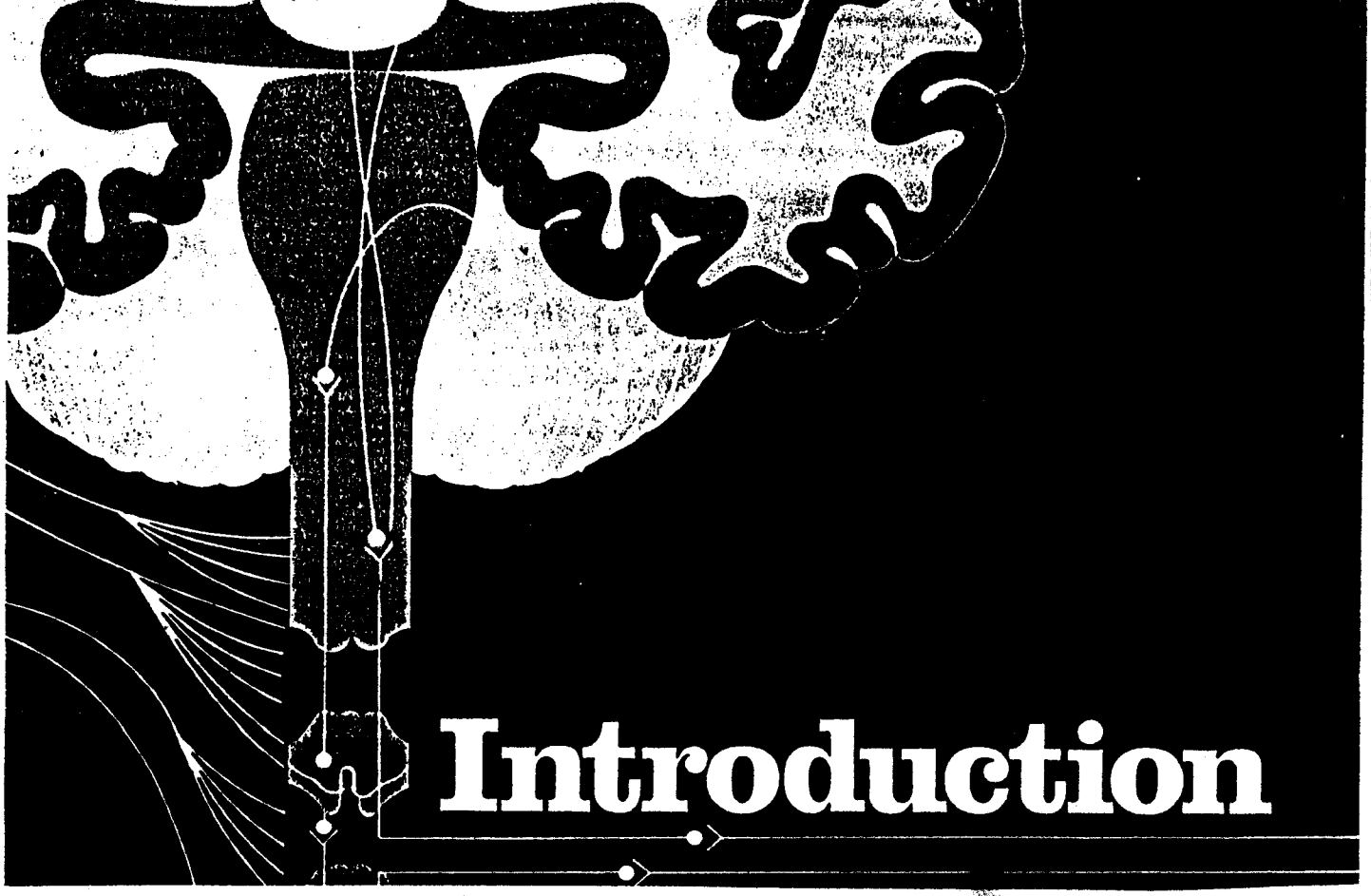


Introduction

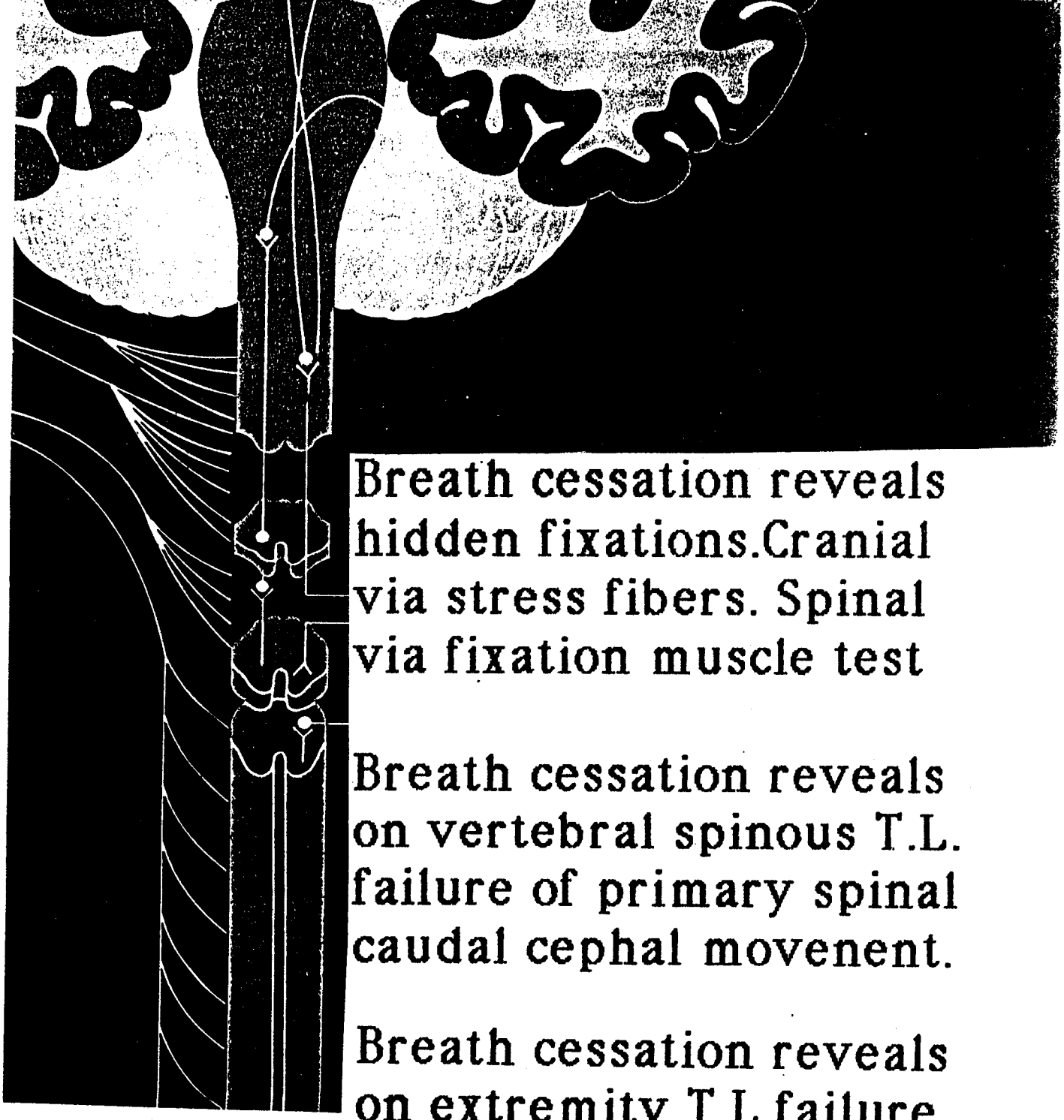
Detection of spinal fixations during respiratory cessation [10 sec,] and the new principle of C. P. M. in orthopedic practice.

Extremity lesion diagnosis during repiratory cessation[10 sec.].

Autoimmune antibody titer neutralization by glycine [w.f.] and by cystine [w.l.] and by glutamine [ch.] in glutathione.

G. J. Goodheart D.C. Research Dir.

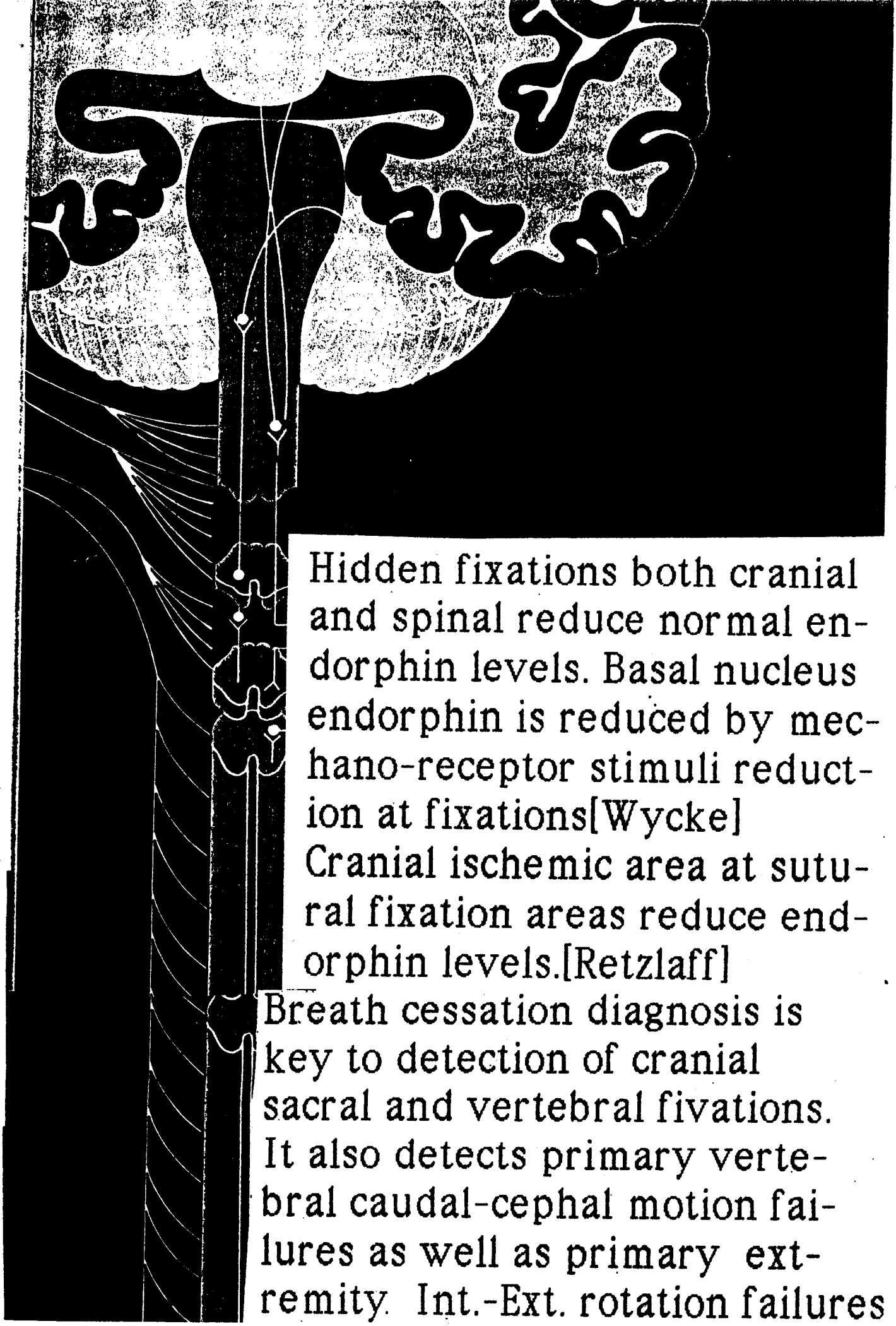
June 89.



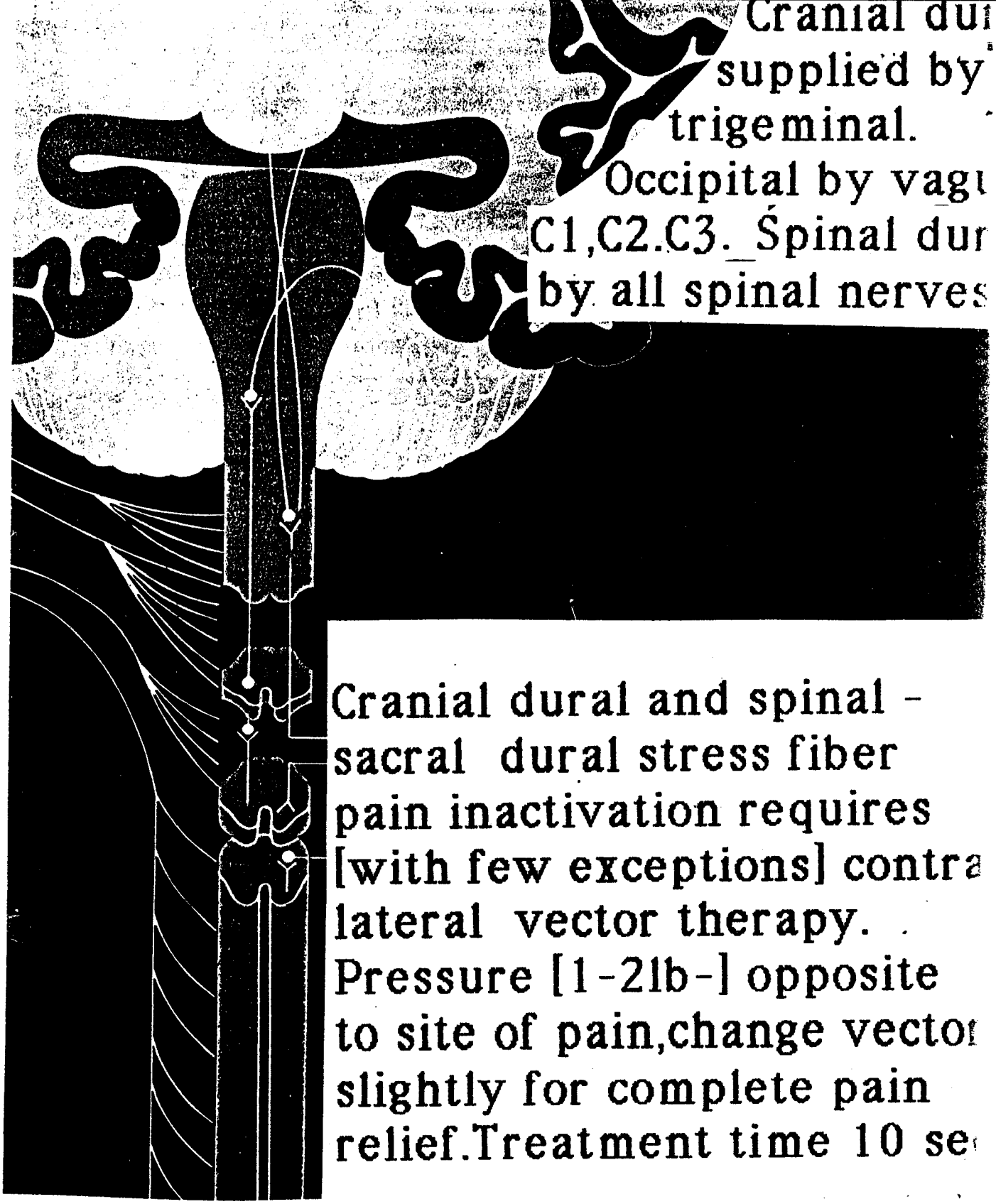
Breath cessation reveals hidden fixations.Cranial via stress fibers. Spinal via fixation muscle test

Breath cessation reveals on vertebral spinous T.L. failure of primary spinal caudal cephal movenent.

Breath cessation reveals on extremity T.L,failure of normal primary R.O.M. in external and internal rotation.



Hidden fixations both cranial and spinal reduce normal endorphin levels. Basal nucleus endorphin is reduced by mechano-receptor stimuli reduction at fixations[Wycke]
Cranial ischemic area at sutural fixation areas reduce endorphin levels.[Retzlaff]
Breath cessation diagnosis is key to detection of cranial sacral and vertebral fivations. It also detects primary vertebral caudal-cephal motion failures as well as primary extremity. Int.-Ext. rotation failures

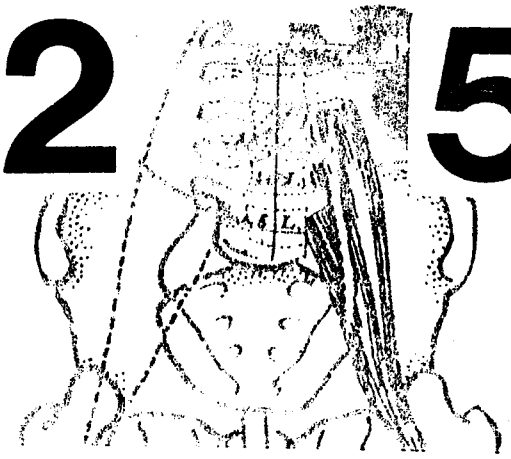


Cranial dur
supplied by
trigeminal.
Occipital by vagu
C1,C2.C3. Spinal dur
by all spinal nerves

Cranial dural and spinal - sacral dural stress fiber pain inactivation requires [with few exceptions] contra lateral vector therapy. Pressure [1-2lb-] opposite to site of pain,change vecto slightly for complete pain relief.Treatment time 10 se

APPLIED KINESIOLOGY

2 5



TO FIND THE SOLUTION TO AN UNSOLVED CLINICAL PROBLEM

RECOGNIZE AN UNSOLVED CLINICAL PROBLEM

THINK
contemplate
speculate

REVIEW SCIENTIFIC LITERATURE
historical background

ASK INTELLIGENT QUESTIONS
why? how? what? which?

FORMULATE AN HYPOTHESIS
a provisional supposition

PLAN RESEARCH PROTOCOL
strategy of the investigation including methods and controls

SEEK COLLABORATION
multidisciplinary research

APPLY FOR FUNDING
clear statement of proposal
peer review

CONDUCT INVESTIGATION
to test validity of the hypothesis

COLLECT AND ANALYSE DATA
statistical significance
serendipity

INTERPRET DATA
theory must fit the facts

DRAW VALID CONCLUSIONS
based on factual data
logic and reasoning

ANSWER ORIGINAL QUESTION
why? how? what? which?

PRESENT RESULTS AT A MEETING
for scientific discussion

PUBLISH SCIENTIFIC PAPER
peer review

APPLY NEW KNOWLEDGE
to the clinical problem

ONE GOOD RESEARCH CYCLE BEGETS ANOTHER

DETECTION OF SPINAL FIXATIONS DURING RESPIRATORY CESSATION AND THE NEW PRINCIPLE OF C.P.M. IN ORTHOPEDIC PRACTICE

In 1971 Dr. John Upledger was assisting a neuro surgeon in a spinal dural surgical procedure. "I was assisting a neurosurgeon in the removal of an extradural calcification from the posterior aspect of the dural tube in the midcervical region. Our goal was to remove the calcified plaque without incising or disrupting the integrity of the dura mater. My task was to hold the dural membrane still with two pairs of forceps while the neurosurgeon removed the plaque without cutting or damaging the underlying dural membrane. But the membrane would not hold still. I was embarrassed because I could not carry out such a simple task. The fully anesthetized patient was in a sitting position. I had no difficulty in reaching or seeing the operating field. There were no excuses.

It became apparent that the movement of the dural membrane was rhythmical at about 8 cycles per minute. This rhythmic activity was independent of the patient's breathing and cardiac rhythms. It was another physiological rhythm. It appeared to be an ebb and flow of the fluid which is contained within the dural membrane. Neither the neurosurgeon, the anesthesiologist, nor I had ever observed this phenomenon before. My curiosity was piqued. I could find no relevant information in the conventional medical or physiology literature.

The patient had been suffering from a dystrophy of the skin of both feet. He had been unable to walk because the skin on his feet continuously turned black, cracked and peeled off. It was very painful. Following the removal of the calcified plaque his condition of about eighteen months duration improved. Three months after the operation, his feet appeared to be normal. The plaque itself was the result of a systemic echinococcus infection which had produced cystic formations in the liver and brain. Medical treatment for these problems was successful. The extradural plaque was a residual effect of the infection."

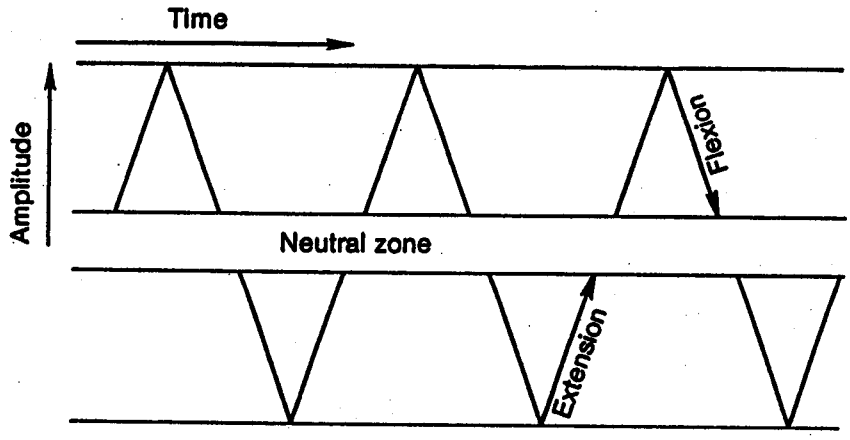
The craniosacral system is characterized by rhythmic, mobile activity which persists throughout life. This craniosacral motion occurs in man, other primates, canines, felines, and probably all or most other vertebrates. It is distinctly different from the physiological motions which are related to breathing, and different from cardiovascular activity as well. It may be the underlying mechanism of, or closely related to, the Traube-Herring phenomenon, which has been observed but not yet adequately explained. Craniosacral rhythmic motion can be palpated most readily on the head. With practice and the development of palpatory skills, however, it can be perceived anywhere on the body.

The normal rate of craniosacral rhythm in humans in between 6 and 12 cycles **per minute**. (This is not be be confused with Alpha rhythm from the brain, which is between 8 and 12 cycles **per second**.) In pathological circumstances, we have observed craniosacral rhythmic rates of less than 6 and more than 12 cycles per minute.

Under reasonably normal circumstances this rhythmic activity appears at the sacrum as a gentle rocking motion about a transverse axis located approximately one inch anterior to the second sacral segment. The rocking motion of the sacrum correlates rhymically to a broadening and narrowing of the transverse dimension of the head. As the head widens, the sacral apex moves in an anterior direction. This phase of motion is referred to as **flexion** of the craniosacral system. The counterpart of flexion is **extension.** During the extension phase, the head narrows in its transverse dimension. The sacral base moves anteriorly while the sacral apex moves posteriorly.

During the flexion phase of the craniosacral motion cycle, the whole body externally rotates and broadens. During the extension phase, the body internally rotates and seems to narrow slightly. A complete cycle of the craniosacral rhythmic motion is composed of one flexion

and one extension phase. There is a **neutral zone** or relaxation between the end of one phase and the beginning of the next phase of each cycle. The neutral zone is perceived as a slight pause which follows upon the return from the extreme range of one phase, and before the physiological forces move into the opposite phase of motion (ILLUSTRATION 1).John Upledger, D.O. Cranio-Sacral Therapy, Eastland Press.



Representation of Craniosacral Motion — Normal

Diagnose the need, supply the need and observe the result has become the byword in Applied Kinesiology. The unique success that has accompanied the breath cessation technique of diagnosis in cranial stress fiber dysfunction has now been extended to the detection and correction of spinal fixation and pelvic and sacral fixations. Extremity diagnosis also has become possible utilizing the same principle.

I am indebted to my good friend, Hans Boehnke D.C. for the original work of orthopedist and brilliant thinker Robert B. Salter M.D. Dr. Salter has pioneered in animal and human studies in the use of continuous passive motion in the rehabilitation of orthopedic problems. Salter believes that the new concept of continuous passive motion "C.P.M." may well have a significant application in the immediate post operative care of humans with a variety of joint disorders and injuries.

It is my belief that the segments of the spine are in continuous active motion when undisturbed by fixation. This motion seems to be the ten to fourteen times per minute that Viola Fryman and others spoke of. The body attempts to cover any fixations by respiratory costal sterno vertebral motion. The actual presence of fixations have in the past been determined by specialized muscle testing. Naturally this was done during normal respiration. We continue to use this method. We now add during the immediate testing breath cessation. The incidence of undetected fixations in difficult problem cases has been very high and breath cessation is necessary for their detection and for the evidence of their response to fixation correction. Corrections are done in the usual manner. Wheat Germ Oil or Octocosanols are routinely prescribed. Clinical response has been excellent in terms of pain reduction and increase in lost function of viscera as well as joint and muscle and tendino-ligamentous situations.

The evidence is very strong from four different sources, Wycke, Greenman, Dvorak, Dvorak, Manual Medicine, Therapy 1988, Thieme, New York, that significant stimulation of the mechano receptors causes presynaptic inhibition of the nociceptor afferent impulses at the level of the posterior horn of the spinal cord. In four scientific studies, mainly in Holland, encephalins are believed to be involved in this inhibitory process.

It is therefore reasonable to assume the lack of motion in the B.C. type fixation would induce the lack of normal endorphin production from normal vertebral motion which must be augmented in this type of hidden fixation by the costo sternal and costo vertebral motion associated with respiratory rates rather than primary craniosacral vertebral motion. The steady restoration of function and normal R.O.M. ranges following correction of the B.C. fixation speaks for itself. The spontaneous therapy localization of practically all the B

and E points during breath cessation for the diagnosis of cranial tender points, and the temporary disappearance of the cranial tender points during simulated gait patterns also speak volumes for the involvement of muscle meridian activity especially the bladder meridian beginning at the lateral nasal area. Certainly the wide variety of cranial respiratory patterns [insp, exp, nasal versus oral respiration, half a breath inspiration or expiration] would lead one to see the potental dural involvements. The dura as you know has trigeminal innervation with the exception of the occipital area which has vagal and C1 C2 and C3. Release of endorphins via the limbic and caudal reticular activating system broadens these concepts. The release of the previously mentioned spinal andorphins by mechano receptor inhibition of nociceptor afferents takes place in the slowest drainage area of vertebral spinal cord circulation according to Barry Wycke the eminent British neurologist. Normal mechano receptor activation suppress pain at type one, type two and possibly at type three mechano receptor sites. Lack of normal motion would obviously reduce normal mechano receptor activation with normal motion not to mention the activity associated with abnormal but necessary motion. Check for B.C. fixation types in all phases, prone, sitting, and standing in difficult cases. Usually prone alone suffices.

In Martindale's original 1955 monograph he said "by carefully checking the effects of the complete elimination of lesioning within the various group spinal lesions that I find to be rather constant, some interesting observations have been made and others are in the process of being made.

Beginning with group spinal lesion C1-C3, I shall enumerate some of these observations, but while making these observations, it was found to be equally important to observe what disease conditions do not tend to exist when no lesioning of particular group spinal lesions has apparently ever existed.

Group spinal lesion C1-C3 appears to play a dominant role in the production of lesions of the cranial bones. The complete elimination of this group spinal lesion is usually followed by the release of cranial bone lesioning throughout the cranium. In fact, the completeness of the reduction of this group spinal lesion may usually be evaluated by the degree of mobility that has been restored to the cranial bones.

I have never observed a patient who was subject to head colds, where this lesion was absent. Its correction, in the presence of a head cold, is usually followed by prompt improvement.

The head is normally the best drained structure of the body. Drainage of lymph and blood, is assisted by gravity at all times, except when one lies down.

Most everyone is aware of the way in which the nasal passages tend to be occluded when a person with a severe head cold lies down. Whichever side he lies on, gravitational drainage usually permits breathing through the upper nostril.

In the presence of group lesion C1-C3, the associated muscular contractures of the cervical spine, nullify, to some extent, this gravitational advantage, thus probably hindering nature in her attempts to overcome or prevent head infections of any kind.

I have never observed an arthritic patient who did not have lesioning of group lesion C1-C3, and it has usually been very severe. Arthritic patients usually improve promptly with the elimination of this lesion, a most difficult one to eliminate in arthritic patients.

Individuals who do not have lesioning at C1-C3 are usually rarely tired, and their energy is apt to be abundant.

Group spinal lesion C4-T2 presents some most interesting observations. One of the most important of these, is the part that this group lesion evidently plays in the development and maintenance of hypertensive disease. I have yet to see a case of hypertension come into my office, since I became aware of this relationship, approximately four years, who has not had most severe lesioning in this group.

Complete elimination of group lesion C4-T2 in hypertensive patients, has, in my experience, been invariably followed by a return of blood pressure to normal or very nearly so. By normal I mean that the blood pressure comes down to 130/85 or better. It is very common to see a blood pressure of 190/100 come down to 120/70 even in old age.

Cases in which only modification of lesion C4-T2 could be obtained, because of arthritis in the vertebrae or because the patient did not come in long enough for me to completely eliminate the lesioning, have invariably shown some improvement in blood pressure levels that seems to correspond to the degree of modification in the lesioning of the group.

I shall doubtless encounter cases of hypertensive disease that do not respond to this treatment, but such cases will be the result of less common causes than my series.

Group spinal lesion C4-T2, seems to have a very marked tendency to cause immobility of the ribs, and it may play a leading part in the development of rib lesions.

A tendency to abdominal ptosis has been observed in group lesion C4-T2. This is greatly benefited by the elimination of such lesioning.

In fact, the marked effect that this lesion appears to have in producing abdominal ptosis, leads me to think that this spinal region may give origin to nerves that have to do with fascial tone.

Group spinal lesion T3-T7, appears to play a vital part in the production of coronary artery disease. The effect upon the pain of coronary insufficiency, when this lesion is eliminated, is so striking, that I believe no observer could fail to be impressed.

I have observed no case of coronary thrombosis that has not been severely lesioned in group T3-T7. The finding is so constant, that I am firmly convinced that in the absence of this lesion, no coronary disease unassociated with general vascular disease, would exist.

Group spinal lesions involving both T3-T7, and T8-T12, appear to have a variety of influences upon the abdominal viscera, but due to the overlapping nerve supply, an accurate evaluation of the effects of each group is most difficult.

Studies related to the effects produced by group spinal lesion L1-L5 are continuing, but no clear-cut conclusions have been drawn.

The apparent tendency for group spinal lesion T8-T12 to trigger group spinal lesion L1-L5 and the sacro-iliac joint lesioning that is usually present in lumbar lesioning, must be considered, when one attempts to evaluate the effects of lesioning of the lumbar spine and sacro-iliac joints."

CRANIAL SACRAL SPINAL PRIMARY RYTHMIC MECHANISM

The dura mater and its attachments determine the functional capability of the cranial sacral respiratory mechanism. The dura has two layers in the cranium. The outer (endosteal layer) attaches to all of the ridges of the inner cranial vault, serves as the periosteal covering of the inner surface of the cranial bones, and lines the foramen of the base of the skull and the opposing surfaces of the cranial sutures It also acts as a cuff around the blood vessels that supply the cranial bones, forms the sheaths around the cranial nerves and attaches to the rim of the foramen magnum. The inner (meningeal layer) adheres tightly to the outer layer and forms the falx cerebri, tentorium cerebelli, falx cerebelli, diaphragma sellae, and the venous sinuses. The dura then extends downward also attaching to the foramen magnum via adherence to the periosteal layer. Once he cranial dura exits the foramen magnum, it becomes the spinal dura and is again attached to the odontoid process and the posterior ring of the atlas. It forms a loose connection with the posterior longitudinal ligament and again firmly attaches to the second sacral tubercle, as well as the first, second and third sacral foramen. Beyond the second sacral foramen, the dura becomes closely invested with the connective tissue of the filum terminale. The two then continue downward and attach to the dorsum of the first coccygeal vertebral segment."

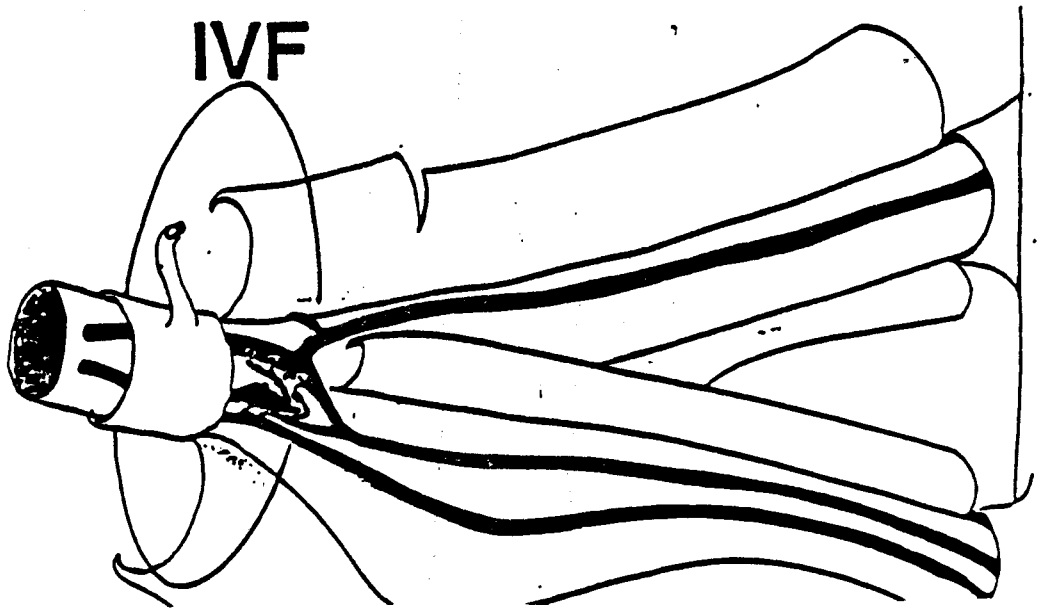
"The dural arrangement and attachments protect and facilitate the smooth pressure-stat pumping of the cerebral spinal fluid by the brain and spinal cord. This is accomplished by a rhythmic contraction and relaxation of the neuroglia (glial cells), particularly the astrocytes. The normal rhythm is thought to be 10 to 14 times per minute. This delicately controlled action is termed, "The Cranial Sacral Spinal Primary Rythmic Mechanism."

The dural attachments and the articular motion of the cranium, atlas 2 and 3 cervicals and sacrum are of paramount importance to the brain

and spinal cords ability to move and replenish the cerebral spinal fluid (CSF) in and around the central nervous system (CNS). This insures proper neuron firing.

Disturbance in the articular function of the cranial bones, atlas and 2 or 3 cervical or the sacrum result in dural torque. There must be compensation for this malfunction or a neurologic deficit results. At any spinal level, torquing of the dura may result in; paravertebral muscle changes with motor unit changes, CSF pooling and efferent nerve impulse bombardment to the associated visceral or somatic structures. Compensation is achieved by a return of afferent impulses to the spine which ultimately results in alterations of the multifidus and rotates muscles causing compensatory vertebral rotation and <u>hopefully</u> resultant relief of CSF pooling. Failure here results in pain, disease and pathology."

DURAL CUFF SCHEMATIC



"The dura covers the anterior and the posterior nerve roots, becoming the dural cuff at the intervertebral foramen (IVF), where it adheres to the periosteum of the vertebrae. It then becomes continuous with the epineurium. Each nerve root is supplied with arteries, veins, and lymphatics which extend from the spinal cord. Just medial to the (IVF), arachnold granulations drain cerebral spinal fluid from the nerve roots. In this area the cerebral spinal fluid is reabsorbed into the lymphatics. This is similar to the way that CSF is absorbed in the venous sinuses of the cranium. Dural torque of the nerve root interferes with the CSF flow and results in nerve root congestion and swelling." *

*David Denton D.C.

The filium terminale attaches to the posterior of the coccyx.

The pia mater is a delicate connective tissue membrane and it carries the rich network of blood vessels throughout both brain and cord. It attaches to the nerve tissues where the minute blood vessels penetrate the piaglial membrane. A concentration of fibers alongside the spinal pia mater forms the denticulate ligament. The pia mater attaches to the coccyx by the central ligament of the spinal cord."

A frequent corolary pattern exists between the piriformis right or left and the breath cessation cranial tender point technique. Testing the piriformis is usually negative on routine testing and the right piriformis participates well in the P.L.U.S. pattern. Slow or rapid contraction or slow or rapid stretching produces marked weakness which responds well to strain counterstrain techniques. The piriformis is the exception to posterior muscle extension and expiration techniques. The piriformis requires in the prone position marked flexion to eliminate the pain on palpation at the belly of the involved piriformis and requires a spread finger unidirectional pressure by both spread fingers to eliminate palpatory pain. Spread finger unidirectional pressure is in line with muscles origin and insertion and may be towards or away from origin or insertion. Treatment greatly aids dural torque reduction and associated pain patterns in multiple Dvorak and Dvorak spinal areas, especially cervical and femoral areas. A left or right lateral sway is the postural indicator.

The pressure state model of choroid plexus C.S.F. production being higher than anachroid villi absorption seems a logical explanation of D.S.F. pressure and the widening movements of the head with inspiration and sacral anterior - (forward tip movement) and narrowing of the head on expiration with sacral tip moving posterior, backward.

This occurs 10-14 cycles per minute. Visualize a football or motorcycle helmet being the skull and visualize the helmet wearer's head being the brain within the skull supended by dura - temtorium and falx etc. Tender points found supine may indicate excessive backward movement towards the table by normal gravity pulling on the "straps" of the interior of the helmet suspensory arrangement. The non presence of pain on the posterior of the head in supine and sudden appearance of pain at posterior when prone would validate this concept. The disappearance of anterior and posterior pain following treatment would further the concept of dural membrane tension balancing. These "straps" are "stress fibers" of the dura matter, horizontal, vertical; transverse, circular and spinal groups.

The periosteal cranial dura is firmly attached to and penetrates the sutures and forms the outer cranial periostium. The cranial dura is also firmly attached to the foramen magnum entire circumference and the upper anterior rim of the atlas. The cranial dural membrane is firmly attached to the foramen magnum and becomes the lining or endostium of the vertebral canal. The brain's dural membrane is firmly attached to the foramen magnum and is firmly attached to the posterior of the axis and the third cervical vertebrae. Dentate ligaments loosely secure the cord until the firm cord attachment at the anterior of the second sacral segment below is the filum terminale non neural attachment to the posterior portion of the first coccygeal segment. Spinal length variations, dural torque factors are all elements to be considered. Cranial tender points are greatly though temporarily abolished by gait configuration in supine position (left arm right leg forward - test for pain at tender points, - right arm, left leg forward - test for tender points. Usually left leg forward with right arm temporarily abolishes all tender points. Removal of dural spinal torque must be accomplished along with removal of cranial dural tension and cervical mechano receptor balancing. The cranial dura and the brain dura though duplicate "skull caps" are firmly attached to each other - the vertebral canal is lined with endosteal

(periostium) dura which is duplicated by the spinal dura but in contrast the veretebral canal dura and the spinal cord dura are only firmly attached at the posterior aspect of the second and third cervical segment and then below by only the loose denate ligaments until the anterior portion of the second sacral segment and then finally by the non neural filum terminale attachment at the posterior of the first coccygeal segment. This closed tubal attachment relationship can then be changed as you would imagine by spine length and spinal curve changes producing dural tension and or torque.

The nutrient of choice for the breath cessation cranial dural tension pattern seems to be either the omega three or the omega six fatty acids. Black currant seed oil (omega six) seems to be the most frequent requirement, T.I.D. It temporarily abolishes palpatory pain response and negates breath cessation induced weakness of stress muscle indicator

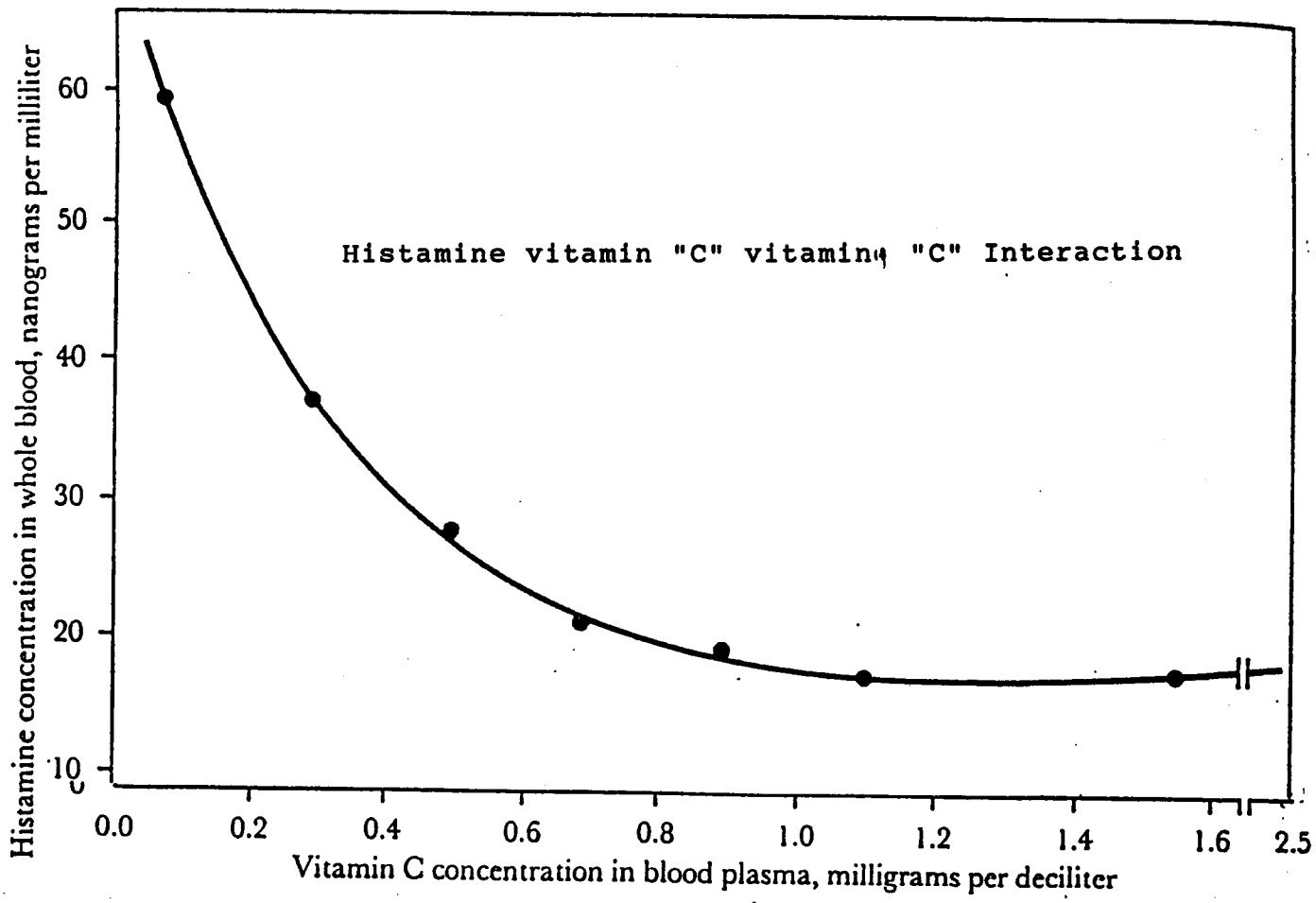
SIMPLIFIED FOOD ALLERGY AND AUTO IMMUNE TEST

"Betaine is another relatively neglected factor in foods. Chemically, it is trimethylglycine, another probably essential derivative of a nonessential amino acid, glycine. It combines with hydrochloric acid to form betaine-hydrochloride which releases the hydrochloric acid on solution in water, so it has been used for years as a means of administering hydrochloric acid in capsule form. The betaine was considered inert. Curiously, however, betaine has been found to destroy tetanus toxin on contact, (2) and its administration to a patient who is fighting any infective process seems to break down his resistance to temporarily aggravate the symptoms of the infective disease. It is probable this is the effect of betaine in destroying antibodies that were hindering the development of the infective process. The wiping out of antibodies, maybe is a good thing in many cases, for a new production takes place probably more promptly after this than otherwise. But, the continuous use of betaine could be possibly an unwise procedure. In allergic patients there is a pathological sensitiveness to certain proteins and the effect of betaine in wiping out the antibodies here would be certainly of great benefit. Some believe pernicious anemia and other diseases such as myasthenia gravis are cases where a pathological antibody is destroying vital cells, and betaine here has a theoretical promise of benefits. It is now an established fact that the normal control or organ and tissue growth is maintained in part by the action of antibodies to our own cells and proteins. (3) So it is clear how atrophy can result from an excessive formation of any of these specific antibodies, whether muscle cell or red blood cell.

It is now known that a dosage of excess vitamin B is followed by liver degeneration and cirrohosis. These reactions are really methionine deficiency, aggravated by the increased demand made by the vitamin B. Methionine is one of the materials required by vitamin B to do its

work, and its place can be taken by betaine which is normally a part of the natural vitamin B complex from wheat germ. Methionine, however, probably does not have the effect of inactivating antibodies. Again, one of the derivatives of betaine, glutathione, is more potent than betaine, apparently, in inactivating antibodies, for it is physiologically used for this purpose in the egg. The amount of glutathione in the egg of a hen determines how big the full grown chicken will be, for the number of cell divisions will be the greater before hatching if more glutathione is present to hold down antibody production for a longer time." Royal Lee, D.D.S.

Test strong muscles against histidine amino acid powder on tongue. If weakness of strong muscle occurs-test against source of betaine (Betafood S.P. or beet top source) as well as glutathione. Measure C level of mouth lingual ascorbic acid disappearance time. Measure C level of urine with Ames "Stix" reagent strip for vitamin C. Any level below 40mg percent is low and requires temporary specific food restriction and increase in betaine or glutathione support rather than "C" level increases.



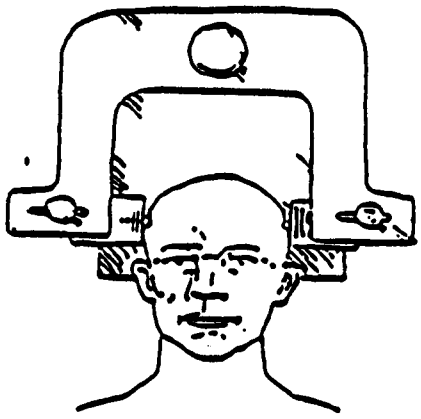
FURTHER DURAL CRANIAL AND DURAL SPINAL ANATOMY

The cranial dura consists of two layers of inner meningeal and outer periosteal and are continuous to and including the foramen magnum. The cranial part is divided into falx cerebri, tentorium cerebelli and the falx cerebelli...the fourth and smallest is the diaphragma sella.

The spinal dura forms a loose sheath around the spinal cord and corresponds to the cranial meningeal layer of the dura. The periosteal dura of the cranium is now represented by the periosteum of the vertebra which lines the vertebral canal. The veins of the spinal dura correspond to the sinuses of the cranial dura. The spinal dura is fixed to the foramen magnum and the second and third cervical vertebrae. The periosteal cranial dura attaches to the superior rim of the atlas and explains why the atlas vertebra responds to movement of a patient's heel (os calcis). The spinal dura attaches to the posterior longitudinal ligament by fibrous slips. The cavity of the durala tube ends at the second sacral vertebra.

HOLOGRAM MODEL OF CRANIAL MOTION

"Motion of the skull bones has been objectively measured. The first study was made by Frymann. When she applied transducers to a subject's head, she found a rhythmic autonomous motion supporting Sutherland's original observation of 10-14 cycles per minute.

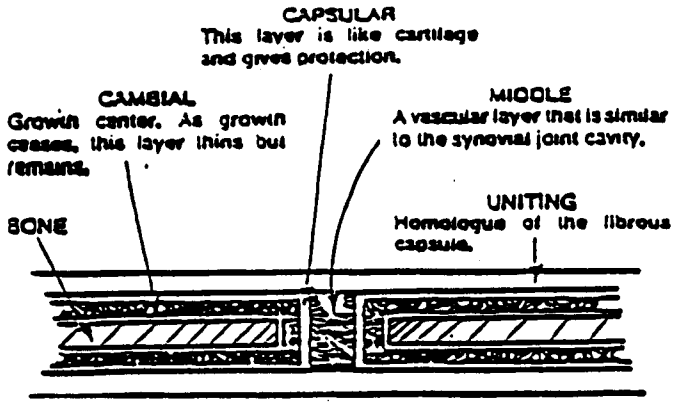


Study of motion with fixed transducers. After Frymann.

Further motion studies have been done to eliminate effects of intervening tissue between the transducer probe and the skull. Michael and Retzlaff attached force transducers to a screw eye placed in the parietal bone of anesthetized monkeys. A cyclic cranial bone displacement of 5-7 cycles per minute was observed that could not be attributed to either respiration of heart rate. With normal function there is predictable movement of the cranial bones. It continues throughout life, cycling 10-14 times per minute, and is called "primary respiratory mechanism." The motion is separate from the heart or breathing rate; however, cranial motion is enhanced by thoracic respiration. Although this influence is always present, relaxed breathing and primary respiration do not always parallel.

The history of a suture is that of a joint designed for motion. Pritchard et al. found five distinct layers of cells and fibers

between the edges of the adjoining bones in human specimens. They concluded that '...history of the sutures suggests that it has two main functions, viz. that it is a site of active bone growth and that it is at the same time a firm bond of union between the neighboring bones, which nevertheless allows a little movement.' The first of five layers making up the suture is bone.



Suture histology, after Pritchard et al.

The cambial layer is the growth center that thins as growth ceases, and the capsular layer is light cartilage that provides protection. The middle aspect in the suture is a vascular layer similar to the synovial joint cavity. Finally, covering the suture is the uniting layer that is the homologue of the fibrous capsule.

UPledger et al. studied specimens taken from living adult skulls at the time of neurosurgical craniotomies. Along with connective tissue, the sutures were shown to have the presence of viable myelinated and unmyelinated nerve fibers and nerve receptor endings.

Biohology is the application of hologram principles in nature. These principles have been demonstrated mathematically in vision, sound transmission, and in the somatosensory, somatomotor, and gustatory systems.

E.H.Land, the Polaroid developer, demonstrated that one area in the visual receptive field can create illusions in the perception of entirely different areas in the same receptive field, giving rise to color effects. This and other visual illusions are explained by the hologram.

The interaction of the nervous system at two locations appears to function like a hologram. Von Bekesy studied the perception of 'pitch' on the skin to relate to the function of hearing.

There is much evidence to indicate that the holographic brain model is accurate. Dolgoff gives an excellent overview of support for the model. To keep the model in perspective he states, "This analysis does not mean to imply that all brain and neural function can be reduced to holographic process, but that certain processes are most accurately describable by analogy to specific, well-understood, holographic-related processes." An example of processing by two methods is in the auditory system, which functions both hologramically and non-hologramically.

The position of cranial bones in relation to each other can be observed on accurately positioned x-rays. Cranial nerve V angles over the petrous apex of the temporal bone. Gardner measured the height of the petrous apex by x-ray and found that trigeminal neuralgia occurred three times more often on the side of the high petrous apex than on the low side.

Cranial distortion may change the dental occlusion by mandibular movement change or by disrelation of the cranial bones. Malocclusion is a common complaint following an auto accident with whiplash dynamics to the neck and head. Many doctors fail to appreciate this because the patient often does not discuss it with the orthopedically-inclined physician. If the patient discusses the matter with a dentist who is not knowledgeable about cranial motion,

the teeth may be equilibrated to match the distorted skull; this locks in the cranial faults and makes correction difficult or impossible without further dental attention. Baker found a 0.0276" increase in the distance between the second molars following cranial manipulation.

It is important to understand all of the aspects that can be involved in dysfunction of the stomatognathic system. A patient may come to a chiropractor for a whiplash-type injury to the cervical spine. This condition often relates with cranial faults that may in turn cause malocclusion. Correcting the cranial faults may correct the malocclusion; however, if the condition has been present for a considerable time the teeth may have changed position by the natural process of remodelling, thus locking in the cranial faults. In this case it may be necessary to consult a dentist for a bite plane, and perhaps eventual equilibration of the teet

Ninety percent of the venous blood leaves the cranium by way of the jugular foramen, which is really at the junction of two bones and is surrounded by dura. We developed the concept that there are two types of cranial faults. One is a subluxation which needs the inspiration assist or expiration assist or half a breath in or half a breath out or the universal cranial fault or the nasal sphenoid or the pineal mandibular spread--the technics we have discussed in the past which have been based more or less on a sort of subluxation concept; the other is a fixation complex.

We have discussed this before, but in OSTEOPATHIC MEDICINE for July 1978, John Upledger, D.O. and Ernest Retzlaff, PH.D., both of the Department of Biomechanics of the College of Osteopathic Medicine of Michigan State University, and John Vredugol, Assistant Professor of the College of Human Ecology of Michigan State, state that:
"Traditionally anatomists have taught that the suture articulations of adult humans are fused and hence immoveable. Recent histological work done by us (meaning Upledger, Retzlaff and Vredugol) would contradict

this view with specimens taken from living adult skulls at the time of neurosurgical craniotomy. Hence those tissues studied resemble more closely the invivo circumstances."

By use modified staining technics, the authors have been able to demonstrate the presence of viable myelinated and unmyelinated nerve fibers, nerve receptor endings and potentially functioning vascular network and collagen elastic fiber complexes within the adult human cranial suture.

CRANIAL SUBLUXATIONS AND CRANIAL FIXATIONS

On difficult Melzack-Wall situations we suggested that you use therapy localization while the patient did breath holding. This was a useful procedure for finding a difficult Melzack-Wall situation if we were attempting to relieve pain and it didn't reveal itself in the usual fashion.

Previously we also talked about the E.I.D. pattern, putting the "Eyes into the Distortion," and therapy localizing under an E.I.D. basis.

In the Cranial Bowl" that Sutherland wrote, he cites the evidence that spinal fluid is basically an electrolyte. If you will think that the falx cerebri is basically a membrane dividing the brain into the left and right halves, and if you remember what we talked about when we spoke of Davis & Rawls' concept that the body is pisitive on the right and negative on the left on the anterior, the reverse being true on the posterior--if you can postulate that concept--then assume that breathing is the generator for the battery that is the brain. This will explain why some people can "leave their lights on all night" so to speak, and still have enough energy to "start their car in the morn ing," something like those Sears television commercials; and other people have to keep everything going just to stay alive.

We have found that the electrolyte factor of the spinal fluid is an element in the maintenance and the production of a better response to treatment, so we have approached it in this particular fashion.

To continue, they say the suture is now known to possess some neural structures necessary for nerve reflex activity. Sensory input into the nervous system with motor activity can take place if it is apparent that a distortion of the functional relationships between the sutural osseous boundaries may produce abnormal neurogenic activity as well as abnormal sutural activity.

In view of the aforementioned findings, it seems obvious that restoration of sutural mobility is desirable. Several mechanisms underlie sutural dysfunction. One which has been almost completely overlooked is hypertonus or contracture of the temporalis muscle, and other cranial and facial muscles.

Here again, develop the concept that there are fixations and subluxations; and again develop a concept that the cranial bones with their sutures have a resemblance to an electrical situation of condensor, or as it is sometimes known, a capacitor.

Condensors are classified according to the materials used to insulate them. There are air conductors that have an air space between aluminum plates, as in radio sets; there are glass connectors that store charges of live voltage; there are mica condensors that have plates made of mica to insulate the conductors, which are used for high voltage; and there are electrolytic condensors that have two metal conductors in a liquid or moisture retaining material.

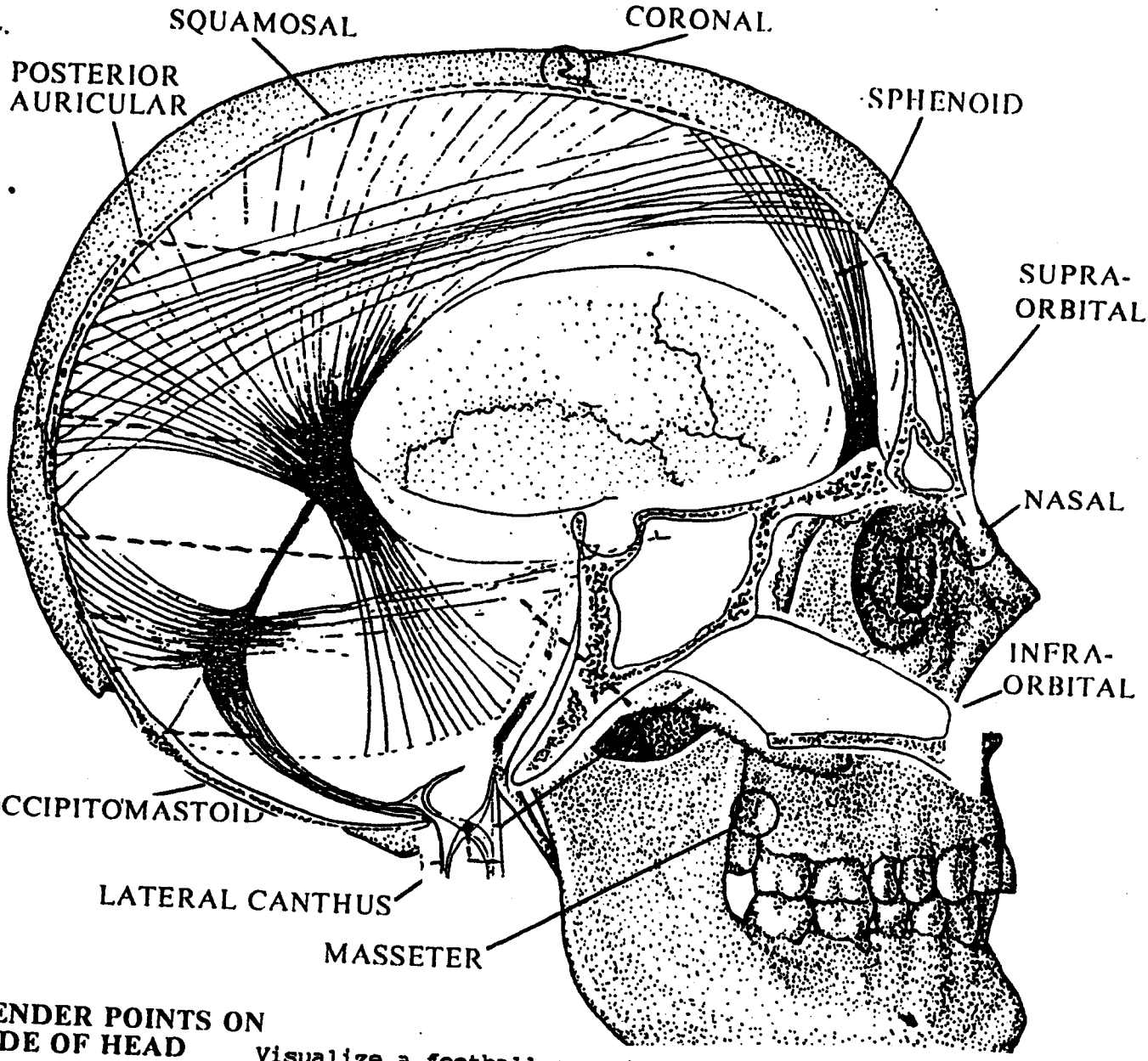
Under an electrical charge the atoms and liquids form ions and help form an electrical charge.

If you take the concept of a capacitor and relate it to too close an approximation or too far a separation of the cranial bones, and then transfer that to a spinal fluid concept of the spinal fluid being basically an electrolyte, and then transfer that to the concept of the right side being positive and the left side being negative, you can readily see that the sutures themselves may be approximated or may be separated by muscular activity, and thus alter A.M.C. holographic feedback activity. The concept being that the skull is like a capacitor or a condensor, the cerebral spinal fluid is an electrolyte or the "battery fluid" so to speak, and the breathing is basically the generator. In some people, if you have a "loose fan belt partially disconnecting the generator from the battery" eventually you use more current than the generator puts back in, and "your lights start to dim." The response to treatment, therefore, is minimal. Many people "jump start" their primary cranial motion (10-14 per minute) by costol voluntary respiration (16-20 per minute). When there is sufficient power, as there is many times in children, the response to treatment is sometimes very quick and amazing.

FURTHER DURAL CRANIAL AND DURAL SPINAL ANATOMY

The cranial dura consists of two layers of inner meningeal and outer periosteal and are continuous to and including the foramen magnum. The cranial part is divided into falx cerebri, tentorium cerebelli and the falx cerebelli...the fourth and smallest is the diaphragma sella.

The spinal dura forms a loose sheath around the spinal cord and corresponds to the cranial meningeal layer of the dura. The periosteal dura of the cranium is now represented by the periosteum of the vertebra which lines the vertebral canal. The veins of the spinal dura correspond to the sinuses of the cranial dura. The spinal dura is fixed to the foramen magnum and the second and third cervical vertebrae. The periosteal cranial dura attaches to the superior rim of the atlas and explains why the atlas vertebra responds to movement of a patient's heel (os calcis). The spinal dura attaches to the posterior longitudinal ligament by fibrous slips. The cavity of the durala tube ends at the second sacral vertebra.



TENDER POINTS ON SIDE OF HEAD Visualize a football or motorcycle helmet being the skull and visualize the helmet wearer's head being the brain within the skull suspended by dura - temtorium and falx etc. Tender points found supine may indicate excessive backward movement towards the table by normal gravity pulling on the "straps" of the interior of the helmet suspensory arrangement. The non presence of pain on the posterior of the head in supine and sudden appearance of pain at posterior when prone would validate this concept. The disappearance of anterior and posterior pain following treatment would further the concept of dural membrane tension balancing.

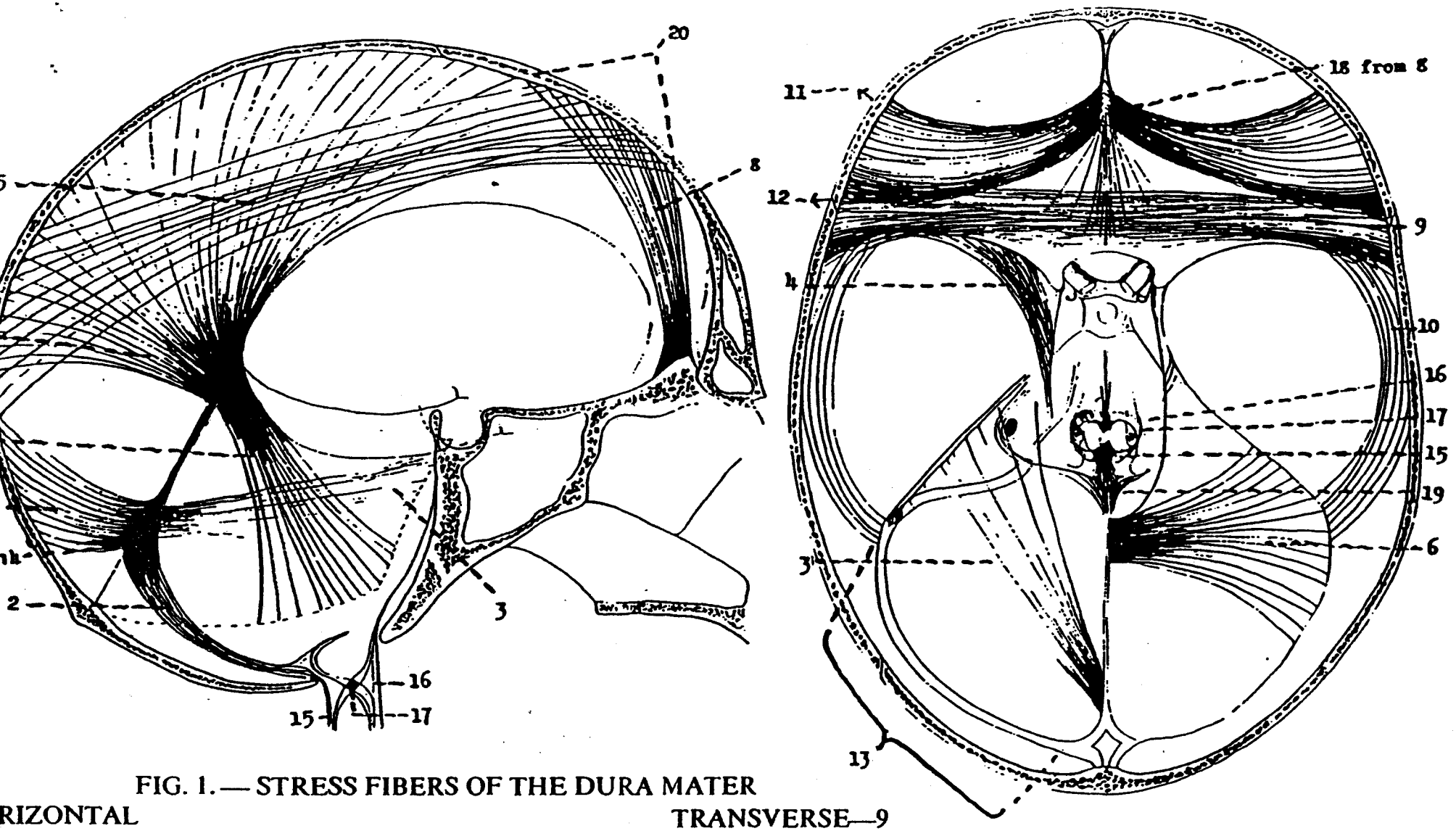


FIG. 1.— STRESS FIBERS OF THE DURA MATER

RIZONTAL
1. Falx cerebri inferior
2. Falx cerebelli—tripod—19 } —from torcular mass
3. Tentorium
4. Sphenoidal
5. Falx cerebri superior

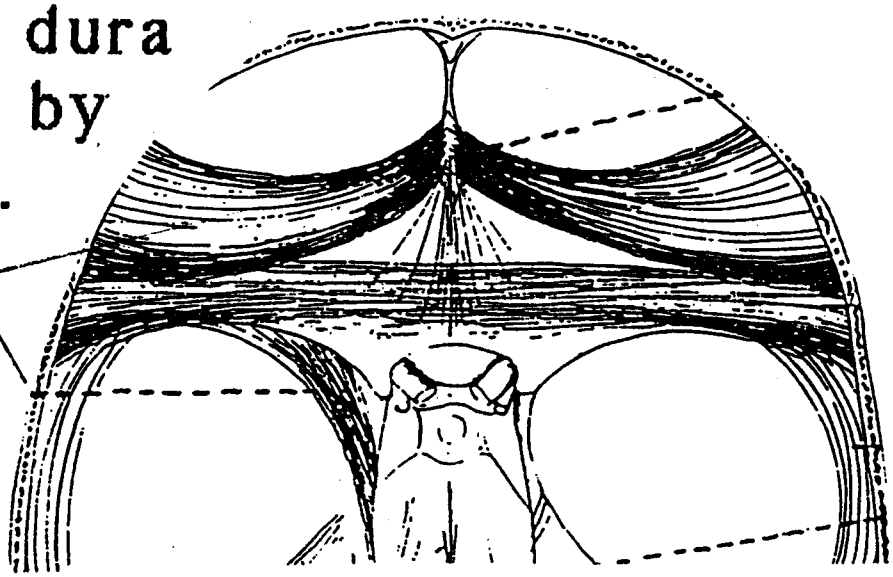
RTICAL
6. Tentorium
7. Falx cerebri posterior
8. Falx cerebri anterior—crista galli tripod—18

TRANSVERSE—9

CIRCULAR
10. Squamosal
11. Vault { anterior / middle / posterior } from metopic area—20
12.
13.
14. Posterior fossa or cerebellar—from torcular mass

SPINAL
15. Posterior—tripod { 16, 17 } lateral fibers intersect

Cranial dura supplied by trigeminal.



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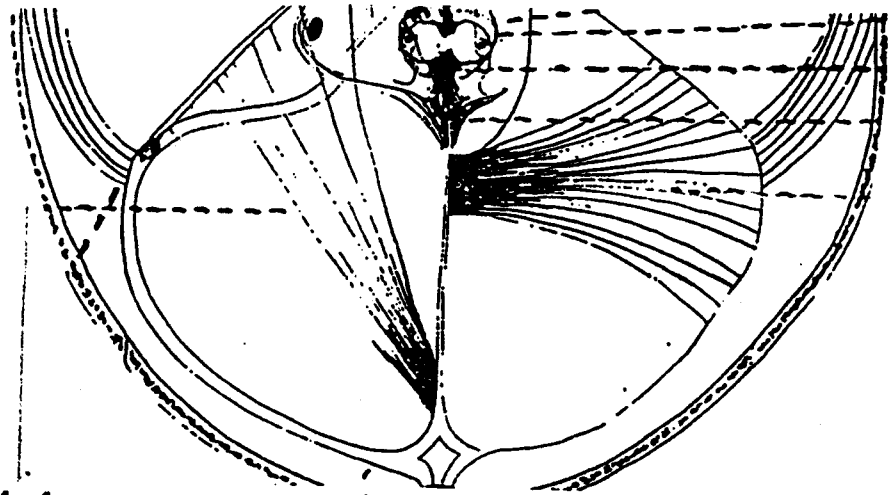
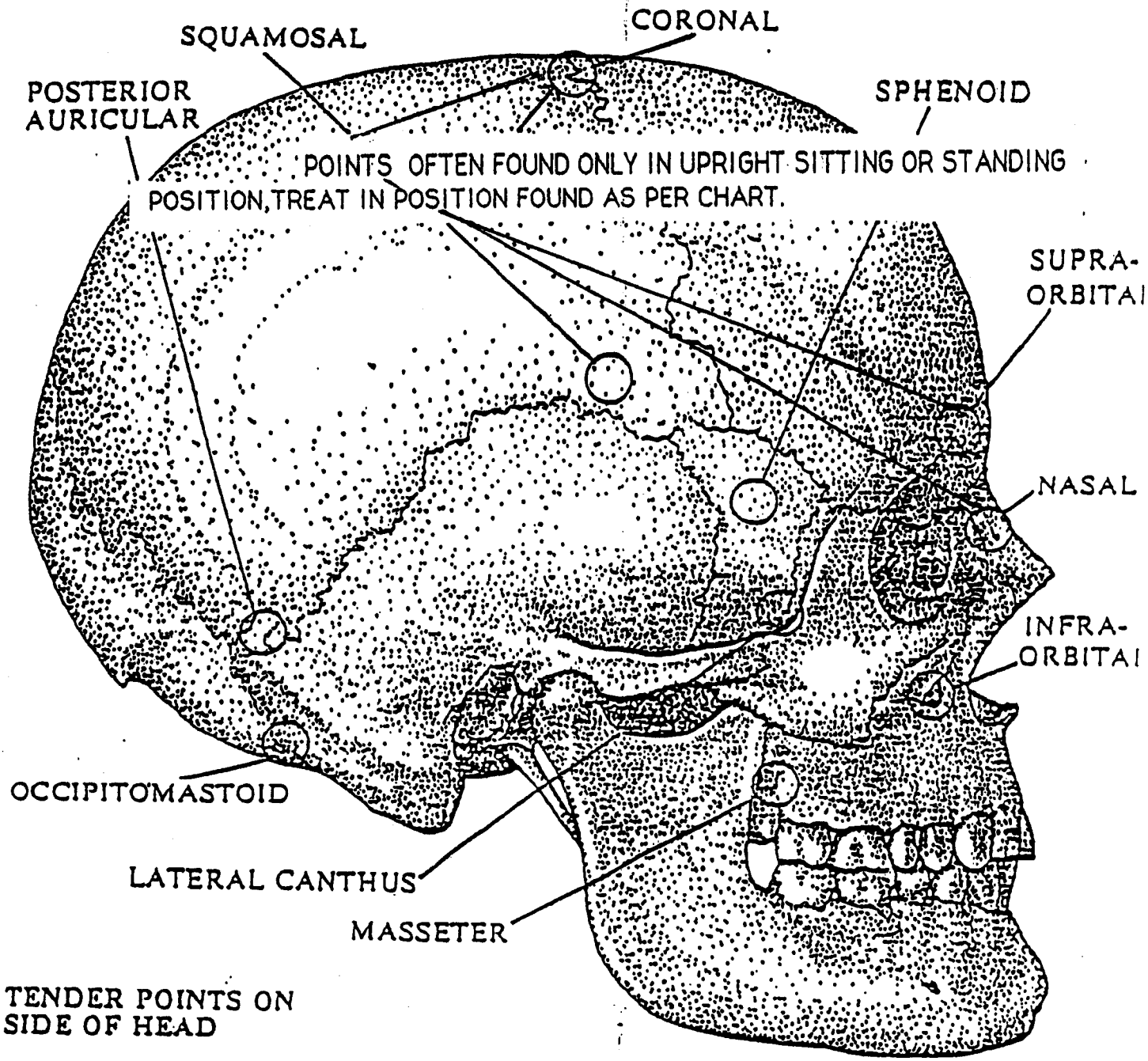


Occipital by vagus,C1,C2.C3.

Spinal dura by all spinal nerves

Cranial Breath Cessation Technic



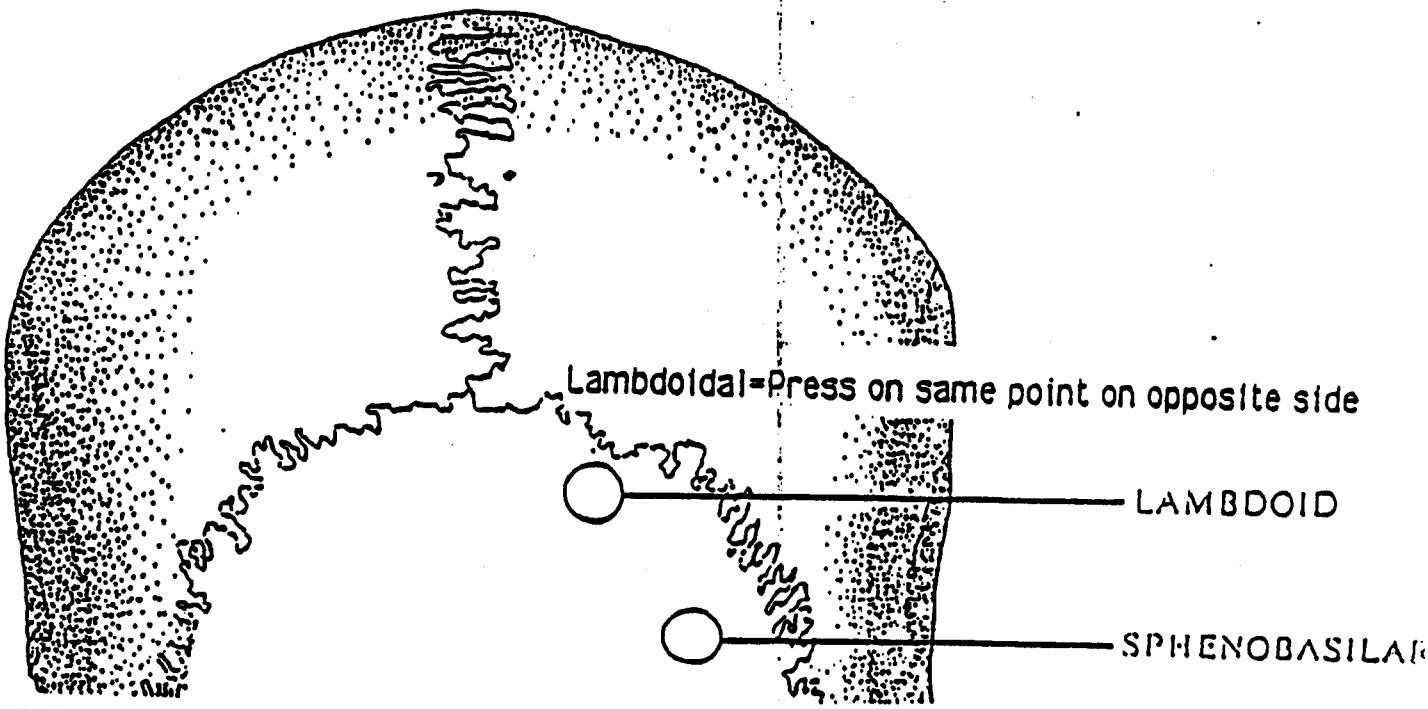
Coronal-Opposite area pressure

Sphenoid-Opposite sphenoid pressure

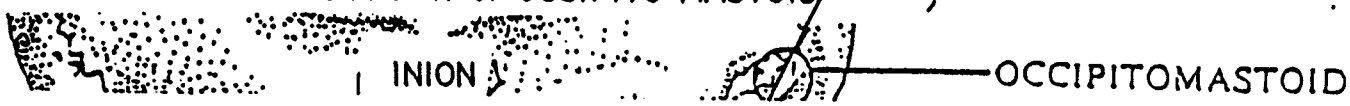
Squamosal-Pull up to vertex

Posterior Auricular-Bend skull sidewayn

Cranial Breath Cessation Technic



OCCIPITO -LAMBDOIDAL MAY REQUIRE MASTOID TORQUE-COUNTER TORQUEOF OPPOSITE MASTOIDS, ASSAY FOR DIRECTION OF TORQUE COUNTER TORQUE BY PAIN RESPONSE OF TENDER POINT OF OCCIPITO-MASTOID



INION , LEFT AND RIGHT OF E. O. P. USE STRAIN AND COUNTER STRAIN OF JONES TECH. OCCIPUT AND FIRST THREE CERVICALS REQUIRE FLEXION AND IINSPIRATION(30 SEC.) TECH DESPITE THE FACT OF POSTERIOR LOCATION

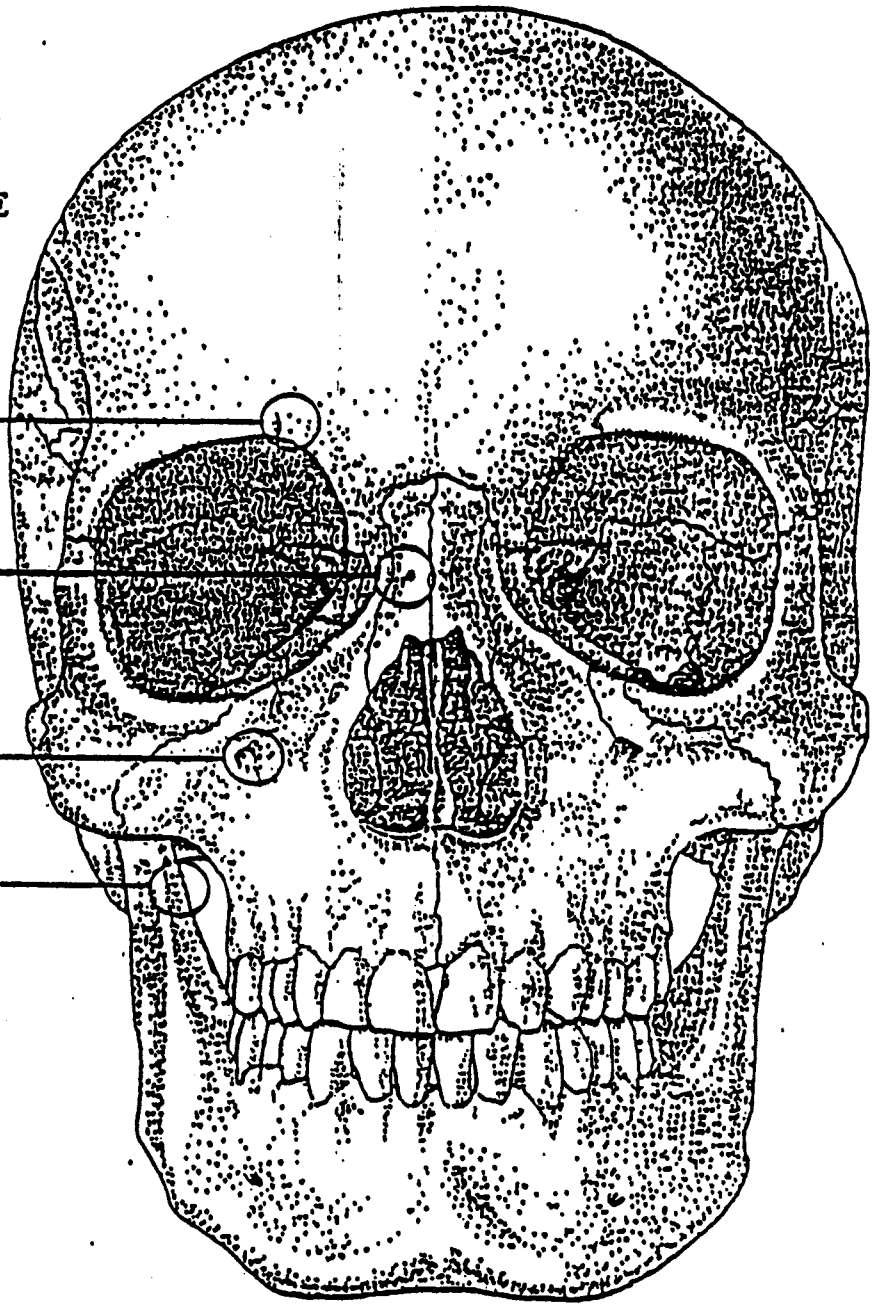
TENDER POINTS ON BACK OF HEAD

ANTERIOR TENDER POINTS USUALLY FOUND PATIENT SUPINE ,POSTERIOR TENDER POINTS USUALLY FOUND.PATIENT PRONE ,EXCEPTIONS OCCUR.

Spheno-Basilar=On right clockwise frontal counter clock wise occipital ,reverse for left

Occipito-Mastoid- Occiput caudal and frontal direction/frontal caudal and towads occiput

TENDER POINTS ON FACE



Infra-Orbital-Pressure posterior-medial

ANTERIOR TENDER POINTS USUALLY FOUND PATIENT SUPINE ,POSTERIOR TENDER POINTS USUALLY FOUND PATIENT PRONE ,EXCEPTIONS OCCUR.

Holographic Breath Cessation

CRANIAL TECHNIC

1. Test sartorious--gracillis, should be strong in clear, if not correct by 51.V.F. factors.

2. Test sartorious--gracillis against breath cessation, should be strong regardless of phase of breath cessation, (insp. or exp.).

3. Test sartorious--gracillis against breath cessation while two handed therapy localization of any and all cranial sutures. Weakness may occur at any or all cranial sutures while breath cessation of 10 to 15 seconds takes place. This tests primary cranial respiratory motion (10-14 per minute).

4. Treat cranial tender points by Jones strain and counter strain technic as per chart diagram. These are cranial muscle indicators determined by trial and error.

5. Following tender point release by appropriate technic, retest against breath cessation and suture therapy localization, Should now exhibit strong sartorious gracillis.

6. Now therapy localize lambdoidal suture with one hand and therapy localize ipsi-later sacro-iliac joint with the opposite hand. If as usual weakness appears on ipsi-lateral hamstring tap lambdoidal suture and sacro-iliac joint not necessarialy in unision. Tap both sides of both areas at one Hertz or faster for 30 to 60 seconds. Re-evaluate.

7. Now test B and E points and treat by tap technic. Over-all clinical response is greatly aided by general sutural tapping at one Hertz or higher.

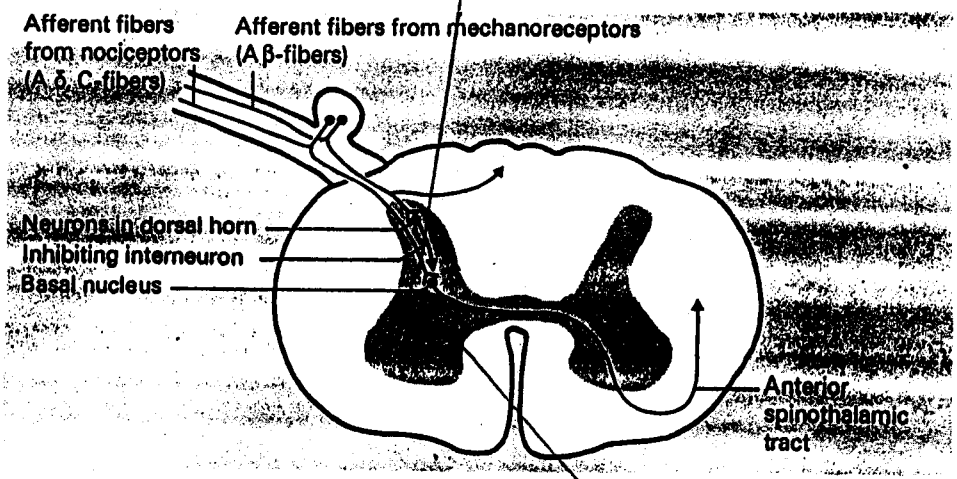
8. In difficult cases tender point eradication is aided by usual flexion inspiration for anterior points and extension expiration for posterior points, SUPRA ORBITAL often requires the (flexion) technic; SPHENOBASILAR also often requres this (extension) technic.

9. Use only indicated muscles for this new but very rewarding approach. Visual hearing and mental acuity have benefitted from this simple approach.

10. B and E points spontaneously respond to this approach, if not treated as usual.

Hidden fixations both cranial and spinal reduce normal endorphin levels.

The evidence is very strong from four different sources, Wycke, Greenman, Dvorak, Dvorak, Manual Medicine, Therapy 1988, Thieme, New York, that significant stimulation of the mechano receptors causes presynaptic inhibition of the nociceptor afferent impulses at the level of the posterior horn of the spinal cord. In four scientific studies, mainly in Holland, encephalins are believed to be involved in this inhibitory process.



. Fibers of nociceptors and mechanoreceptors in the region of the posterior horns (after *Wyke*, 1979a).

Basal nucleus endorphin is reduced by mechano-receptor stimuli reduction at fixation[Wycke]Cranial ischemic areas reduce endorphin levels.[Retzlaff]

Structures Involved

In a fixation complex a minimum of two structures will be involved, and they will have restricted movement between them. Usually three vertebrae are involved in a fixation; however, there may be two or up to five (and possibly even more).

Muscle Weakness

There are specific bilateral muscular weaknesses commonly associated with vertebral fixations (discussed on the next page).

Therapy Localization

When therapy localizing over a vertebral fixation, a previously strong indicator muscle will not weaken unless there is an attempt to introduce motion into the fixation complex. This is done by therapy localizing over the suspected fixation complex while the patient actively moves the spine in that area. In the presence of a fixation, an indicator muscle will weaken. Therapy localization over a fixation will strengthen the bilateral muscle weakness associated with the fixation.

Challenge

There will usually be no reaction to a single-point challenge. Challenge is accomplished by challenging two vertebrae at the same time, usually by pressing in opposite directions on the spinous or transverse processes.

Static X-Ray

Generally no misalignment between fixed spinal structures is observed on x-ray. The mechanism at fault is a lack of motion between contiguous vertebrae, rather than a misalignment causing apparent encroachment on the radix of the nerve.

Motion X-Ray

There will usually be hypokinesis of the spinal fixation complex.

Correction

A fixation requires a two-handed contact or some other method of stabilizing one of the structures while the other is manipulated, because a single-handed contact just moves the entire fixed complex rather than unlocking the mechanism. Occasionally a fixation complex is unlocked with a single-handed contact; however, such results are due more to luck than calculated correction.

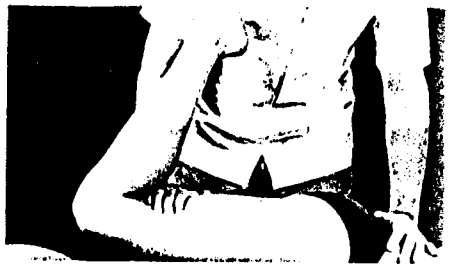
Bilateral Muscle Weakness of Fixations

When a fixation complex is present in the spinal column, it has a specific bilateral muscle weakness. The bilateral weakness observed by Goodheart[25] reveals many spinal fixations which would otherwise be overlooked.

Fixation levels associated with muscles which test weak bilaterally are as follows:

1. Occipital fixation — bilateral psoas muscles.
2. Upper cervical fixation — bilateral gluteus maximus muscles.
3. Lower cervical fixation — bilateral popliteus muscles.
4. Cervicodorsal fixation — bilateral deltoid muscles (rarely, bilateral serratus anticus muscles).
5. Thoracic fixations — bilateral teres major muscles.
6. Dorsolumbar junction fixation — lower trapezius muscles.
7. Lumbar fixation — neck extensors test weak when tested together bilaterally.
8. Sacral fixation — neck extensors test weak bilaterally when each group is tested individually.
9. Sacroiliac fixation — neck extensors test weak on one side only, and no other factor is found for the dysfunction.

3—32. Psoas



3—30. Gluteus maximus

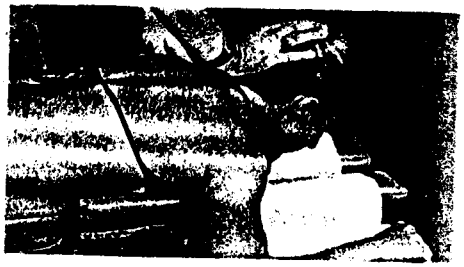
3—31. Popliteus

3—27. Middle deltoids

3—28. Teres major

3—29. Lower trapezius

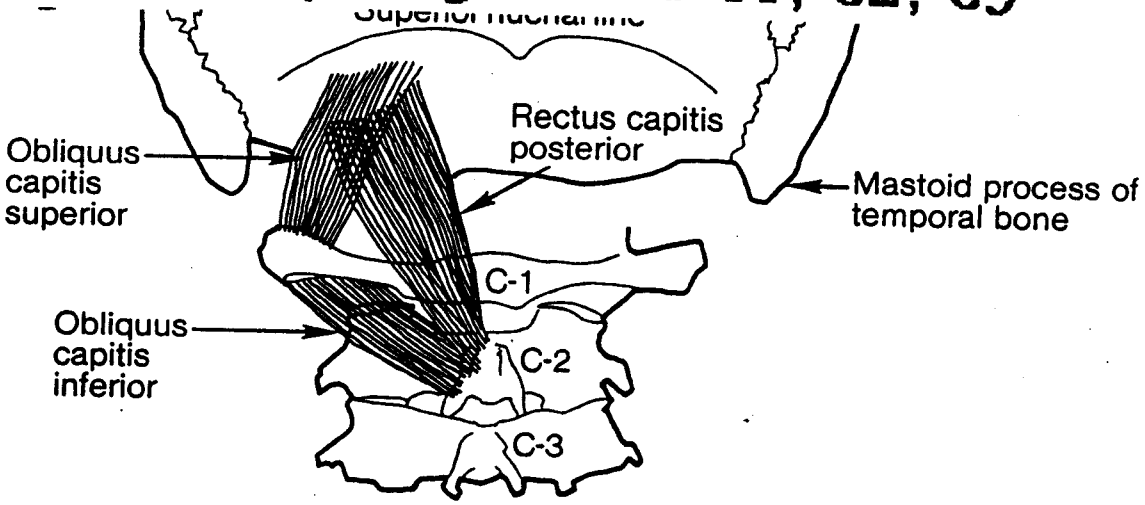
3—26. Neck extensors — right



3—25. Neck extensors — group

Hidden fixations both cranial and spinal reduce normal endorphin levels. Basal nucleus endorphin is reduced by mechano-receptor stimuli reduction at fixations[Wycke]
Cranial ischemic area at sutural fixation areas reduce endorphin levels.[Retzlaff]

Cranial dura supplied by trigeminal, occipital region by vagus and C1, C2, C3

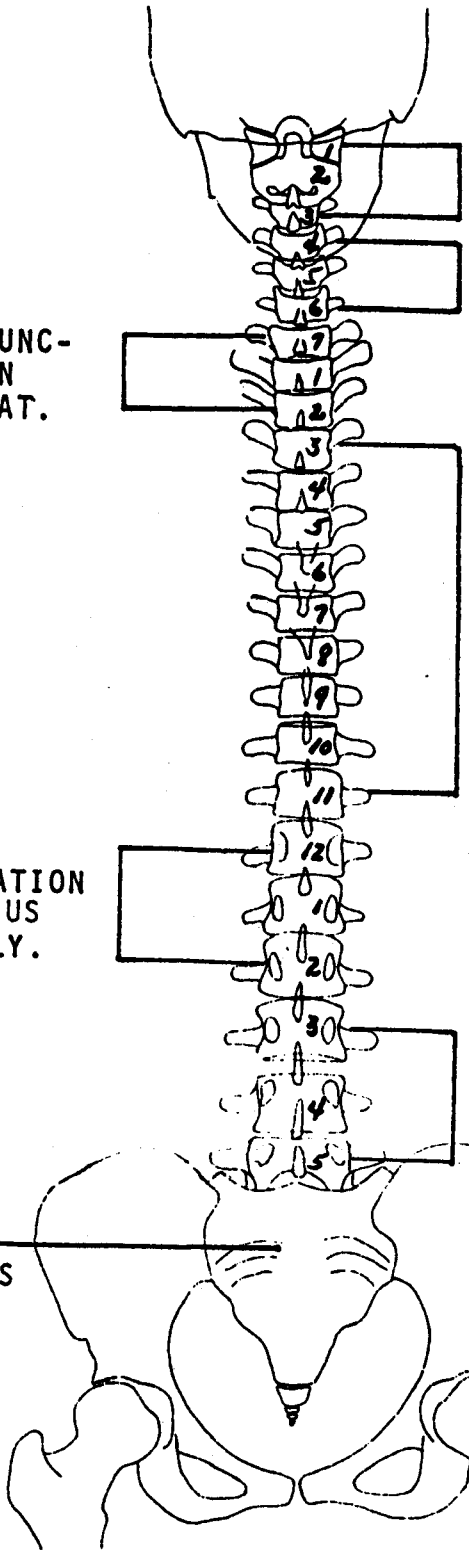


FIXATION CHART

CHECK DURING RANDOM BREATH CESSATION

IN DIFFICULT CASES CHECK IN SITTING MODE

AND CHECK IN STANDING MODE



UPPER CERVICALS IN FIXATION WHEN GLUT. MAX. WEAK BILAT.

LOWER CERVICALS IN FIXATION WHEN POPLITEUS WEAK BILAT.

CERVICAL-DORSAL JUNCTION FIXATION WHEN DELTOIDS WEAK BILAT.

DORSALS IN FIXATION WHEN TER MAJOR IS WEAK BILATERALLY.

DORSAL-LUMBAR FIXATION WHEN LOWER TRAPEZIUS IS WEAK BILATERALLY.

ANY FIVE LUMBARS

LUMBARS IN FIXATION WHEN NEC EXTENSORS ARE WEAK BILATERAL

SACRAL FIXATION WHEN NECK EXTENSORS ARE WEAK BOTH LEFT AND RIGHT.

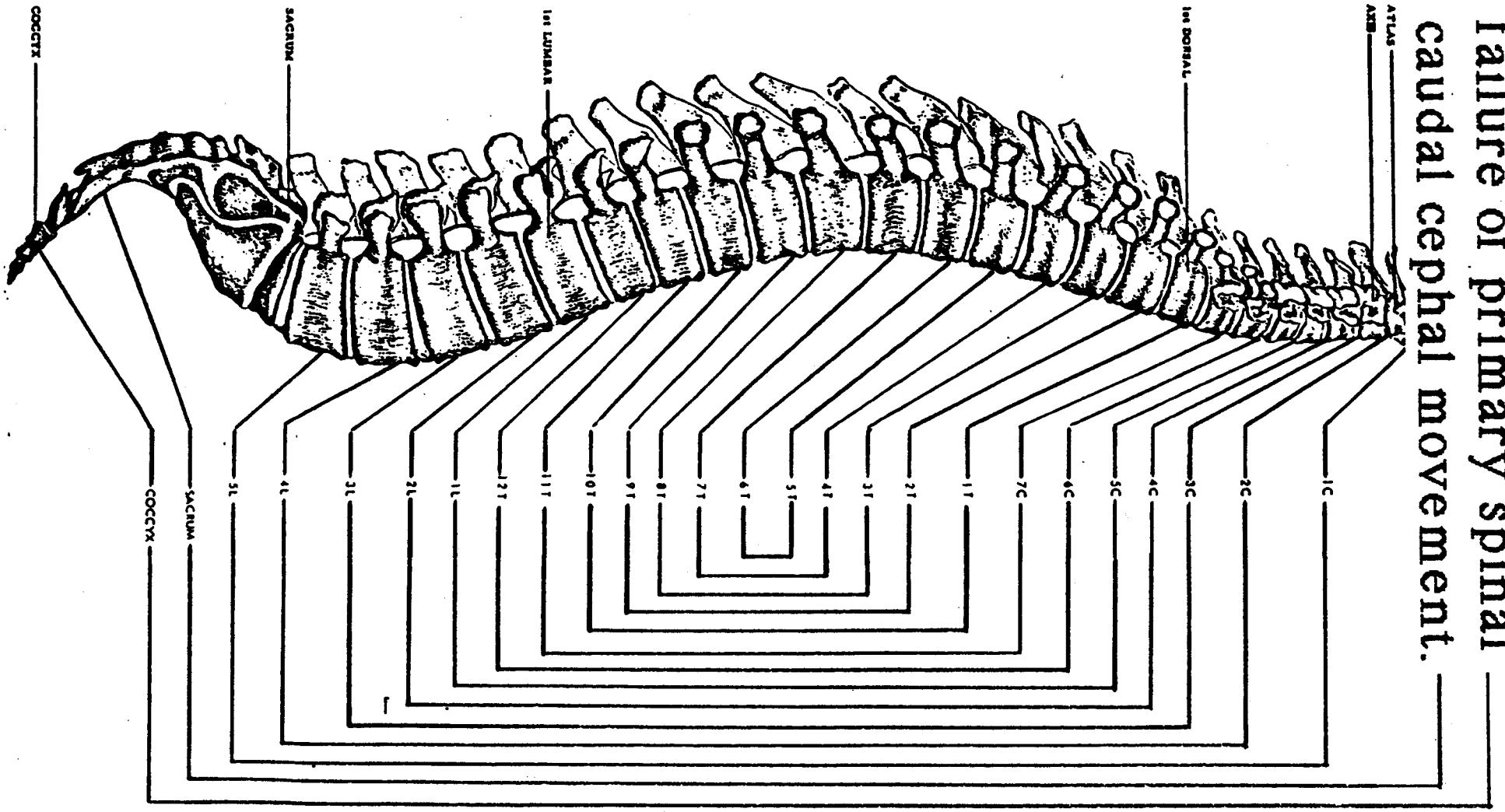
ILIAC FIXATION POSSIB WHEN ONE NECK EXTENSO IS WEAK. IF THERE IS RESPIRATORY PATTERN, THE PIRIFORMIS WILL B WEAK ON SIDE OF ILIAC FIXATION.

VERTEBRAL RESPIRATION MOVEMENT

THERAPY LOCALIZE DURING BREATH CESSATION
IF MUSCLE WEAKENS
TREAT BY RESPIRATORY
MOVEMENT ENHANCEMENT AS
PER DIAGRAM

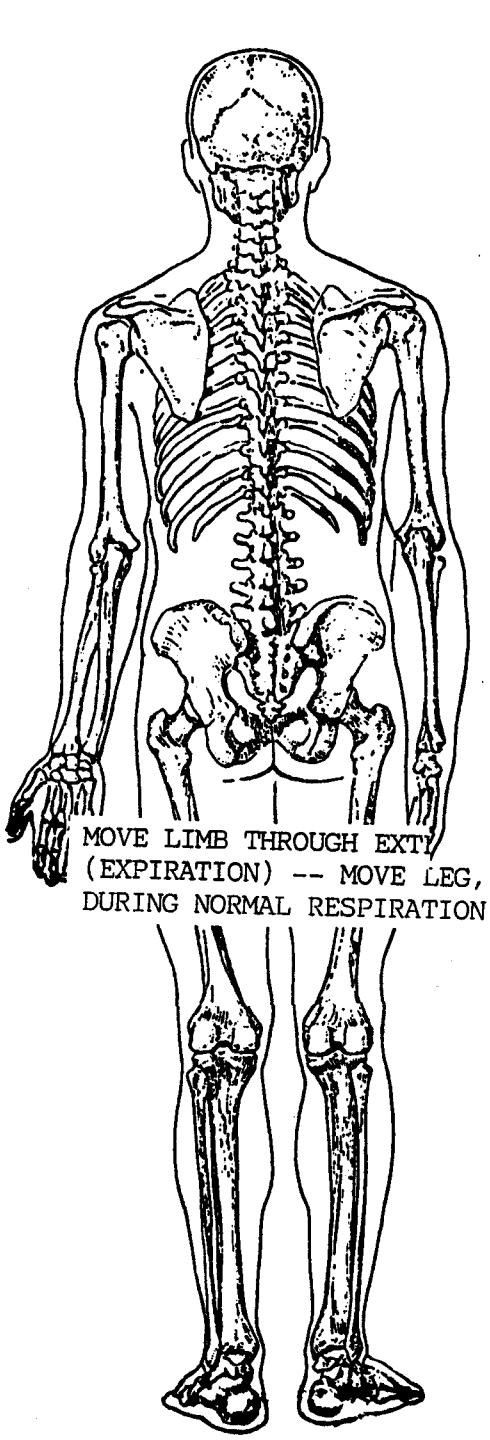
INSPIRATION ← CAUDAL

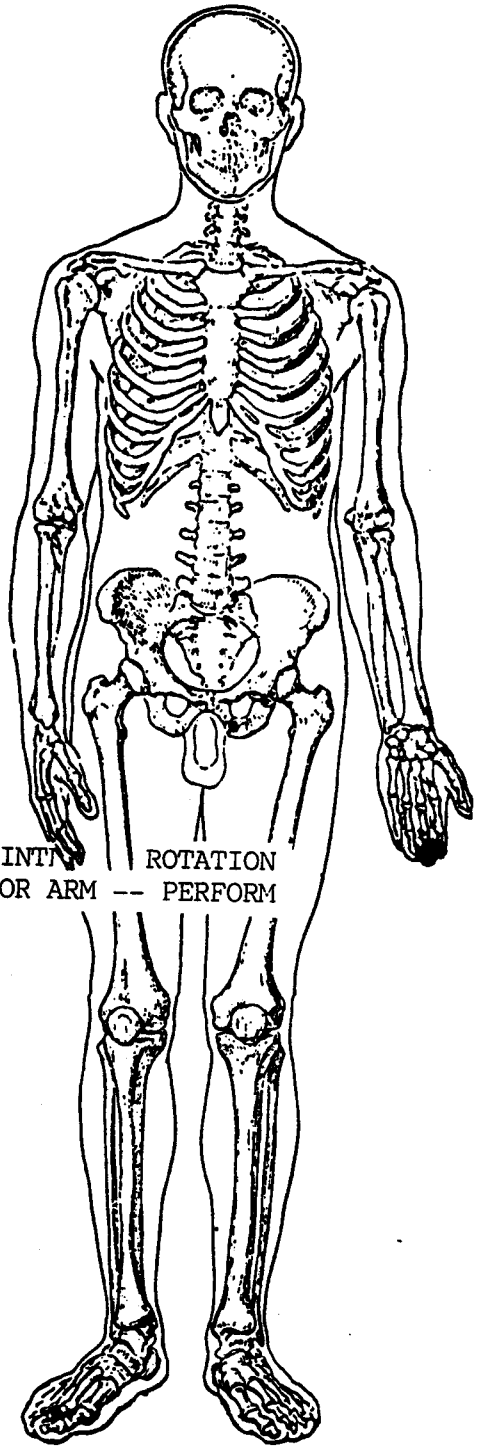
EXPIRATION → CEPHAL



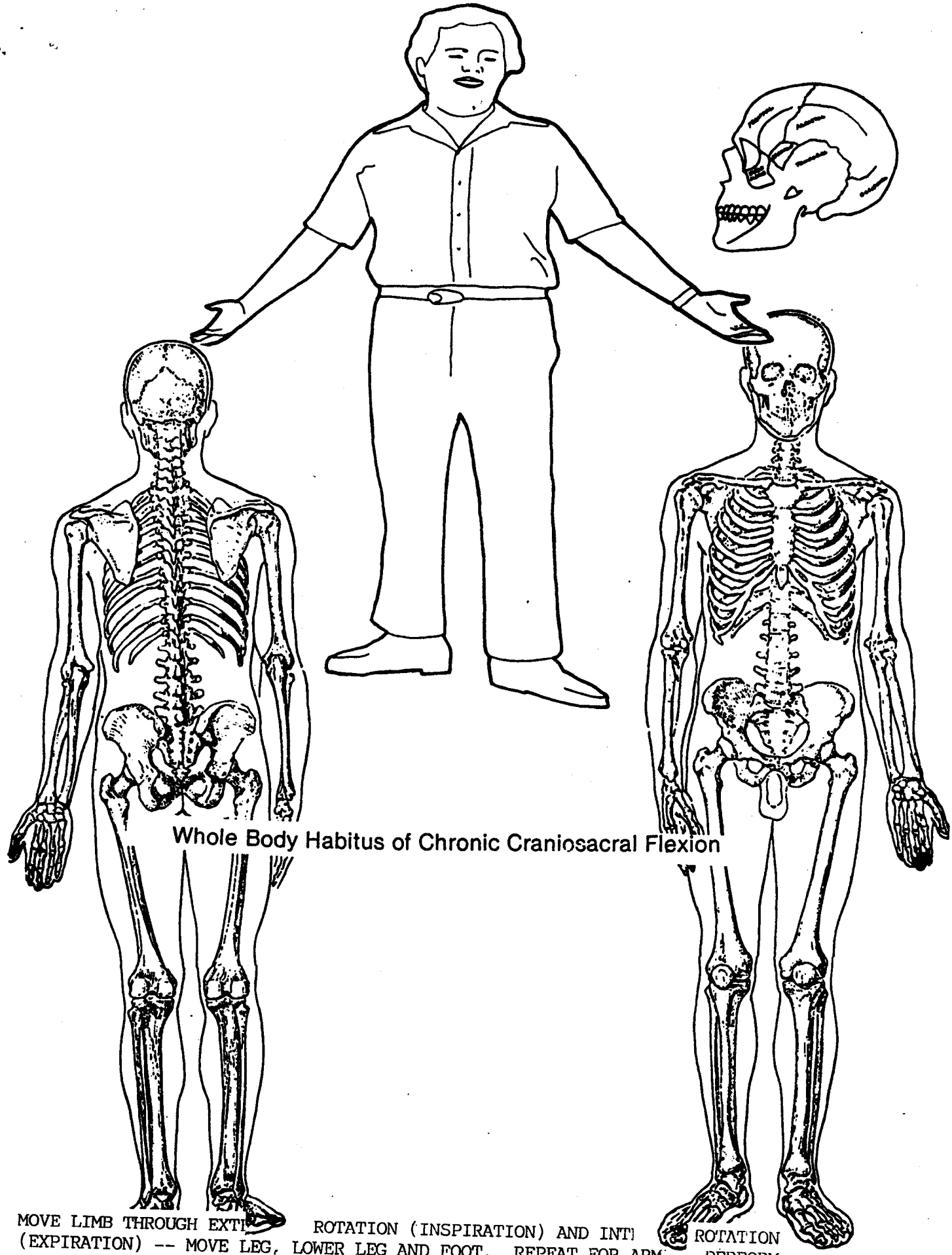
Breath cessation reveals on vertebral spinous T.L. failure of primary spinal caudal cephal movement.

Breath cessation reveals on extremity T.L,failure of normal primary R.O.M. in external and internal rotation.



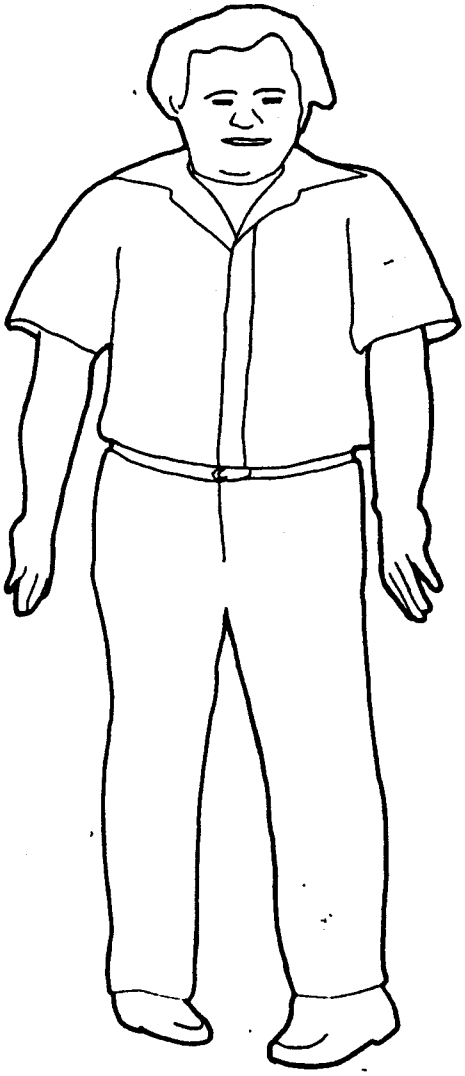


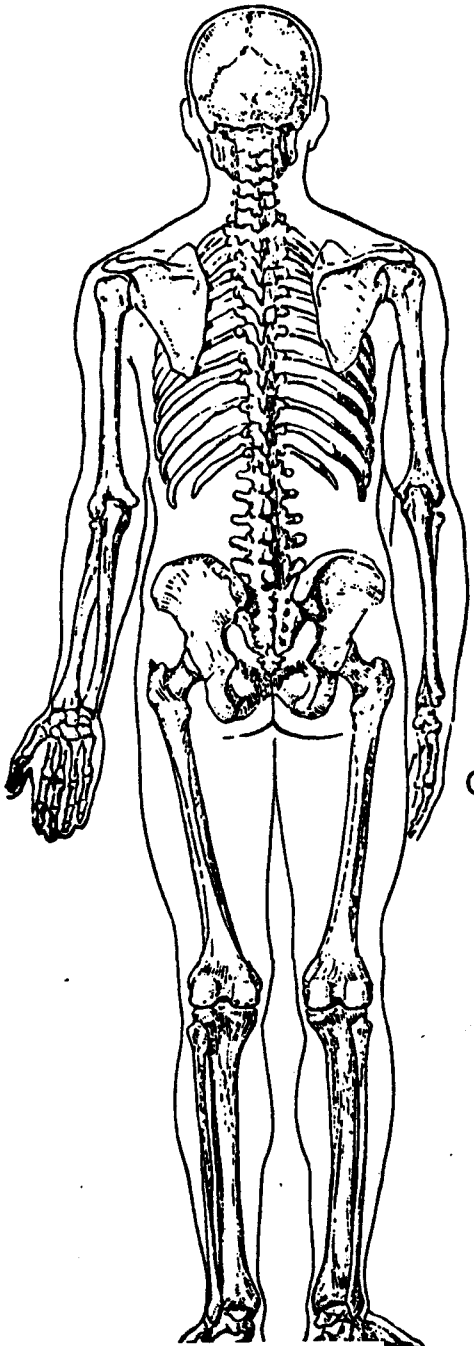
MOVE LIMB THROUGH EXT ROTATION (INSPIRATION) AND INT ROTATION (EXPIRATION) -- MOVE LEG, LOWER LEG AND FOOT. REPEAT FOR ARM -- PERFORM DURING NORMAL RESPIRATION.

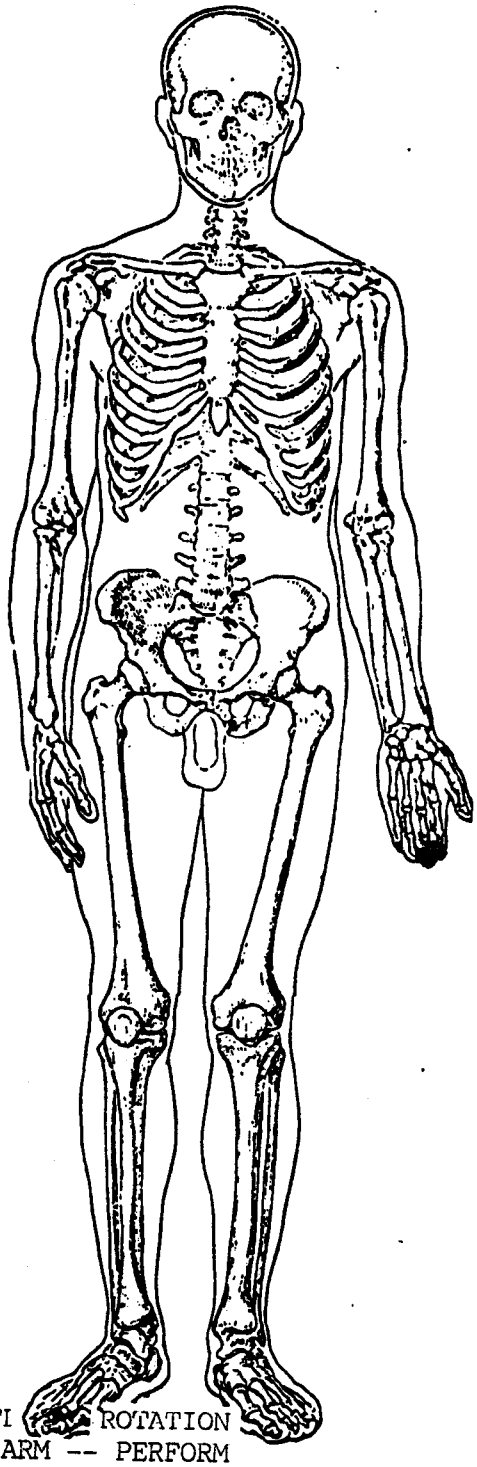


Whole Body Habitus of Chronic Craniosacral Flexion

MOVE LIMB THROUGH EXTE[illegible] ROTATION (INSPIRATION) AND INTE[illegible] ROTATION (EXPIRATION) -- MOVE LEG, LOWER LEG AND FOOT. REPEAT FOR ARM -- PERFORM DURING NORMAL RESPIRATION.







Whole Body Habitus of
Chronic Craniosacral Extension

MOVE LIMB THROUGH EXT[illegible] ROTATION (INSPIRATION) AND INTI[illegible] ROTATION (EXPIRATION) -- MOVE LEG, LOWER LEG AND FOOT. REPEAT FOR ARM -- PERFORM DURING NORMAL RESPIRATION.

Dr. George J. Goodheart
20567 Mack Avenue
Grosse Pointe Woods, MI 48236
(313) 881-0662

CHECK ON BREATH CESSATION AND IN PRONE, SITTING AND STANDING MODES

THE NEW NORMAL OF POSTURAL LATERALITY

THE P_i^* L. U. S* TECHNIQUE

1. Test piriformis in prone and sitting posture, should be strong.

2. Test piriformis in forward flex, backward extend, should be weak on right side. Test sitting, standing, left and right lateral bend. Check against inspiration and expiration.

3. Test lat dorsi and upper trapezius, should be weak left side in forward flex backward extend. Test sitting, standing, left and right lateral bend. Check against inspiration and expiration.

4. Test S.C.M., should be weak on right side in forward flex backward extend. Test sitting, standing, left and right lateral bend. Check against inspiration and expiration.

5. Test iliacus; although both psoas muscles will test strong in forward flex, backward extend, the right iliacus (due to sacral base lateral attachment) will weaken on forward flex and backward extend. Check against inspiration and expiration.

6. If testing above muscles bilaterally does not show conformity to backward and forward flex pattern, suspect in decreasing incidence order;

   Cat one weight bearing, sitting or standing.

   Cat two - three weight bearing, sitting or standing.

   Sacral fixation, sacral subluxation, yaw # 2 weight bearing, sitting or standing. Iliacus malfunction usually right sacral basic contact required. Coccygeal muscle technique rarely required.

   Lumbar 5 fixation or subluxation weight bearing, sitting or standing. Cat three technique frequently required.

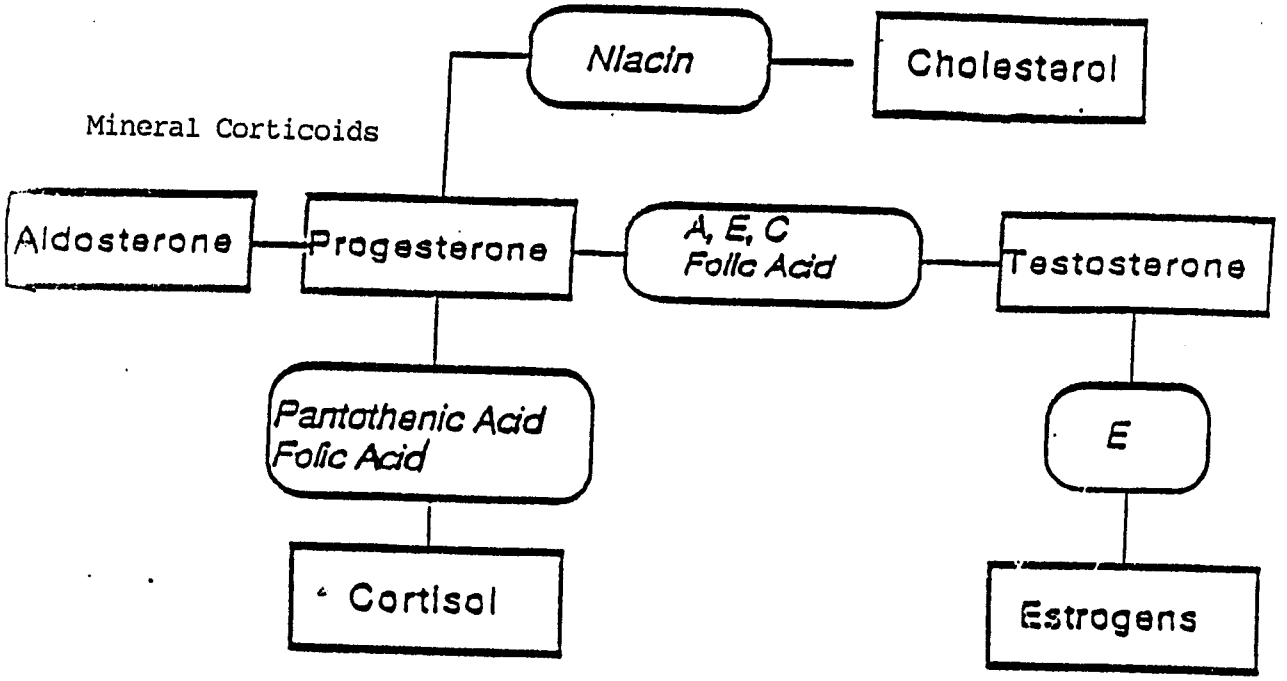
   Occipito-atlanteal fixation or subluxation, or upper cervical subluxation or fixation in weight bearing, sitting or standing. Rarely limbic C-7 1st rib fixation. Cervical compaction; passive versus active range of motion technique is diagnostic.

Correction of above factors should re-establish normal sacral rotation on perpendicular axis, on flex and extend with compensatory 5 lumbar counter rotation with atlas occiput counter torque established by Fred Illi, D.C., 1952 National College Anatomy Lab. Correct all weight bearing problems in weight bearing position if possible.

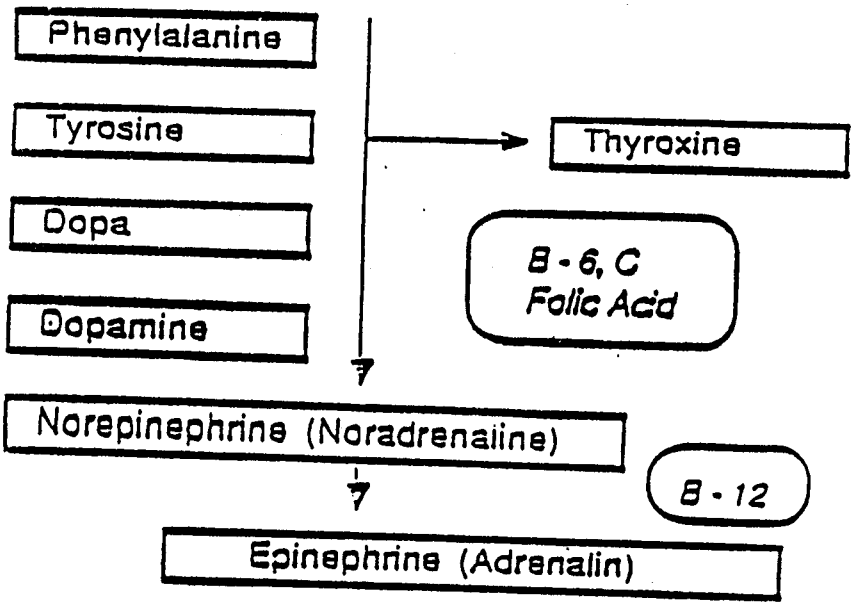
7. Use Electron-Plus, E-Poise or EBA for nutritional support.

8. Set point technique greatly aids spinal length correction.

P_i =	Piriformis (rt) *	Lateral bend high frequency involvement
	i = Iliacus (rt)	
L =	Latissimus Dorsi (lt)	Lateral bend low frequency involvement
U =	Upper Trapezius (lt)	Lateral bend low frequency involvement
S =	Sternocleidomastoid (rt) *	Lateral bend low frequency involvement



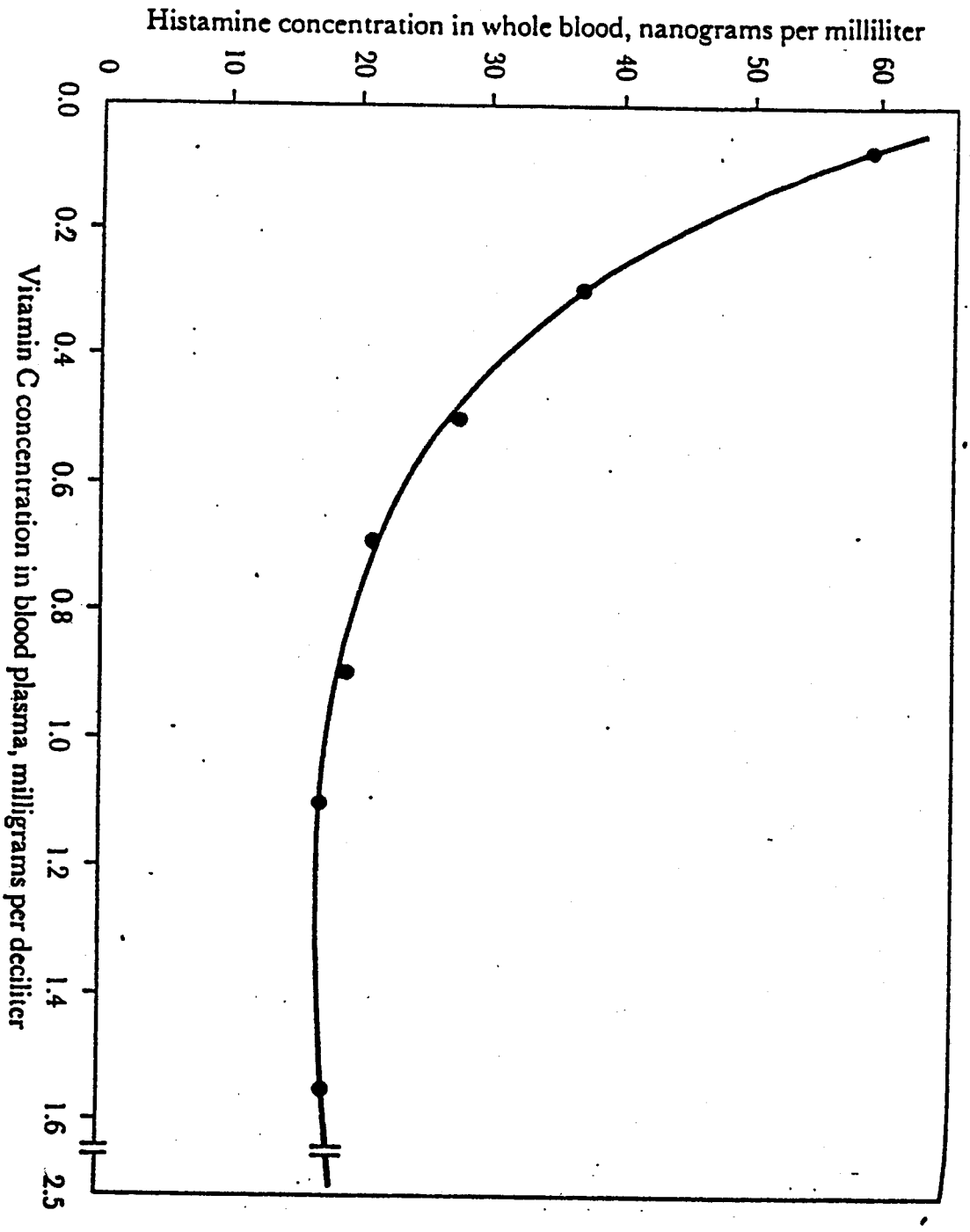
ADRENAL MEDULLA
GRACILIS - NIACIN
PUPIL RESPONSE



ADRENAL CORTEX
SARTORIOUS

BLOOD PRESSURE RESPONSE
ADRENAL - TYROSINE

Cholesterol and Tyrosine based Hormones demonstrating the necessary Co-Factors



Test strong muscles against histidine amino acid powder on tongue. If weakness of strong muscle occurs-test against source of betaine (Betafood S.P. or beet top source) as well as glutathione. Measure C level of mouth lingual ascorbic acid disappearance time. Measure C level of urine with Ames "Stix" reagent strip for vitamin C. Any level below 40mg percent is low and requires temporary specific food restriction and increase in betaine or glutathione support rather than "C" level increases.