal Degenerative disc disease w/ vacuum disc phenanamen L5-S1 Spondylitic ridging severe osteoarthritis of facett joints bilat. further compromise the left L5-S1 foramen. Also L5-S1 disc herniation 3/24/83 L5-S1 bulges minimally but symetrically. There IS NO EVIDENCE OF DISC HERNIATION No change since 3/24/83	Stuart L. Roberts	Danbury, Ct.

Comment: This one we were really proud of. Treated by two chiropractors and one orthopedist, he was a wreck, couldn't stand five minutes -- again the orthopedist did the least harm as the chiropractors were adjusting him 3x a week (terrible!)! We told him even before the CAT Scan we were thinking of the really good surgeons. We had extreme difficulty getting this disc to show -- pulled out all the stops and even used an anti-inflammatory diet. Had to use temporary orthotics to keep his sacrum in place. One day all the pain left qnd the patient even repaired his own roof. The reports by Dr. Roberts are conflicting as he said Left L5-S1 disc herniation 3/24/83 and no evidence of disc herniation but also no change on 6/6/83. He was on vacation and could not be reached for clarification when completing this paper.

CONCLUSIONS

When the NFL and the AFL came together for the first early superbowls, there was a question as to the capability of the AFL teams -- they had not played in the same league with the older NFL teams. The Kansas City Chiefs and the Oakland Raiders showed us with great clarity about their equality. They now play in the same league!

I have proven time and time again that the league we in A.K. are playing in concerning I.V. discs is equal to and superior to the league that is conventional medicine.

When the rest of the chiropractic profession becomes aware of what Applied Kinesiologists can do, if only concerning this one condition, the bulk of non-surgical discs, whether in hospital or office, will be treated by chiropractic physicians. This is, of course, as it should be.

You will note in the sample cases that some CAT Scans showed a normal or slightly bulging disc. It usually takes about a week to get a patient in to a busy hospital schedule, and by that time, they are showing healing and relief from pain. You will also note that patients with healed, verified, herniations can function relatively normally and pain free.

I urge all the members of I.C.A.K. to utilize this valuable diagnostic tool (CAT Scan) to the fullest in the routine care of the IV disc patient. Their confidence in you and your confidence in yourself will be greatly enhanced.

My thanks again to Dr. George Goodheart, whom God continues to Bless.

REFERENCES

Duffy, Dr. Daniel H., "Kinesiological Differentiation of Low Back Syndrome including the Pseudo Disc, True Disc, and the Ramrod Spine", The Digest of Chiropractic Economics, January/February 1978

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