Instructions to Authors

The American Academy of Osteopathy (AAO) Journal is a peer-reviewed publication for disseminating information on the science and art of osteopathic manipulative medicine. It is directed toward osteopathic physicians, students, interns and residents and particularly toward those physicians with a special interest in osteopathic manipulative treatment.

The AAO Journal welcomes contributions in the following categories:

Original Contributions
Clinical or applied research, or basic science research related to clinical practice.

Case Reports
Unusual clinical presentations, newly recognized situations or rarely reported features.

Clinical Practice
Articles about practical applications for general practitioners or specialists.

Special Communications
Items related to the art of practice, such as poems, essays and stories.

Letters to the Editor
Comments on articles published in *The AAO Journal* or new information on clinical topics. Letters must be signed by the author(s). No letters will be published anonymously, or under pseudonyms or pen names.

Professional News of promotions, awards, appointments and other similar professional activities.

Book Reviews
Reviews of publications related to osteopathic manipulative medicine and to manipulative medicine in general.

Note
Contributions are accepted from members of the AOA, faculty members in osteopathic medical colleges, osteopathic residents and interns and students of osteopathic colleges. Contributions by others are accepted on an individual basis.

Submission
Submit all papers to Anthony G. Chila, DO, FAAO, Editor-in-Chief, Ohio University, College of Osteopathic Medicine (OUCOM), Grosvenor Hall, Athens, OH 45701.

Editorial Review
Papers submitted to *The AAO Journal* may be submitted for review by the Editorial Board. Notification of acceptance or rejection usually is given within three months after receipt of the paper; publication follows as soon as possible thereafter, depending upon the backlog of papers. Some papers may be rejected because of duplication of subject matter or the need to establish priorities on the use of limited space.

Requirements for manuscript submission:

Manuscript
1. Type all text, references and tabular material using upper and lower case, double-spaced with one-inch margins. Number all pages consecutively.

2. Submit original plus three copies. Retain one copy for your files.

3. Check that all references, tables and figures are cited in the text and in numerical order.

4. Include a cover letter that gives the author's full name and address, telephone number, institution from which work initiated and academic title or position.

5. Manuscripts must be published with the correct name(s) of the author(s). No manuscripts will be published anonymously, or under pseudonyms or pen names.

6. For human or animal experimental investigations, include proof that the project was approved by an appropriate institutional review board, or when no such board is in place, that the manner in which informed consent was obtained from human subjects.

7. Describe the basic study design; define all statistical methods used; list measurement instruments, methods, and tools used for independent and dependent variables.

8. In the "Materials and Methods" section, identify all interventions that are used which do not comply with approved or standard usage.

Computer Disks
We encourage and welcome computer disks containing the material submitted in hard copy form. Though we prefer Macintosh 3-1/2" disks, MS-DOS formats using either 3-1/2" or 5-1/4" discs are equally acceptable.

Abstract
Provide a 150-word abstract that summarizes the main points of the paper and it's conclusions.

Illustrations
1. Be sure that illustrations submitted are clearly labeled.

2. Photos should be submitted as 5" x 7" glossy black and white prints with high contrast. On the back of each, clearly indicate the top of the photo. Use a photocopy to indicate the placement of arrows and other markers on the photos. If color is necessary, submit clearly labeled 35 mm slides with the tops marked on the frames. All illustrations will be returned to the authors of published manuscripts.

3. Include a caption for each figure.

Permissions
Obtain written permission from the publisher and author to use previously published illustrations and submit these letters with the manuscript. You also must obtain written permission from patients to use their photos if there is a possibility that they might be identified. In the case of children, permission must be obtained from a parent or guardian.

References
1. References are required for all material derived from the work of others. Cite all references in numerical order in the text. If there are references used as general source material, but from which no specific information was taken, list them in alphabetical order following the numbered journals.

2. For journals, include the names of all authors, complete title of the article, name of the journal, volume number, date and inclusive page numbers. For books, include the name(s) of the editor(s), name and location of publisher and year of publication. Give page numbers for exact quotations.

Editorial Processing
All accepted articles are subject to copy editing. Authors are responsible for all statements, including changes made by the manuscript editor. No material may be reprinted from *The AAO Journal* without the written permission of the editor and the author(s).

American® Academy of Osteopathy

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THE AAO JOURNAL
FORUM FOR OSTEOPATHIC THOUGHT
A Publication of the American Academy of Osteopathy

TRADITION SHAPES THE FUTURE • VOLUME 12 NUMBER 4 WINTER 2002

The mission of the American Academy of Osteopathy is to teach, advocate, advance, explore, and research the science and art of osteopathic medicine, emphasizing osteopathic principles, philosophy, palpatory diagnosis and osteopathic manipulative treatment in total health care.

Editorial Section

Peer-Reviewed Section

Component Societies' CME Calendar

and other Osteopathic Affiliated Organizations

2003

February 26 - March 2
Midwinter Basic Course
The Cranial Academy
AZCOM
Phoenix, AZ
Hours: 40 Category 1A (anticipated)
Contact: The Cranial Academy
317/594-0411

May 2-4
Intermediate Face Course
Course Director: Doug Vick, DO
Philadelphia, PA
Hours: 16 Category 1A (anticipated)
Prerequisites: 2 Basic Courses one being SCTF, and 3 years Clinical Practice
Contact: Judy Staser
817/926-7705

May 14-17
Osteopathic Contributions to the Health Perception: The Art and Science of Osteopathy as it applies to the use of optometric lenses, visual dysfunctions, and perceptual strains.
Course Director: Joseph Field, DO
Kennebunkport, ME
Hours: 32 Category 1A (anticipated)
Prerequisites: 2 SCTF approved basic courses in Osteopathy in the Cranial Field
Contact: Joseph Field, DO
207/967-3311

May 30 - June 3
Osteopathy in the Cranial Field
Course Director: Andrew Goldman, DO
Philadelphia, PA
Hours: 40 Category 1A (anticipated)
Contact: Judy Staser
817/926-7705

June 9-10
*Addressing Medical Issues Conference: *OIG Compliance, *Stark Rules, *HIPPA Regulations, *Center for Medicare and Medicaid.*
Pinellas County Osteo Medical Society
Las Vegas, NV
Hours: 12 Category 1A (anticipated)
Contact: Kenneth E. Webster, EdD
717/581-9069

June 14-18
Basic Course
The Cranial Academy
Founders Inn
Virginia Beach, VA
Hours: 40 Category 1A (anticipated)
Contact: The Cranial Academy
317 594-0411

June 19-22
Annual Conference
The Cranial Academy
Founders Inn
Virginia Beach, VA
Hours: 40 Category 1A (anticipated)
Contact: The Cranial Academy
317/594-0411

October 10-13
Research Symposium/SCTF Continuing Studies Program
Indian Lakes Resort
Bloomingdale, IL
The Cranial Academy
Contact: The Cranial Academy
317/594-0411

Fifty Years

The Sutherland Cranial Teaching Foundation, Inc. (SCTF) was established in 1953 by William G. Sutherland, DO and senior members of his teaching faculty. A not-for-profit organization, the foundation was conceived by Doctor Sutherland as a way of providing continuity for his teaching. Doctor Sutherland was the first president of the SCTF. Since his death in 1954, subsequent presidents (deceased) were Howard A. Lippincott, DO, Rollin E. Becker, DO, and John H. Harakal, DO, FAAO. Michael P. Burruano, DO has served as president since 1993. The charter of the SCTF calls for dedication to educational activities. It specifically states its objective as "using its resources to establish the principles of osteopathy in the cranial field as conceived and developed by William Garner Sutherland, to disseminate a general knowledge of these principles and the therapeutic indication for this approach to treatment, to encourage and assist physicians in osteopathy, and to stimulate continued study and greater proficiency on the part of those practicing osteopathy in the cranial field." In anticipation of the 50th Anniversary year of the Sutherland Cranial Teaching Foundation, a Research Symposium is being planned. This event will be held from Friday, October 17, 2003 through Monday, October 20, 2003 at Indian Lakes Resort in suburban Chicago. Joint sponsorship is being provided by the Cranial Academy, an affiliate of the American Academy of Osteopathy, and the SCTF. The program is intended to provide a combination research symposium and an abbreviated SCTF continuing studies program. Michael P. Burruano, DO will oversee the SCTF continuing studies portion of this program, and I will be responsible for the research portion. As a contribution to the significance of the anniversary year, this and subsequent issues of *The AAO Journal* in 2003 will feature archival and contemporary papers concerned with the progression of Doctor Sutherland's thought.

In 1954, the *Journal of the Osteopathic Cranial Association* stated that the purpose of the organization was to:

1. "establish a foundation for the *growth* of Cranial Osteopathy as conceived and developed by Dr. William G. Sutherland.
2. "disseminate a general knowledge of the philosophy of, and the therapeutic indications for Cranial Osteopathy.
3. "establish standards for adequate training in Cranial Osteopathy according to the principles as conceived and developed by Dr. Sutherland.
4. "encourage osteopathic physicians to become adequately trained in the principles and practice of Cranial Osteopathy.
5. "stimulate continued study and greater proficiency on the part of those practicing Cranial Osteopathy."

Note: Rachel H. Woods, DO was serving as president of the Osteopathic Cranial Academy (OCA) at that time. The organization was an affiliate of the Academy of Applied Osteopathy. *The OCA Journal* was an annual publication. The table of contents of the 1954 volume included:

- President Rachel Woods' Letter
- Embryology and Physiology of Fascia: G. E. Snyder, PhD.
- Fascia in the Writings of AT Still: H. I. Magoun, A.B., DO.
- Malalignment of the Sternum and Fascial Strain: Sam Hitch, DO.
- Glenard's Syndrome and the Sutherland Fulcrum: Rollin E. Becker, DO.
- A Pep Talk on Osteopathy: Paul E. Kimberly, DO.
- Orthoptics and Osteopathy: Paul W. Thielking, OD.
- Cranial Thinking and Meniere's Disease: Howard A. Lippincott, DO.
- Migraine and Its Treatment: O.R. Attebery, DO.
- Bulbs: H.G. Grainger, DO.
- Dental Pathology in the Light of Cranial Osteopathy: H. I. Magoun, A.B., DO.

Author's Correction:

At the end of the article "Digging On: Some thoughts on the integration of Russellian Cosmology and Osteopathy" by Paula D. Scariati, DO, MPH, please note the change in address for correspondence:

Paula D. Scariati, DO, MPH
2265 Craft Drive
Blacksburg, VA 24060
Phone: 540/443-9106
E-mail: scariati@ewcom.org

The AAO Journal, Volume 12, No. 3, Fall 2002, pp 31-37

Contributors

Ward, RC. Social Capital and Osteopathic Medicine in Transition.

Well known to members of the American Academy of Osteopathy and respected throughout the osteopathic profession, Doctor Ward presented the 2002 *Scott Memorial Lecture.* A Professor Emeritus, Michigan State University, he discusses the relational glue that holds groups and societies together. His particular focus in this presentation is the distinction between corporate and commercial American osteopathy and institutionally organized osteopathic medicine.

Nelson, KE. The Primary Respiratory Mechanism.

Doctor Nelson presents a review of the Cranial Rhythmic Impulse and the Primary Respiratory Mechanism with respect to the Traube-Hering-Mayer oscillation and associated physiology and biochemistry.

Regular Features

Dig On. The 46th Annual AOA Research Conference, 2002 provides the material for this issue. Four abstracts are summarized which address various aspects of osteopathic cranial research. Topics studied include the effects of CV-4 on muscle symp0athetic nerve activity, cranial rhythmic impulse, effect of OMT on recurrent otitis media and effects of cranial manipulation on visual function.

From the Archives. In keeping with the theme of this issue, we refer to the pioneering work of William G. Sutherland, DO, developer of the principles of *Osteopathy in the Cranial Field.* Recognized as an astute student of Andrew Taylor Still, the work of Doctor Sutherland continues to provide the foundation for practice utilized by osteopathic physicians throughout the world. It is appropriate to reprint in full his 1944 *Untitled Talk***.** The reader will have the opportunity to follow this original thinker in great depth as he discusses the Primary Respiratory Mechanism.

Elsewhere in Print. The problem of unilateral low back pain provides daily challenge to the knowledge and skill of osteopathic practitioners. **Michael T. Cibulka, PT** notes that numerous descriptions of sacroiliac joint movement and dysfunction are also problematic for physiotherapists trying to understand the the sacroiliac joint. Osteopathic authors cited are Mitchell and Beal. **Michael P. Zumpano, PhD** and co-authors provide another perspective on the soft-tissue bridge between the Rectus Capitis Posterior and the Posterior Atlanto-occipital Membrane. Osteopathic authors cited are Greenman and Rechtien.

AAO 2003 CME Calendar

For information, contact
American Academy of Osteopathy, Phone: 317/879-1881
Visit AAO's Web site at: www.academyofosteopathy.org

January 16-19
Manual Medicine/Manipulation for Physicians: Lower Back, Pelvis and Lower Extremities
in San Antonio, TX

February 6-9
Diagnosis and Treatment of Low Back Pain and Introduction to Prolotherapy in Santa Rosa, CA

March 17-19
Visceral Manipulation: Manual Thermal Diagnosis
in Ottawa, Ontario, Canada

March 19-23
2003 Annual Convocation: Education and Research: The Backbone of Osteopathy
in Ottawa, Ontario, Canada

April 26-27
Dr. Fulford's Basic Percussion
in Chicago, IL

May 2-4
Prolotherapy: Above the Diaphragm
in Biddeford, ME

June 27-29
Manual Medicine/Manipulation for Physicians: Upper Back, Neck and Upper Extremities
in Chicago, IL

July 18-20
OMT for Common Organic and Clinical Problems
in East Lansing, MI

August 21-24
13th Annual OMT Update "Application of Osteopathic Concepts in Clinical Medicine plus Preparation for Certifying boards"
at Walt Disney World
in Buena Vista, FL

September 19-21
Unlocking the Cranial Sutures I: Development and Release
in San Francisco, CA

October 11
One-Day Pre-AOA Convention Workshop: OMT in Geriatrics
in New Orleans, LA

October 12-16
AAO Program at AOA Convention
in New Orleans, LA

November 7-9
Prolotherapy: Below the Diaphragm
in Biddeford, ME

December 5-7
Visceral Manipulation: Urogenital
in Fort Lauderdale, FL

Dig On

Anthony G. Chila, DO, FAAO

Recent Research

In regard to Osteopathy in the Cranial Field, the 46th Annual AOA Research Conference, 2002, gave evidence that a number of investigators are pursuing aspects of that model. The following abstracts are cited from *JAOA*, Vol 102, No 8, August 2002:

1. THE EFFECTS OF CRANIOSACRAL CV-4 ON MUSCLE SYMPATHETIC NERVE ACTIVITY: Cutler MJ, Stupski BA, Gamber RG, Smith ML. 437

In this study, 6 subjects participated in each of three trials of randomized order which included CV-4 trial, sham and control. Heart rate, mean arterial pressure and muscle sympathetic nerve activity were measured continuously during each trial. Directly measured efferent mSNA was obtained using standard microneurographic technique at the fibular nerve. The authors concluded that a modest decrease in mSNA is associated with the CV-4 induced "still point".

2. UNDERSTANDING THE CRANIAL RHYTHMIC IMPULSE: Lenahan BJ, Nelson KE, Sergueef N, Glonek T. 438

The investigators hypothesize that the Traube-Hering-Mayer oscillation, measured by laser-Doppler blood flowmetry, is useful in aiding the understanding of the basic physiology of osteopathy in the cranial field. Forty-four subjects were evaluated. It was determined that flowmetry provides frequency and signal power data, the interpretation of which is consistent with concepts of Cranial Rhythmic Impulse (CRI) potency, fast and slow tides, and still points.

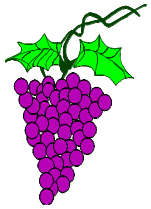
3. EFFECT OF OSTEOPATHIC MANIPULATIVE TREATMENT (OMT) ON RECURRENT ACUTE OTITIS MEDIA IN CHILDREN: Mills MV, Henley CE, Barnes LLB, Carreiro JE, Degenhardt BF, Worden K. 441

This multicenter study utilized a prospective blinded non-placebo controlled study design. Fifty-seven subjects, 3 months to 6.5 years of age were randomized and completed the study. Each subject served as its own control. The "OMT" group received routine medical care and OMT. The "non-OMT" group received routine medical care. After a one-month washout period, the OMT group showed fewer surgical interventions, fewer average monthly episodes of AOM, and fewer average monthly antibiotics prescribed.

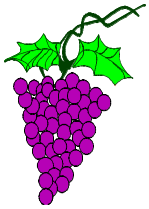
4. THE EFFECTS OF CRANIAL MANIPULATION ON VISUAL FUNCTION-A PILOT STUDY: Greenblatt J, Sandhouse M, Shechtman D, Sorkin R Matterson M, Hardigan P, Snyder A, Shallo-Hoffman A. 448

This study was a double-blind randomized trial. Thirty subjects were given optometric examinations, evaluated for cranial asymmetry and randomly assigned to treatment or control groups. The treatment group was given treatment to correct identified dysfunction, while light pressure without treatment was administered to the control group. Subjects were then reassessed for the presence of cranial dysfunction and subsequently receive repeat optometric examination. Results suggested that visual function can be altered following cranial treatment and that cranial treatment can be useful in certain ocular conditions.

Diagnosis and Treatment of Low Back Pain and Introduction to Prolotherapy



Santa Rosa, California
February 6-9, 2003



John C. Glover, DO, Program Chair

The program anticipates being approved for 22 hours of AOA Category 1-A CME credit pending approval by the AOA CCME.

Course Description: Level II

The course will focus on the mechanical causes of low back pain. Differential diagnosis will be discussed and several different models of evaluation will be presented. The physiological basis, indications and contraindications, evaluation, and treatment utilizing different manipulative models will be presented. The participants will be introduced to the concept of ligament laxity and tendon instability and treatment with prolotherapy.

Learning Objectives:

At the end of each session, participants should:

- Differentiate the different causes of low back pain (mechanical, nerve root & disk, spinal & systemic pathology)
- Choose the appropriate manipulative model for the patient and the problem on the basis of indications and contraindications.
- Differentiate the different manipulative models on the basis of physiological mechanisms
- Understand the physiological basis, mechanisms of action, and indications/contraindications of prolotherapy
- Utilize a palpatory screen to evaluate ligamentous laxity
- Utilize a postural/structural model of evaluation to determine the use of osteopathic manipulative treatment
- Practice treatment of findings using multiple models (balanced ligamentous tension, counterstrain, facilitated positional release, Still technique, etc.)
- Observe/practice selected injection techniques used for treatment of low back pain of ligamentous origin
- Evaluate the lumbar spine, pelvis and lower extremity for sources of low back pain
- Understand the role of the viscera in low back pain
- Discuss issues relating to coding and billing

Course Location and Hotel Information:

Hilton Hotel, Sonoma County
3555 Round Barn Boulevard
Santa Rosa, CA 95403
Reservation Telephone: 707/523-7555
Room Rate: $109.00
Reservation Cut-off Date: January 7, 2003

Program Time Table:

Thursday, February 6 ..... 5:00 pm – 10:00 pm
Friday, February 7 ..... 7:00 am – 1:30 pm
Saturday, February 8 ..... 7:00 am – 1:30 pm
Sunday, February 9 ..... 7:00 am – 1:30 pm
(each day includes (2) 15 minute breaks)

Afternoons are open to experience wine-tasting at local vineyards

Registration Form

Diagnosis and Treatment of Low Back Pain & Intro to Prolotherapy

February 6-9, 2003

Full Name _____

Nickname for Badge _____

Street Address _____

_____

City _____ State_____ Zip_____

Office phone # _____ Fax #: _____

E-mail: _____

AOA # _____ College/Yr Graduated _____

(AAO makes every attempt to provide meals that will meet participant's needs. However, we cannot guarantee to satisfy all requests.)

Registration Rates

	On or Before 1/6/03	After 1/6/03
AAO Member	$630	$730
Intern/Resident/Student	$530	$630
AAO Non-Member	$1,000	$1,100

(Non-members – see membership application on page 14)

AAO accepts Visa or Mastercard

Credit Card # _____

Cardholder's Name _____

Date of Expiration _____

Signature _____

From the Archives

Untitled Talk

Contributions of Thought, Sutherland Cranial Teaching Foundation, 1967, pages 101-115, William Garner Sutherland, DO

This talk was given without notes during a course of instruction at the Des Moines Still College of Osteopathy and Surgery in 1944. The talk was transcribed from either shorthand notes or a recording. A mounted disarticulated skull, separate cranial bones and many anatomical charts were on display as was customary in Dr. Sutherland's courses of instruction in cranial osteopathy. These and the actual demonstration of technics are frequently referred to in the talk.

At the time of my talks before the Academy of Applied Osteopathy meeting in Chicago, a member of the profession put two questions to me: "Is the cranial concept a religious one"? and "Where did you find the bug to think out this stuff"?

While meditating on a promise to write an autobiography, that it seems there will never be time to fulfill, I have wondered if I might devote the opening chapter to a consideration of these two questions. My answers at the time were as follows:

If the recognition by Dr. Andrew Taylor Still of God as creator of the human body is religious then the science of osteopathy, in concept, is religious. If the science of osteopathy is religious then the cranial concept in osteopathy is religious. The science of osteopathy is a specialty and those who practice that specialty are specialists. The cranial concept itself is not a specialty. It is osteopathy and the credit belongs to Doctor Still.

The concept of the science of osteopathy came during a sad period in Dr. Still's life when he had lost members of his immediate family. The experience was related to his loss of faith in orthodox medical methods. The new concept came at an hour when a sincere prayer went up to his Maker for guidance. Dr. Still studied the living human body in great detail and developed a knowledge of its anatomical-physiological mechanisms that became the keynote of his phenomenal skill in diagnosis and technique. In all of his lectures and talks he never neglected to refer to his Maker, the Maker of the human body.

"To the dreamer who can work and to the worker who can dream, life surrenders all things."

That might be modified to read,

"To the dreamer who will dig and to the digger who can dream, the science of osteopathy provides possibilities superior to all other therapeutic methods."

Where did I find "the bug" to think out this "cranial stuff?" Some forty-six years ago, while a student at the American School of Osteopathy in Kirksville, Missouri, my attention was drawn to the bones of a disarticulated skull that Dr. Still had on display among other anatomical specimens. This thought came, like a bolt from the blue: "Beveled like the gills of a fish; indicating articular mobility for a respiratory mechanism." Because of my doubt of the possibility of such mobility that guiding thought became a compelling whip stimulating me to dig and find out. It became "the bug" in my system.

In my study of the intricate articular surfaces on the cranial bones, I found that every detail on those articular surfaces indicated mobility for a respiratory mechanism. In the continued study, I eventually began experiments on my own skull even to the point of creating cranial lesions for the study of their effects. Some of these effects were quite serious, but they helped to show the way.

Someone has said, "To the dreamer who can work and to the worker who can dream, life surrenders all things. " That might be modified to read, "To the dreamer who will dig and to the digger who can dream, the science of osteopathy provides possibilities superior to all other therapeutic methods.

You already know from Dr. Kimberly's lectures covering the anatomy of this region that you are going to have many dreams and that there is reason for meeting with oth-

ers in monthly study groups to dig into this subject further. Remember that Dr. Still said in reference to osteopathy, "We merely have a grip on the tail of the squirrel in the hole of the tree. "Much of the osteopathic squirrel is still within "the hole of the tree." The cranial concept is but a portion of the whole. There are undreamed possibilities in the science of osteopathy as conceived by Dr. Still. Each dream may initiate the working out of a hypothesis. It is necessary to have a beginning.

We learn of the creation of man that, "The Breath of Life," not the breath of air, "was breathed into the nasals of a form of clay and man became a living soul." I consider the breath of air as one of the material elements utilized by man in his walk about here on earth. The human brain is a motor; the Breath of Life is a spark of ignition to the motor, something that is not material, that we cannot see.

In my hypothesis, I have described what we call the primary respiratory mechanism. This mechanism includes the brain, the cerebrospinal fluid, the intracranial membranes and the articular mobility of the cranial bones; and also the spinal cord, the intraspinal membranes, the same cerebrospinal fluid and the involuntary mobility of the sacrum between the ilia. Critics have pointed out to me that there are no muscles attached to the sacrum and the ilia to provide for articular mobility between them. Yet mobility between them has been demonstrated. It has also been pointed out that there are no muscles attached from bone to bone in the cranial structure to provide articular motion between them. It is therefore apparent that the mobility of the sacrum between the ilia and the mobility between the cranial bones is not provided by muscular agencies. It is not the voluntary articular mobility that is motivated by muscular action. The mobility of the cranial mechanism and also the mobility of the sacrum between the ilia is an involuntary movement and the whole functions as a unit during the periods of respiration. The mechanical interpretation of the design of the articular surfaces of the cranial bones, such as the beveled articular surfaces, indicates mobility related to a respiratory mechanism. This does not refer to the respiratory mechanism concerned with the breath of air. As all the physiologic centers of the human body, including the respiratory center, are located in the floor of the fourth ventricle, a primary respiratory mechanism that includes all the elements already named would be primary to thoracic respiration through the center of respiration.

The cranial concept is but a portion of the whole. There are undreamed possibilities in the science of osteopathy as conceived by Dr. Still. Each dream may initiate the working out of a hypothesis. It is necessary to have a beginning.

As a beginning in the study of the primary respiratory mechanism, consider a cast of the ventricles of the brain and spinal cord. Notice its bird-like form. Here we have the body of the bird and the cerebral aqueduct with the fourth ventricle and the central canal of the spinal cord resemble the tail of the bird. Notice, where these lateral ventricles are attached. They are located, as one would find the wings of a bird attached, at the superior anterior border of the third ventricle. Thus we can use the lateral ventricles as an illustration of the wings of the bird that flies. We will take this cerebral hemisphere and put it around that lateral ventricle in its normal position. This develops the wings of the bird. Now, put the breath of Life in there with the spark that ignites the motor and visualize the convolutions of the hemisphere expanding. What does the bird do when it flies? The wings move outward posteriorly during the inhalation period of respiration. Now watch the third ventricle. See the third ventricle dilating in a V-shape manner. Note that the floor of the ventricle moves upward and that the roof stretches out.

What is attached on the floor of the third ventricle? The infundibulum, which runs down to the little pituitary body riding in the sella turcica, or the saddle. The body is not riding freely in the saddle, but is strapped down into the sella turcica by dural membrane. The infundibulum draws that little body upward at the posterior area of the sphenoid during inspiration. Consequently the anterior end of the sphenoid goes downward. The brain does not require muscular agencies for the movement of its structure within the cranium. It lifts the little pituitary body upward and tips the sphenoid into a nosedive during the period of inhalation. What happens during the exhalation period? Birds fly, light on trees and fold their wings down. The upper area of the third ventricle where the wings are attached moves inward, the roof of the third ventricle crowds together, the floor drops down and the little pituitary body drops downward while the anterior end of the sphenoid elevates.

Hilton stated in *Rest and Pain**, ". . . the central parts of the base of the brain . . . rest upon this collection of cerebrospinal fluid which forms for it a most beautiful, efficient and perfectly adapted water-bed;" To this

*Hilton, John. Rest and Pain. 1863. new and revised edition. 195O. p. 24. J. B. Lippincott Co. Philadelphia

➔

I add, not only rests, but rocks its cranial articular cradle. There are two "water-beds," the cisterna interpeduncularis and the cisterna magna.

In the study of the spinal column you learned that the ligaments hold the vertebrae together and allow a range of mobility. You might call them check ligaments or reciprocal tension ligaments. Direct your attention to the reciprocal tension membrane here in the cranium, the falx cerebri and the tentorium cerebelli. I have told you that the membrane continues down the spinal column, hanging like a hollow tube, with firm attachment only at its upper area around the foramen magnum and one or two of the upper cervical vertebrae and at its lower area to the sacrum. Here, in the falx cerebri and the tentorium cerebelli, we have the reciprocal tension membrane between poles of articular attachment in the cranial mechanism. As the sphenoid is lifted during inhalation the reciprocal tension membrane lifts the petrous portions of the temporal bones into external rotation from the median line. At the same time we see the foramen magnum move forward lifting the intraspinal membranes and drawing the sacrum posteriorly between the ilia. During exhalation the reciprocal tension membrane moves the opposite way; the posterior part of the sphenoid drops down, the petrous portions rotate internally and the intraspinal membrane drops the sacrum anteriorly between the ilia.

Beneath the dural membrane we have the arachnoid membrane beneath which the cerebrospinal fluid fluctuates within the brain, around the brain, around the spinal cord, like a hydraulic brake mechanism in an automobile. It has an intracranial force. The emergency brake system in an automobile can really stop the car. The cerebrospinal fluid is not only an hydraulic mechanism but also has chemicals within it similar to those found in arterial blood plus something else - elements of which more may be known in the future. The arterial stream may be supreme but the cerebrospinal fluid is in command.

Out on the battlefields today terrific explosions are creating heavy vibrations. In many instances these affect the membranes of people in the environment locking them down over the little cerebral lakes of cerebrospinal fluid. We will be meeting this effect in our practices of the future.

The Breath of Life, not the breath of air, was still present. It was the spark that ignited the motor. I merely "cranked the starter mechanism" of material respiration.

These cases will have the same problem as one I will cite that arose from a combination of toxic and physical causes. The instance occurred on the shore of Lake Erie where there is a long stretch of shallow water. The man had been imbibing moonshine liquor of poor quality, for it was in the days of prohibition. He had waded out into the water and was suddenly taken with a meningeal shock where the depth was hardly above his knees. His companion carried him back to shore where they worked over him with methods of respiratory resuscitation even though there was no water in his lungs.

I was a guest in a nearby cottage and we hastened to the scene upon hearing the commotion. The man was blue and stiff as a cadaver with no sign of respiration. He appeared to be dead. I grasped his temporal bones and threw them into external rotation. A warm sensation appeared and respiration began. I released my grasp and respiration ceased. Bystanders called out, "Why doesn't someone send for a doctor? " I repeated the technic and the same warm sensation resulted as respiration returned. The man turned his head and spoke to his sister. The Breath of Life, not the breath of air, was still present. It was the spark that ignited the motor. I merely "cranked the starter mechanism" of material respiration.

The ventricles are expanding during inhalation. Visualize that body of cerebrospinal fluid fluctuating through the fourth ventricle during inhalation as well as through the third and the two lateral ventricles. The ventricles dilate during this period. During exhalation the ventricles contract and the fluid fluctuates in the opposite direction, fluctuating also around the brain and the spinal cord. It is a hydraulic mechanism, which we utilize with respiration in the reduction of cranial lesions.

We have an articular mobility in the cranial base, which is formed by bones that ossified in cartilage. This is the cranial bowl and mobility would be impossible here without some compensation in the vault whose bones are formed and ossify in membrane. This compensation is achieved by two features. One is the provision for sutural movement indicated in the serrated design of the articulations between the bones that form the cranial vault. The other is the flexibility throughout the structural portions of these bones that form in membrane. The diploe has two walls. The inner wall is smooth and the outer is rough. There is fluid between the two walls. Thus, to repeat, the bones that are formed in membrane provide compensation to articular mobility between the bones of the cranial base that are formed in cartilage.

Here we have a schematic sketch designed to clarify the movement of the sphenoid and occiput. The sphenoid, including the sella turcica, is

shown as a wheel with spokes. As the sphenoid circumducts, or revolves, the various locations on the wheel move as suggested by the spokes. The movement of the sphenoid is not a backward, nor a forward movement. The occiput, too turns like a wheel. The two wheels turn at the same time. During inhalation the sphenoid wheel turns anteriorly and the occiput posteriorly. Thus you see the sella turcica and the anterior end of the basilar process of the occiput both moving upward. During exhalation just the opposite occurs. The sphenoid bone turns posteriorly and the occiput anteriorly. Thus the sella turcica drops downward, the basilar process drops downward and the jugular foramen and the foramen magnum turn with the wheel, as represented by the spokes.

The junction of the sphenoid and the basilar process of the occiput is an arch. It is somewhat like one of those bridges on the Chicago River that opens up and closes down, both sides together. Although it moves downward it remains an arch as it lowers. This is an important point in visualization when it comes to cranial technic. This junction has been likened to a symphysis. It is most important as an area in the cranial mechanism, an area which you cannot feel directly but which you must visualize. It is like the area of the vertebral bodies in that you cannot palpate the body of a vertebra but you have the mental picture. Your sense of touch observes the transverse processes and that observation tells you of the position of the body. You can learn to tell the position at the junction between the body of the sphenoid and the basilar process of the occiput by the sense of touch. This is not difficult although it may seem so to you right now.

Now we come to the study of the two temporal bones as they join in the movement of the cranial base. First we consider their form and location between the sphenoid and occiput and then we note what the study of their articular surfaces tells us about the mechanics of their motion as the sphenoid and occiput circumduct into flexion and extension at the sphenobasilar symphysis. This mental picture will give us the understanding of the normal motion going on all the time in the cranial bowl. From this mechanical understanding of the normal we will be able to observe and interpret the variations and abnormalities to be understood when we meet them in our patients. For we must have a working diagnosis before we come to consider lesions of this area and technics for correcting them. Two basic motions that occur in the cranial base other than flexion and extension are sidebending and torsion. The temporal bones enter into these significantly. In fact I sometimes think of them as mischief-makers.

The temporal bone moves like a wobbling wheel. If you hold one in your hand with one finger on the tip of the petrous portion and another at the base of the mastoid process and then contact the zygomatic process with the other hand you will observe that when you pull down on the zygomatic process the petrous portion rotates externally, that is, away from the medial line. As I lift up on the zygomatic process you see that the petrous portion rotates internally toward the medial line. Now you note that the petrous portions are located on a diagonal that points forward into the head. Place them into the cranial base between the occiput and the sphenoid and fit the grooves on them to the tongues on the sides of the basilar process of the occiput and you have the picture for motion. When the sphenoid and occiput move into flexion during the inhalation period the petrous portions rotate externally. When the sphenoid and occiput turn into extension the petrous portions rotate internally.

When the petrous portion of a temporal bone rotates externally this mastoid portion moves outward and this mastoid process moves inward. That is, in external rotation of the petrous portion we find the mastoid portion more prominent on the outside of the skull and the mastoid process less prominent. When the petrous portion rotates internally it is the other way around. Then the process moves outward and the portion inward. Thus we have evidence on the outside of the skull of the comparative rotations of the petrous portions on the inside of the skull. This evidence can be palpated and used in the construction of a mental picture of the positions in the sphenobasilar area. Because of the tongue and groove articulations between the basilar process of the occiput and the petrous portions of the temporal bones, the relation between them is direct. From the evidence on the outside as to the rotation of the petrous portions, we can interpret the position of the basilar process of the occiput. The mechanism of the movement between the occiput and the temporal bones is quite intricate and needs detailed study. In some degree the temporal bones move with the occiput because they are carried by it on the jugular processes. The strange part lies in realizing that when the basilar process, as a spoke on the occipital wheel, turns, the petrous portion of the temporal turns along with it. Yet, at the same time there is a motion between the two bones that resembles the motion between a fruit jar and its cap.

The circumduction of the occiput not only turns the basilar process but also the foramen magnum and the jugular processes. We view the jugular processes as a combined pivot and fulcrum process, an arrangement on which the petrous portion is fixed. Thus, if the basilar process moves the pivot moves along with it, yet that

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pivot allows the tip of the petrous portion to move forward and the lower area of the mastoid portion to turn backward. This occurs in inhalation or flexion at the sphenobasilar symphysis. As the basilar process turns backward during exhalation the pivot moves along with it and the tip of the petrous portion moves backward as the lower area of the mastoid portion turns forward. As with the fruit jar and its cap, when you turn the jar in one direction and the cap in the other, while the pivot and the petrous portion move forward the basilar process moves in one direction and the petrous portion in the opposite. You can now see why we have that concavity right back of the jugular processes and the pivot, and why the lower area of the mastoid portion is convex.

During inhalation the anterior end of the sphenoid makes a nosedive, that is, moves down as the area of the sella turcica moves up. What happens at the sphenosquamous articulation? We find that the upper half of the greater wing is beveled externally and the upper half of the squamous portion of the temporal bone is beveled internally and then at a niche at about the infratemporal line the beveling changes so that the lower half of the greater wing is beveled internally and the lower half of the temporal is externally beveled. In the early days of my explorations, I was able to disarticulate the temporal bone from a skull that I owned, by prying it apart with a small blade of a penknife. Because of my mental picture of this mechanism I knew how to take it apart. If you will place the fingers of one hand beneath the mastoid process and spring in on the greater wing of the sphenoid with the other hand, or better with another finger of the same hand, you will feel the action that this sphenosquamous design permits. It is not difficult.

To feel the intricate movement between the occiput and the temporal bones first feel along the occipitomastoid suture and locate each bone. In getting acquainted with the area use light palpation and locate the groove on the undersurface medial to the mastoid process, then feel the pulse of the occipital artery. The articulation is medial to this. Draw your fingers along it at the same degree of palpation. You can detect the movement like the fruit jar and its cap of which I spoke. The movement is somewhat like a crease in a piece of cardboard. It moves outward and inward. Do not press in and obstruct the movement. This is an intricate mechanism and your touch must be very light in order to sense the movements and interpret them in making your diagnosis. You do not do this to fix a suture. You feel it.

When you have the feeling it is necessary to visualize the mechanism and how it works in order to understand what you feel and learn to know the difference between the normal and abnormal from experience. You have the pivot going forward and the petrous going along with it during inhalation and yet the petrous is turning in the opposite direction at the same time. You have the pivot and fulcrum moving backward in exhalation and yet the temporal bone is turning in the opposite direction. As you study this mechanism you will find that this is a lateral and inferior area. But when you examine this pivot it is more of a superior articular surface. A little turning point that means a lot in that mechanism. The pivot moves forward and the temporal bone turns on the lateral surface. That picture must be in mind when you come to a lesion. You are twisting it in the concave and convex surface.

What is a cranial lesion? Suppose we use flexion as an example. If the sphenobasilar symphysis had moved a little beyond its normal range of movement in the direction of flexion and become fixed in that position there would be lack of mobility that would prevent the movement in the direction of extension. For diagnosis you would have all the appearances that go with the flexion position and when you tested for motion you would find that the area could move in the direction of flexion but not in the direction of extension. So you would call it a flexion lesion. The opposite would be the case with an extension lesion. We will also discuss sidebending and torsion lesions of the sphenobasilar area. Thus for diagnosis and technic we will have these four sphenobasilar patterns to work with. There are these factors to think about: the mechanics of movement that the anatomical design of the articular surfaces permits; what each of the four patterns looks like and feels like on the outside of the head; how to test for motion in making a working diagnosis and how to use that diagnosis in applying a technic that will correct the lesion in the easiest and most successful way.

What happens in flexion of the cranial base?

We have seen that when the occiput turns as a wheel the basilar process is turned forward and the jugular process and foramen magnum are also turned forward. The petrous portions of the temporal bones rotate externally and we have the mastoid portions more prominent on the outside of the skull and the mastoid processes inward. The upper area of the temporal bones is turned forward and the area at the parietomastoid articulation is outward.

The sphenoid wheel also turns at the same time and as the anterior end goes into its "nosedive" the greater wings move forward. This movement carries the eyeballs forward, the angles of the frontal bones outward and there is thus a receding at the metopic suture where the two frontal bones join. We find that the ethmoid notch in the or-

bital plate of the frontal is widened posteriorly and the ethmoid bone is moved backward. The lateral parts of the ethmoid contain a collection of air chambers and the fragile turbinates curl and uncurl during inhalation and exhalation like the leaves of a tree. In the midline you have the perpendicular plate of the ethmoid articulating with the ethmoidal crest on the front of the sphenoid body and with the vomer, which in turn articulates with the body of the sphenoid at the rostrum. The vomer runs out over the palatines and maxillae like a plowshare and with the perpendicular plate of the ethmoid makes up the bony part of the nasal septum.

As the sphenoid circumducts there is movement between the ethmoid and the vomer, a little gliding movement. The pterygoid processes hang down below the body of the sphenoid and as we follow their movement as spokes of the wheel we find that they go downward and backward. They turn, as you know, in grooves or tracks on the back of the little palatines. The palatines fit into the maxillae so that the pterygoid processes turn the maxillae through them outward and posteriorly, the same as the angles of the frontal bones are turned. You will find that the teeth, the upper incisors, will have a receding movement the same as the frontal bones. As the greater and lesser wings of the sphenoid form part of the orbital cavities and as four of the extrinsic muscles of each eyeball have their origin around the optic foramen between the roots of the lesser wing, the forward movement of the greater wings makes for a forward position of the eyeballs in flexion.

The greater wing of the sphenoid articulates with the zygomatic bone in the lateral wall of the orbit. As the wing comes forward in flexion of the cranial base it tips the zygomatic bone outward. As the zygomatic process of the temporal bone is moved in external rotation of the petrous portion it also aids in external rotation of the zygomatic. The sphenoid does not as a rule articulate with the maxilla and neither does the temporal bone, but they do articulate with the zygomatic, which in turn articulates with the maxilla. In the orbital cavity there is the sphenomaxillary fissure which we see can provide for a widening and narrowing of the cavity.

In the flexion type lesion at the sphenobasilar area we have what we call a wide type of skull with the wide orbital cavity and the forward eyeball. This gives you an observation cue for diagnosis. You palpate for motion to confirm your diagnosis through observation. In the extension type lesion we have the case where the physiologic motion in the direction of extension has gone beyond the normal range at the sphenobasilar symphysis. You may find it in cases of bronchitis and asthma. The petrous portions have rotated internally and so you find the mastoid portions and the mastoid angles of the parietals to be medial and the mastoid processes prominent. The effect is a narrow shape of the skull. The sphenoid is backward, the frontal inward and the ethmoid notch is narrowed posteriorly. The maxilla is up and drawn inward and the zygomatic bone has turned inward thus narrowing and deepening the orbital cavity. The eyeball is receded into the deepened space. Here you have a factor to consider when thinking about the shape of the eyeballs as related to near and distant vision.

You see that patient coming in with a narrow skull and receding eyeballs. Observation indicates the sphenobasilar extension type. Palpation tells the story. Why? Because you have the anatomical-physiological understanding and the ability to apply the keynote of Dr. Still's technique, namely, thinking, feeling, seeing, knowing fingers. Osteopathy still has undreamed of possibilities to yield. It is not a speciality in the sense of applying to just one region of the body but it does provide the opportunity, should you wish, to specialize osteopathically in the treatment of eyes, ears, nose and throat.

Now to consider the sidebending type of sphenobasilar lesion. In a sidebending rotation of the sphenobasilar junction with the convexity to the left, the greater wings of the sphenoid would be tipped so that the right wing would be higher than the left while the sphenobasilar junction would rotate down on the left. At the same time the occiput tips upward on the right and downward on the left side. The basilar process is included in this so that it is tipped upward on its right side and downward on its left side: on the side, not the end. Whenever the basilar process tips up on one side and down on the other side in this way, the effect on the position of the petrous portions of the temporal bones is direct. The petrous portion on the high side is carried into the position of internal rotation and the petrous portion on the low side is carried into the position of external rotation. So, again, the evidence as to the position of the petrous portion of the temporal bones tells you about the position of the basilar process of the occiput. On the face there will be a wider orbit and a forward eyeball on the side of the higher great wing and a narrower orbit with a receding eyeball on the side of the lower great wing. Thus, with sidebending to the left the great wing being high on the right the wider orbit and the forward eyeball will be on the right. Sidebending rotation at the sphenobasilar to the right would be just the opposite.

Here we have what we call a torsion lesion, meaning here a twist at the sphenobasilar junction. In this case the greater wing is high on the right and the basilar process is high

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on the left. You see that the basilar process is tipped in the opposite direction to the tip of the sphenoid. Now, whenever the basilar process is tipped up on its side you will always find the petrous portion in internal rotation on that side. In this case the left petrous portion is in internal rotation. With the greater wing high on the right we have the wider orbit and the forward eyeball on the right and the externally rotated temporal on the right. On the patient's left side we have the narrower orbit with the receding eyeball and the internally rotated temporal.

Now look at these differences as they can be seen in the general effect they make on the contours of the head as a whole. In the sidebending type you will see that the side of the head, from front to back, on the side where the sphenoid and occiput are high, is flatter, even concave in some instances, and on the other side it is longer and fuller, even convex to a marked degree in some instances. This can be verified by palpation. With this picture in mind you then make your real working diagnosis by the feel for mobility.

Now you can't get your feel down to the area of the sphenobasilar junction itself but you can grasp the sphenoid in front and the occiput in back and turn each of them gently, carefully, so as to get the range of mobility or possible motion. If you find that the range of mobility is more in extension then it is the extension type of lesion. If you find that the greater wing moves up farther on the right and the occiput farther on the left then it is the torsion type with the greater wing high on the right. If you find that the greater wing moves up farther on the right and the occiput also moves up farther on the right then you have the sidebending type with the convexity to the left. With the necessary anatomical-physiological knowledge your fingers can determine the type you are dealing with.

In technic we endeavor to follow Dr. Still's methods. That is, getting the point of release with no jerking and then allowing the natural agencies to return the bones to their normal relations and positions. What are the natural agencies? The ligaments, not the muscles, are the natural agencies for this purpose of correcting the relations and positions at joints. Dr. Still's application of the technic is the gentle exaggeration of the lesion that allows the natural agencies to draw the bones into place. Dr. Still has taken my hand in his and allowed me to feel the lesion as it was being exaggerated and then as the natural agencies pulled the bones back into place. There is reason for applying that technic in the cranial mechanism. The difference between spinal technic and cranial technic is like the difference between an automobile mechanic and a watchmaker. We do not force anything into place in the reduction of the lesion. We have something more potent than our own forces working always in the patient towards the direction of the normal.

What are the normal agencies in the cranium? They are:

1 – The brain - the motor of respiration.
2 – The cerebrospinal fluid.
3 – The reciprocal tension membrane.

When we exaggerate the cranial lesion to the point of articular release we have the patient cooperate through respiration. That is exhaling as deeply as possible and then holding the breath as long as possible. When unable to hold it longer there is a sudden inhalation over which the patient has no power. It moves in the normal direction and we find that the motor causes the cerebrospinal fluid to fluctuate in the membranes. Have you seen a force pump? Often that is the change that occurs in a motor. It is always operating in a direction toward the normal. That is intracranial force.

Note-At this time Dr. Sutherland was demonstrating the methods he was speaking of and the members of the class were located two to a table about the room as they practiced what he was teaching – The verbal description of the general idea of cranial technic is to be found on page 67 in *The Cranial Bowl*.*

In application the flexor profundus digitorum muscles are used for leverage. The origin of these muscles is in the forearm and the action is in the fingers. I liken these muscular agencies to the handles of a pair of pliers. The fingers are interlaced and I pull or draw with the digits. The force is guided along the index finger, then along the little finger, then the middle fingers. One finger does one thing another does another while lifting the parietal bones upward. In this lift you lift these angles of the parietals outward and upward. At the same time you can lift the mastoid angle and pull it forward by exercising the index finger.

In this test you place one palm over the occiput with the fingers extended. Be certain that you are on the occiput and not on the temporal bone. The other hand grasps the greater wing of the sphenoid. Rotate the greater wing anteriorly and the occiput posteriorly, gently and carefully, to see how much movement there is in the upward movement at the sphenobasilar junction. Do not tilt the head. You are moving the sphenoid and occiput only. Some of you place your hand beneath the sacrum to see if you can detect evidence of the motion at the sphenobasilar symphysis. Now throw the sphenobasilar junction downward, just the opposite of what you did to produce flexion and repeat the movement into flexion. Notice the temporal bone when it rotates externally, moving along at the same time that the bottom part turns backward

Sutherland, William G. The Cranial Bowl. 1939. Mankato, Minnesota. Free Press Co.

and the squamous part forward; the mastoid process going inward. Place the index finger over the parietomastoid suture. Have the patient inhale deeply. Note the action that takes place. Visualize mentally the picture of the cerebellum moving with the foramen magnum and the body of cerebrospinal fluid in the cisterna magna. See the cerebellum expanding - the fourth ventricle expanding. The cerebrospinal fluid is in command. The tentorium cerebelli is above the roof of the fourth ventricle. Here you have the temporal bones, the petrous portion in external rotation. During inhalation see the membranous connection with the cerebellum and the tentorium.

Throw the sphenobasilar into extension and note the petrous portion rotate internally. The pivot on the occiput goes backward and so does the foramen magnum. The intraspinal membranes drop down and the base of the sacrum falls anteriorly. As the occiput turns in this direction it carries the petrous portion backward. The mastoid portion is inward and the lower area goes forward as the squamous portion moves posteriorly. In exhalation the mastoid process will be outward. In inhalation the mastoid process is inward, the parietomastoid suture is prominent and the mastoid portion is outward by comparison with the process.

Try to bring the left greater wing up as you also bring the occiput up on the right. This tests the sphenobasilar position of sphenoid up on the left and the basilar process up on the right. Detect the mobility at the sphenobasilar junction. Now try sphenoid up on the right and basilar process up on the left. Next try the sidebending rotation. Move the sphenoid up on one side and the sphenobasilar junction rotates to the other. Test the sidebending rotation to the right. Turn the sphenoid and occiput up on the left. This rotates the sphenobasilar junction to the right.

Now that you have the picture of the rotation of the temporal bones you can learn to fluctuate the cerebrospinal fluid by alternate rotation of the temporals. In this method the tentorium cerebelli fluctuates the cerebrospinal fluid not only in the cranium but also in the entire spinal column. You can use the method for testing the mobility of the temporal bones. You can throw the petrous portion into external rotation and by changing the pull you can throw them into internal rotation. Thus you tell the mobility of the temporal bones. Do not compress. With the same sense of touch, drop onto the lambdoidal suture. You will sense a movement somewhat like a crease in a cardboard . . . Use those thinking, feeling, seeing, knowing fingers!

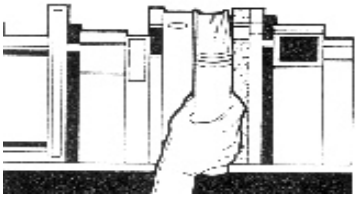
Swedenborg, two hundred years ago, said there is movement of the brain. Have we anything totally new? No.

Now, have your patient hold his breath out. Visualize the compression of the fourth ventricle. The patient is doing more compressing than you are. Then there comes a sudden involuntary inhalation. Then a sensation of warmth is felt through the area of the third and fourth ventricles. The movement of the diaphragm changes. When that point is reached all the fluids of the human body change, including those of the eyeballs, toes, fingers, heart, et cetera. You start the motor that runs this. A branch of the arterial inflow runs up through the body of the cerebrospinal fluid, up on the roof of the third ventricle, out onto the walls of the lateral ventricles and another on the roof of the fourth. These go to make up the choroid plexuses. They lie on the outside of the neural tube and within the arachnoid membrane. The choroid plexus on the roof of the third ventricle stretches out during inhalation.

In thinking about the choroid plexus, realize that Dr. Still was a thousand years ahead of us in his mental picture. What does he mean when he says that the brain is God's drug store, having in it opiates, acids and every other drug thought necessary for human happiness and health? If you become a mechanic of the cranial mechanism by correcting a cranial lesion you then become the pharmacist. There is no end to this thought. It is not a new thought. Swedenborg, two hundred years ago, said there is movement of the brain. Have we anything totally new? No.

The juices of the body are most important. Especially the cerebrospinal fluid. I call attention to the fact that the little pituitary body is surrounded by a wall of blood on every side. Speransky would have recognized a movement of the brain and cranium as a respiratory mechanism if he had gone further in his experiments.

In my experiments I had to use my own skull. Why? Because it was I who had to possess the personal knowledge. I found that not only did the cranial articulations have mobility but that there is also movement of the intracranial and intraspinal membranes and, best of all, a fluctuation of the cerebrospinal fluid. I also found that I could feel that fluid in the cranium of my patient. Take a can of water, and give it a shake and set it down. Place your hand on the can and you will feel the water within it fluctuate. You can feel such a movement in the cranium. You know what you are feeling for. When I used a parietal lift on my head the effect was as though someone were milking my fingers, toes, et cetera.❐



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An Osteopathic Approach to Ear, Nose and throat Patients: The Contributions of William C. McCarty, DO, was published as a "Book Review" by Anthony G. Chila, DO, FAAO in the fall issue of *The AAO Journal*. This book can be purchased from the Matthews CCOM Bookstore, Phone: 630/515-6143. The cost is $13.65 plus shipping/handling which varies depending on location of shipment. The Matthews Bookstore accepts Visa, MasterCard, Discover and American Express.

Social Capital and Osteopathic Medicine in Transition

Robert C Ward, DO, FAAO

Summary

Social capital is the relational glue that holds groups and societies together. Both corporate and individually practiced osteopathic medicine are intimately involved in these high stakes relationships. Social capital shapes the norms, as well as the quality and quantity of societal, institutional, and social interactions, including all private, public, and personal relationships. In general, social networks use social capital to facilitate coordination and cooperation among groups and individuals by increasing their potential efficiency. Horizontal social capital relationships are characterized by social networks that influence individual, family and local community productivity and well-being. The health-related implications are readily apparent. Local governments, schools, churches, and service organizations, as well as families, are good examples. Vertical social capital relationships are characterized by inter-institutional dealings that control the behaviors of organizations, and, by extension, their constituencies. Institutional and governmental politics are good examples. Another example is "the greater osteopathic medicine health care network", with its increasingly complex and influential, world-wide influence through directly affiliated and semi-autonomous stake-holders.

The following discusses a few social capital concepts from a number of standpoints, including the role of AT Still as he set the both the American and international osteopathic social capital networks in motion.

The Scott Memorial Lecture is funded by the Academy for delivery to students at the Kirksville College of Osteopathic Medicine (KCOM). The lecture was established by the family of Drs. J.H.B. and Katherine McLeod Scott to demonstrate "the original and basic techniques as first developed by Dr. Still in founding osteopathy."

Greetings

First, I must thank the American Academy of Osteopathy and KCOM for the honor of sharing some of my thoughts with you today. Forty-nine years ago I sat where you are. Not only does this seem impossible, but, importantly, I could never have predicted a career path that gives me the privilege of sharing this important occasion with you. Joining you on the Truman State University campus is extra special. I have been a Harry Truman fan from the time he took office in 1945, while I was in junior high school.

Dean Osborn tells me that this is the first time the Scott Lecture has been given on the occasion of the White Coat Ceremony. For this opportunity, I am doubly pleased, since better than half my 32 year academic career has been and continues to be spent working with pre-clinical, clinical, and post-doctoral students like yourselves.

Introduction

During my musings for our time together, it occurred to me for the first time that through an accident of history and genetics, my life within osteopathic medicine began a mere 15 years following the death of AT Still.

My gosh! I have spent my whole life within the osteopathic profession; 70 of its 128 years. This works out to almost 55% of its total existence. By virtue of having been born into an osteopathic family, my brother and I spent our formative years discussing the profession's future around the family dinner table. It was as natural as eating and sleeping. Enmeshed in the osteopathic web, we could not possibly have escaped. Osteopathic medicine, with its political ups and downs was always a topic for conversation. For better or worse, classic osteopathic principles and their applications were seldom mentioned.

Since our parents were native Canadians, we seldom discussed American history; not even the Civil War (an obvious breech of osteopathic etiquette). On the other hand, we learned

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lots about Canadian history with its splendid amalgam of British and western frontier roots.

Growing up in the suburbs north of Detroit, Michigan, prevailing osteopathic topics dealt with hospitals and hospital development, osteopathic practice rights, including exclusion of DO's from hospital staffs and military service. Eventually, third party reimbursements took center stage and remain there today.

In retrospect, our family was witness to the extraordinary and dramatic evolution of both corporate and commercial American osteopathy and its evolving, often controversial, *social capital* that was every bit as exciting as anything the profession has experienced before or since.

And so, today, I have chosen to spend a few minutes discussing our complex and evolving osteopathically-oriented *social capital relationships.* I do this hoping that some of the ideas will help us look to the profession's future with more clarity and optimism.

After 45 years of clinical practice and teaching, I have come to understand that corporate and commercial American osteopathy stand separate and distinct from institutionally organized osteopathic medicine.

For the most part, both their corporate and commercial activities operate more or less ambiguously in local and general community settings. Sometimes the osteopathic identity is clear. Often, it is not. On the other hand, institutional osteopathic medicine uses its considerable social capital to specifically identify itself as a distinct, socially cohesive group. I am not sure these comparative differences are much appreciated.

Do they make a difference? You bet they do!

Do they specifically involve osteopathic principles and their many applications? Personally, I think they do. Realistically, however, this is a tough question to answer in straightforward terms. This is because I regularly work with both corporate and commercially oriented osteopathic colleagues, who either quietly or openly disagree with this point of view.

Why is this so?

After decades of reflection and experience with fellow professionals of every possible osteopathic persuasion, I am convinced that *fundamental perceptions of osteopathic principles and their practices continue to challenge everyone in the greater osteopathic network,* both nationally and internationally. Everyone seems to believe they are important, but, for better or worse, believing and acting are very different behaviors.

The problems are particularly perverse in this, the early part of the 21st century as shifting standards for osteopathic principles' applications assume new and unanticipated roles.

AT Still started it all

How these often contrasting *social capital relationships* can work together for the profession's common good is on ongoing dilemma that, perhaps unwittingly, began with Still, himself.

For example, he advocated better surgical and obstetrical practices, as well as state-of-the art psychiatric care. At the same time, he raged against the use of most drugs and vaccines and advocated the use of manipulative treatment for virtually everything.

Typically, Still declared that in order to practice "good" osteopathy, a social capital notion, all one needs to have is a thorough knowledge of structural anatomy and its many functions. For many, particularly for many of his "regular" physician students who were familiar with pharmaceuticals, his position was difficult to accept.

On the other hand, he also emphasized concepts of structure and function that speak to the importance of a healthy life style as well as appropriate interventions for illness and disease, *social capital* concepts acceptable to almost everyone.

Another, important example: Still hired highly qualified physicians like the Edinburgh Scotsman William Smith who taught anatomy, and J Martin Littlejohn, the ASO's first dean and teacher of physiology and psychiatry. It is arguable as to where and when Littlejohn got his MD degree, but that is beside the point. An early trend was underway.

Still and Littlejohn often disagreed about curricular content.[1] Before long, these disagreements were to have national and world-wide consequences.[2]

With the benefit of hindsight, their substantial disagreements amounted to an osteopathic social capital fight that continues, even today, in some sectors. In social capital terms, their eventually strained relationships led to the spread of osteopathic education and practices to the major metropolitan centers of Chicago in 1900, and, eventually, to the establishment of the British School of Osteopathy in London in 1917.

My evolving interpretation of his position is twofold. First of all, Still was deeply concerned about the routine use of obviously ineffective poisons for a wide variety of medical illness. Additionally, he was instinctively anxious about corporate and commercial osteopathy's future. Institutional osteopathy, in the form of the American Osteopathic Association and the Association of Osteopathic Colleges, was growing quickly and becoming politically more powerful.

As general osteopathic social capital picked up speed, Still's charismatic leadership inevitably gave way to perceived needs of the larger osteopathic community, licensure and full practice rights, in particular.[3]

And so, after this lengthy preamble, what is social capital, and how is it defined?

Social Capital and social cohesion

A term borrowed from the fields of economics and political science, *social capital* is the relational, i.e., behavioral, and economic glue that holds like-minded groups and societies together. Social capital, itself, works through relational networks of horizontal and vertically organized relationships.

Social capital and social cohesion work together. Large measures of each create and sustain support networks and relationships of all types. Corporate osteopathic medicine easily fits the definition.

What is horizontal social capital?

Horizontal social capital refers to the social glue that holds individual and local relationships together. Forms of horizontal capital may be the primary reason you chose an osteopathic career.

Examples of cohesive horizontal social capital relationships are: friendship networks, families, clans, tribes, and congregational religious networks. Perhaps it was a particular osteopathic physician, or maybe you had a particularly good relationship with a colleague or friend who introduced you to the profession. The possibilities are endless. Importantly, that individual or individuals will always be remembered, both individually and corporately.

For example, earlier today, Dean Osborn told me about his decades old relationships with five former classmates from right here in Kirksville. They often studied and socialized together. In some cases they trained together after graduation. Now they maintain contact on a more or less regular basis, both individually and institutionally through their organizational work.

If, on the other hand, social capital and social cohesion are persistently incomplete, related social networks are often forced to restructure or ultimately fail. Alienation and divorce are common examples. Clearly, the implications are huge for individuals and corporations alike.

What about vertical social capital?

Vertical social capital accumulates within and among formal institutions, as well as less formal, but connected, corporate relationships. Obvious examples are public agencies, insurance companies, and organizations. Examples are The American Osteopathic Association, The American Association of Colleges of Osteopathic Medicine, the National Board of Osteopathic Medical Examiners, and the American Osteopathic Hospital Association. Collectively, each represents any number of complex formal and informal, interdependent, institutional social capital relationships. The American Medical Association and the rest of mainstream medicine play increasingly high profile roles in this evolving state of osteopathic affairs.

What about social capital failure?

Persistently incomplete or dysfunctional horizontal and vertical capital networks are also at risk for failure. Obvious current examples are personal alienation and divorce, as well as the devastating economic and social fallout experienced by numbers of American corporations and their employees.

How do these concepts relate to 21st century osteopathic medicine?

In my view, both social cohesion and slowly accumulating vertical social capital, have kept corporate osteopathic medicine together for a number of under appreciated reasons. This has not always been the case across the country, however.

An example: For over 50 years, the social capital of both corporate and institutional osteopathic medicine was sustainable in only a few states. Most notable were Missouri, Michigan, Pennsylvania, and California. With the exception of Michigan, each of the other three had strong educational and clinical training programs. Michigan ultimately sustained itself with a state-wide network of independently administered community hospitals that began in Detroit.

This reality slowly began to change in unexpected ways during and after World War II. Favorable federal and state-by-state legislative changes opened many new pathways for the profession. Spearheaded by the AOA, the driving principles were federal support for osteopathic hospital construction and unrestricted medical/osteopathic practice acts. At first unintended, a surprisingly important social capital feature was the profession's ability to economically educate and train large numbers of community-based general practitioners.

Corporate osteopathic medicine continued its growth in other ways. In the 1960s, some 20 years after WW II, new college development took off with the establishment of the first publicly funded osteopathic college at Michigan State University. At the same time, several other institutionally based activities were operating in concert, including hospital, college and post-doctoral accreditation under the (social capital) auspices of the American Osteopathic Association, the American Osteopathic Hospital Association, and the American Association of Colleges of Osteopathic Medicine.

Perversely, and to the dismay of many, traditional osteopathic principles, the basis for the birth and early growth of the profession, received little in the way of serious academic and clinical attention during this growing period. In many ways, fundamental osteopathically-oriented social capital was given lower priority, perhaps even squandered, in the rush for licensure, hospital development, and recognition for general practice as a central osteopathic theme.

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In retrospect, it is clear that several social capital strategies were operative. All were successful, but often in unpredicted ways. For example, originally independent osteopathic hospitals have, for the most part morphed from free-standing institutions into branches of large, multi-institutional mixed-staff entities. In social capital terms, these megamergers are economically useful corporate social capital structures for clinical and educational and research activities. They also represent access to the socially diverse system represented by mainstream allopathic medicine.

How the profession was nearly lost

Significantly, an internal breakdown of social cohesion and misapplication of accumulated social capital by a small group of powerful DO's in California created one of the greatest crises in the profession's history.

Beginning in the 1930s and into middle of the 20th century, corporate osteopathic medicine in California became the largest arm of the profession. By 1950, the state's major teaching facility included one half of the total beds of the Los Angeles County hospital, and students from all six osteopathic schools regularly fought for training slots.

California's social capital blunder occurred when a number of powerfully connected osteopathic physicians decided they would be better off without the "greater osteopathic profession." As we know, they seriously misjudged the social cohesiveness of their colleagues across the United States.

In social capital terms, their politically motivated moves, an example of manipulating *vertical social capital relationships*, were not sustainable. Importantly, there was a major social disconnect between California's power elite and the rest of the American osteopathic network.

Two relational networks picked up the pieces. The first, was a small group of dedicated practitioners working with a politically astute group of committed lawyers and lay people; horizontal relationships to the rescue. The second was institutional osteopathic medicine in the form of the American Osteopathic Association, with its political, financial, and legal resources. Vertically organized social capital also to the rescue.

Evolving social capital becomes both personal and institutional

To illustrate my points in more personal terms, I have chosen to speak of two of my student experiences as a way of outlining some of the complexities. Both were personally instructive and took place almost 47 years ago when I was a third year student at your sister school in Kansas City. Each incident turned out to be a life-changing event, but for very different reasons. Of course, I didn't realize it at the time. Almost five decades later, it seems clear to me that *the concept of evolving social capital* forms a core for each.

The first episode involves student clinical performance at the bedside, a patient-doctor interaction. The second, unwittingly, involved some of us with osteopathic medicine's corporate evolution from so-called "cult" to inclusion in American medicine's mainstream.

Episode One: horizontal social capital develops at the patient's bedside.

In 1955, a number of third-year students sometimes worked as private duty aides to earn extra money. My new patient, an 81year old widower, lived alone and was mostly bedridden from a severe stroke that paralyzed along the whole of his right side. He spent most days propped up, leaning to the right. Not surprisingly, he was depressed from a combination of isolation, lack of mobility, and severe right shoulder and neck pain. For pain control, he was taking strong narcotics that did little but make him sleepy and constipated.

To my inexperienced clinical eye, his plight seemed entirely reasonable: right sided paralysis, pain from lying on the same side, why not? After two or three evenings together, I impulsively asked him if he would let me examine his neck and shoulder using my steadily improving, but still primitive, osteopathic palpatory and treatment skills. He readily agreed. Much to my surprise, there were extensive and easily identifiable musculoskeletal findings in his cervical soft tissues and spinal mechanics.

To make a long story short, application of softly applied osteopathic treatment methods on two separate days took care of his neck pain and got him off narcotics. Today the methods are called functional indirect techniques. At the time, our Kansas City faculty merely labeled them as forms of osteopathic treatment. From then on, I was hooked. Osteopathic manipulative care became an instant educational priority and has been incorporated with my clinical and teaching work ever since.

In the context of this discussion, significant horizontal social capital was banked for future reference, that, in literal terms, has come in handy thousands of times over.

Episode Two: The Cline Committee of the American Medical Association links itself with *evolving vertical institutionally based osteopathic social capital*

My second incident occurred a few months later, when two or three members of our class were asked to make a command performance before the American Medical Association's Cline Committee. A five member *ad hoc* inspection team, the group was inspecting the pre-clinical and clini-

cal curriculums in three of the five osteopathic colleges. Both Kansas City and Kirksville participated, along with the Chicago College of Osteopathy.

After meeting with administrators, our student group was asked to individually demonstrate history, physical exam, and differential diagnosis skills with an unknown patient. During our time together, team members showed considerable interest, particularly in our palpatory skills. In the process, they asked gently straightforward questions and obviously were accustomed to dealing with novice clinicians. The whole experience was actually quite pleasant.

A few weeks later, the Committee filed a favorable, but not unanimous report with the AMA House of Delegates. Their vote was 4-1 in favor of recognizing osteopathic medicine as a legitimate and medically qualified group, albeit with substandard facilities. It was impossible to disagree with this latter judgment.

The House of Delegates was thrown into an uproar, to put it mildly. Ultimately, it was decided to reject the majority report and continue the policy of denying recognition, i.e., removing the "cultist" label from corporate osteopathy had to wait.

In the context of this discussion, it is easily seen that banked vertical social capital relationships among the AMA membership was (at least temporarily) more powerful than that of either its blue ribbon committee or the osteopathic profession.

With the benefit of decades-long hindsight, however, we now know that favorable treatment for corporate osteopathic medicine eventually became inevitable. An obvious current example of change is the presence of hundreds of osteopathic physicians with dual memberships in both the AOA and the AMA.

Long range social capital issues face osteopathic medicine, both individually and corporately.

Now that osteopathic medicine has come so far, a number of long-range, socially vexing problems continue to demand the profession's attention. What are to be reasonable and realistic goals for the following?

>Applications of osteopathic principles and practices in a context of accelerating integration with mainstream American medicine.

>The use of carefully-crafted osteopathic manipulative treatment in a context of total patient care increasingly driven by evidence-based practices. This problem includes the increasingly complicated interactions among labor-intensive primary care health care delivery systems, including demands that more patients be seen in less time. Examples of successful adaptations to these demands are practices that integrate mid-level practitioners of all kinds; clearly an accelerating trend that mitigates against the use of palpatory diagnosis and manipulative treatment.

What can one person do?

So, what is one to do? After a lifetime in the profession, it seems to me that we need to continuously commit evolving osteopathic horizontal and vertical social capital to aspects of our work that make a real difference with patients. In my biased view, this means committing oneself to learning and clinically using palpatory diagnosis and manipulative treatment in a wide variety of settings.

This is exactly what Dr. Still did after several years of ruminating over his future and what it might look like. Anyone with even a passing knowledge of his life's story should realize that he was particularly clever at developing and sustaining varieties of social capital through a combination of charismatic religious fervor, adept patient care, a deep core knowledge of anatomy, and political will. In a very real sense, he was a renaissance man.

What might this mean for the profession's future?

Of course, no one knows, but trends can be seen. An obvious example is the growing diversity and cultures of our student bodies across the profession, our long-term future stake-holders. On this basis alone, both individually practiced and corporate osteopathic medicine needs to continuously evaluate, re-think and nurture its fundamental concepts. Fortunately, efforts along these lines are occurring, both within the osteopathic college network, the National Board of Osteopathic Medical Examiners, and the American Osteopathic Association.

Conclusion

Obviously, Andrew Taylor Still was and continues to be a role model for many. In many ways, he was clairvoyantly ahead of his time. In others, he was glaringly unable to see the future. It is to his credit that those who followed have adapted, sometimes reluctantly, to the profession's changing social capital demands. In retrospect, I suspect that many institutionally-based osteopathic choices were different from those Still, himself, would have made. He was, after all a charismatic leader with little tolerance for consensus, the glue that ultimately sustains all social capital. On the other hand, his feisty personal traits and prescient common sense continue endearing him to many. After all, that is why we gather here today.

There is much more to say, but our time together is short. It is a privilege and pleasure to share some of my thoughts with you on this important occasion. May good fortune and personal fulfillment go with you as you begin your osteopathic journey.

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1. Berchtold, TA, 1975, To Teach to Heal, to Serve! The story of the Chicago College of Osteopathic Medicine; the first 75 years (1900-1975), Chicago College of Osteopathic Medicine, Chicago, p.7

   In this note, Berchtold states: "Although he was not unhappy in his work at ASO, his [Dr. J. Martin Littlejohn's] insistence that 'physiology is the gateway by which this immense field of Osteopathy is to be entered,' rather than blind allegiance to the theory that manipulation and anatomy were the sole basics, brought about opposition to his (Littlejohn's, ed.) views."

2. Author's note: Personal correspondence with Dr. Barbara Peterson, past editor of the Journal of the American Osteopathic Association, and an authority on osteopathic history, is as follows: "It may be significant that Still's definition of "anatomy" included function as well as structure. I have a hunch that "physiology" in Still's mind was associated with the introduction of foreign substances in the body. According to Berchtold, Littlejohn, while at Kirksville, did research on the effects of morphine, quinine, iron, and arsenic in animals, probably in connection with …osteopathic…lesions induced under anesthesia. Maybe …their… disagreements were related to such studies as these. Who knows" ?

3. Peterson, BE, 2002, Major Events in Osteopathic History, in Ward RC, Foundations for Osteopathic Medicine, 2nd edition, pp 22-26, Lippincott, Williams and Wilkins, Philadelphia

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The Primary Respiratory Mechanism

Kenneth E. Nelson, DO, FAAO, FACOFP

Introduction

William Garner Sutherland[1] proposed the primary respiratory mechanism (PRM) that unites the fundamental physiology, cellular metabolism, of the most distant regions of the human body into a coordinated holism. Magoun[2(p.34)] summarized this aspect of the PRM as the "dynamic metabolic interchange in every cell (of the body) with each phase of (its) action." Sutherland, following the methods of Still, based his hypothesis upon a rigorous study of anatomy. He proposed an extremely subtle mechanism that is particularly difficult to prove, or from the perspective of the nulhypothesis, to disprove. Although much research has been performed to elucidate the proposed components of the PRM, the underlying mechanism remains undefined.

Unproven hypotheses may be lent credibility by corroborative experiences. To any physician who has employed Sutherland's methods to treat a patient, or to any patient who has benefited from such treatment, there is corroboration beyond doubt. Such anecdotal evidence, however, is insufficient for individuals who demand rigorous scientific documentation.

Early in the history of osteopathic education, A. T. Still was purported to have felt that anatomy was the one subject an osteopath ought need to know. Later he stated that the student of osteopathy must know, in addition, physiology and chemistry.[3(pg. xvi)] Consequently, Sutherland, who was an early student of Still's, developed his ideas from the perspective of applied anatomy,[4(p.5)] for at the time, the science of anatomy was significantly more developed than the sciences of physiology and biochemistry.

Physiology and biochemistry are now highly advanced and it is appropriate that we now look into these disciplines for evidence that corroborates Sutherland's hypothesis. If the proposed mechanism of Cranial Osteopathy is congruent with scientifically established phenomena, it must be accorded a higher level of credibility. Additionally, the recognition of a body of scientifically validated material will assist future research into Sutherland's discovery.

Over the years, many authors have commented upon the similarity of the Traube-Hering (TH) oscillation (Figure 1) to the cranial rhythmic impulse (CRI), the palpable manifestation of the PRM.[5-9] The Traube-Hering oscillation was initially recognized in 1865 when Traube[10] reported the measurement of a fluctuation in pulse pressure with the frequency of respiration that persisted after respiration had been arrested. In, 1869, Hering[11] confirmed Traube's discovery. Several years later, in 1876, Mayer[12] identified a similar, but slower rate, oscillation. Collectively, these phenomena are now known as the Traube-Hering-Mayer (THM) oscillation.[13] Components of the THM have been measured in association with blood pressure,[10,13-19] heart rate,[13,19,20] cardiac contractility,[21] pulmonary blood flow,[22] cerebral blood flow and movement of the cerebrospinal fluid,[23-25] and peripheral blood flow including venous volume and body temperature regulation.[13,17,19,26] These oscillations are the result of a complex interaction between the sympathetic and parasympathetic components of the autonomic nervous system and renin-angiotensin upon the cardiovascular system, and they are an integral aspect of homeostasis.[17]

Neural activity producing and coordinating the THM oscillation emanates from the floor of the fourth ventricle in the nucleus of the tractus solitarius (NTS). There are lateral pressor areas in the NTS responsible for vasoconstriction, and medial depressor areas responsible for vasodilatation. These areas within the NTS exhibit inherent automaticity.[26] The vagus, cranial nerve X, arises from the medulla immediately adjacent to the NTS and contributes to the THM oscillation through its cardio-inhibitory efferent fibers. A complex interrelationship of tonic activities, reflecting phasic input from the brainstem and the humoral effect of renin-angiotensin, gives rise to the THM oscillation.

Recognizing that Sutherland's proposal is a masterpiece of insight, we

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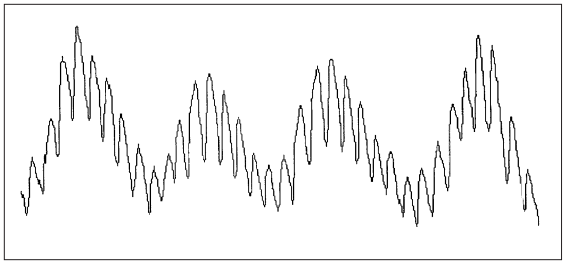
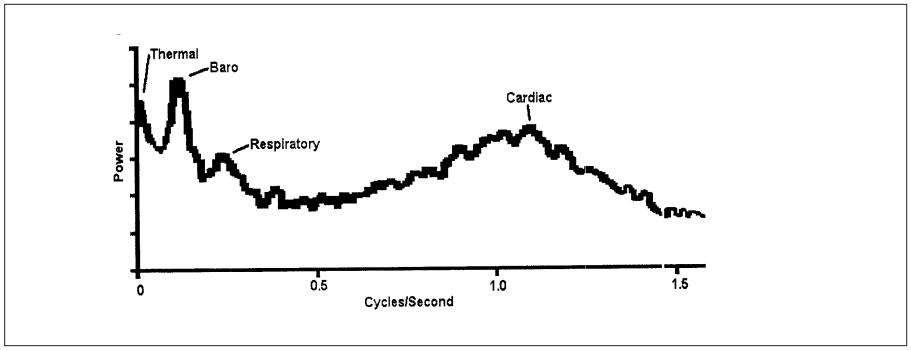


PRM Figure 1: Four Traube-Hering oscillations, in a thirty-second recording of fluctuations in blood flow velocity (heart rate 70 bpm, Traube-Hering rate 8 cpm) measured with laser-Doppler flowmetry,

are still confronted by the question of how it works. Sutherland [4(p.13)] described the PRM as consisting of four features. The first feature is the fluctuation of the cerebrospinal fluid (CSF). The second is the reciprocal tension membrane, which is the dura mater that acts to balance the system by uniting its boney components. The third is the motility of the central nervous system (CNS), and the forth is the articular mobility of the cranial bones and of the sacrum between the ilia (later authors have separated this last feature into two components). It is the first and third components of Sutherland's mechanism that may be considered in the context of the THM. In the following paper, I will attempt to explain the CRI and the PRM, which are discussed in the context of the THM oscillation and related physiology and biochemistry.



PRM Figure 2: Power spectrum analysis (Fourier, Transform) of blood flow velocity demonstrating distinct frequency peaks at 1 cpm (Mayer, thermal oscillation, CRI slow tide, 0.01 Hz), 6 cpm (Traube-Hering, baroreflex oscillation, CRI fast tide, 0. 1 Hz), 15 cpm (respiratory oscillation, 0.25 Hz) and 66 cpm (heart rate, 1. 1 Hz)

The rate of the CRI

The CRI has a traditionally agreed upon rate of 10 to 14 cycles per minute.[2,27-30] Using various methods, many researchers have measured the CRI on human subjects[5,9,28,31-36] and animals.[37,38] In these studies, rates have been reported for the CRI of between 3 and 14 cycles/min.

Becker[27] stated that the CRI consists of two components, a "fast tide" (8-12 cpm) and a "slow tide" (0.6 cpm). Frymann,[5] using a pressure transducer applied to the head to monitor the CRI and a plethysmograph applied to the finger or arm, noted that cranial motion recordings coincided with appendicular volume changes. The plethysmograph also demonstrated "long slow cycles from 50 to 60 seconds" duration which were thought not to be related to cranial changes. Upledger,[35] using plethysmography to monitor the CRI, identified "a frequency of 9-11 cpm" and an "even slower frequency of 1-2 cpm".

The fast Fourier transform spectral analysis of blood flow velocity (Figure 2) reveals three significant peaks at frequencies below the peak for heart rate. These are, from highest to lowest frequency, representative of respiration, Traube-Hering, and Mayer oscillations. The Traube-Hering component demonstrates a frequency range from 5 to 10 cycles per minute and is associated with baroreflex activity.[17-19,39,40,41] Because the response time for the parasympathetic nervous system is shorter than that of the sympathetic nervous system, only the parasympathetic system reacts rapidly enough to directly mediate Traube-Hering activity above 6 cpm (0.09-0.17 Hz).[18,19]

The Mayer component of the THM oscillation demonstrates a frequency range from 0.5 to 2.5 cycles per minute (0.008 to 0.04 Hz),[17-19,26,39,40] and is associated with thermal regulation.[17,26,42,43] The sympathetic and parasympathetic nervous systems are both directly responsible for the modulation of cardiovascular fluctuations in the range of the Mayer low frequency oscillation. This component of the THM oscillation is further regulated by activity of the renin-angiotensin system.[18,19]

Using laser-Doppler blood velocity flowmetry, we have shown that the Traube-Hering component of the oscillation is simultaneous with the fast tide of the CRI.[9] We measured the ratio at 2:1 between Traube-Hering and CRI, which indicates a slower rate (3-7 cpm) for the CRI than has been reported in most osteopathic literature. This rate is, however, consistent with the rate for the palpated CRI measured by Norton.[36]

Lockwood and Degenhardt[44] further analyzed Frymann's data and demonstrated cycle to cycle variability that bears striking resemblance to frequency modulation demonstrated in the laser-Doppler blood velocity flowmetry measurements of the THM.[9]

Frymann,[2 (p. 322)] referring to her landmark studies measuring the CRI, is quoted as follows: "The cranium is not only an elastic rather than a rigid container, but appears to at least at times involve itself in at least three distinct oscillatory motions. First, an oscillation having the same period as the breathing of the subject. Next, an oscillation having a period of five or six seconds, independent of the breathing cycle, i.e., the former normally does not occur as a harmonic of the latter. Lastly, a very slow cycle from one to several minutes duration. There is little doubt that the second of the distinct oscillations is the Sutherland wave . . ." Thus, it is logi-

cal to argue of the THM oscillation that the Traube-Hering oscillation is the "Sutherland wave", or "fast tide", of the CRI and that the Mayer oscillation is Becker's "slow tide".

A whole-body phenomenon

The CRI and the THM oscillation share the quality of being demonstrable throughout the body. The CRI is palpable in all areas of the body.[2,5,6,29-31] The THM oscillation effects all tissues of the body through their impact upon the entire circulatory system. They have been measured simultaneously in the right index finger, right second toe, and pinna of the right ear.[41]

It is important to note that although the THM is discernable throughout the body, it does not consistently affect all areas measured simultaneously.[41] This property may explain the difficulties experienced when two examiners have attempted to concomitantly palpate the CRI.[45]

The relationship between the CRI and pulmonary respiration

Pulmonary respiration is recognized as closely associated with, yet independent of, the CRI.[1,2,5,6,29,30,46,47] Respiratory cooperation of the patient is often employed in association with cranial treatment.[1,2,27,48-51] Cranial manipulation has been said to effect respiration,[47,51] and spontaneous deep sighing respiration has been reported coincidental with the therapeutic end point.[29] Fourier analysis of the THM shows that the low frequency oscillations, Mayer, Traube-Hering and respiration, are distinctly separate components.[13,18,19,39,52] The Mayer and Traube-Hering components, however, are closely linked to, and may be modulated by, pulmonary respiration through the phenomenon of frequency entrainment.[53,54] Entrainment occurs when two systems are oscillating at close frequency, one to the other. The dominant frequency will force the second oscillation to assume, in synchrony, the same frequency as the dominant input.[17,26,55-57] MacPartland[8] has suggested further that entrainment might play a significant role in cranial treatment, for example CV-4, directed at modification of the CRI.

Fluctuation of the cerebrospinal fluid and motion of the central nervous system

The first and third features of the PRM, the fluctuation of the CSF and the inherent mobiIity of the CNS, are readily explained in the context of the THM oscillation. Sutherland stated: "According to my present hypothesis . . . the brain involuntarily and rhythmically moves within the skull. This involuntary rhythmical movement involves dilation and contraction of the ventricles, during respiratory periods. The ventricle dilation and contraction in turn effects cerebrospinal fluid circulatory activity; and the circulatory activity effects movement of the arachnoid and dural membranes; and through the special reciprocal tension membrane. . . effects mobility of the basilar articulations."[1(p.51-52)] . . . "The hypothesis does not include dilation nor contraction of the spinal canal. The spinal canal merely moves upward and downward. . . The cerebrospinal fluid throughout the vertebral column fluctuates by way of the arachnoid membrane; the membrane being hung from above, with only one attachment, and that at the sacrum."[1(p.52-53)]

Motion of the brain[58] and CSF,[58-59] in synchrony with the cardiac cycle, has been demonstrated utilizing magnetic resonance velocity imaging. The images show that, during cardiac systole, there is a net inflow of blood into the brain, causing it to expand in volume and move in a complex fashion. Because of the lesser mobility of the skull, this volume change causes the central portion of the brain and the brainstem to be displaced in a caudal direction. The CSF in the lateral ventricles of the cerebral cortex moves medially into the third ventricle. The CSF in the third ventricle moves in a caudal direction into the fourth ventricle to allow for CSF oscillation the fourth ventricle acts as a "mixing chamber". An amount of CSF equal to the volume change of the brain, displaced from the ventricles and intracranial subarachnoid space into the spinal canal, moves through the spinal subarachnoid space in a caudal direction, thereby increasing pressure in the dural sac surrounding the spinal cord. During diastole, because of the lower intracranial pressure, there is recoil of the caudal displacement of the brain, and the CSF motion reverses direction. For this to occur effectively, the system must demonstrate capacitance. The spinal dural sac acts as the required capacitor.

As blood flow velocity and pressure has been demonstrated to fluctuate at the frequency of the Traube-Hering oscillation, so too, using ultrasound, volume oscillations at the rate of Traube-Hering waves have been measured in the brains of conscious healthy humans.[60] In patients with normal pressure hydrocephalus, intracranial pressure, measured via a catheter inserted into the lumbar subarachnoid space, showed pressure fluctuations at the same frequencies as the THM oscillation.[25] The Traube-Hering component has been labeled the C waves and demonstrates an amplitude from barely discernible to 20 mm. Hg. Lower frequency waves, at the frequency of Mayer waves, with an amplitude as great as 50 mm Hg are identified as B waves. Thus, it may be presumed that the motion of the brain and the CSF, in synchrony

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with the cardiac cycle, continues in similar fashion at the rate of the Traube-Hering oscillation.

Utilizing exposure times that were too slow to assess cardiac and Traube-Hering synchronous motions, computerized tomography has been employed to observe movement of the lateral and third ventricles in the normal brain. It was shown that the brain, rostral to the foramen of Monro, demonstrates a complex rolling peristaltic motion with a rate (26 sec to several minutes in duration) in the range of the B waves or the Mayer component of the THM.[61]

The pulsating brain, can therefore, be considered to act as a pump, energized, at least in part, by the volumetric fluctuations of circulating blood and CSF. As the intracranial blood volume increases, within the skull CSF moves through the ventricular system and is displaced into the extracranial subarachnoid space, increasing the amount of CSF in the spinal dural sac capacitor. As intracranial blood volume decreases, the tension of the spinal dural sac facilitates the return of CSF into the skull, Thus, synchronous with, and, at least partially because of the THM oscillation, the CSF may well be described as "ebbing and flowing".

The primary respiratory mechanism

The PRM is described as the driving force associated with the activity of cellular metabolism.[1,2,30] Magoun[2(p.34)] specifically states: "By this means every cell in the body receives not only the inspired oxygen but also the nutrition, the enzymes, the hormones and whatever else contributes to high level wellness. Included in this internal respiration is the elimination of waste metabolites through the proper emunctories."

The THM oscillation is intimately involved in the regulation of peripheral blood flow and, consequently, tissue perfusion. Circulatory and body core temperature homeostasis is a result of the THM oscillation.[13,17,19,26] A hypothetical explanation for the PRM, therefore, can be devised by employing our understanding of the THM oscillation.

Heart rate, blood pressure and blood flow velocity fluctuates at the THM frequencies. Thus, the peripheral vascular system is entirely under the influence of the THM oscillation.[10,13,17-26,39-43] Cellular respiration is dependant upon effective tissue perfusion, a manifestation of the peripheral circulation. A model for the PRM can consequently be constructed based upon the physiology of peripheral circulation. It is appropriate, therefore, to consider how each component of the system, the arterial resistance vessels, the capillary bed interface with the interstitium, cellular respiration, the lymphatic return, and the venous capacitor and return, contributes to the function of the PRM.

The arterial resistance vessels: The arterial system is the active location of blood pressure modulation. This occurs to a great extent through baroreflex control of arterial vasomotor tone. The Traube-Hering oscillation is a direct manifestation of baroreflex activity.[13,19,62,63] Stretch receptors in the arch of the aorta, at the bifurcation of the brachiocephalic artery, in the common carotid arteries, and in the carotid sinuses continuously monitor systemic arterial pressure. Sensory neurons transmit information regarding the status of the blood pressure (increasing or decreasing) to the NTS in the floor of the fourth ventricle. Neural activity producing the THM oscillation emanates from the NTS. Within the spinal cord, myelinated vasoconstrictor fibers under the control of the NTS, descend to the thoracolumbar sympathetic ganglia. From there unmyelinated post-ganglionic fibers carry vasoconstrictor activity to the periphery. The pressor and depressor areas of the NTS exhibit inherent automaticity.[39] Even though oscillating sympathetic activity demonstrates frequency content from just above O.o cpm (0 Hz) to heart rate, 60-180 cpm (1-3 Hz) in humans, the arterial vasculature responds to modulation of sympathetic stimuli as low-pass filters with significant gain only to frequencies below 9 cpm (~0.15 Hz).[64] This response of rhythmic tonic activity, reflecting phasic input from the brainstem, gives rise to the THM oscillation of blood flow velocity and pressure.[14]

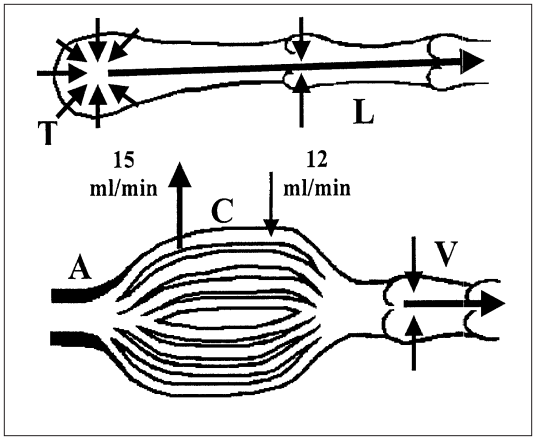
The capillary bed interface with the interstitium: Capillary, and consequently cellular, perfusion is determined by interplay between central regulation and local requirements. Blood flow, through the peripheral tissues demonstrates periods of underperfusion alternating with increased perfusion, such that there is flow through individual capillaries that satisfies local metabolic tissue demands. The variation in tissue perfusion has been demonstrated to occur in discrete locations, in groups from 10 to 15 capillaries. The control of this local fluctuation in blood flow velocity is, therefore, presumed to be at the level of the supply arterioles.[65]

Local blood flow oscillations occur independently, at the 7-10 cpm frequency; however, these oscillations are probably coordinated by the central Traube-Hering oscillation through entrainment of frequency.[17,26,55-57] Thus, although regulated by local tissue requirements, the oscillating arteriolar vasomotion is synchronized by the NTS.

Arteriolar motion drives fluid into the interstitium by physical activity. Periodic changes in arteriolar diameter implicit to vasomotor activity, when combined with the length of the active vessel, produces an equivalent displacement of the tissue mass and its fluid.[66,67] Additionally, in the cap-

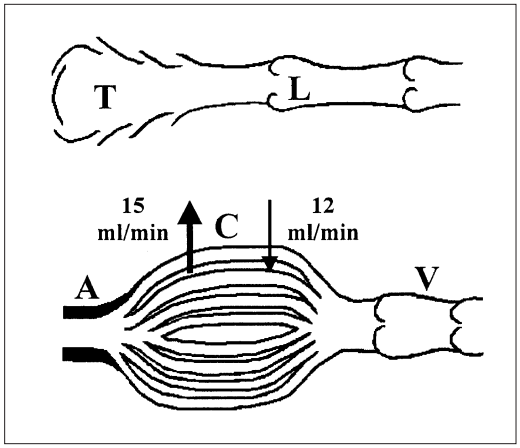
Forces moving fluid out of the vascular compartment:	
Mean capillary pressure	17.0 mm Hg
Mean negative interstitial pressure	7.0 mm Hg
Interstitial fluid colloid osmotic pressure	4.5 mm Hg
Total outward pressure	28.5 mm Hg
Forces drawing fluid into the vascular compartment:	
Total colloid osmotic pressure	28.0 mm Hg
The summation of these forces results in a net outward force of:	0.5 mm Hg

PRM Figure 3: Starling's equilibrium: The direction and rate of transfer between plasma in the capillaries and fluid in the interstitial matrix depends upon; the hydrostatic pressure on each side of the capillary wall, the osmotic pressure of protein in the plasma and in the interstitial fluid, and the properties of the capillary wall as a filtering membrane. The hydrostatic pressure varies as a manifestation of the THM oscillation.

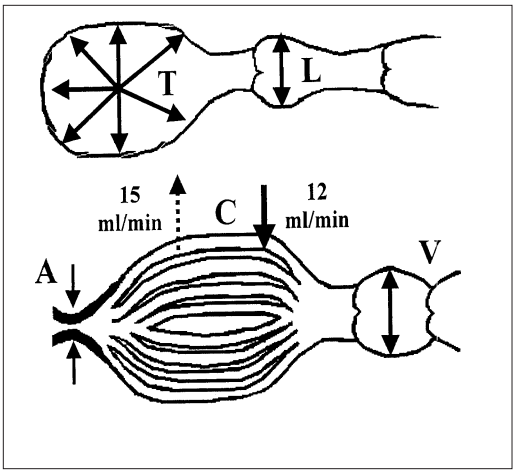


PRM Figure 6: As arterioles dilate, increased blood flow results in increased intracapillary hematocrit and pressure. Consequently interstitial pressure increases, compressing the lymphatic and venous vasculature thereby driving lymph and blood centrally.

PRM Figure 4: The vascular system and the interstitial matrix; arteriole (A), capillary bed (C), venule (V), terminal lymphatic (T), lymphangion (L). Starling's equilibrium requires that for every 15 ml of fluid that enters the interstitial matrix from 12 to 13.5 ml returns to the arteriovenous system, leaving 1.5 to 3 ml to be returned centrally via the lymphatics.



PRM Figure 5: With arteriolar constriction, intracapillary hematocrit and pressure, and consequently interstitial pressure, decreases to approximate venular pressure. This enhances fluid return into the capillaries and also results in distension of the lymphatic vasculature and venules.



illary beds, local contractile responses to distension have been demonstrated in some tissues.[14(p.127)]

This oscillation also has significant effects upon Starling's equilibrium (Figures 3 and 4). Arteriolar vasomotion causes capillary hematocrit changes in such a way that during periods of vasodilatation, with increased blood flow velocity, there is increased local hematocrit and, therefore, increased transcapillary pressure gradients (Figure 5). During the alternate vasoconstrictive phase, the hematocrit decreases, and intracapillary pressure drops to the level of adjacent venules facilitating reabsorption from the interstitium (Figure 6).[65]

Cellular respiration: Oscillating "oxygen availability waves" in the central nervous system have been recognized for years in both animals[68-71] and humans.[72-75] Following these studies, it has been shown that cortical metabolism oscillates at a mean rate of 9.58 cpm.[76] This was demonstrated by reflectance spectrophotometry of the cortical cytochrome oxidase redox state. Associated local fluctuations in blood volume were

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shown to occur. These data suggest that the cyclic increases in cortical oxidative metabolism represent the primary local oscillatory process, followed by reflex hemodynamic changes that effect local tissue perfusion, and in this specific study, intracranial blood volume. Although this process does not occur synchronously throughout the brain, it is not unreasonable to conclude that the relatively close frequencies of the cytochrome oxidase redox state and the Traube-Hering oscillation also allows the two processes to become entrained, thus linking local and central control of tissue perfusion.[17,26,55-57]

The work on cytochrome oxidase redox state and associated arterial vasomotion was published several years before the recognition of the vasomotor effect of nitric oxide (NO). Among other sites, NO is generated in arteriolar walls by endothelial nitric oxide synthase. The generation and release of NO occurs as the result of shear forces exerted by the flow of the blood against the vessel wall. As vasoconstriction increases the shear forces, NO release results in vasodilatation. NO is also released into the interstitial space where it enters the cell, binds to and reversibly inhibits mitochondrial cytochrome oxidase, with resultant reduction of cellular oxygen consumption. The NO-mediated inhibition of cellular oxygen consumption may be modulated in part by the redox state of cytochrome oxidase in the mitochondria.[77] It is also of interest to note that NO has been demonstrated to act as a mediator of the baroreflex in the NTS.[78]

The lymphatic return: Sutherland noted an asso1ciation between the PRM and lymphatic circulation. "Compression was applied around the head with the intent to limit all basilar activity. The experiment resulted in an immediate change of the movement of the diaphragmatic respiratory mechanism, as well as an indication of a change throughout the systemic lymph channels; the indication in the lymph activity being greater in its manifestation than the author has thus been able to secure through the application of the lymphatic pump method."[1(P.55)]

As blood flows through the capillaries, fluid filters out into the interstitial space. In a resting adult, approximately 15 ml/min (Figure 4) leaves the vascular space and 12 to 13.5 ml/min (or 80-90%) returns. This results in 1.5 to 3 ml/min, or 2 to 4 L/24 hr, carrying 80-200 gm of protein that remains in the interstitium and, therefore, must be removed by the lymphatic system each day.[79(p.5)]

Effective lymphatic drainage necessitates the efficient movement of fluid through the interstitial space. As much as 99.9% of interstitial fluid exists in a gel-like state. Proteoglycan filaments form weak cross links with each other, with collagen fibers, and with protein molecules to give the interstitium its gel-like consistency. Consequently, fluid does not flow freely through the interstitial space.[79(p.6)] The interstitial gel, however, demonstrates elasticity.[79(p.15)] An oscillation of this gel matrix with a more fluid sol state in synchrony with the PRM has been proposed.[80] Tissue fluid pressure is a determinant of fluid transfer between the blood and tissue spaces and between tissue spaces and terminal lymph vessels.[79(p.16)] It is probable that Traube-Hering mediated pressure changes induce fluid movement through the interstitial matrix.

The key requirement for lymphatic filling is a volume change of the terminal lymphatic. (Figure 4) The cells of the terminal lymph vessels are arranged in an overlapping-shingle fashion that allows interstitial fluid to enter the vessel. Due to the presence of one-way valves within the proximal lymphatic vessels, volume increases in the end lymphatics can occur only when fluid traverses the interstitium and crosses the lymphatic endothelium. The hydrostatic pressure in the end lymphatic vessels is presumed to be similar to that in the interstitial fluid, with transient differences between the two compartments being quickly equilibrated.[79(p.62)] Negative interstitial pressure (-7 mm Hg) of Starling's equilibrium (Figure 3) causes the interstitial fluid to be pulled into the terminal lymphatic vessels. The negative interstitial pressure is maintained by removal of protein from the interstitium by the lymphatics.[79(p.19)] Additionally, intermittent motion in the tissues associated with the Traube-Hering-mediated arteriolar vasomotion causes pulses of fluid to move into the terminal lymphatics.[79(p.7)] The Traube-Hering-driven fluctuating intracapillary pressure and hematocrit further adds to this mechanism.

Once the terminal lymphatic becomes filled to capacity, the overlapping cells approximate one another, augmented by the drop in interstitial pressure from arteriolar vasomotion and fluctuating intracapillary pressure and hematocrit (Figure 5). This prevents fluid return to the interstitial space.

Lymph is propelled centrally by Traube-Hering-induced movement of surrounding tissues and by contraction of the lymphatic endothelial cells (Figure 6). Vascular distension may be the stimulus that releases prostaglandin H_2 and thromboxane that act as mediators in lymphatic vasomotion.[81,82] Lymph endothelial cells contain actin or actomyosin filaments that are capable of causing the cell to contract.[79(p.25)] Lymph vessels have been shown to demonstrate spontaneous contractions varying from 1 to 30 cycles per minute.[79,83-91] Olszewski[84] reported spontaneous lymphatic vascular contractility at a rate of 1 to 9 (ave. 4) cycles per minute that was independent of arterial pulse rate, respiration, and body movements.

Lymphatic vessels proximal to the terminal lymphatic consist of a series of individual units, lymphangions (Figure 4). A lymphangion is that portion of a lymphatic vessel between two adjacent one-way valves. The presence of valves in the lymphatic vessels and the low resistance along this pathway, insures that any volume decrease of the end lymphatics must occur because of the displacement of fluid centrally.[79(p.58)] Each lymphangion is also capable of spontaneous independent contractility. The pacemaker for contraction appears to be located in the lymphangion wall just proximal to the valve.[85] Although lymphangions may contract randomly, they function more efficiently when contracting synchronously. Lymphatic vessels tend to develop synchronous activity easily.[71] Again, Traube-Hering-driven entrainment probably insures optimal efficiency of this aspect of the mechanism.

The venous capacitor and return: Of the fluid entering the interstitial space from the capillary bed, 10 to 20% returns to the general circulation via the lymphatic system, but the majority, 80 to 90%, re-enters the capillary bed and exits the region via the venous system.

The venules (Figure 4) are of relatively greater diameter, with thinner muscular walIs, than arterioles. They are innervated by the sympathetic nervous system. Their walls can contract and relax, thereby contributing greatly to capacitance of the vascular system and the regulation of tissue perfusion, [14(p.111),72] and providing a physical mechanism for the observed frequency modulation of the THM waveforms.[9,52] The venules (post-capillary resistance vessels) help to regulate capillary hydrostatic pressure and thereby effect fluid exchange in the capillaries.[14(p.118)] As precapillary arterioles constrict, there is a resultant intracapillary hematocrit decrease and a drop in intracapillary pressure to the level of adjacent venules facilitating reabsorption from the interstitium.[65] It is probable that post-capillary resistance control lies in the larger venules up to 300 micrometers in diameter.[14(p.127)]

The thin walled veins act as a capacitance system, holding up to 80% of systemic blood.[14(p.150)] Reflex changes in sympathetic tone affect the caliber of the veins and, thereby, the size of the venous capacitor as well as, to some degree, the compliance of their walls.14(p.124) Resultant changes in the venous capacitance will impact both the nature (selection of coupled frequencies) and the degree (magnitude of the coupling) to which frequency modulation contributes to changing the THM waveform and any associated systems.

As the vascular system fluctuates with the Traube-Hering oscillation and, in concert with arterial resistance, the venous capacitor is contracting slowly and regularly.[13,19,92] This fluctuation facilitates fluid movement through the interstitium. It facilitates lymphatic circulation, and it facilitates the return of venous blood to the heart. Vasomotion resulting from the Traube-Hering oscillation accounts for the negative interstitial pressure of Starling's equilibrium. (Figure 3) General anesthesia and certain drugs (calcium channel blockers) disrupt the Traube-Hering oscillation. This results in the development of peripheral edema.[65]

Up to this point we have considered most everything in the context of the Traube-Hering oscillation. The venous system is, however, intimately involved in thermal regulation. Thermal regulation is under control of the sympathetic and parasympathetic components of the autonomic nervous system in concert with renin-angiotensin.[17,26,42,43] It is a manifestation of the Mayer oscillation, the frequency 0.5 to 2.4 cpm (0.008 to 0.04 Hz,[17-19,26,39,40]), and irregularity of which bears a striking similarity to Becker's "slow tide."[27] It consists of a controlled shifting of blood between the compliant splanchnic veins, that typically contain up to 30% of blood volume, and the cutaneous veins.[93]

Conclusion

Comparison of CRI measurements and descriptions of the PRM from the osteopathic literature with along current information about the THM oscillation demonstrates much more than coincidental similarity. It is proposed that there is sufficient evidence to conclude that the Traube-Hering, baroreflex, oscillation is the Sutherland wave, or "fast tide" of the CRI, and that the Mayer, thermal reflex, oscillation is the "slow tide" of the CRI described by Becker. It follows therefore that the PRM can be logically explained in the context of the THM oscillation and associated physiology and biochemistry. Utilizing the THM to understand the PRM offers a holistic model. It unites the CNS with every cell in the body through the sympathetic and parasympathetic branches of the autonomic nervous system and the cardiovascular system.

The floor of the fourth ventricle provides the frequencies, 5.4-10.2 cpm (0.09-0.17 Hz. fast tide) and 0.5-2.4 cpm (0.008-0.04 Hz, slow tide). The THM oscillation synchronized with the metabolic requirements of individual brain cells provides, at least in part, for motion of the CNS that, in turn, drives the circulation of the CSF.

The PRM, however, must include more than just an oscillation of the CNS. It is a total body phenomenon, and is proposed to occur as follows. The heart, under the central influence of the brainstem, beats with a rhythm, the frequency of which fluctuates at the component frequencies of the THM. It pumps blood that arrives in ***all*** of the capillary beds in the body via arteries and arterioles whose walls are contracting at those same frequen- ➔

cies. Blood pressure, capillary blood flow rate, capillary hematocrit, and venous capacitance are all oscillating at these frequencies.

The THM oscillation is less constrained as the blood passes through the capillaries and enters the venous system than in the thicker walled arterial system. The capacitance of these thin walled vessels allows for significantly greater volume fluctuations with proportionate displacement of adjacent structures. The arteriolar and venular vasomotion and blood pressure and hematocrit fluctuation that results from the THM oscillation aids in the distribution, and mixing of extravascular fluids and facilitates, mechanically, the passage of fluid through capillary and lymphatic walls.

Locally, the metabolism of the individual cell, regulated by the respiratory activity of its mitochondria, is oscillating independently, with essentially the same frequency as the Traube-Hering component of the THM. Cellular respiration regulates local arteriolar tone. Oscillating hematocrit and blood flow velocity, in turn results in oscillating shear forces effecting the vascular epithelium with resultant modulation of NO synthesis. Increasing NO liberation results in local vasodilatation and inhibition of mitochondrial activity.

Capillary hematocrit, and consequently Starling's equilibrium, fluctuates under this influence. Cellular metabolic exchange occurs within the interstitial gel medium. Fluid, that will not move as freely through this gel as it can in a purely liquid medium, is pumped in and out of the intravascular compartment and through the interstitium by the oscillating pressures. The central THM blood flow and pressure oscillations act to entrain the local metabolically-induced oscillations.

Fluid, proteins and particulate matter, not returned to the arteriovenous system are removed from the interstitium via the lymphatics. End lymphatic filling and fluid transport through subsequent lymphangions is subject to spontaneous vascular contractility. Again, efficiency of the system is enhanced through entrainment by the central THM oscillation.

Fluid returned to the capillaries is transported back to the heart through the thin walled veins. The capacitance of the veins permits variable sequestration of blood in the periphery. Oscillation of the venous system at the rate of the Traube-Hering oscillation facilitates efficiency of blood return to the heart. Oscillation at the Mayer frequency, specifically between the splanchnic and cutaneous veins, maintains body core temperature.

The coordinating effect upon the lymphatic and venous systems by the Traube-Hering mediated oscillation might be looked upon as a "peripheral heart" functioning to efficiently return lymph and blood to the heart. The beating of the heart (70 beats per min) and that of the Traube-Hering "peripheral heart" (8 cycles per min) can be seen in Figure 1.

Thus, local and central control mechanisms act synergistically to satisfy the metabolic demands of the peripheral tissues. Locally, the activity of the musculature of the vascular bed is modified and integrated by changes in the composition of the extracellular fluid. Neural control is exercised via specialized sensory endings of peripheral afferent cells within the integrative centers of the central nervous system. Response occurs to varying levels of oxygen, carbon dioxide and hydrogen ion concentration and temperature of the blood and extracellular fluid. Or as Magoun proposed of the PRM, the THM oscillation facilitates "dynamic metabolic interchange in every cell, with each phase of action."

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Book Review

Reviewer: Anthony G. Chila, DO, FAAO

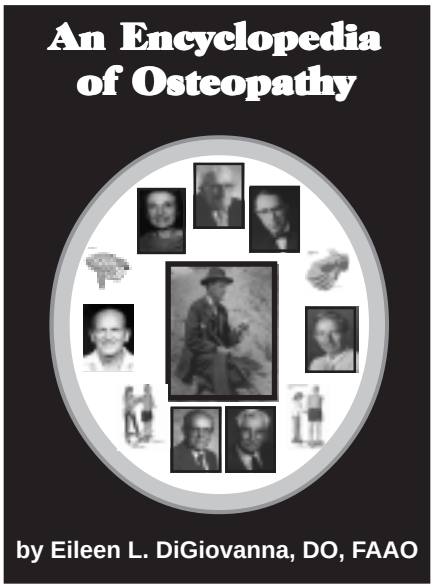
An Encyclopedia of Osteopathy

Author: Eileen L. DiGiovanna, DO, FAAO
Published by: American Academy of Osteopathy; Indianapolis, IN 46268

An encyclopedia is " A book or set of books containing articles on various topics, usually in alphabetical arrangement, covering all branches of knowledge or, less commonly, all aspects of one subject" (The Random House Dictionary of the English Language: 2nd Edition, Unabridged).

The *Glossary of Osteopathic Terminology*, a dictionary of commonly used osteopathic terms, has been available to the osteopathic profession since 1980. The editor and contributors of *An Encyclopedia of Osteopathy* indicate that the present volume "attempts to bring these terms and more, as well as the brief biographies of some outstanding members of the profession, into an encyclopedic format." In considering this admonition, the reader will note an extensive array of personalities who were responsible for shaping osteopathic thought and practice during the 20th Century.

Retrieved archival photographs of selected individuals enhance such information. At the same time, then, attention to any given page may be providing definition of a term(s) interspersed with a biographical vignette of an individual(s) whose contribution was significant. As an example, page 48 in this encyclopedia presents the following: Halladay Flexible Spines; Halladay, H. Virgil, DO; Hannibal, Missouri; Harakal, John H., DO; Hazzard, Charles, DO; Health Policy Fellowship. Perusing and utilizing this information enables the osteopathic practitioner to learn



about Doctor Halladay and to appreciate his contribution to the study of regional, inter-segmental and total spinal motion through the use of flexible spines. It is also revealed that the use of such spines was discontinued because the chemical preservation formula contained arsenic and mercury. To learn that Andrew Taylor Still and his son Harry practiced in Hannibal, Missouri for six months in 1884-85 is to be reminded of Still's references to the years of development of his theory and practice of osteopathy prior to the opening of the American School of Osteopathy in 1892. This town is also associated with the author Samuel L. Clemens (Mark Twain), a strong supporter of osteopathy. Doctor John Harakal's work with the Cranial Academy, Sutherland Cranial Teaching Foundation, Educational Council on Osteopathic Principles and its Glossary Revision Committee is recalled. The influence of Doctor Charles Hazzard's texts on the derivation of Still Techniques leads into the contemporary scene. The Health Policy Fellowship, a year-long certificate program offered at Ohio University and Michigan State University, promotes professional leadership and/or influence in such diverse areas as the health professions work force, economics, managed care, and telecommunications/telemedicine.

This first edition of *An Encyclopedia of Osteopathy* does demonstrate very well the attainment of its goals. Having done this, it is apparent that ongoing effort will be necessary to refine the scope of its complementary relationship to the *Glossary of Osteopathic Terminology***.** The osteopathic profession will benefit from such continued effort by having access to "a book or set of books containing articles on various topics, usually in alphabetical arrangement, covering all aspects of one subject."❐

The 108 page Encylopedia of Osteopathy can be ordered through the American Academy of Osteopathy: Phone: 317/879-1881, Fax: 317/879-0563 or visit the AAO's Web site at: www.academyofosteopathy.org)

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Elsewhere in Print

Cibulka MT. Understanding sacroiliac joint movement as a guide to the management of a patient with unilateral low back pain. Manual Therapy (2002) 7(4), 215-221

The sacroiliac joint continues to be one of the most misunderstood joints in the body. It is a synovial joint and as such it is subjected to the same inflammatory and infectious conditions that affect other synovial joints (Bernard & Cassady 1991). Its deep location, limited movement and irregular anatomy add to the misunderstanding of this joint. Although the sacroiliac joint is often considered in low back pain, its importance remains controversial. No less than eight different sacroiliac joint movements and eight different types of sacroiliac joint dysfunction have been described by various authors (Mitchell et al. 1979; Beal 1982; Woerman 1982; Aitken 1986; Wells 1986; Bernard & Kirkaldy-Willis 1987; Lee 1989; Oldrieve 1996, 1998; Shaw 1992). Thus, it is no wonder that physiotherapists have difficulty understanding this joint. Consequently, a poor or confused understanding of how the sacroiliac joint moves will not assist the physiotherapist trying to determine whether sacroiliac joint dysfunction is present or not.

Zumpano MP, Jagos CS, Hartwell-Ford, S. A Cadaveric Survey Exploring the Variation, Prevalence, Sex Bias, and Tissue Type of the Soft-Tissue Bridge Between Rectus Capitis Posterior Minor and the Posterior Atlanto-occipital Membrane. JNMS: Journal of the Neuromusculoskeletal System, 10:133-140, 2002

The purpose of this investigation was to determine the variation, prevalence, tissue type, and sex bias in the soft-tissue bridge between rectus capitis posterior minor (RCPMi) and the posterior atlanto-occipital membrane (PAO). Seventy-five cadavers (27 females and 48 males) were surveyed using a traditional layering dissection of the posterior cervical musculature. When RCPMi was revealed, its superior attachment was detached and the muscle was reflected inferiorly to determine if it was attached to the underlying PAO. If a soft-tissue bridge was identified, the fibers found within the bridge were classified by visual inspection into three categories: tendon-like, muscle-like, and fascia-like. A fourth category of no attachment was also noted. These results show that RCPMi was present bilaterally in 93% of all cadavers surveyed (89% of the female cadavers and 96% of the male cadavers). On the right side, a soft-tissue bridge was present 67% in males and 78% in females. On the left side, the soft-tissue bridge was present in 69% of the males and in 82% of the females. The number of male cadavers possessing tendon fibers in a soft-tissue bridge was 56% on the right side and 55% on the left side. In females, the number of cadavers possessing tendon fibers in a soft-tissue bridge was 44% on the right side and 64% on the left side. In males, muscle fibers were present in the soft-tissue bridge in 34% of the cadavers on the right side and in 36% on the left. In females, muscle fibers were found in the soft-tissue bridge in 43% of the cadavers on the right and in 36% on the left. A Pearson Chi-square test ($p<.05$) was performed to determine: 1) if gender was associated with the presence of the soft-tissue bridge and 2) if a fiber type within a soft-tissue bridge, as denoted by the authors, was associated with gender. The results of the Chi-square analyses indicated that there were no significant associations of gender in either test.

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