THE AAO
FORUM FOR OSTEOPATHIC THOUGHT
JOURNAL
Official Publication of the American Academy of Osteopathy®
TRADITION SHAPES THE FUTURE • VOLUME 14 NUMBER 3 SEPTEMBER 2004

Osteopathic Medical Students Author OMM Case Studies

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.

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.

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

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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.

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TRADITION SHAPES THE FUTURE • VOLUME 14 NUMBER 3 SEPTEMBER 2004

A PEER-REVIEWED JOURNAL

The Mission of the American Academy of Osteopathy® is to teach, advocate, and research the science, art and philosophy of osteopathic medicine, emphasizing the integration of osteopathic principles, practices and manipulative treatment in patient care.

IN THIS ISSUE:

AAO Calendar of Courses

2004

September

30 - Oct 2 — *Emotional Diagnosis and Release (Barral Approach)*
Osteopathic Center for Children, San Diego, CA

October

3- 5 — *Unlocking the Cranial Sutures (The Face)*
Osteopathic Center for Children, San Diego, CA

November

6 — *Modifying Delivery of OMT in an Allopathic Environment*
San Francisco, CA

7-11 — *AOA Convention*
AAO Program: OMT for Cervical, Lumbar and Pelvic Pain Syndromes: A Study in Applied Anatomy
Karen M. Steele, DO, FAAO, Program Chair
San Francisco, CA

12-14 — *Prolotherapy: Below the Diaphragm*
UNECOM, Biddeford, ME

December

4-5 — *Facilitated Positional Release*
NSUCOM; Ft. Lauderdale, FL

2005

January

28-30 — *Winter OMT Update: Application of Osteopathic Concepts in Clinical Medicine plus Preparation for Certifying Boards*
TUCOM-NV; Henderson, NV

February

18-20 — *Clinical Applications of Muscle Energy in Primary Care*
Midwestern University/AZCOM; Glendale, AZ

March

13-16 — *Advanced Visceral Manipulation: Visceral Approach to Cranial and Peripheral Nerve Dysfunction*
Featuring: Jean-Pierre Barral, DO, MROF
Reno /Lake Tahoe, NV

16-20 — *2005 Annual Convocation: The Hand: The Instrument of Our Distinction*
Charles J. Smutny, DO, FAAO, Program Chair
Reno /Lake Tahoe, NV

April

9-10 — *Dr. Fulford's Basic Percussion: A Systematic Approach to the Whole Body*
Midwestern University/CCOM; Chicago, IL

May

13-15 — *Prolotherapy: Above the Diaphragm*
UNECOM; Biddeford, ME

June

17-19 — *Visceral Approach to Cardiopulmonary Dysfunction*
UNECOM; Biddeford, ME

July

29-31 — *Muscle Energy: Three Visions*
Midwestern University/CCOM; Chicago, IL

August

19-22 — *15th Annual OMT Update: Application of Osteopathic Concepts in Clinical Medicine plus Preparation for Certifying Boards*
The Contemporary at Walt Disney World®
Buena Vista, FL

September

16-18 — *Clinical Application of Principles of Ligamentous Articular Strain in Primary Care*
UMDNJ-SOM; Stratford, NJ

October

22 — *Rapid OMT: Increase Your Reimbursement in an Ambulatory Setting*
Orlando, FL

23-27 — *AOA Unified Convention:*
AAO Program: Osteopathy in the Specialties: A Hands-on Approach
Kenneth L. Lossing, DO, Program Chair
Orlando, FL

November

11-13 — *Prolotherapy: Below the Diaphragm*
UNECOM; Biddeford, ME

December

2-4 — *Lymphatic Approach to the Viscera*
AZCOM; Glendale, AZ

View from the Pyramids

Anthony G. Chila

What is Osteopathy?

Andrew Taylor Still offered his answer to this question in *The Philosophy and Mechanical Principles of Osteopathy* (p. 18):

"What is osteopathy? It is a scientific knowledge of anatomy and physiology in the hands of a person of intelligence and skill, who can apply that knowledge to the use of man when sick or wounded by strains, shocks, falls, or mechanical derangement or injury of any kind to the body. An up-to-date osteopath must have a masterful knowledge of anatomy and physiology. He must have brains in osteopathic surgery, osteopathic obstetrics, and osteopathic practice, curing diseases by skillful readjustment of the parts of the body that have been deranged by strains, falls, or any other cause that may have removed even a minute nerve from the normal, although not more than the thousandth of an inch. He sees cause in a slight anatomical deviation for the beginning of disease. Osteopathy means a knowledge of the anatomy of the head, face, neck, thorax, abdomen, pelvis, and limbs, and a knowledge why health prevails in all cases of perfect normality of all parts of the body. Osteopathy means a studious application of the best mental talents at the command of the man or woman that would hold a place in the profession."

With perhaps slight rephrasing, the same question occupied the attention of the recent meeting of the Osteopathic International Alliance (OIA). The Second Invitational Conference for the OIA was held June 5-6, 2004 at Toronto, Canada. Seven countries (Canada, England, France, Germany, New Zealand, Switzerland and the United States were represented by osteopathic physicians, non-physician osteopaths and other health professionals. This organization is educational and not-for-profit, seeking to assemble national organizations of osteopathic physicians as well as non-physician osteopaths. Among its intentions are: facilitation of communication and sharing of ideas and expertise; assisting foreign nations in the development of standards of training and legal aspects of training and practice. At present, three levels of membership are contemplated: Full membership for organizations governmentally recognized and representing the whole of licensed or legally recognized osteopathic physicians or non-physician osteopaths; Associate membership for governmentally recognized authorities overseeing the regulation of osteopathic medicine as well as emerging non-physician osteopathic groups working toward governmental recognition; Partner membership for supporting organizations contributing to advancing the work of the OIA. The outcome of the OIA effort in addressing its currently mapped functions and processes remains to be seen. At the same time, future challenges will include issues of governance, vote, assembly, delegation. Good will in the continuing activities of this alliance will surely help facilitate its desire to become an organization of organizations. (For more complete reporting of this meeting, see *The DO*, August 2004: 35-41).

"There are other fields of research. May my grand army march on. If we cannot have the pure osteopathic principles taught in our schools, I hope the faithful will rally around the flag and we will build an international school that will offer no compromise unless it is the golden truth. D.O. means DIG ON."

A. T. Still, MD, DO

Even as attendees wrestled with these questions at Toronto in 2004, it can be shown that Andrew Taylor Still was sufficiently prescient to recognize the necessity for daring exploration. In *Philosophy of Osteopathy* (p. 24), he writes: "I felt that I must anchor my boat to living truths and follow them wheresoever they might drift. Thus I launched my boat many years ago on the open seas, fearlessly, and have never found a wave of scorn nor abuse that truth could not eat, and do well on." In a document dated August 1, 1915 and signed "A.T.Still", he indicated that: "There are other fields of research. May my grand army march on. If we cannot have the pure osteopathic principles taught in our schools, I hope the faithful will rally around the flag and we will build an international school that will offer no compromise unless it is the golden truth. D.O. means DIG ON."❐

Contributors

Kenneth E. Nelson, Nicette Sergueef, Thomas Glonek. Cranial Manipulation Induces Sequential Changes in Blood Flow Velocity on Demand. In previous studies, the authors have demonstrated correspondence of the palpable Cranial Rhythmic Impulse (CRI) to a low frequency component of the Traube-Hering-Mayer (THM) oscillation. They have also demonstrated that cranial manipulation induces change in the THM oscillation when compared to sham treatment. This study continues their work, demonstrating that these changes can be repeatedly induced on demand. **(p. 15)**

Tammy Gregg and Stuart F. Williams. Chronic Fatigue Syndrome: The Misunderstood Disease. The authors present a case study exploring this complicated and often misunderstood disease. They emphasize that no one, simple solution can be offered for treatment. This point is made by acknowledging that this entity should be understood as a conglomerate of multiple research strategies. In focusing on the value of Osteopathic Manipulative Treatment, they appear to be addressing this entity as a "neuroendocrineimmune dysfunction syndrome". **(p. 20)**

Paula Rossi and Jerry L. Dickey. Chronic Pelvic Pain Syndrome. The authors present a case study of a common syndrome occurring in males aged 25-45 years. An acknowledgment of the current National Institutes of Health (NIH) classification of prostatitis syndromes is the springboard for their appeal to use the principles of diagnosis and treatment espoused by Andrew Taylor Still. In explaining their management, they demonstrate comprehensive integration of diagnosis and treatment. **(p. 23)**

Josalyn M. Mann and Karen M. Steele. The Effect of Osteopathic Manipulative Treatment on Gait Disturbance in Multiple Sclerosis Patients. The authors examine 5 patients diagnosed as having Multiple Sclerosis (MS) and able to ambulate without the aid of a crutch, walker or cane. Their study focused on gait instability and incorporated the use of Osteopathic Manipulative Treatment. Treatment was individualized for each patient, allowing each patient to serve as his/her own control. Sham treatment was not included. The mixed results reported may reflect interexaminer reliability issues, possibly requiring more training/agreement on criteria. **(p. 27)**

Sean McMillan, William T. Crow, Charlotte H. Greene. Lymphatic Manipulative Pump Research: A Brief Review of Literature. The authors seek to explore research validation of a commonly performed manipulative procedure, noting the lack of clinical studies. Through the use of Internet search engines, they selected articles related to (a) immune system responses and (b) changes in lymph flow rates. In their discussion, they incorporate a plea for the generation of more studies to acquire statistical support for the use of this procedure. **(p. 32)**

Regular Features

DIG ON. Parallel and Distinct. The original thought of Andrew Taylor Still continues to be influential in shaping the character of the practice of medicine. In his writing, he acknowledges "standard authors", but cites relatively few sources. In this portrayal, the parallel lives of S. Weir Mitchell (1829-1914) and Andrew Taylor Still (1828-1917) provide an opportunity to view the distinctive contribution of each in moving from the 19th into the 20th centuries. **(p. 8)**

From the Archives. The American Academy of Osteopathy® has identified with the Pyramids since 1992. History certainly indicates the presence of such structures for a much longer period of time. Perrin T. Wilson, DO describes his encounter with the Pyramid of Cheops in 1931 (*Osteopathic Magazine*; March 1931, 25-26). Incidentally noted in this publication: the office of the American Osteopathic Association was located at 430 N. Michigan Ave., Chicago, IL. [Courtesy of Christian Fossum, DO (UK)]. **(p. 10)**

Book Reviews. *Getting at the Root: Treating the Deepest Source of Disease.* Andrew Lange, a Naturopath and Homeopath, offers a vision for the incorporation of modern allopathic and vitalistic approaches. *Physicians of the Soul*. Robert M. May offers a twentieth anniversary analysis of teachings of Lao Tzu, Moses, Jesus, Gautama Buddha, Krishna, Muhammad and the Native American holy woman White Buffalo Woman. *The Medium, the Mystic, and the Physicist.* A re-release of this classic by psychologist Lawrence Le Shan is accompanied by his new preface and his conviction that his 1966 thesis about parapsychology remains intact today. **(p. 34)**

Elsewhere in Print. *New Thinking About Migraine* (Roger Cady, MD) offers a very contemporary perspective on medical management of this problem. An osteopathic perspective about migraine headache would consider a rationale for manipulative intervention in any of several phases of manifestation of this problem. The thought process expressed in *Foundations for Osteopathic Medicine* (2nd Ed; 439, 679) would clearly provide a parallel and distinct contribution to patient care. A thoughtful collation of both presentations would offer a very comprehensive management scheme for those patients afflicted with this problem. **(p. 35)**

CME Credit. In response to reader requests, *AAOJ* will offer CME Credit to readers completing the enclosed quiz. At this time, 1 Hour II-B Credit will be offered, with request for upgrade as AAOJ qualifications are reviewed by the American Osteopathic Association.

Component Societies' CME Calendar

and other Osteopathic Affiliated Organizations

October 1-3, 2004
Arizona Clinical CounterStrain
Arizona Academy of Osteopathy
Faculty: Edward Goering, DO and Harmon Myers, DO
OMM Lab at MWU/AZCOM
Hours: 13 Category 1A (anticipated)
Contact: Wm. Devine, DO
623/572-3350 or 623/572-3351

October 8-10, 2004
NeuroFasical Release Conference West
Faculty: Stephen Davidson, DO
Arizona Academy of Osteopathy
Phoenix, AZ
Hours: 24 Category 1A anticipated
Contact: Wm. Devine, DO
623/572-3350 or 623/572-3351

October 16-17, 2004
Advanced NeuroFascial Release
Arizona Academy of Osteopathy
Faculty: Stephen Davidson, DO
OMM Lab at MWU/AZCOM
Hours: 16 Category 1A anticipated
Contact: Wm. Devine, DO
623/572-3350 or 623/572-3351

October 17-20, 2004
Biodynamics Phase III: The Long Tide and the Dura
Franconia, NH
CME: 21.5 Category 1A (anticipated)
Contact: James Jealous, DO
207/778-9847

November 1-4, 2004
Biodynamics Phase V: The Embroyological Face
Franconia, NH
CME: 21 Category 1A (anticipated)
Contact: James Jealous, DO
207/778-9847

November 7-10, 2004
Biodynamics Phase III: The Long Tide and the Dura
Franconia, NH
CME: 21.5 Category 1A (anticipated)
Contact: James Jealous, DO
207/778-9847

November 14-17, 2004
Biodynamics Phase IV: The Midline
Franconia, NH
CME: 23 Category 1A (anticipated)
Contact: James Jealous, DO
207/778-9847

December 3-5, 2004
23rd Annual Winter Update
Indiana Osteopathic Association
Indianapolis, IN
CME: 20 Category 1A (anticipated)
Contact: IAO
317/926-3009

Dig On

Anthony G. Chila

Parallel and Distinct

Silas Weir Mitchell (1829-1914) was the leading American neurologist of his time. A graduate of the Jefferson Medical College (1850), he studied in Paris and was influenced by Claude Bernard. His life span, contemporary with Andrew Taylor Still (1828-1917), provides an opportunity to gain insight into the character of 19th Century American medicine, and the contribution of each.

Fielding H. Garrison's *An Introduction to the History of Medicine* (4th Edition, 1929, p. 512) notes that "The advancement of scientific medicine in the second half of the 19th century was characterized by the introduction of a biological or evolutionary view of morphology and physiology, out of which came the sciences of cellular pathology, bacteriology, and parasitology, new modes of seeing disease and its causes, which had in them the germ of novel methods of treatment by means of sera and vaccines." As will be shown, Mitchell and Still reflected these attributes of Garrison's description from different perspectives.

Among his many contributions, Mitchell is noted for: pointing out the coordinating functions of the cerebellum; demonstrating that the knee-jerk can be reinforced by sensory stimulation; studying gunshot and other injuries of peripheral nerves, including the earliest distinct accounts of ascending neuritis; providing the first descriptions of causalgia, red neuralgia, postparalytic chorea; being the first to emphasize the importance of eye strain as a cause of headache; introducing the "rest cure" (Weir Mitchell) treatment of nervous disease; studying the effect of meteorological changes on traumatic neuralgias, particularly in old amputation stumps; contributing original studies of phantom limbs, disorders of sleep and freezing of his own ulnar nerve.

In 1892, contributing to the history of medicine, Mitchell published an accurate history of instrumental precision. In the same year, Still copyrighted *The Philosophy and Mechanical Principles of Osteopathy* and the American School of Osteopathy came into existence. The sophisticated Philadelphian (Mitchell) and the Frontier Doctor (Still) were, from different perspectives, anticipating medical progress in the 20th Century. For Still, this was expressed in *The Philosophy and Mechanical Principles of Osteopathy* (pp. 11-12), as Demand for Progress:

"The twentieth century demands that advance in the healing arts should be one of the leading objects of the day and generation, because of the truth that the advancement in that profession has not been in line with other professions. … This work is written for the student of osteopathy; written to assist him to think before he acts, to reason for and hunt the cause in all cases before he treats; for on his ability to find the cause depends his success in relieving and curing the afflicted. With the posted osteopath all the old systems of treating diseases are relegated to the waste basket and marked 'Obsolete'. He must remember that the American School of Osteopathy does not teach him to cure by drugs, but to adjust deranged systems from a false condition to the truly normal, that blood may reach the affected parts and relieve by the powers that belong to pure blood. The osteopath must remember that his first lesson is anatomy, his last lesson is anatomy, and all his lessons are anatomy."

This portrayal clearly indicates that these two original thinkers overlapped in their life spans and made significant original contribution in accord with the environment of 19th Century American medicine. Each proceeded from a distinctly different base of thought. We do not have evidence to suggest that each was aware of the other's work. It is only conjecture to ask what the character of American medical education and practice might have been if their respective effort had been given the opportunity for mutual sharing and elaboration of thought … Osteopathic Medicine and Surgery?❐

Winter OMT Update

"Application of Osteopathic Concepts in Clinical Medicine"

plus Preparation for Certifying Boards

Henderson, Nevada • January 28-30, 2005

Ann L. Habenicht, DO, FAAO, Program Chair

"LAST OMT UPDATE
before May 1, 2005
AOBNMM Application Deadline"

Course location:
Nevada's NEW School of Osteopathic Medicine

The program anticipates being approved for 20 hours of AOA Category 1-A CME credit pending approval by the AOA CCME.

Course Objectives: Level III

This Academy program was designed to meet the needs of the physician desiring the following:

- OMT Review - hands-on experience and troubleshooting
- Integration of OMT in treatment of clinical cases
- Preparation for OMT practical portions of certifying boards
- Preparation for AOBNMM (American Osteopathic Board of Neuromusculoskeletal Medicine) certifying/licensing boards
- Information on CODING for manipulative procedures
- Good review with relaxation and family time

***Prerequisites*:** The participant should have a basic understanding of functional anatomy and (1) Level II course.

Program Time Table:

Friday, January 28 ..... 8:00 am - 5:30 pm
Saturday, January 29 ..... 8:00 am – 5:30 pm
Sunday, January 30 ..... 8:00 am –12:30 pm
(Friday & Saturday include (2) 15 minute breaks and a (1) hour lunch; Sunday includes a 30 minute break.)

Course Location:

TUCOM/Nevada
874 American Pacific Dr.
Henderson, NV 89014

Hotel Accommodations:

Holiday Inn Express Hotel & Suites
441 Astaire Drive, Henderson, NV 89014 • 702/990-2323
1.3 Miles northwest from course location

Hampton Inn & Suites Henderson
421 Astaire Drive, Henerson, NV 89014 • 702/992-9292
1.3 Miles northwest from course location

For other hotels in area, check out the internet:
www.orbitz.com or www.hotels.com

Registration Form
Winter OMT Update
January 28-30, 2005

Full Name _____

Nickname for Badge _____

Street Address _____

_____

City _____ State_____ Zip_____

Office phone # _____

Fax #: _____

E-mail: _____

By releasing your Fax number/E-mail address, you have given the AAO permission to send marketing information regarding courses via the Fax/E-mail.

AOA # _____ College/Yr Graduated _____

I need AAFP credit ❒ I require a vegetarian meal ❒

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

Registration Rates

	On or Before 12/28/05	After 12/28/05
AAO Member	$550	$650
Intern/Resident/Student	$450	$550
AAO Non-Member	$765	$865

(Non-members – see membership application on page 23)

AAO accepts Visa or Mastercard

Credit Card # _____

Cardholder's Name _____

Date of Expiration _____

Signature _____

From the Archives

Osteopathy with a Background of 5,000 Years

Perrin T. Wilson
Osteopathic Magazine, March 1931, pg 25-26.

One glorious moonlight night, I started up the left hand corner of the Pyramid of Cheops, seen in the background of the picture. The Pyramid of Cheops is a solid mass of masonry and contains approximately 2,300,000 blocks of limestone – each weighing over two tons on the average. Its base covers 13 acres and its height is approximately 451 feet.

At first, I scrambled up the enormous blocks of stone quite nonchalantly, but before I had climbed 50 feet I was affronted with a particularly high block which made me stop and wait for my Arab guide to give assistance. Even if the 4-foot step had not stopped me, I would soon have had to rest to recover my breath. Seven times before I reached the top it was necessary to stop until my heart slowed down.

After pulling myself over the last stone, I lay flat on my back with my arms spread out waiting to get enough breath to move. Suddenly as I lay there 451 feet above the desert looking straight up into a soul-filling sky it flashed across me that even 5,000 years ago the fundamental law on which osteopathy depends worked for those poor hundreds of thousands of slaves who cut the stones from a quarry 12 miles away, moved them to

the edge of the desert and erected that pyramidal pile of limestone to house the dead body of their king. Back in those ancient times nature strove to rid the body of the various diseases which attacked it. I could usualize those poor overburdened workers straining and pulling at stones, which seemed too cumbersome for human hands to move. I could see many strained backs in the area where the nerves go to the heart, the lungs, the liver, the kidney, or other of those vital centers along the spine, which when irritated, reduce the recuperative powers of the organ. What a difference an osteopathic physician would have made in the comfort of those lives!

My breath was again normal – I stirred myself, arose and walked around on the top looking first off across the desert to see the twinkling light of a camping party, then looking down close at the City of the Dead, and again towards the City of Cairo and the River Nile. Reluctantly I left the calm, the beauty, and the charm of that setting and slowly clambered back to earth. Never will that night be forgotten; nor will I ever forget to be thankful for the benefits of osteopathy.❐

Letters to the Editor

Dear Editor:

Scientific validation of a given observation occurs when two independent research laboratories demonstrate the same findings, particularly if the methodologies are different. The science of osteopathy in the cranial field has been significantly advanced with the demonstration of the effect of cranial manipulation upon intracranial fluid dynamics (Moskalenko and Kravchenko, *AAOJ,* Summer 2004). The authors have shown that cranial manipulation, venous sinus technique, specifically increases the spectral power of slow fluctuations in intracranial fluid content with a rate in normal individuals between 7 and 9 cycles per minute (0.115-0.150 Hz), as measured by transcranial bio-impedance (see Figure on page 12). These data are of great significance because they corroborate our findings that cranial manipulation, consisting of equilibration of the global cranial motion pattern and the cranio-cervical junction, significantly increases the low frequency, 0.1-0.15 Hz, oscillation in blood flow velocity as measured by laser Doppler flowmetry (see Table on page 13).[1]

It must, however, be noted that Moskalenko and Kravchenko state that changes in intracranial fluid content are driven by vasomotion, that they state is of uniquely intracranial in origin, while our findings are measurement of peripheral vasomotion, the Traube-Hering oscillation, a manifestation, at least in part, of baroreflex physiology. Moskalenko and Kravchenko state that the former represents the primary respiratory mechanism (PRM), while the latter does not. We are not in agreement with this particular opinion.

The presence of a 0.1-0.15 Hz frequency oscillation is demonstrable in; bloodflow velocity, blood pressure, cardiac rhythm, muscle sympathetic tone and intracranial fluid content.[2-5] These are all arguably related to baroreflex physiology and regulated in part by the vasomotor center in the nucleus of the tractus solitarius (NTS) from its location in the floor of the fourth ventricle.[6] There is a similar frequency vascular oscillation, identified by Vern et al., that occurs independently on a cellular level in the CNS.[7] It results from the oscillation of mitochondrial cytochrome oxidase between its reduced and oxidized state. The mitochondrial activity (cellular respiration) is linked to the vasomotor activity in a complex feedback system. Oscillating systems of similar frequency will become synchronous through entrainment, with the dominant oscillation determining the pace.[8] The dominant oscillation in this instance is most probably the activity of the NTS. To propose that the independent and therefore random cellular oscillations entrain the NTS would be rather like the tail wagging the dog.

It has been said that osteopathy became truly holistic when Sutherland proposed cranial function and dysfunction. It is an error to consider cranial osteopathy as a system that is isolated from the remainder of the body. If osteopathy without cranial osteopathy is a decapitated system, so also cranial osteopathy without consideration of the entire body could be said to be "decorporated."

The Oxford English Dictionary defines *mechanism* as: "The structure or mutual adaptation of parts in a machine, or anything comparable to a machine, whether material or immaterial. (In early use chiefly with reference to natural objects.)" Magoun clearly states of the primary respiratory mechanism: "To summarize, the primary respiratory mechanism is considered to include the innate motility of the central nervous system, which coordinates with the observable fluctuation of the cerebrospinal fluid fluctuation, under the guidance and restraint of the reciprocal tension membranes, to produce motion in the linked craniosacral mechanism, and the two-phase rhythmic cycle throughout the body, which, though minimal, is none the less important. This cycle manifests as the cranial rhythmic impulse and represents a dynamic metabolic interchange in every cell, with each phase of action."[9]

The Traube-Hering oscillation is not the PRM, or even the CRI. It is a component manifestation of the mechanism, and as such is, as is the mitochondrial cytochrome red-ox oscillation identified by Vern et al., a window through which we may peer to study and elucidate the PRM. It is particularly consistent with a holistic theory of medicine that we can observe the therapeutic effect of cranial manipulative procedures not only by transcranial bio-impedance, but also in the peripheral capillaries via laser-Doppler flowmetry. And that in both cases the effect is increase of the power of components with the same spectral frequency.

As has been said of the PRM; "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."[10] This is a mechanism that encompasses the entire body. It is the goal of the physician to identify and eliminate impediments to its function, through the diagnosis and treatment of somatic dysfunction. The component parts of the PRM are most efficient when functioning in synchrony, when the power of these low-frequency oscillations are consequently amplified. This is most likely obtained by entrainment.[11,12]

We applaud Moskalenko and Kravchenko for their excellent work. They have identified that the "source of physical forces for wave phenomena are periodical fluctuations of tone of brain blood vessels, because only blood vessels are provided by contractile elements

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– smooth musculature." But we believe that the intracranial aspect of the PRM is, like the Traube-Hering oscillation, but one component of the entire mechanism. We therefore must express a word of caution. Do not lose sight of the holistic integration of human physiology, the osteopathic big picture.

Respectfully submitted
Kenneth E. Nelson, DO, FAAO, FACOFP
Nicette Sergueef, DO (France)
Thomas Glonek, PhD

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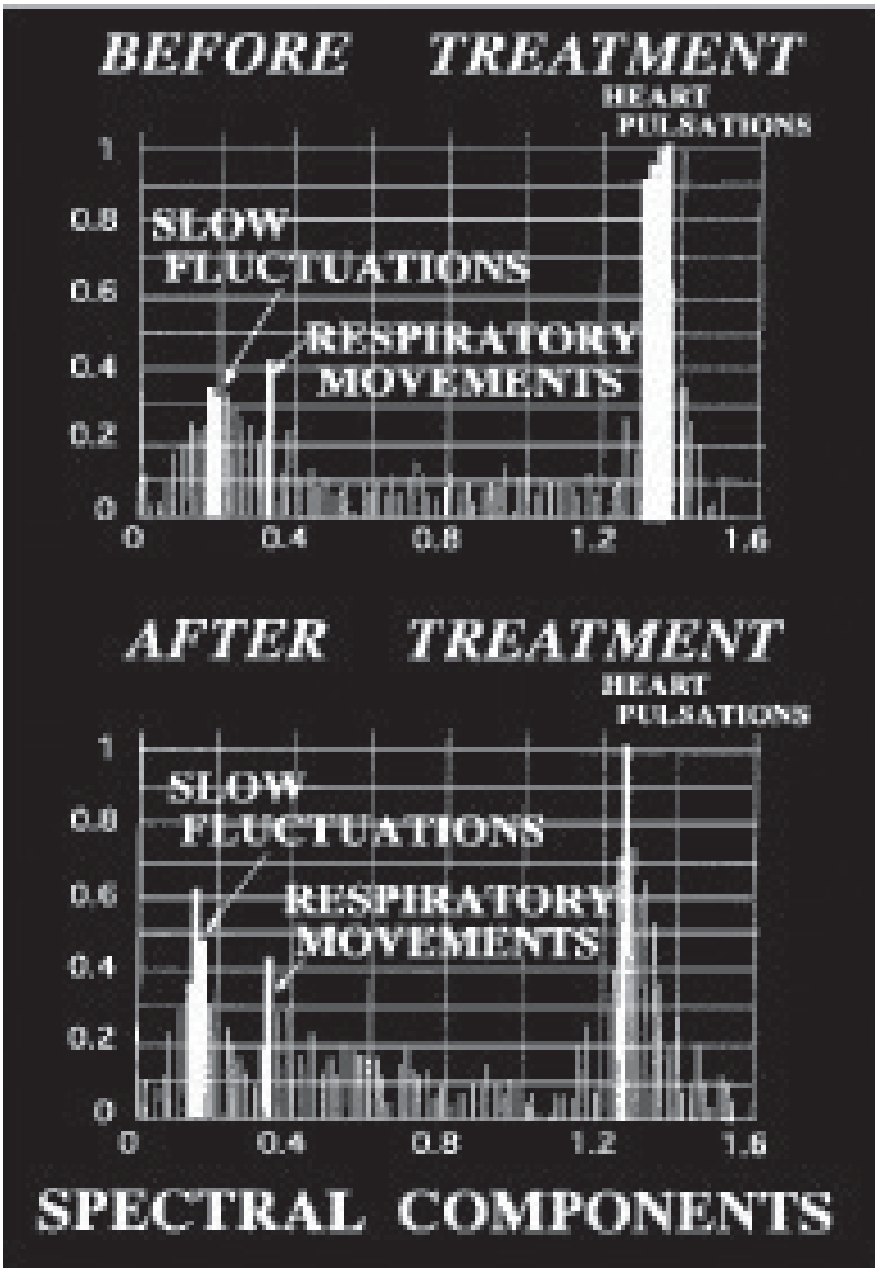

Frequency-somain spectrum from Moskalenko and Kravchenko, The AAO Journal, *Summer 2004. p38.*

Table. Tabulation of frequencies of the various signals (ultra-slow fluctuation, the Mayer wave; slow fluctuation, the Traube-Hering wave; pulmonary respiration; cardiac pulse) measured within blood-flow velocity by laser-Doppler flowmetry. This demonstrates the increased power of the slow fluctuation, Traube-Hering wave and decreased power of the ultra-slow fluctuation, Mayer wave, following cranial manipulation.

Signal	Intervention							
	Palpation Only (N = 13)				Cranial Treatment (N = 10)			
	Doppler Record Segment	Mean Power	Paired Difference (Before – After ± S.D.)	Significance of the Difference	Doppler Record Segment	Mean Power	Paired Difference (Before – After ± S.D.)	Significance of the Difference
0.05-0.08 Hz	Before	42.93	3.36 ± 5.69	0.054	Before	47.79	9.30 ± 5.65	0.001
Meyer	After	39.58			After	38.49		
0.1-0.15 Hz	Before	39.83	-0.27 ± 3.85	0.805	Before	47.40	-3.90 ± 4.40	0.021
Traube-Hering	After	40.10			After	51.30		
0.2-0.3 Hz	Before	27.54	0.34 ± 3.23	0.715	Before	29.72	-0.30 ± 2.89	0.747
Resp	After	27.20			After	30.02		
1-1.5 Hz	Before	37.92	0.78 ± 4.15	0.511	Before	41.11	0.41 ± 4.67	0.788
Cardiac	After	37.14			After	40.70		

Prof. Yuri Moskalenko's response:

Dear Editor:

In reference to the letter concerning our recent paper, we should like first of all to emphasize that we are in full agreement that functioning of the majority of physiological systems is accompanying by slow rhythmic processes, some of them were described a number of decades ago. Indeed, there are famous slow fluctuations of arterial pressure, named Traube-Hering and Mayer waves; slow fluctuations of heart activity: heart rate deviations; local cerebral and peripherical blood flow fluctuations; slow waves of intracranial pressure, which are correlated with brain blood volume, and others. All of these kinds of fluctuations are characterized by similar frequencies. Therefore, comparison of statistical parameters of these fluctuations, using principle of spectral and Fourier analyzing, indicates, that probability of their role in forming of background of PRM are approximately equal. So, basing on such data it is the most difficult to conclude, which kind of fluctuations either intracranial or extracranial nature, play the major role in forming of PRM, as a physiological mechanism, definition of which generally follows from the Oxford English Dictionary and its adaptation for osteopathy, given by Dr. Magoun (see ref. 10)*, as mechanism, responsible for "dynamic interchange in every cell". Dr. Magoun's expression was recently confirmed by investigations of CSF movements between brain ventricles and its surface (see ref. 26).

This was an argument, why in our investigations, idea of which was directed to make clear principles of functioning and structural organization of PRM, we selected the way of multichannel simultaneous recordings of intracranial and extracranial circulatory processes. Physiological background for this methodology follows from our previous investigations with simultaneous recordings of slow fluctuations of intracranial pressure, local cerebral blood flow, local oxygen availability in brain tissue, systemic arterial pressure, heart and respiratory rates, in chronic experiment with animals (rabbits, cats, monkeys … see ref. 23), as well as results of human observation with brain implanted electrodes in patients with neurological

disturbances (see ref. 16 and 20). It was shown, that although all these fluctuations look similar, in every particular experiment or human observation every rhythmic process is characterized by some peculiarities. These peculiarities permit to identify the specifics of every fluctuation in particular experiment.

Simultaneous spectral analyzing of head bio-impedance with fronto-mastoid electrode position, and transcranial dopplerography of basement segment of the Middle Cerebral Artery permits to conclude that the intracranial processes are the major factor, responsible for PRM. However, it does not mean that extracranial factors did not take part in origin of this complicated physiological mechanism. The latter follows from Figure 5 of our paper (see also Figure 1 in the letter to Editor), which demonstrate, that after application of venus sinus technique, increase of slow fluctuations of intracerebral origin and absent similar changes at simultaneously recorded transcranial dopplero-graphy (see ref. 18), although indices of systemic circulatory systems (representing, in particular on Figure 5, by deviations of heart rate) are significantly changed. For more precisely analyzing of the comparative role of extra- and intracranial factors in background of PRM, some functional tests have been used (see ref.18 of our paper). The use of functional tests such as inhalation of gas mixture with 7% CO_2 or 5% O_2 inhalation, Tilt-table and Stookey maneuvers, have shown, that the direction of changes of indices, reflecting slow fluctuations of cerebral and CSF circulation is special for every particular functional test, because every test evoked particular physiological response. These investigations definitely showed, that responses of indices, reflecting slow fluctuations of intracranial nature are sufficiently differ from indices, reflecting slow fluctuations of extracranial circulation.

Thus, all our data support the statement, that waves, which are responsible for PRM, have intracranial origin. It means, that physical forces, which are responsible for skull bone motions, representing PRM, are "generated" inside cranium. However, it does not make cranial osteopathy "decorporated" or general osteopathy "decapitated", because it would be a great mistake to exclude of extracranial component in background of PRM, which is based on dynamic interaction of functions in the united body. This principle is fully applied for control processes in cerebrovascular system and it was described in our paper as synergetic interaction of different control links, including neurogenic mechanism. Of course, this interaction could involve extracranial structures and functions. For example, sympathetic innervation of brain blood vessels have extracranial (Ganglium Stellatum) and intracranial (Locus Coeruleus) origin. Because sympathetic efferent innervation is special for small blood vessels up to precapillar level, of both intracranial and extracranial ones, sympathetic nerve system may be one of linkage, which responsible for similarities of slow fluctuations, recorded by head bioimpedance and laser flowmetry.

We agree with the authors of the letter, that not all problems, connected with fundamental basis of PRM are clear now. By our mind, it looks very useful further investigations of correlation between of slow fluctuations on brain tissue level -oxygen availability fluctuations in brain tissue (see ref. 16), which is correspond to Dr. Vern data, (see ref. 32), with fluctuations, total for all cranial cavity and their correlation with slow fluctuations of extracranial origin, in particular which are recorded by laser flowmetry. The special attention it is reasonable to paid also to investigations of relations between slow fluctuations intracerebral and extracerebral circulations and fluctuations, which are special for other physiological systems, for example, fluctuations of posture control. Such investigations, which should be based on simultaneous recording of number of physiological processes, could provide the most important information, concerning peculiarities of functioning of PRM, as multicomponent and complicated physiological mechanism. It promises the significant future not for only osteopathy but also for other branches of medicine.

We are deeply thankful to Drs. Nelson, Sergueef and Glonek for their valuable remarks, which are important for our future study.

Prof. Yuri Moskalenko, DSci, DO(Hon)
and Dr. T. Kravchenko, MD, PhD, DO

* All references were taken from our paper, published at June (2004) issue of *The AAO Journal*.❐

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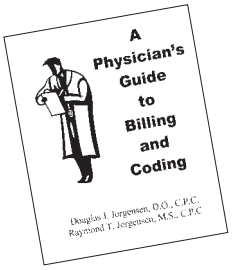

Acting on a recommendation from the Publications Committee, the Board of Trustees has approved the addition of a new publication to the Academy's inventory of resources on osteopathic medicine. The AAO is now the exclusive distributor of this publication within the osteopathic medical profession.

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Cranial Manipulation Induces Sequential Changes in Blood Flow Velocity on Demand

Kenneth E. Nelson, Nicette Sergueef, Thomas Glonek

Abstract

Primary Objective: *To demonstrate that when cranial manipulation is applied as a therapeutic intervention, the dominant, 0.1 Hz frequency, Traube-Hering (TH) component of blood flow velocity that is related to baroreflex activity is specifically amplified. Further, when intervention is stopped, the flowmetry record reflects the change in intervention. In this instance, the timing of cranial treatment depends only upon a pre-established protocol.* **Methods:** *Using laser-Doppler flowmetry to quantify the TH and other components of the blood flow velocity oscillation, we compared flowmetry records of 15 subjects before and immediately following cranial manipulation. The timing of the treatment/non-treatment sequence was established prior to manipulative intervention.* **Results:** *Selected continuous record segments from within treatment and non-treatment portions of the experimental flowmetry records were converted to frequency-domain spectra via a Fourier-transformation (FT). From the FT data, difference spectra were computed by subtracting the spectrum acquired during a non-treatment segment from the spectrum of adjacent treatment-period records. The resultant difference showed that cranial manipulative treatment enhanced the magnitude of the 0.1 Hz component and increased the fundamental heart rate. No other prominent changes with treatment were observed.* **Conclusions:** *Flowmetry shows that cranial manipulation may be used to alter the 0.1 Hz blood flow component of the TH oscillation according to a pre-determined protocol. Thus, cranial manipulation may be used to alter blood flow according to specific interventional directives.*

Key words: *osteopathic medicine, cranial osteopathy, primary respiratory mechanism, cranial rhythmic impulse, cardiovascular system, laser-Doppler flowmetry, Traube-Hering-Mayer oscillation*

Introduction

Using laser Doppler flowmetry, we have demonstrated that the palpable experience of the cranial rhythmic impulse (CRI) corresponds to the low frequency Traube-Hering (TH) component of the Traube-Hering-Mayer (THM) oscillation in blood flow velocity.[1] Further, we have established that cranial manipulation, as compared to sham treatments consisting of cranial palpation only, induces change in the THM oscillation.[2] In this study we show these changes to be repeatedly induced upon demand.

Hypothesis

The application of cranial osteopathic manipulative treatment will have measurable effect upon the dominant 0.1 Hz, TH, component of the THM oscillation and that effect can be repeatedly induced upon demand according to a predetermined schedule.

Methods

Fifteen healthy adult subjects of both sexes (IRB-approved informed consent obtained) participated. Blood flow velocity was measured using the Transonic Laser-Doppler Monitor BLF21 Series. The laser-Doppler probe was placed in the midline upon the forehead of each subject. The subject was allowed to lie quietly upon an OMT table for a 3-minute equilibration period. A baseline blood flow velocity record, of 5 to 7 minutes was then obtained. Following this, 5 to 7 minute periods of cranial manipulation, alternating with 5 to 7 minute periods cranial palpation only, were carried out. The timing of the treatment/non-treatment sequence was established prior to the manipulative intervention. The total recording time for each subject was approximately 35 minutes.

Cranial manipulation was employed utilizing an incitant technique.[3] The physician's fingers were interlaced loosely beneath the occipital squama, and the thumbs were extended along and medially to the occipitomastoid sutures in a hand placement similar to that employed when performing compression of the fourth ventricle. Augmentation of the CRI was induced with a firm pumping action in synchrony with cranial flexion and extension.

Results

All 15 subjects demonstrated similar changes in blood flow velocity in response to cranial manipulation. The examples sited here are the most striking. Figure 1 presents the compressed laser-Doppler-flowmetry, relative blood velocity waveforms, of two subjects treated by cranial manipulation at designated 5 minute (Subject 1) and 7 minute (Subject 2) intervals. The sinusoidal wave is the THM oscillation as it is expressed in terms of blood flow. The heartbeat is not

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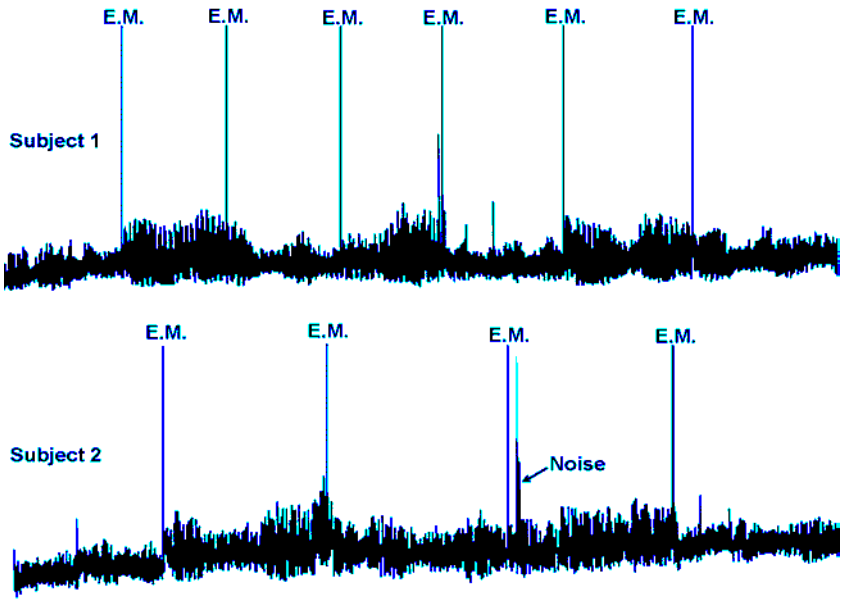


Figure 1: Compressed laser-Doppler-flowmetry, relative blood velocity waveforms, of two subjects treated by cranial manipulation at designed 5 minute (Subject 1) and 7 minute (Subject 2) intervals. Event marks (EM) indicate points in time when cranial manipulation started and stopped.

discernable at this degree of record compression and appears as record noise. The vertical lines are event marks (EM) indicating the points at which cranial manipulation of the subject was either started or terminated. The vertical amplitude denotes signal power and is equivalent in both records. Each record begins with a rest period, after which cranial treatment is administered (first event marks) for the predetermined periods of time. Treatment is terminated (second event marks), after a second rest period, the process is repeated, etc.

Figure 2 represents expansion of the laser-Doppler flowmetry records of subject 1 showing the THM oscillation. The top record shows the initial resting segment followed by the first treatment segment; the bottom record shows the analogous segment pair beginning at 18 min. Cranial treatment amplifies the power of the oscillation. The power of the cardiac component of the waveform, which appears as fine oscillations superimposed over the THM waveform, is essentially unaffected by the cranial treatment regimen.

On the next page, Figure 3 (top and middle) shows Fourier transformation (FT) magnitude spectra of two contiguous segments (18.1 to 28.2 min) of the flowmetry record of subject 1. Plotted is component intensity as a function of component frequency. The top spectrum was obtained from the resting segment (18.1 - 22.9 min), the middle spectrum from the treatment segment (22.9 - 28.2 min). Each spectral peak corresponds to a component of the selected flowmetry segment and has been assigned by others to specific physiological functions as follows: M (Mayer, thermal regulation) component; TH (Traube-Hering, baroreflex) component; R (respiratory) component; H (heart) component.[4]

The abscissae in Figure 3 denote the frequency at which each component is oscillating. For components M, TH, and R, the frequency remains nearly constant with treatment. The frequency of the cardiac component H, however, increases. The ordinates denote relative component strength. With treatment, the TH and M components increase in intensity while the R and H components remain essentially unchanged.

Figure 3 is a magnitude difference spectrum, the spectrum resulting from the point-by-point subtraction of the resting-segment FT spectrum (top) from the treated-segment spectrum (middle). The components of this spectrum denote that which has changed as a result of cranial treatment. From these data it can be seen that:

1) the component strength of the TH, 0.1Hz, frequency oscillation in blood-flow velocity increased.

2) the heart rate increased from approximately 70 to 82 beats per minute following cranial manipulation while its component strength remained relatively unchanged.

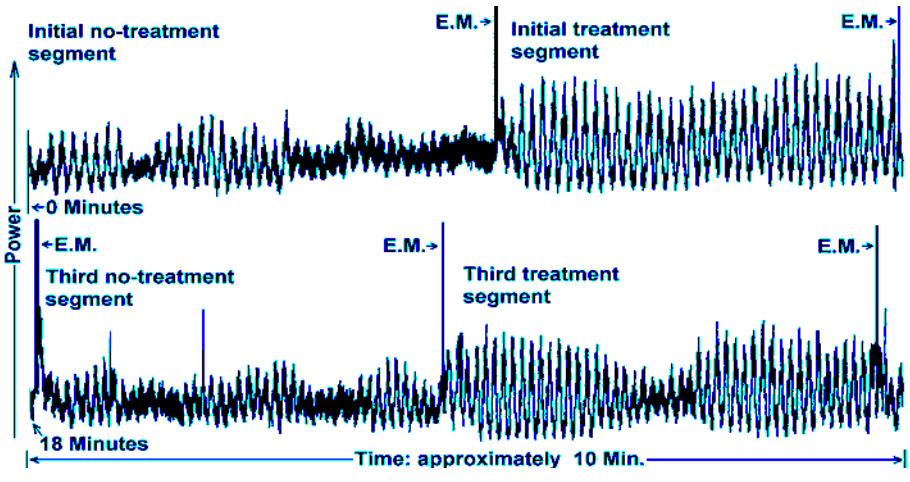
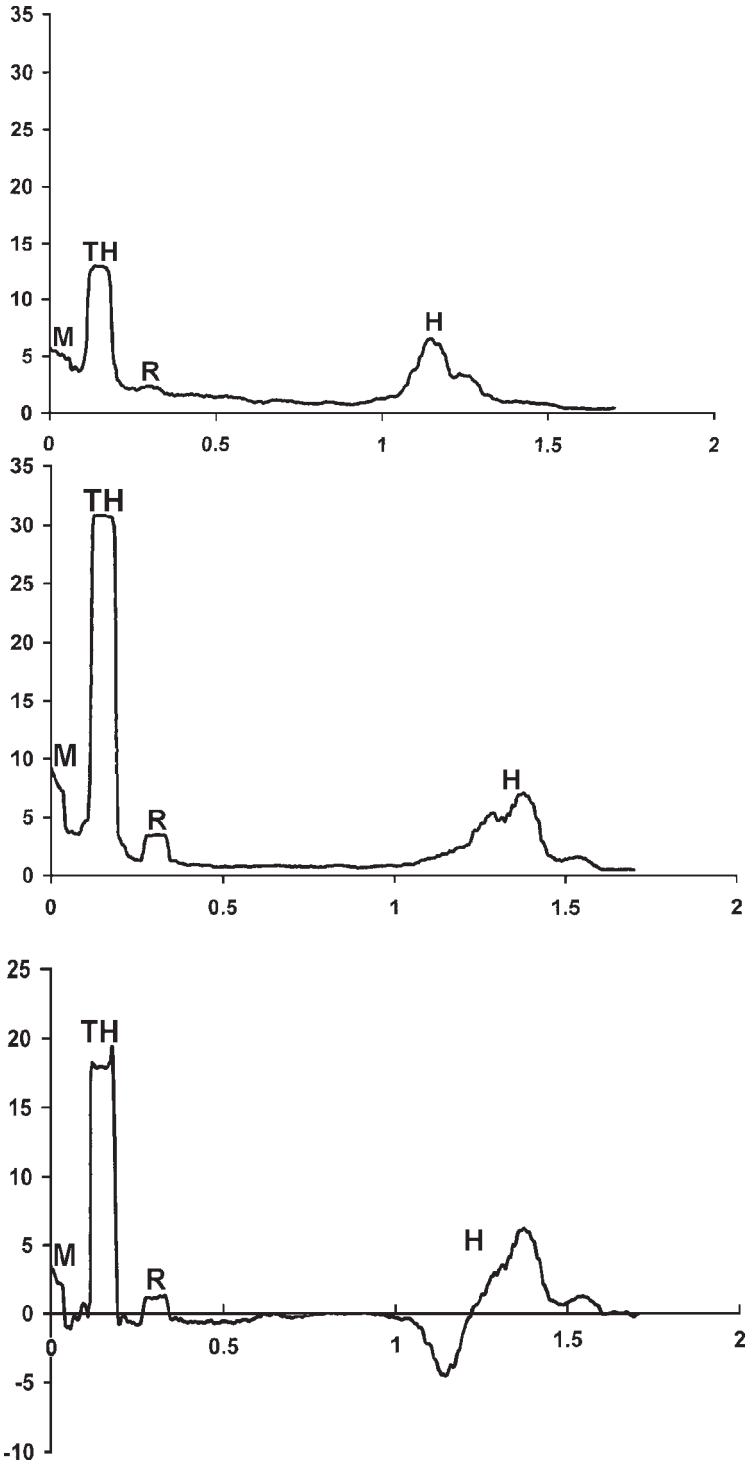


Figure 2: Expansion of the laser-Doppler flowmetry records of subject 1 (Figure 1) showing the THM oscillation. The top record shows the initial resting segment followed by the first treatment segment; the bottom record shows the analogous segment pair beginning at 18 min. Cranial treatment amplifies the power of the oscillation.



Top: Fourier-transformation Subject 1: Third non-treatment segment. Middle: Fourier-transformation Subject 1: Third treatment segment. Below: magnitude difference spectrum, the spectrum resulting from the point-by-point subtraction of the resting-segment FT spectrum (top) from the treated-segment spectrum (middle). The components of this spectrum denote that the component strength of the TH, 0.1Hz, frequency oscillation in blood-flow velocity increased and that the heart rate increased, resulting in the "sinusoidal" appearance of H, from approximately 70 to 82 beats per minute following cranial manipulation while its component strength remained relatively unchanged.

Conclusions

Cranial manipulation can alter the physiologic parameters of blood flow velocity. Manipulation increases the TH, baroreflex, component most markedly and the M, thermal, component to some degree. This results in a marked augmentation of the palpable CRI at the same frequency as that of the of the TH, baroreflex, component of the flowmetry record. The effects of cranial manipulation occur within a few seconds. Likewise the flowmetry record returns to near baseline levels within fractions of a minute after the intervention is stopped. Occasionally, following cessation of the intervention an exponential decay of the augmented flowmetry oscillation is observed. Thus, it is concluded that the marked, short-term effects of cranial treatment have a measurable half-life of a few seconds. The Fourier analysis reveals that the flowmetry record does not return precisely to baseline following intervention but exhibits a small residual effect with a considerably longer half-life. This residual component may be that which in part accounts for the observed therapeutic effects of cranial manipulation.

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CME QUIZ

The purpose of the quiz found on the next page is to provide a convenient means of self-assessment for your reading of the scientific content in the article, *Cranial Manipulation Induces Sequential Changes in Blood Flow Velocity on Demand* by Kenneth E. Nelson, Nicette Sergueef, and Thomas Glonek.

For each of the questions, place a check mark in the space provided next to your answer so that you can easily verify your answers against the correct answers that will be published in the December 2004 issue of the *AAOJ*.

To apply for Category 2-B CME credit, transfer your answers to the AAOJ CME Quiz Application Form answer sheet on the next page, then mail the bottom half of the form with your AOA number ONLY to the AAO as indicated. The top half of the form should be sent to the American Osteopathic Association in Chicago. The AAO will record the fact that you submitted the form for Category 2-B CME credit and will forward your test results to the AOA Division of CME for documentation.

Errata:

The article in the June 2004 issue of the AAOJ by Tony Sinay and Simon Geletta was published with an incorrect title. The word "abstract" was inadvertently added to the end of the original title of: *Health Promotion and Disease Prevention (HPDP) Programs of Osteopathic Hospitals: A Comparative Analysis.*

On page 24 the first graphic should be: $\hat{OR} = \frac{r}{s}$

Our apologizes to Drs. Sinay and Geletta.

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Authors: *Kenneth E. Nelson, DO, FAAO, FACOFP; Nicette Sergueef, DO (France); Thomas Glonek, PhD*

Publication: *Journal of the American Academy of Osteopathy*, Volume 14, No. 3, September 2004, pp 15-17.

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1. In this study, incitant cranial manipulation has been demonstrated to most markedly effect the amplitude (spectral power) of which component of bloodflow velocity as computed from the Fourier-transform (frequency-domain) spectrum?

___A. Mayer (0.008-0.03 Hz) 0.5 to 2 cycles per minute.
___B. Traube-Hering (0.1-0.15 Hz) 6 to 9 cycles per minute.
___C. Pulmonary respiration (0.2-0.3 Hz) 12 to 20 cycles per minute.
___D. Heart rate (1.0-1.5 Hz) 60 to 90 cycles per minute.

2. Incitant cranial manipulation appears to

___A. Decrease the rate of the Mayer wave.
___B. Increase the rate of the Traube-Hering wave.
___C. Decrease the respiratory rate.
___D. Increase the heart rate.

3. The Traube-Hering oscillation manifest in bloodflow velocity is thought to be due to

___A. Thermal regulation.
___B. Baroreflex physiology.
___C. Hypoxia.
___D. Chemoreceptor stimulation.

4. In this study, augmentation of the CRI was induced

___A. With a firm pumping action in synchrony with cranial flexion and extension.
___B. By applying steady external rotation of the temporal bones.
___C. With a rocking force applied to the sphenobasilar synchondrosis.
___D. By CV4.

5. In this study, amplification of the Traube-Hering component of bloodflow velocity by cranial manipulation was demonstrated to be more than happenstance because

___A. Timing of cranial treatment and concomitant Traube-Hering changes corresponded to a preestablished protocol.
___B. An inverse relationship between Traube-Hering amplitude and cranial manipulation exists.
___C. With a firm pumping action in synchrony with cranial flexion and extension, the amplitude of low-frequency bloodflow velocity oscillations decreased.
___D. It occurred whether or not cranial manipulation was employed.

Answer sheet to September 2004 AAOJ CME quiz will appear in the December 2004 issue.

June 2004 AAOJ CME quiz answers:
1. C
2. D
3. B
4. C
5. C

Chronic Fatigue Syndrome: The Misunderstood Disease

Tammy Gregg, Stuart F. Williams

Introduction

Chronic fatigue syndrome (CFS) is a syndrome that is not always well understood. Many times, a patient with this disease can be misdiagnosed as having a mental problem, rather than a physical one. CFS can be a serious physical disease and one, which deserves more attention in the medical community.

To begin to understand CFS, one must first know how to define CFS. The Center for Disease Control (CDC) defines CFS as:

1) Persistent or relapsing clinically evaluated chronic fatigue of new or definite onset. It cannot be the result of exertion and is not alleviated by rest. It must result in deficits in previous levels of occupational, educational, social, or personal activities.
2) The patient must also have four of the following symptoms lasting greater than six consecutive months and which cannot predate the fatigue
   a. Self-reported impairment in short-term memory or concentration severe enough to cause substantial reduction in previous levels of occupational, educational, social, or personal activities
   b. Sore throat
   c. Tender cervical or axillary lymph nodes
   d. Muscle pain
   e. Multijoint pain without joint swelling or redness
   f. New headaches
   g. Unrefreshing sleep
   h. Post-exertional malaise lasting more than 24 hours

The doctor must also be aware that CFS is a diagnosis of exclusion. The fatigue cannot be due to any undiagnosed or untreated disease (such as hypothyroidism), or to any mental disorder (such as major depressive disorder, anorexia, schizophrenia, etc), substance abuse, or severe obesity. If an illness has been treated effectively and the patient is still fatigued, then a diagnosis of CFS must be considered.[1]

Further understanding of CFS is accomplished by knowing who is affected by this disease. A study conducted by Jason et al. illustrates the prevalence of CFS. CFS is a more common condition than previously thought. It affects 422 per 100,000 or 836,000 people in the US. CFS is significantly more common in women than men (522 vs. 291 per 100,000). The study also showed that CFS is more common in Latinos and African Americans compared to Caucasians. The most common age group affected is 40 to 49 year olds. Marital status, social status, educational attainment, or occupational status is not a major factor in determining who will be afflicted with CFS.[2]

Case Report

C.S. is a 45-year-old Caucasian female. She began suffering from symptoms in November of 1987. She had just started seminary school when she became ill with flu-like symptoms, such as low-grade temperature, myalgia, headaches and lymph node swelling. Lab work conducted at this time showed an increase in her CMV titer. She remained bedridden for 6 months, but made a full recovery in 15 months.

Then in 1991, C.S. became ill again from a viral relapse. She experienced the same type of symptoms as before, but this time they were more intense and lasted for a longer period of time. She became housebound and bedridden for one year. In 1992, she was able to return to school but could only take one class at a time until she earned her degree. By 1994, C.S. had recovered 90 percent of her previous strength and energy, but noticed that she had developed a push-crash symptomatology (she could push herself to do what she wanted, but then crashed afterwards). She also noticed that she would still fatigue easily. Despite this, she began her career as a full-time minister.

In March of 1996, C.S. again relapsed. This relapse had even more pain and fatigue. Her headaches were worse than before. In this episode, she found herself with such a severe lack of concentration that she could no longer balance her own checkbook, despite being a math major in undergrad. C.S. was once again bedridden for a year. She has still not fully recovered from this episode. Six months after this last episode began; C.S. was diagnosed with Chronic Fatigue Syndrome (CFS).

Her tests included the exercise bike test and blood work. The exercise bike test showed that her cortisol did not rise during her biking. The blood work showed that she had decreased NK cells, increased alpha-interferon, increased 25A-RNaseL (precursor to RNaseL) and increased RNaseL (whose presence indicates the person is "fighting" a viral infection). There was also evidence of HHV-6 and Mycoplasma fermentans incognitos infections. Her Rheumatologic exam was negative.

Before all of this began, C.S. was a healthy individual. She did not get sick often, but if she did get sick with a virus, it always seemed to take longer for her to recover than those around her. Her past surgical history includes wisdom teeth removal done under local anesthetic. C.S.

has one brother who has had two episodes of fatigue that have lasted less than six months each (not CFS).

C.S. is undergoing intensive therapy with supplements to improve digestion and return balance to the important nutrients in the body. Immunomodulation is used to enhance her immune system to fight off the chronic viral infection. Liver and gut health is improved to promote the good bacteria living within the gut, which also helps to aid in absorbing the nutrients that she takes orally. Craniosacral techniques and myofascial unwinding are also used to aid in her recovery.

Review of Literature

Many studies have been conducted in an attempt to define how and why people become inflicted with CFS. One such study was developed by Buchwald, et al. on a population of people in the Lake Tahoe and surrounding areas. The participants experienced a rapid onset of a flu-like illness that was both chronic and debilitating. Many of the patients were regularly bedridden or shut in. Other symptoms experienced on a daily basis were difficulty in concentration, depression, headaches, adenopathy, arthralgias, paresthesias and rash. These symptoms often lasted months or even years and were often problems that had not been experienced by the residents before. After conducting different serological studies on the blood and MRI studies of the brain, a very unique picture was forming. The most common virus found to be actively replicating was human herpes virus type 6 (HHV-6). The MRI depicted abnormal signal intensity in the white matter usually indicative of an inflammatory process of the CNS. This can be associated with diseases such as Multiple Sclerosis, but can also be caused by infection with a virus, such as HHV6.[3]

A second study conducted by Suhadolnik, et al. reaffirmed the theory that CFS could be caused by a viral infection or reinfection. During a viral infection, the 2-5A Synthetase/RNase L pathway becomes activated. The 2-5A synthetase converts ATP to 2'5'-oligoadenylates (2-5A). This activates a latent endoribonuclease (RNaseL) to degrade cellular and viral single-stranded RNA. Three important points were reflected in this study. First, the predominant form of 2-5A seen in CFS patients was in the active form. Second, the bioactive 2-5A intracellular concentration were 220 fold in CNS patients than controls. Third, the terminal functional enzyme of this antiviral defense pathway (RNaseL) was also elevated in CFS patients.[4]

Another study again reaffirmed the belief that CFS could be caused by a virus. Caligiuri, et al. demonstrated that CFS patients have an abnormally low level of NK cells, specifically $NKH1^{+}T3$. $NKH1^{+}T3^{-}$ phenotype is normally the most numerous of the NK cells. Instead of $NKH1^{+}T3^{-}$, CFS patients tend to have more $NKH1^{+}T3^{+}$, normally very low in the cell population. In addition to the numbers, CFS patients have impairment of NK activity. Thus, these patients have a difficult time in destroying cells invaded by viruses.[5]

Other signs and symptoms associated with CFS might not be from a viral cause. A different theory is that the Hypothalamic-Pituitary-Adrenal axis might be defective in patients with CFS. Dinan, et al. showed that CFS patients had a blunted release of ACTH from the pituitary in response to ipsapirone challenge. There are several theories to explain this phenomenon including: 1) decreased responsivity of serotonin receptors in hypothalamus, 2) under-productivity of the corticotrophs, 3) decreased responsivity of CRH receptors on corticotrophs.[6] Another study analyzed the cognitive impairment symptoms experienced by many CFS patients. Johnson et al. determined that many CFS patients had impaired performance on the Paced Auditory Serial Addition Test (PASAT). This showed that the patients had difficulty with complex tasks requiring concentration and demanding simultaneous processing of several cognitive activities at one time.[7] Lastly, sleep appears to also be disturbed in patients with CFS. They show disturbances in sleep initiation and maintenance when compared to healthy controls.[8]

Treatments

Since there are many different theories of causes of CFS, the treatment plan for any CFS patient must be able to address the different etiologies that can present at once in any given patient. Masuda et al. showed that a multi-disciplinary approach to treating CFS was more effective than a one-treatment regimen. In this study, the therapy consisted of three separate stages. Stage one was drug therapy, rehabilitation, and counseling. Stage two was cognitive-behavioral therapy and family therapy. Stage three was exercise therapy. After the treatment, many of the patients were able to return to work, especially those who were post-infectious CFS.[9]

Other treatments used for CFS focus on a single symptom. For example, it was found that treatment with poly (I)•poly(C_{12}U) for several weeks decreased the giant cell formation seen in CFS patients by restoring normal lymphokine balance or directly inhibiting the viral reactivation.[4] Low dosages of hydrocortisone have been shown to significantly reduce the self-rated fatigue and disability in CFS patients.[10]

Another important treatment to aid in the recovery of CFS patients is cranial osteopathy, or the primary respiratory mechanism. During the inhalation part of the cycle, the hemispheres will move upward, the third ventricle dilates and lifts the pituitary body to elevate the saddle of the sphenoid while tipping the anterior end downward. Concurrently, the ethmoid drops down causing external rotation of the petrous portion of the temporal bones. The opposite will then occur with exhalation.[11] If the doctor were to manipulate the cranial bones to allow for better movement during this cycle, it would allow for the pituitary to function in a more efficient manner and would help to relieve the headaches felt by many patients afflicted with CFS.

Discussion

C.S. was diagnosed with viral induced CFS. From the data gathered on her, she was positive for both HHV-6 and Mycoplasma fermentans incognitos, with decreased levels of NK cells, increased expression of the 25A-RNaseL pathway, and increased alpha-interferon. These results supported her diagnosis of viral induced CFS. Her cortisol levels remained low during times of stress, discovered during

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the exercise bike test, another common finding in patients with CFS as seen in the literature. Her other symptoms of headache, pain, impairment in cognitive function, and her lasting fatigue place her in the CDC definition of CFS.

C.S.'s treatment is extensive. She takes many different supplements, including some to maintain a healthy gut, others to give the body the nutrients it requires (sometimes through injections), and some to help digest and absorb the nutrients in the diet. She is also taking Klonopin, Doxepin and Neurontin to help regulate her sleep patterns. To fight the virus HHV-6, she takes Isoprinosine. C.S. is prescribed different growth hormones to help her body repair and rebuild her organs. Lastly, C.S. underwent craniosacral work with a licensed professional to help alleviate her painful and debilitating headaches. In the process, she discovered that the treatments also gave her an overall feeling of wellness. She also experienced more energy after her treatments.

Summary

Chronic Fatigue Syndrome is a complicated and often misunderstood disease. There is no one, simple solution for treatment. Instead it requires dedication, hard work and understanding from the physician, the patient, and their family. The clinical research so far has been limited to following only one specific entity of CFS at a time. It becomes the physician's responsibility to understand and utilize the different research methodologies to find the best treatment for their patient. CFS is not defined in terms of only one issue, but a conglomerate of everything the research has uncovered. The only avenue left to explore is how OMT and cranial osteopathy can help these patients. Since there is a documented deficiency of the pituitary gland to secrete ACTH under stress, then perhaps cranial osteopathy can help to increase the functional capacity of this organ. If nothing more, by utilizing cranial osteopathy, we can help to alleviate the headaches these patients feel without having to rely heavily on medications.

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Chronic Pelvic Pain Syndrome

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Introduction

Prostatitis, in itself, has been classified by the NIH into acute bacterial prostatitis, chronic bacterial prostatitis, chronic pelvic pain syndrome with and without inflammation, and asymptomatic inflammatory prostatitis (as shown in Table 1). Prostatitis and benign prostatic hypertrophy are common diagnosis among men aged 50 and up. So what do you do when a 30-year-old man presents with urinary hesitancy and pelvic pain? Tests on urine and prostatic fluid would be performed, but what if all results came back negative? If we remember A. T. Still's second principle stating mechanical or structural disturbances in the body interfere with functioning of certain parts of the body, we may explore other avenues for patients of this younger age group with the above symptoms.[1] A basic structural exam of the lower thoracic spine, lumbar spine, pelvis, and sacrum may reveal TART changes, which could lead us in additional directions besides just the basic prostate exam and prescription of antibiotics for 4-6 weeks. In doing an osteopathic evaluation, going back to Still's third principle, fixing the structural disturbance with osteopathic techniques can help the body overcome and compensate for any functional disturbances we may find.[1]

Case Presentation

D.R. is a 47-year-old African-American male who presented to the manipulation clinic for pain "in the groin area". The patient's medical history included benign prostate enlargement (which had been present on and off for 12 years), chronic bronchitis, Type II Diabetes Mellitus, seasonal allergies, tuberculosis diagnosed in 1997, and recently diagnosed degenerative joint disease of the lumbar spine. Surgical history included two prior disc fusions of the lumbar spine at unknown levels 10 years ago. Socially, a history of alcohol abuse and 30-pack year smoking history (the patient reportedly quit in 1997) was elicited. The patient's family history was significant for Type II Diabetes Mellitus and hypertension. Currently, the patient reports taking cetirizine, occasional use of an albuterol inhaler, an anti-tuberculin medication, which he could not name, and etodolac for 2 months.

On presentation to the clinic, D.R.'s chief complaint was right groin pain. He reported a long history of back and groin pain which had become worse in the last 2 years. The areas of pain included the lower back bilaterally, radiating around to the groin and occasionally included testicular pain. The patient reported that the pain was always present with no relief of pain with any position, and, in fact, the pain was made worse by sitting or walking for long periods and when bending at the waist to pick thing sup. The pain was described as a burning, achy pain, which occasionally became sharp. Although D.R. stated etodolac curbed the pain to 3/10, he rated his pain 9/10 on

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TABLE 1 NIH classification of prostatitis syndromes.3,4

Category		Symptoms	Treatment
I	Acute bacterial prostatitis	chills, fever, perineal and low back pain, irritative urinary symptoms	Antibiotics (usual bacteria: E. coli, enterobacter, or pseudomonas)
II	Chronic bacterial prostatitis (CBP)	chronic perineal aching and or low back pain, irritative voiding symptoms	Same bacteriology as acute, antibiotics must be lipid soluble with minimal plasma protein binding.
IIIA	CPPS, inflammatory (formerly chronic non-bacterial prostatitis)	Urinary complaints with vague pelvic/perineal pain, negative cultures, positive white blood cells in semen, prostatic secretions, and/or urine	
IIIB	CPPS, non-inflammatory (formerly prostatodynia)	no inflammation; negative cultures; pelvic pain in the perineal, penile, or suprapubic area; no irritative voiding symptoms, but hesitancy, urgency, and weak stream are common; no white cells in semen, prostatic secretions, or urine	
IV	Asymptomatic inflammatory prostatis	No subjective symptoms; incidental finding on prostatic biopsy or by white cells in prostatic fluid.	

presentation. The patient denied radiation to either leg or lower extremity paresthesias. Urinary symptoms included urgency and frequency, but not incontinence or pain with urination. The patient stated he did not recognize any hesitancy, decrease in stream of urine, or interruption of flow.

On physical exam, D.R. showed 5/5-muscle strength in lower extremities, 2+ patellar and Achilles reflexes, negative straight leg test, and intact sensation of the lower extremities. Somatically, a non-neutral sacrum of left on a right oblique axis along with a non-neutral L5 vertebra to the right was discovered. An anterior innominate was elicited as well. No dysfunction in the public bones was appreciated/ however extreme tenderness was noted by the patient during palpation. In the thoracic area, T10-12 were sidebent right, rotated left.

Treatment began with HVLA to the thoracic spine to correct T10-12. After correction of the thoracic spine, pubic decompression followed. The patient reported decreased sensitivity to palpation after this procedure. A supine-indirect method using respiratory force was begun on the sacrum, followed by prone-indirect method to finally overcome the sacral dysfunction. At this point, the patient's left leg was positioned off the table flexed at the knee and hip and muscle energy was employed with the patient attempting to extend his leg at the hip.

At the end of the manipulations, D.R. stated his pain had decreased from 9/10 to 3/10. He stated he thought that the pubic decompression was significantly helpful in immediately decreasing his pain. D.R. was given a sheet of low back exercises, explained revese-Kegle exercises with emphasis on maintaining the relaxation phase for 3-5 seconds (as opposed to concentrating on the contraction phase) and told to do these exercises 4-times daily.

Journal Review

Chronic pelvic pain syndrome (CPPS) is a common urologic occurrence in men less than age 50 and accounts for a large number of urological visits each year.[2] Other terms for CPPS include prostatitis, proctadynia, pelvic myalgia, proctalgia, levator ani syndrome, diaphragma pelvis spastica, and coccygodynia.[2,6] Most patients present to the urologist with the diagnosis of chronic prostatitis after unsuccessful treatments by their PCP with antibiotics, alpha-blockers, and anti-inflammatories.[2]

Although the exact etiology of CPPS is unknown, many theories exist including dysfunctional high pressure voiding (contraction of the external urethral sphincter during voiding causing a rise in prostatic urethral pressure which causes reflux of urine into the prostatic parenchyma, provoking inflammation and pain), autoimmune disorder, neuromuscular disorder, and interstitial cystitis.[2,4] Hyothesis of microorganisms which are non-culturable, including C. trachomas, have not panned out. Since some patients improve with prostate and/or pelvic floor massage, spasm of the pelvic floor musculature may be responsible for the pain.[7] Along those lines, another theory states that pain due to chronically contracted, fatigued pelvic muscles and pelvic muscle spasticity may compromise local immune defenses which, along with urine reflux into the prostatic ducts, may trigger an inflammatory response in the prostatic tissue.[6,13]

An additional issue lies in the fact that small, unmyelinated and slow C pain fibers in the pelvis respond to thermal stimuli and most autonomic signals and visceral pain. Inflammation and internal chemical stimuli (such as bradykinin and cytokines including interleukin-1 beta and TNF) cause recruitment of C fibers, magnifying pain.[4,7] This chronic nociceptive bombardment of the central regulatory voiding circuits via somatic pelvic floor afferents triggers a cascade of neural events that culminate in neurogenically mediated inflammation with an up regulated sacral arch.[13]

Urodynamic studies may show abnormal reflex reactivity and hypersensitivity, hypertonia of the sphincter region and dyssenergic pelvic floor behavior with voiding (as a result of increased alpha-adrenergic activity).[2,10] Detrusor hypocontractility and higher prostate tissue pressure are noted as well.[9] One study reviewed the use of transurethral needle ablation (TUNA), which is efficacious for BPH and effects alpha-adrenergic receptors and sensory nerves, on patients with CPPS.[10] No statistical difference was found between TUNA patients and sham patients, but all patients received 20 minutes of cyctoscopy in the prostatic urethra which may have led to improvement of sham patients due to treatment of prostatic uretha obstruction.[10]

As noted in table 2, malignant and bacterial disease must first be ruled out prior to diagnosis of CPPS.[3] The basic workup would include palpation of the prostate for texture, size, tenderness, and presence of nodules. In CPPS, the prostate is not tender, but movement of the parapsoriatic fascia is painful. Prostatic fluid exam will be normal. Rectal exam evaluates the sphincter ani circular muscles for tone and tenderness with trigger points observed at the attachment of the rectus abdominus to the pubic bone, inguinal canal obliques, subpubic adduc-

TABLE 2 Differential diagnosis of CPPS.[4]

Bladder conditions
Carcinoma-in-situ or carcinoma
Interstitial cystitis
Bladder outlet obstruction
Bladder irritation by colonic carcinoma or diverticulitis
Seminal vesicle abnormalities
Seminal vesicle calculi
Seminal vesicle cysts (e.g. associated with ipsilateral renal agenesis)
Miscellaneous
Occult inguinal and femoral hernias
Inguinal ligament enthesopathy
Lumbosacral arthropathy
Recurrent genital herpes simplex

tor longus insertion, pubococcygeus insertion intersecting with prostatic endopelvic fasic, obturator internus muscle at Alcock's canal.[2]

Classically, patients will complain of pain that is a dull, poorly circumscribed ache at various regions within the pelvis, reminiscent of visceral type pain versus somatic pain which is sharp and well-circumscribed.[7] The pain is aggravated by sitting and accompanied by suprapubic pressure, perineal or penile pain sometimes involving the testicles, supropubic/retropubic areas, inguinal and sacral regions, and variable urinary symptoms. This pain works in a cycle: pelvic floor tension myalgia begins with continuous habitual contraction of the muscles of the pelvic floor (levator ani and short external rotators of the hip) because as pain increases, pelvic muscle contraction increases.[1,4]

Looking at this from an osteopathic perspective, we note that CPPS may be considered a myofascial pain condition caused by central neurogenic mechanisms.[13] As pelvic floor myofascial trigger points are not only a source of pain and voiding symptoms, but also a trigger for neurogenic bladder inflammation via antidromic reflexes, the use of pelvic floor manual therapy decreases pelvic floor hypertonus, effectively ameliorating the symptoms of urgency and frequency.[11] The pelvic diaphragm's normal function is to remain relaxed and work in synchrony with the abdominal diaphragm to ensure efficient return of lymph flow, and as patients with CPPS are chronically contracted, decreased lymph flow causes inflammation of the prostate leading to tissue edema, disturbed microcirculation, and hypoxia resulting in pain.[9,14]

Discussion

We must examine the body as a whole to further treat this syndrome. Somatic dysfunction of the sacral, sacroiliac, pelvic, and lumbosacral attachment points may be associated with bladder symptoms, sphincter control, and pain the perineum due to spasms of the pelvic diaphragm or the prostate.[14] Since the pubic bone is connected to the prostate by the pubovprostatica fascia, a pubic shear or pubic compression somatic dysfunctions can produce symptoms of frequency and urgency.[14] From a sympathetic approach, the innervation to the prostate exits from T11-12 and the bladder sphincter from T11-L3.14 Finally, Chapman's point of tenderness along the iliotibial band may indicate dysfunction or disease of the colon, broad ligament, or the prostate.[14]

Anatomically, the prostate gland is surrounded by Denonvillier's fascia, the endopelvic fascia, and the puboprostatic ligament.[14] Muscularly, the inferior boundary of the pelvis is the muscular pelvic diaphragm composed of the coccygeus and levator ani muscles. The prostate fits in the anterior defect of the pelvic diaphragm above the superior fascia of the deep pouch of the perineum.[14] Knowing this anatomic information aids in designing a treatment plan. Since chronically tight and inflamed tissues are at least partially causal for CPPS, it makes sense that treatment aimed at releasing fascia and relaxing muscles would be of great benefit. Exercises shown in figure 1 performed 4 times daily will also aid the patient in muscular re-education which relaxes the muscles of the pelvic floor, particularly the levator ani.[6]

Conclusion

Chronic pelvic pain syndrome is a common syndrome of men age 25-45. This syndrome consists of vague pelvic pain, urinary symptoms of hesitancy and/or urgency, and a non-tender prostate. Sometimes described as an abacterial prostatitis (class III), CPPS is a diagnosis of exclusion when all testing is negative. Lower thoracic, lumbar, pelvis, and sacrum must be examined to find areas of tissue texture abnormality, asymmetry, restriction of motion, and/or tenderness. Myofascial considerations must be taken as well. We must remember A. T. Still's principles when evaluating patients as visceral issues may, in fact, have somatic causes.

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The Effect of Osteopathic Manipulative Treatment on Gait Disturbance in Multiple Sclerosis Patients

Josalyn M. Mann, Karen M. Steele

Abstract

Multiple Sclerosis is a debilitating neurological disease that affects a large portion of our population. Gait disturbance is a common symptom that these individuals suffer. In this outcome study, the author examined whether or not osteopathic manipulative treatment would affect gait disturbance. Both subjective and objective results showed a trend in improvement of gait stability. Lack of statistical significance may have been due to a small sample size (N=5) or with interrater reliability issues. Though no statistical significance was proven, multiple sclerosis patients still reported an increase in quality of life following osteopathic manipulative treatment.

Key words: multiple sclerosis, gait disturbance, spinal facilitation, somatic dysfunction, observational gait analysis

Multiple sclerosis (MS), a debilitating neurological disease, affecting females more than males, involves a demyelinating process. Usually striking between the ages of 20 and 40 years of age, the cause of the disease is idiopathic. Several theories exist in an attempt to provide an explanation for the disease. Among these indefinite causes, some believe an autoimmune process may be responsible for stimulating the disease, while others point to a viral entity as the culprit.[1]

The disease is extremely variable in nature as sufferers experience a range of symptoms and can be classified one of several different patterns. Currently, pharmacologic therapy is used to help treat the progression of the disease and control symptoms. Additionally, approximately 30 percent of MS patients report trying alternative and complementary medicine.[2,3] Alternative treatment encompasses a wide range of practices, including massage therapy, dietary monitoring, acupuncture, relaxation techniques, and manipulation.[3,4,5,6,7,8,9] While osteopathic manipulative treatment (OMT) is not a commonly listed treatment employed by MS patients, several articles have suggested its beneficial effects on locomotion, gait, efficiency of movement, and strength.[9,10] Since OMT utilizes a variety of techniques, it would seem to be an appropriate management tool for symptoms.

Of the symptoms MS patients suffer, gait disturbance appears to be often changing with one day bringing stability and the next delivering an unstable, sometimes spastic or foot drop gait.[11] Many components combine to allow gait to occur. Among them, motor nerves, muscles, bones, ligaments, and tendons are prominent features. The motor nerves responsible for the motion of gait range from T12-S3,[12] while other structural or postural adaptations may also play a role in locomotion.

Abnormal stimulation from various sources can cause the body's spinal nerves to act unregulated. The muscles controlled by these aberrant nerves may react by becoming taut. Osteopathic medicine recognizes the tense muscles as one component of somatic dysfunction. Sometimes the spinal nerves responsible for such somatic dysfunctions can be held in a condition referred to as spinal facilitation which occurs when a group of neurons are held in an incomplete excitational state.[13] In this situation, the amount of afferent stimulation needed to actually produce an impulse becomes lessened.[13] The Educational Council on Osteopathic Principles (ECOP) included as part of the definition of spinal facilitation that it is a "theory regarding the neurophysiological mechanisms underlying the neuronal activity associated with somatic dysfunction."[13] Irvin Korr, PhD also wrote, "Facilitation of motor pathways leads to sustained muscular tensions, exaggerated responses, postural asymmetries, and limited and painful motion."[14] It is through this cascade of neural facilitation creating musculoskeletal somatic dysfunction that it is theorized OMT can help normalize the nervous system and improve physiologic function. As a degenerative disease, MS attacks the nervous system leading to symptoms affecting the musculoskeletal system, i.e. spasticity, tremor, and gait disturbance.[15] If OMT can assist in normalizing the nervous system in MS patients through the musculoskeletal system, then gait disturbance may also improve. ➔

Methods

Five patients, whom were able to ambulate without the aid of a crutch, walker, or cane, were recruited through surrounding community MS support groups. Each of the patients attended a three-hour session held at the West Virginia School of Osteopathic Medicine in Lewisburg, WV. During this time, they completed subjective questionnaires regarding their disease process and gait instability. The participants were also examined, ambulated, and received OMT.

Pre-OMT subjective questions included a survey of current medications and treatments, age at initial MS diagnosis, pattern of disease, history of OMT, and gait instability. Patients were asked to assign a number to their instability, with zero representing completely stable and ten being severely unstable. A physical exam was completed that concentrated on spine and lower extremity range of motion, muscle strength testing of the lower extremities, and an osteopathic examination. Other components of the exam were used to arrive at Kurtzke's expanded disability status scale score[16] by utilizing the functional evaluation *(Figure 1). All patients qualified by receiving a score of six or less. After the examination, patients walked the length of 70 feet with a turn in the middle. This was performed twice as the patients were recorded once by an anterior-posterior camera and again from a lateral view.

The OMT utilized in this project included indirect techniques, with the exception of muscle energy. Techniques that may have more directly stimulated the lymphatics were excluded, as requested by the Institutional Review Board. Following treatment, patients were examined to assess for structural and tissue texture changes. Once this was accomplished, the participants ambulated once more. Post-OMT subjective data asked patients to evaluate how they felt treatment had affected their condition and to again rate their gait instability. At the completion, each patient was given a seven-day diary to record adverse effects that may have resulted from OMT. On day seven, each patient was interviewed by phone to conclude if any adverse effects had indeed taken place. The patients were also asked to evaluate if they believed OMT had helped them get through the week, and if they might consider using OMT as a part of their future treatment plan. The entire project was approved by the Institutional Review Board.

Upon completion of the project, the gait videotapes were digitized and randomized for analysis. A final copy was provided to each of the two blinded, independent reviewers. Each analyst is on faculty at the West Virginia School of Osteopathic Medicine. The gait analysis *(Figure 2) reviewed multiple anatomical and functional parameters that would affect stability. In the end, the analysis was summarized by a more global assessment of patient stability. Each MS patient was assigned a total point value *(Figure 3) for their pre- and post-OMT ambulation based on a comparative score that a normal, healthy patient might achieve.

Results

A paired t-test was performed and found no statistically significant results based on all individual gait parameters; however, the global assessment of patient stability proved to demonstrate a trend of improvement. Patient subjective data showed dramatic improvement, as four of five patients reported better ability to walk, less fatigue, an increase in range of motion, and an overall feeling that they were able to function more easily throughout the week after OMT.

Discussion

Subjective data in this project shows improvement in patient symptoms and quality of life, while the objective figures do not correlate. The mixed results obtained may reflect interrater reliability issues. Although a meeting between the principal investigator and analysts occurred in an attempt to eliminate predictable problems, it appears that more training may be necessary. An overview of the analysis demonstrates that the two independent analysts did not agree on the conclusions of most parameters, except gait stability. One explanation for this disagreement may have been because the two analysts come from different professional backgrounds. As an anatomist, one analyst may have emphasized more of an anatomical model, while the other, a practicing osteopathic clinician, may have used more clinical judgement in the evaluation. A suggestion for the future may include having the analysts review the gait videotapes produced from this project again. By examining them and arriving at final decisions together, they might establish more consistency in findings.

Another cause for the mixed results yielded in this project may be due to the low number of subjects (N=5). For the rural surrounding area and exclusion criteria (diagnosis of MS and ambulation without the aid of a crutch, cane, or walker), recruitment resulted in a low patient population. In the future, a larger number of subjects would be desired.

While this study is meant to demonstrate the usefulness of OMT, some may criticize that exact techniques to be used were left undefined in this project. By individualizing treatment for each patient, true somatic dysfunction was able to be addressed instead of standardizing what to treat. It seems most appropriate to attempt individualized treatment because in the end, this project tests a concept, not a treatment technique. Many studies that use OMT also include sham treatment in the protocol to help measure results against. This project did not need to include sham treatment since each patient served as his or her own control through a gait comparison of pre- and post OMT. The question asked addressed what the effect of OMT on gait disturbance in MS patients is, not whether it was a matter of having a touch versus non-touch stimulus that affected gait disturbance. If a larger scale study proved statistically significant, then the issue of whether gait disturbance improves with OMT and/or sham treatment could be addressed at that time.

Finally, since this study utilized the observational gait analysis, the results are not as valid as they could have been using the gold standard, a 3-D platform analysis found in a gait lab. Although the 3-D analysis is more universally accepted, it appeared nearly impossibIe to furnish the study with this method. Financial and time constraints, along with a rural geographical location led to using a more accommodating means of gait analysis.

In conclusion, although objective re-

* *Figures are located on pages 30-31*

sults did not yield statistical significance, OMT seemed to dramatically help these MS patients. This study shows that MS patients describe relief with gait disturbance and other symptoms following OMT. Treatment provided an increase in quality of life as reported in their improved gait stability, mobility, strength, and even self-esteem.

Acknowledgement

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FIGURE 1

Functional Evaluation for Expanded Disability Status Scale Rating

Pyramidal

1. Observe for atrophy present ___yes ___no
2. Any tremor present ___yes ___no
3. Detection of increased tone (spasticity) ___yes ___no
4. Abnormal muscle strength testing (<4) ___yes ___no
5. Drift present ___yes ___no

Scale:

- *0 Normal*
- *1 Abnormal signs wihtout disability*
- *2 Minimal disability*
- *3 Mild or moderate paraparesis or hemiparesis; severe monoparesis*
- *4 Marked paraparesis or hemiparesis; moderate quadriparesis; or monoplegia*
- *5 Paraplegia, hemiplegia, or marked quadriparesis*
- *6 Quadriplegia*
- *V Unknown*

Cerebellar

1. Finger-to-nose; Heel-to-shin are jerky, uncoordinated ___yes ___no
2. Rapid alternating movements are non-rhythmic ___yes ___no
3. Romberg test:
   - pt. unsteady with eyes closed ___yes ___no
   - pt. unsteady with eyes open ___yes ___no
4. Gait instability ___yes ___no

Scale:

- *0 Normal*
- *1 Abnormal signs without disability*
- *2 Mild ataxia*
- *3 Moderate truncal or limb ataxia*
- *4 Severe ataxia, all limbs*
- *5 Unable to perform coordinated movements due to ataxia*
- *V Unknown*
- *X Follows number (3) if weakness interferes with testing*

Brain Stem

1. Nystagmus present (CN III, IV, VI) ___yes ___no
2. Extraocular weakness present (CN III, IV, VI) ___yes ___no
3. Speech non-fluent (CN XII) ___yes ___no
4. Inability to swallow (CN IX, X, XII) ___yes ___no

Scale

- *0 Normal*
- *1 Signs only*
- *2 Moderate nystagmus or other mild disability*
- *3 Severe nystagmus, marked extraocular weakness, or moderate disability of other cranial nerves*
- *4 Marked dysarthria or other marked disability*
- *5 Inability to swallow or speak*
- *V Unknown*

Sensory

1. Decrease in touch sensation ___yes ___no ___mild ___mod ___severe ___# of limbs
2. Decrease in position sense ___yes ___no ___mild ___mod ___severe ___# of limbs
3. Decrease in vibration ___yes ___no ___mild ___mod ___severe ___# of limbs
4. Decrease in pain sensation ___yes ___no ___mild ___mod ___severe ___# of limbs

Scale:

- *0 Normal*
- *1 Vibration or figure-writing decrease only in 1 or 2 limbs*
- *2 Mild decrease in touch or pain or position sense and/or moderate decrease in vibration in 1 or 2 limbs; or vibratory decrease alone in 3 or 4 limbs*
- *3 Moderate decrease in touch or pain or position sense, and/or essentially lost vibration in 1 or 2 limbs; or mild decrease in touch or pain and/or moderate decrease in all proprioceptive tests in 3 or 4 limbs*
- *4 Marked decrease in touch or pain or loss of proprioception, alone or combined, in 1 or 2 limbs; or moderate decrease in touch or pain and/or severe proprioceptive decrease in more than 2 limbs*
- *5 Loss of sensation in 1 or 2 limbs; or moderate decrease in touch or pain and/or loss of proprioception for most of the body below the head*
- *6 Sensation essentially lost below the head*
- *V Unknown*

Bowel and Bladder

1. Urinary hesitancy exist ___yes ___no ___mild ___mod
2. Urinary urgency exist ___yes ___no ___mild ___mod
3. Urinary retention exist ___yes ___no ___mild ___mod
4. Urinary incontinence exist ___yes ___no ___rare ___frequent
5. Loss of bladder function ___yes ___no
6. Loss of bowel function ___yes ___no

Scale:

- *0 Normal*
- *1 Mild urinary hesitancy, urgency, or retention*
- *2 Moderate hesitancy, urgency, retention of bowel or bladder, or rare urinary incontinence*
- *3 Frequent urinary incontinence*

4 In need of almost constant catheterization
5 Loss of bladder function
6 Loss of bowel and bladder function
V Unknown

Visual (or Optic)

1. Scotoma present ___yes ___no ___left ___right ___both
2. Visual acuity (corrected) ___left ___right ___both
3. Decrease in visual fields (CN II) ___yes ___no ___mod ___marked

Scale:

0 Normal
1 Scotoma with visual acuity better than 20/30
2 Worse eye with scotoma with maximal visual acuity of 20/30 to 20/59
3 Worse eye with large scotoma, or moderate decrease in fields, but with maximal visual acuity of 20/60 to 20/99
4 Worse eye with marked decrease of fieldls and maximal visual acuity of 20/100 to 20/200; grade 3 plus maximal acuity of better eye of 20/60 or less
5 Worse eye with maximal visual acuity less than 20/200; grade 4 plus maximal acuity of better eye of 20/60 or less
6 Grade 5 plus maximal visual acuity of better eye of 20/60 or less
V Unknown
X Added to grades 0 to 6 for presence of temporal pallor

Cerebral (or Mental)

1. Mood alteration present (euphoria, depression) ___yes ___no
2. Mini-mental Status Examination score
   - mild decrease in mentation 21 ___yes ___no
   - moderate decrease in mentation 10-20 ___yes ___no
   - marked decrease in mentation 9 ___yes ___no
3. Dementia present ___yes ___no

Score:

0 Normal
1 Mood alteration only (Does not affect DSS score)
2 Mild decrease in mentation
3 Moderate decrease in mentation
4 Marked decrease in mentation
5 Dementia or chronic brain syndrome-severe or incompetent
V Unknown

Other

1. Hyperreflexia present ___yes ___no
2. Babinski present ___yes ___no
3. Chaddock present ___yes ___no

Scale:

0 None
1 Any other neurologic findings attributed to MS
V Unknown

FIGURE 2

A Multiple Sclerosis Patient Gait Analysis

Ambulation speed=distance/time _____m/s
Stance phase normal @ 60% ___yes ___no
Swing phase normal @ 40% ___yes ___no

Extended heel strike ___yes ___no
Extended toe off ___yes ___no

Single limb support>double limb support ___yes ___no

Smooth, fluent progression ___yes ___no
Head centered over body ___yes ___no

Shoulder girdle level ___yes ___no

Pelvic Girdle level ___yes ___no
Pelvic total rotation (via iliac crest) _____ _____
<15 degrees >15degrees

Arm swing symmetric ___yes ___no
Leg swing directly forward ___yes ___no

Rhythmic stride ___yes ___no

Foot drop present ___yes ___no
LE spasticity present ___yes ___no

Patient stability _____ _____ _____ _____
Completely Stable | Minimally Unstable | Moderately Unstable | Severely Unstable

FIGURE 3

A Multiple Sclerosis Patient Gait Analysis Score

	+1	0
Ambulation speed=distance/time	~1.5 (m/s)	<1.5(m/s)
Stance phase normal @ 60%	___yes	___no
Swing phase normal @ 40%	___yes	___no
Extended heel strike	___no	___yes
Extended toe off	___no	___yes
Single limb support>double limb support	___yes	___no
Smooth, fluent progression	___yes	___no
Head centered over body	___yes	___no
Shoulder girdle level	___yes	___no
Pelvic Girdle level	___yes	___no
Pelvic total rotation (via iliac crest)	_____	_____
	<15 degrees	>15 degrees
Arm swing symmetric	___yes	___no
Leg swing directly forward	___yes	___no
Rhythmic stride	___yes	___no
Foot drop present	___no	___yes
LE spasticity present	___no	___yes
Patient stability	____+1	_____0
	Completely Stable	Minimally Unstable	Moderately Unstable	Severely Unstable
	____+1	_____0

Total: _____/17
(where there are a total possible 17 points in a healthy, normal individual)

Lymphatic Manipulative Pump Research: A Brief Review of Literature

Sean Mc Millan, William T. Crow, Charlotte H. Greene

Abstract:

Lymphatic manipulative pump (LMP) is an osteopathic technique that has been utilized with much success for decades. During this time, there have been numerous clinical reports of it's efficacy, however, scientific research to substantiate the findings have been lacking. Research that has been done documenting the effect LMP has upon the body has been promising and this review of the literature highlights current trends in this field, as well as calls for a look ahead to the future of LMP research.

Introduction

Osteopathic palpatory diagnosis and manipulative treatment (OMT) has been a cornerstone of osteopathic medicine since the 19th century. Their clinical efficacy has been reported since the influenza pandemic of 1918.[1] The ability to influence flow and dynamics within the human body for self-healing is central to the training of osteopathic medical doctors throughout the world. However, the lack of controlled studies of every aspect of diagnosis and treatment has led to skepticism and challenges to the validity of this approach.

One of the simplest techniques universally taught and utilized by DO's involves the principle of lymphatic manipulative pump (LMP). Examples of LMP techniques include: thoracic pump, splenic pump, pedal pump, and pectoral traction. Dr. A.T. Still, the founder of osteopathic medicine, stated, "Let the lymphatics always receive and discharge naturally, if so we have no substance detained long enough to produce fermentation, fever, sickness, and death."[2] Lymph, the final component of the lymphatic system, is a clear proteinaceous fluid that travels throughout the body. The primary cellular components of lymph are: lymphocytes, clotting factors, and large particles, such as bacteria and smaller viruses. During fluid circulation within the body, lymph is filtered through lymph nodes allowing access to the immune system. This immune response contributes to the removal of potential pathogens and restores normal immunological functioning. The goal of OMT is to maintain fluid dynamics by regulating and normalizing diaphragmatic movement and lymph drainage using alternating forces of positive and negative pressures.[3]

> *"Let the lymphatics always receive and discharge naturally, if so we have no substance detained long enough to produce fermentation, fever, sickness, and death."*
>
> Andrew Taylor Still, MD

A brief review of the literature documenting lymphatic manipulative pump (LMP) research was undertaken to analyze pertinent clinical data. Principal Internet search engines were utilized employing the following keywords: 1) lymphatic pump; 2) OMT; 3) thoracic duct flow; and 4) osteopathic research. The resulting articles were surveyed and divided into the following broad categories: editorials,[3-7] clinical reports, and reports of scientific studies. The clinical reports and scientific studies were categorized according to these subsets: a) immune system responses,[8-11] b) ENT and respiratory related treatments,[12-14] c) post-surgical use,[15,16] d) use related to pregnancy,[17,18] autonomic effects of LMP,[19-22] and e) changes in lymph flow rates.[23-26] Articles were then selected from subset A (immune system responses) to summarize findings in this area because it is currently the focus of the most active research; and subset E (changes in lymph flow rates) because of the lack of understanding of the mechanism by which LMP increases lymph flow rates throughout the body. This group of articles highlight current trends of the past decade in lymphatic research, and possibly shed light as to the direction where research could be heading.

Effects of LMP on the Immune Response

One of the most studied areas over the past ten years was the effect of LMP on stimulating the immune response. Research in this area began to move to the forefront in aftermath of a 1987 thoracic lymphatic pump review article by Amalfitano[3], in which the need for documentation of the effects of OMT on the immune response was cited. In 1998, Jackson[9] et al. published "The effect of lymphatic and splenic pump techniques on the antibody response to hepatitis B

vaccine". In their research it was hypothesized that OMT would facilitate the movement of lymphatic fluid and may enhance the immunologic response to infection or injected antigen. It was noted that patients receiving OMT had higher antibody titers than those who did not after six weeks and it was concluded that there was an enhanced immunological response in OMT recipients.

Hampton et al. twice published in 1998 on the subject of basophilia occurrence following lymphatic pump techniques. In the initial study,[10] male medical students were divided into 2 groups: those who received splenic pump and pectoral traction, and those who received no OMT. In the treatment pool, 5 ml of venous blood were drawn prior to treatment, and again at 15, 30, 60, 120, and 240 minutes postmanipulation. The control population had blood drawn according to the same schedule. The subjects who received treatment were given 1 minute of splenic pump followed by pectoral traction for 3 minutes. Upon examination of the blood samples, there was an increased basophil count over the baseline levels in all of the patients who received LMP treatment. The control group, all of who received no LMP, exhibited no increase in basophil levels. Later in 1998 Hampton et al. published another paper with similar methodology that supported their earlier study.[11]

On the contrary, a 2001 study by Dugan et al.[8] entitled "Effect of lymphatic pump techniques on the immune response to influenza vaccine" produced results that disagreed with the findings of Jackson et al. The aim of the Dugan et al. study was to demonstrate that OMT facilitated lymph movement and immunological response in the people over age 55, but no significant differences in antibody titers between those receiving OMT versus those who did not were found.

Effects of LMP on Lymph Flow Rates

While the tenet that LMP treatment increases healing of patients due to the increased facilitation of lymphatic drainage has been clinically supported for nearly 80 years; there has been a lack of data to confirm the theoretical mechanism of action. Only within the past couple of years has there been published reports of attempts to correlate LMP and lymph flow rates within the body.

Olszewski explored the contractility patterns of normal and pathological lymphatic changes in human legs.[25] Lymphatic flow patterns were monitored as the leg or foot was taken though a variety of movements by cannulizing subcutaneous lymph vessels. One of these movements was similar in theory to the pedal pump technique. By rhythmically flexing the foot at a rate of 30 per minute, the effects exerted on lymph vessel systolic pressures and pulse frequency could be recorded. No increase in mean systolic pressure from resting baseline was demonstrated, however, an increase in lymphatic pulse frequency ($P<0.025$) was noted.

Dery, Winterson, and Yonuschot at the UNECOM have reported multiple studies analyzing the effects of OMT on the lymphatic systems in rats. They first examined the rate of lymphatic flow in healthy rats that were treated with lymphatic pump manipulation (LMP).[23] Utilizing a florescent probe bound to albumin to monitor flow, the treatment groups received 5 minutes of LMP per hour. A 10% increase of lymphatic movement resulted in treated rats versus the untreated controls.

The effects of lymph flow enhancing treatment (LFET) on a part of the body remote from the regions of lymph uptake into the initial lymphatics were examined in a later study.[24] Flow was assessed using a fluorescent probe placed into a potential space in the lower extremity. Each rat in the treatment group received 5 minutes of manipulation consisting of bilateral finger pressure applied to the lower ribs, followed by a light tap to the sternum. Results indicated that mechanical pressure distant from lymph formation site enhanced lymph uptake.

Finally, a 2003 study by Knott et al. measured the amount of thoracic duct flow in mongrel dogs before and after receiving various LMP techniques.[26] Treatments included thoracic, abdominal, and pedal pump. A Transonics flow transducer was placed over the thoracic duct and used to measure changes in lymph flow rates. Each dog received two pump treatments of thirty seconds duration. The investigators concluded that there was a substantial increase in mean thoracic flow during the treatments. The average rate of lymph flow through the thoracic duct increased by about 1.5 ml/min during thoracic and pedal pump treatment, and by roughly 3 ml/min. when abdominal pump therapy was administered.

Discussion

In 1946, Dr. M. D. Young, DO wrote an article in *The Osteopathic Profession* entitled "Lymphatic Pump Speeds Flow". In this article he spoke of the healing benefits to the human body by performing any number of pump techniques. In summarizing the various techniques, Dr. Young stated the following: "All of the methods mentioned here have worked out beneficially in clinical practice. May we someday fully evaluate them scientifically."[6] Fifty years later, those sentiments were echoed by Degenhardt and Kuchera. Their review closed with the following comment: "Research evaluating the effects of manipulation on lymph flow and its role in facilitating recovery needs to become a priority for centers of osteopathic research."[27] Indeed the osteopathic research community has only begun to do just that. A review of the literature has revealed numerous studies that examine LMP, ranging the use of LMP as a means to measure antibody movement, to the examination of healing rates in patients suffering from influenza. However, only a few of these studies looked at the mechanism of LMP as a healing technique, which is to say, "if LMP works by increasing the rate of lymphatic flow and drainage, by how much does it do so?"

While each of the studies mentioned above represent a positive step in fulfilling the need for quantitative data, the need for further study remains. Extrapolating results from rodent and canine studies assumes applicability to humans. Furthermore, the effect LMP has on the movement of intracellular and extracellular fluid has received little attention. In this age of evidenced based medicine, it seems only logical to build upon these studies in an attempt to acquire statistical support for the validity of LMP and provide avenues to improved patient treatment.❐

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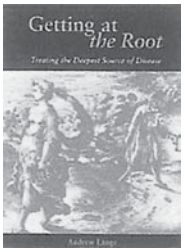
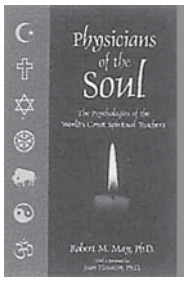
Book Reviews



Getting at the Root: Treating the Deepest Source of Disease

In receiving care from a health practitioner, true healing means more than just suppressing physical symptoms. A skilled healer takes into account the whole person, not just the physical structure of the body. Through a combination of case studies and expositions on his theories of healing, naturopath and homeopath Andrew Lange reveals his vision for a medicine that can incorporate the best of modern allopathic techniques alongside more vitalistic approaches. Just as the original root of the word "healing" points to the notion of "making whole", Lange strives to heal the state of modern medicine itself, so that all may benefit. (North Atlantic, 2001)



Physicians of the Soul

Engaging and relevant, Robert M. May's analysis of the teachings of Lao Tzu, Moses, Jesus, Gautama Buddha, Krishna, Muhammad – and new to this twentieth anniversary edition, a chapter on Native American holy woman White Buffalo Woman – is an absolute delight to read. May is a lecturer and teacher on the topics of religion, philosophy, psychology, and mythology, and he correlates the continuum of these wisdom teachings to their place in modern psychology. As inspiring as it is informative, the book is an excellent work for scholar, teacher, student, and seeker alike., (White Cloud Press 2003).

The Medium, the Mystic, and the Physicist

Truly a classic in the field of the noetic sciences, this book from psychologist Lawrence LeShan has inspired intelligent people to stand up to the nay-say-ers and dogmatic hyperskeptics who claim that evidence for psychic phenomena is pure flim-flam. First published in 1966 after LeShan's quest to debunk parapsychological claims ended up pointing him towards evidence for psychic abilities that he could not refute, this book has been re-released with a new preface from LeShan. The fact that he has not altered the contents of his work indicates that what he found true nearly 40 years ago still stands true today. (Helios Press, 2003).

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Elsewhere in Print

New Thinking About Migraine
Cortlandt Forum: June 25, 2004; 33-41

Cady, R (The author, a family practitioner, is director of the Headache Care Center in Springfield, MO, and a member of the board of directors for the National Headache Foundation).

The author indicates that the understanding of migraine has undergone an evolution during the past 15 years such that it is now a recognized neurologic condition. It is estimated that 28 million Americans are afflicted by this condition, nearly 18% of women and 6% of men. Migraine is discussed as a progressive neurologic process, each of four phases amenable to treatment capable of stopping a complete evolution.

Premonitory or prodrome phase

Diffuse symptoms characterize this phase (fatigue, anxiety, neck pain, mood changes, food cravings, nasal congestion, facial pressure, alteration in sensory processing). Cognitive impairment can also occur. Risk factors may include stress, menses, hormonal changes, diet and environmental factors. Individual specificity often relates to the latter two of these risk factors. Appropriate proactive intervention during this phase can include biofeedback, relaxation techniques, exercise or self-removal from the risk environment. Oral or nasal triptan medications can be moderately effective.

The aura

Visual scotoma and unilateral paresthesias may comprise an aura which can last from five to 60 minutes. Headache usually, but not always, follows. Few effective treatments are acknowledged for aura, but can include OTC analgesics, NSAIDs or triptan medications.

Mild headache phase

A spontaneously resolving headache without significant associated symptoms is generally regarded as a tension-type or sinus headache. There is potential for progression to moderate-to-severe throbbing pain accompanied by nausea and sensory sensitivity. If the mild headache consistently precedes a high-impact headache, triptan medications offer the most effective treatment. Pharmacologically, triptans are serotonin receptor agonists that are selective for a particular 5-hydroxytryptamine receptor subtype which mediates cranial vasoconstriction, reverses inflammatory changes, and dampens sensory conduction of trigeminal input into the brain stem.

Moderate- to-severe phase

Throbbing head pain is associated with nausea and sensory sensitivity, any minor activity making the pain worse. Central sensitization occurs and the potential exists for escalation into an allodynic pain state. This phase can last 24 hours, or persist from hours to several days. Treatment is often more palliative than abortive. The FDA has recently approved rofecoxib for the treatment of acute migraine. In clinical trials, reduction of associated symptoms was significant, as was the need for rescue medications. Common side effects included dizziness, somnolence and dyspepsia, although rofecoxib was generally well-tolerated.

Chronic migraine

A small number of patients may develop chronicity, defined as attacks occurring on more than 15 days a month. Chronicity may be related to both stressful life events and medication overusage, the latter because interventions utilized are only marginally effective.

Prevention

Limits should be set on acute medication usage to avoid analgesic or medication overuse headaches. Therapies prescribed should be migraine abortive rather than palliative. The use of triptans during the mild phase demonstrates nearly doubled efficacy compared with waiting until pain escalates to the severe phase. Regular meals, good sleeping habits, avoidance of caffeine and stress reduction can significantly lower the risk for migraine.

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ONE-DAY PRE-CONVENTION WORKSHOP

How to do OMT without an "OMT" table!

(Treating in an outpatient setting)

November 6, 2004 • San Francisco, California

Ann L. Habenicht, DO, FAAO
Program Chair

The program anticipates being approved for 8 hours of AOA Category 1-A CME credit pending approval by the AOA CCME.

COURSE DESCRIPTION:

Many physicians work in an outpatient setting with high tables or carts on which to examine and treat the patient. This presents a challenge: how can the patient be affectively treated with OMT? This course is designed to offer solutions for treating patients in an outpatient setting without an OMT table. The course is designed for primary care, urgent care and emergency medicine physicians who want to treat their patients with OMT but have been frustrated by the office equipment. Participants will treat on chairs and inadequate height tables. OMT utilizing high velocity/low amplitude, facilitated positional release, Still, muscle energy and myofascial release techniques will be included. Common patient complaints will be addressed.

LEARNING OBJECTIVES:

1. To perform quick and efficient OMT for common outpatient complaints.
2. To demonstrate ability to treat utilizing inappropriate height tables and carts.
3. To demonstrate ability to treat utilizing chairs.

PROGRAM TIME TABLE:

Saturday, November 6 ..... 8:00 am - 5:00 pm

(lunch on your own)

HOTEL INFORMATION:

Hotel information will be published by the American Osteopathic Association in the *Journal of the American Osteopathic Association (JAOA)* and *The DO* magazine early summer 2004.

For registration information, please contact:
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