THE AAO
FORUM FOR OSTEOPATHIC THOUGHT
JOURNAL
Official Publication of the American Academy of Osteopathy®
TRADITION SHAPES THE FUTURE • VOLUME 14 NUMBER 4 DECEMBER 2004

"How many of you will commit malpractice?"

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.

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

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

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

3. Include a caption for each figure.

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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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THE AAO JOURNAL
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TRADITION SHAPES THE FUTURE • VOLUME 14 NUMBER 4 DECEMBER 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.

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AAO Calendar of Courses

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

Remembering Doctor Still

Andrew Taylor Still suffered a stroke in 1914 from which he never fully regained his speech. Declining health eventually resulted in his passing on December 12, 1917. He was buried at Kirksville, MO on December 14, 1917. It is appropriate that acknowledgment be given as a reminder of the osteopathic profession's debt to the contribution and vision of its founder.

George W. Riley, DO was a graduate of the American School of Osteopathy, Class of 1904. He also served as President of the American Osteopathic Association, 1917. Excerpted below are some of Doctor Riley's recollections of his student days and the Old Doctor[1]:

"Although I had completely severed my business relations in New York and had come to Kirksville with every intention of studying osteopathy, nevertheless, I spent my first two weeks in visiting classes, talking with members of the faculty, with students, with patients going to and from the infirmary, with citizens of the town, trying to rid my mind of that element of doubt, aye 'doubting Thomas' – like trying to find the pierced side wherein I might place my hand and clinch my faith in this momentously new venture I was about to undertake.

Finally my mind was satisfied and I entered into the work with a zest and faith seemingly impossible for one who had been beset with such hesitancy.

The members of our class (June, 1904) came from all sections of the country – from village, farm, and city. I presume every member of the class was over twenty-three years of age, maybe twenty-four, many far beyond that age, and probably everyone had learned the value of a dollar by the sweat of his brow. Everyone gave the impression of knowing what he was there for, of knowing, 'what the shootin' was all about.' This possibly is most pronounced in more mature classes who probably look with greater zeal on the practical goal sought than the more common cultural aim of classes composed of younger students.

I was very much impressed with the spirit of osteopathy that pervaded not only the whole life of the student body, but that of the citizenry of the town as well. This is easily accounted for when we take into consideration the fact that there came into the little city of Kirksville, each term, a combined outside population of between 600 and 1000 students and patients, whose aim and interest in life, for the time being, was osteopathy and what it could do for afflicted humanity. This number had to be taken into the homes of the citizens. Their interests therefore pervaded the whole life of the town. As a result we all ate, drank, and slept with osteopathy.

We, as a class, were particularly fortunate in having the subject, principles and practice of osteopathy, under Dr. Guy Hulett. Our diplomas were the last signed by that great teacher, cut off in the early dawn of what gave promise of being a remarkable career. He was a conscientious believer in the science and a man thoroughly grounded in the fundamentals of osteopathy. His was perhaps the keenest, most logical, and scientific mind of the younger members of the profession of that day. A superior student himself, he possessed to an unusual degree the faculties of a real teacher. It was always a joy to watch the look of approval on the face of the Old Doctor when listening to a lecture by Dr. Hulett. His death removed from the profession one of its keenest, most brilliant intellects.

We again were fortunate, indeed more fortunate than we perhaps realized, in having frequent visits to our class of our revered Old Doctor. In a spirit of sympathetic humility I feel sincerely sorry for all those of the profession who never enjoyed the privilege of knowing him personally, or of hearing his rare epigrammatic thought-provoking remarks, and those seemingly inspired diagnoses of his.

Frequently he would come into the classroom, always unannounced, listen attentively to the lecture or discussion and maybe after a few minutes pass out as quietly as he had entered. More often, however, he would make some quaint observation that would forever fix in our minds the fundamental osteopathic principle underlying the subject under discussion. …Perhaps the outstanding impression I have of him was his eager, passionate desire for every student to become thoroughly imbued and grounded in the fundamentals of osteopathy. That was his great goal, his supreme ambition. The one disturbing thought that he seemingly had sometimes, was that insufficient emphasis was being given these fundamentals in our class work. He wanted each student to know these principles and to know that he knew them."

1. Hildreth, AG: *The Lengthening Shadow of Andrew Taylor Still*. Macon, MO. 1938. 415-417.

Contributors

Dennis J. Dowling. Don't Raise Your Hand – Put it On The Patient. The 2004 Scott Memorial Lecture addresses essential osteopathic practice in the context of a significant contemporary social occurrence: Malpractice. In recognizing the changes in educational environment for osteopathic education during the past twenty years, the author makes the case that the best defense for practice continues to be the application of the hand to the patient. **(p. 10)**

James A. Lipton and Michele L. Neil. A Case Study of Left Adhesive Capsulitis Presumably Resulting from Previous Treatment with Protease Inhibitors. The authors present an unusual case study in which circumstantial evidence suggested association of the chief complaint with prior administration of agents for protection from an HIV infected source. In this instance, reconciliation of diagnostic findings and clinical presentation rested on the eventual casual reporting of an overlooked incident in the medical history. **(p. 17)**

Yvette Somoano and Stefan Hagopian. An Osteopathic Resolution of Neurocardiogenic Syncope. The authors' presentation of this complaint (Neurocardiogenic Syncope) demonstrates the value of application of osteopathic thought. The clinical history suggested that a complex series of imbalances had occurred over many years, probably associated with the cranial base and upper thorax. The rationale provided for the successful use of osteopathic manipulative intervention is striking given the variety of less than successful conventional medical interventions. **(p. 20)**

Rafael Zegarra-Parodi. Could Joint Hypomobility Alter Optimal Proprioceptive Information? The author provides an opportunity for expanding thought in this review of contemporary attitudes about Joint Complex Dysfunctions (JCD). An osteopathic perspective begins with an expression by John Martin Littlejohn. The continuing search for understanding of complexities associated with JCD has its current expression as *Somatic Dysfunction*. In today's research environment, other disciplines are offering observations and interpretations, which may be helpful. **(p. 25)**

Regular Features

DIG ON. A Program Of Research. Albert E. Guy, DO practiced in Paris, France and Mount Vernon, New York. A small sampling of his extensive writing during the years 1930-1933 indicates that he was very proactive in suggesting an organizational plan for research for the osteopathic profession. **(p. 8)**

FROM THE ARCHIVES. The name of George Malcolm McCole is not frequently recognized in today's teaching of osteopathic theory and practice. Seventy years ago this Montana practitioner offered a classification of osteopathic spinal lesions which remains useful today. Of particular note from that era is the descriptor *The Osteopathic Greater Lesion Complex*. **(p. 9)**

BOOK REVIEW. Principles of Manual Medicine. It is unusual to have the opportunity to follow the contribution of thought of a single author through three consecutive editions of a textbook. In the case of Philip E. Greenman, DO, FAAO, this fact encompasses fifty years of practice and contribution to major developments in osteopathic education, here and abroad. **(p. 32)**

ELSEWHERE IN PRINT. Do CAM therapies work for pain management? Authors Robert Bonakdar, MD and David E. Bresler, PhD address the fact that existing approaches to disease and pain do not work for all patients. Four Complementary Alternative Medicine approaches are discussed. In an additional citation, the pioneering work of Earl H. Gedney, DO (Hypermobile Joint) is recognized. **(p. 33)**

CME CREDIT. In response to reader requests, *AAOJ* will offer CME Credit to readers completing the enclosed quiz. At this time, 1 Hour 2-B Credit will be offered, with request for upgrade as *AAOJ* qualifications are reviewed by the **American Osteopathic Association**. **(p. 24)**

Component Societies' CME Calendar

and other Osteopathic Affiliated Organizations

January 8, 2005
Osteopathic Dissection Project of the Head & Neck
Central California Osteopathic Study Group
Soquel, CA
CME: 7 Category 1A (anticipated)
Contact: Bonnie Gintis, DO
831/477-1200

January 20-23, 2005
16th Annual Osteopathic Winter Seminar and National Clinical Update
Pinellas County Osteopathic Medical Society
St. Pete Beach, FL
CME: 27 Category 1A (anticipated)
Contact: Kenneth E. Webster, EdD
727/581-9069 or
866/254-8798

January 21-24, 2005
Biodynamics Phase II: The Fluid Body
Portland, OR
CME: 23 Category 1A (anticipated)
Contact: James Jealous, DO
207/778-9847

January 28-31, 2005
Biodynamics Phase VI: The Embroyological Health
Portland, OR
CME: 24.75 Category 1A (anticipated)
Contact: James Jealous, DO
207/778-9847

February 11-13, 2005
Energetically Integrated Osteopathic Medicine: The Life, Thought and Work of Robert Fulford, DO as Interpreted by Zachary Comeaux, DO, FAAO
WVSOM; Lewisburg, WV
CME: 12 Category 1A (anticipated)
Contact: Zach Comeaux, DO, FAAO
304/647-6356

February 16-20, 2005
Midwinter Basic Course Osteopathic in the Cranial Field
Course Director:
Richard A. Feely, DO, FAAO
The Cranial Academy
Tampa, FL
CME: 40 Category 1A (anticipated)
Contact: The Cranial Academy
317/594-0411

February 26, 2005
OMM for the Family Practitioner
Arizona Academy of Osteopathy
Glendale, AZ
CME: 7 hours Category 1A (anticipated)
Contact: William Devine, DO
623/572-3350

March 5-8, 2005
Biodynamics Phase I: Biodynamics
Kona, Hawaii
CME: 21.5 Category 1A (anticipated)
Contact: Thomas Shaver, DO
207/778-9847

April 1-4, 2005
Biodynamics Phase IV: The Midline
Topanga, CA
CME: 23 Category 1A (anticipated)
Contact: Stefan Hagopian, DO
207/778-9847

April 2-5, 2005
Biodynamics Phase II: The Fluid Body
Franconia, NH
CME: 23 Category 1A (anticipated)
Contact: Donald Hankinson, DO
207/778-9847

April 17-20, 2005
Biodynamics Phase III: The Long Tide and the Dura
Franconia, NH
Faculty: James Jealous, DO and Donald Hankinson, DO
CME: 22.5 Category 1A (anticipated)
Contact: James Jealous, DO
207/778-9847

May 8-11, 2005
Biodynamics Phase VI: The Embroyological Health
Franconia, NH
CME: 24.75 Category 1A (anticipated)
Contact: James Jealous, DO
207/778-9847

May 15-18, 2005
Biodynamics Phase VII: The Health Alone
CME: 22 Category 1A (anticipated)
Franconia, NH
Contact: James Jealous, DO
207/778-9847

May 20-22, 2005
Crash Recovery The Long Road Home: Treataing Victims of Motor Vehicle Accidents and Brain Injuries
UMDNJ/SOM, Stratford, NJ
CME: 17 Category 1A (anticipated
Contact: The Cranial Academy
317/594-0411

May 22-25, 2005
Biodynamics Phase I: Biodynamics
Franconia, NH
CME: 21.5 Category 1A (anticipated)
Contact: Thomas Shaver, DO
207/778-9847

Dig On

Anthony G. Chila

A Program of Research

The decade of the 1930s was a period of significant contribution to medical practice expressed through extensive writing by osteopathic physicians. From that period of time one can easily construct a pantheon of distinguished practitioners and teachers who offered much direction and guidance for the continuing development of the osteopathic profession. Among those names: Burns, Conley, Downing, Hildreth, and a host of others not all remembered or whose written contributions are now out of print. In pursuing the development of Andrew Taylor Still's philosophy, the American Osteopathic Association established the Committee on Research in 1939.

Albert E. Guy, DO wrote extensively about Vertebral Lesions and Vertebral Mechanics. Some of his most important observations were published in *The Journal of the American Osteopathic Association* during the years 1930-33. As a practitioner, he observed that "The practice of osteopathy has fully demonstrated its worth, but the development of its theory, so urgently needed *in view of the advance of others*, has not progressed very far beyond the *dicta* of A.T. Still. Guy proposed the following considerations for osteopathic research[1]:

1. The vertebral column viewed as an entity
2. The vertebral unit, composed of two adjacent articulated vertebrae
3. The intervertebral disk; the annulus lamellosus; the nucleus pulposus; the exonuclear lesions
4. The intervertebral ligaments; their innervation; their control functions
5. The apophyseal articulations; the mechanics of their displacements
6. The deep vertebral musculature; its innervation
7. The contents of the intervertebral foramen
8. The nerve sinu vertebral; its function as vaso-regulator of the blood supply and drainage of the meningeal tissues and of others located in the vertebral canal and in the intervertebral foramina
9. The supporting or connective tissue; its hygrometric properties; its role in edematous and inflammatory processes in relation with the lymphatic circulation
10. The purpose and physiological effects of the abrupt and intentional separation of the apophyseal articulations, with incidental "popping"; the physics of the latter
11. The analysis of the maintenance of the relative displacement of two adjacent vertebrae, or so-called "lesion", in a position within the normal range of motion, nevertheless permitting a certain amount of mobility
12. The pathological effects of the maintenance in a fixed position, either normal or abnormal, of the articulations of two adjacent vertebrae; the remedial procedure through osteopathic manipulation for such a condition
13. The analysis of the effects of osteopathic manipulation of soft tissues; drainage of the latter; abatement of congestive conditions; activation of arterial circulation; reduction of acidosis, hence of irritation both to the nerve terminals and to the trunks, and consequent appeasement of superficial and deep-seated tenderness
14. The costovertebral articulations; the influence of their disordered conditions upon the nutrition of the costal tissues, ligaments, musculature, bones, marrow and its hematopoietic functions; study of the development of eruptive disturbances such as herpes zoster, of costogenic anemias and toxemias, of the influence of the latter upon the genital functions

Guy's challenge in offering this program was expressed as "In the field of research concerning the intimate parts of the spine it was our heritage to lead; shall we be contented with merely joining?" Given the growth of the osteopathic profession since that time, how far have we progressed?

1. *Academy of Applied Osteopathy Yearbook*. 1949. pp. 72-73.❐

From the Archives

Chapter VII, Table IV, Four Great Classes of Osteopathic Spinal Lesions

George Malcolm McCole, DO: *An Analysis of the Osteopathic Lesion*, 1935, pp. 35-39

Osteopathic spinal lesions are classified according (1) to their *cause* and (2) with respect to their *age*.

According to cause there are two great classes of spinal lesions, (1) **Traumatic** and (2) **Reflex**. With respect to state of development or age of the lesion at the time of observation there are two other great classes, (3) **Acute** and (4) **Chronic**.

In practice, however, classification is not so simple as the following discussion shows.

The (1) **Traumatic Spinal Lesion**, instantly upon its formation, has reflex symptoms caused by impulses from spinal cord centers in relation. The traumatic lesion may be either acute or chronic.

The (2) **Reflex Lesion** is caused by impulses abnormal in intensity or continuousness, which impulses arrive from a distant part. These stimuli first reach the cord centers of the segment and from there are distributed to the surrounding joint muscles and blood vessels. After its formation the reflex lesion of itself sends abnormal impulses into the cord centers of the segment thus increasing the volume of stimuli distributed from these centers.

The (3) **Acute Lesion**, soon after formation, begins to develop chronic attributes from fibrous infiltration. The acute lesion may be either traumatic or reflex.

The (4) **Chronic Lesion** always retains some acute symptoms, which with slight provocation definitely increase. The chronic lesion may be either traumatic or reflex. There is no actual dividing line between the acute and the chronic.

The speed with which an acute lesion takes on chronic attributes is governed by the reactions of the individual, the life he leads, and the burdens he requires his spine to bear.

Acute Lesion at Site of Chronic Lesion

The acute lesion is often found at the site of a chronic lesion, which has impaired the muscular, and ligamentous control of the joint and caused it to be susceptible to fresh insult. A chronic lesion, hitherto unnoticed, may flare up with acute symptoms and thus come under observation.

Chronic Lesion is the Result of Acute Lesion

The chronic lesion is often the result of several acute lesions having at various intervals succeeded one another in a certain segment. Nature unaided, apparently removes many acute lesions, but the effect of inflammation repeatedly occurring in the tissues of a certain joint is cumulative, and the formation of fibrous tissue in muscle fasciculi and in ligament-fibers is stimulated. This formation is called the 'fibrous contracture" of the chronic lesion.

The Causative Lesion

The traumatic lesion is sometimes called the causative lesion, and it is. However, it must be remembered that *the reflex lesion becomes causative as soon as it is formed. If it were not, it would not be an osteopathic lesion.*

Reflex Symptoms Complicate Traumatic Lesions

When a lesion of a certain spinal segment (acting through efferent reflex nerve pathways) causes disease in a part remote from the spine, that diseased part sends abnormal nerve impulses streaming back (over afferent nerve pathways) to the lesioned segment, to set up reflex tensions in the muscles of that segment. Therefore, reflex lesion symptoms develop wherever there is a traumatically lesioned segment – the vicious circle.

Again: When abnormal nerve impulses are being sent out from a spinal cord segment, we think of them as going to parts remote from the spinal joint. These abnormal impulses are distributed to the short, deep, segmental muscles surrounding the joint, just as they are sent to tissues remote from the spine. In fact, the distribution of abnormal nerve impulses seems to be more promptly concentrated in the close-by short segmental joint muscles than is the distribution to more distant tissues, which are occupied with other kinds of duties and where there is better opportunity to integrate.

Bone Malposition

In the traumatic lesion the bone is usually held seated in position of abnormal side-bent rotation. Thus, bone malpositions are more frequent in traumatic than in reflex lesions. For the same reason the traumatic lesion is apt to be unilateral rather than bilateral.

Compound Lesions

Therefore, although lesions are divided into four great classes, they are all in some degree compound. All osteopathic spinal lesions tend toward the same pathology, irrespective of cause or time elapsed since formation.

continued on page 19

2004 Scott Memorial Lecture

Don't Raise Your Hand – Put it on the Patient

Dennis J. Dowling

How many of you wish to commit malpractice in the future? I see that no hands were raised.

As defined in the Merriam-Webster dictionary, Malpractice is:

1: a dereliction of professional duty or a failure to exercise an accepted degree of professional skill or learning by one (as a physician) rendering professional services which results in injury, loss, or damage

2: an injurious, negligent, or improper practice[1]

Legally, there are several elements to malpractice. In most states, in order to prove medical malpractice, a physician's duty to act is judged according to certain standards of care. Next, there is a breach of that standard of care. Usually, this results in an injury and a causal connection between the breach of care and the injury can be established. A doctor must act in a reasonable and prudent fashion as would anyone possessing the same or similar skills or knowledge. The expectation is that the same treatment intervention would be given by another physician under similar circumstances. The consideration of the caregiver's location and the standard of care to a similar community has shrunk due to the explosion in information availability.

There is also another element utilized in malpractice situations known as "loss of chance". The *loss of chance* doctrine in medical malpractice actions refers to "the injury sustained by a patient whose medical providers negligently deprived the patient of a chance to survive or recover from a health problem, or where the malpractice lessened the effectiveness of treatment or increased the risk of an unfavorable outcome to the patient."[2]

Legally and clinically, the patient is deprived of a chance for successful treatment. That decreased chance for successful treatment more likely than not resulted from the doctor's negligence. In other words, the doctor could have done something and did not. Please remember that errors can be ones of commission, where something adversely is performed, and of omission, where something could or should have been done but was not. A doctor who fails to do therapeutic interventions for which he or she has been trained is depriving the patient of the greatest chance to recover. Worse yet would be if it was not only neglected, but knowingly withheld.

"How many of you will commit malpractice?"

Now that these definitions of legality have been explained, I will ask the question again: "How many of you will commit malpractice"? There is a subtlety there. I asked the first time "How many of you *wish* to commit malpractice in the future"? Now, I am asking the question to determine how many of you think that you will commit malpractice. You may not even realize that your future behavior may be considered malpractice. Do not worry, I am still asking a rhetorical question. I do not expect any of you to raise your hands. We do know that in today's litigious American society that it is likely that for every three of you, one will be sued. Being sued does not mean that malpractice has been committed. That is not my question. My question is more in regards to determining the fact that by your actions, or, more to the point, inactions, patients will suffer because they have not been given optimal treatment. Some will suffer because they lost a chance to get better or survive. It will be your responsibility as an osteopathic physician to assist them and failure to do so may place them at a critical disadvantage. I am interested in determining your desire to provide the best for your patients. No matter which stage of training, we have all made the commitment to become practitioners of the osteopathic medical profession. That means more than getting a degree; it is a responsibility that must be undertaken with eyes wide open. When I took the *Osteopathic Oath* I swore to "be mindful always of my great responsibility to preserve the health and life of patients; to retain their confidence and respect, both as a physician and a friend, who will guard their secrets with scrupulous honor and fidelity; to perform faithfully my professional duties; and to employ only those recognized methods of treatment consistent with good judgment and with my skill and ability, keeping in mind always nature's laws and the body's inherent capacity for recovery." This is the oath taken by all graduates of osteopathic medical schools.

There are those who say that osteopathic manipulative medicine is unproven. They will include, the health insurance industry, hospital organizations, the community and unfortunately, even your colleagues and teachers, DOs as well as MDs. If you are one of them, then the course in osteopathic manipulative medicine will only be an obstacle to hurdle. That is sad. If you believe in OMM, then you will be put into the un-

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enviable position of nearly constantly defending its effectiveness. We no longer appear to need to defend ourselves as physicians in the arena of medicine, but must do so in regards to our unique aspects. What is it that others feel needs to be proven? It is as if the standard of what else is being done in the name of medicine has been thoroughly proven. At one time, the then U.S. Office of Technology Assessment (OTA) reported that, "Only 10 to 20 percent of all procedures currently used in medical practice have been shown to be efficacious by controlled trial."[3] However, osteopathic manipulation has been studied. Much of it was in step with the principles of statistical analysis of the time. We must remember that the practice of osteopathy beyond a single practitioner is only 112 years old and the use of single- and double-blind studies is only a half-century old. The term "evidence-based medicine" is being used frequently nowadays, and that is a good thing. We should control the care of patients in a way that has been demonstrated to cause no harm and, hopefully, help. However, evidence-based medicine is best suited for pharmaceutical studies and less so to other types of care and intervention. Evidenced-based approaches are actually a study of studies. We hope that everything that we do for the benefit of the patients actually benefits them.[4] Any medication, surgery, and any other intervention, including manipulation, can help or injure. A colleague, Kenneth Johnson, DO uses a quote that "all treatments cause harm; some cause good." Our goal is to maximize and apply the good as much as possible. Despite being used by students and others with little experience, there are very few instances where the application of OMT has caused any irreversible side-effects.

When compared to 20 years ago, there are nearly twice as many osteopathic schools, three times as many graduates, and very few osteopathic hospitals. Our graduates are spending the large majority of time in their undergraduate and post-graduate training in hospitals where there is little or no supervision for the application of osteopathy. However, the use of OMT and the application of osteopathic principles and practice are related to ongoing learning of OMT after graduation, continued interest during internship and residency, and emphasis placed upon these by the training institutions.[5] Yet the evidence is mounting that the osteopathic approach is not only safe, but effective. If you as a physician could do something simple and decrease the need for intravenous antibiotics, decrease hospital stays by days and makes it possible for patients to go home days earlier than they would have otherwise, would you not do it? Hospitals are no places for sick people. That sounds like a contradiction, but it is a sad ironic recognition that even in modern days, hospitals are dangerous places for the very people they are designed to serve. It is a fact with which you will too soon be confronted. Medication and other errors as well as nosocomial (hospital-acquired) infections are just a few of the dangers that claim the lives or injure tens of thousands per year. A chance to be home, to get rest at night or even during the day, to eat the food that one chooses, to be more easily with family and friends, and other benefits can be accrued by leaving the hospital. Anyone who has been in a hospital knows that a hospital is one of the worst places to get rest. What could be done? There is good enough indication that osteopathic approaches assists in earlier discharge from the hospital setting with a concomitant decrease in medications. We have Dr. Noll, who is on faculty here at KCOM to thank for this and several other studies substantiating osteopathic manipulative medicine.[6,7] Another small study of patients with pancreatitis showed a mean reduction of 3.5 days of hospital stay with the treatment group receiving only 10-20 minutes of an OMT standardized protocol.[8] Think of the savings in money alone by multiplying this by each day by approximately $1300-2500.[9,10] Think of the lives saved.

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Would you spend a few minutes with a patient and enhance his respiratory status? OMT has been compared favorably to the use of incentive spirometry following surgery.[11] Incentive spirometry is a plastic device given to patients, especially post-operatively, to use to increase their ability to breathe more deeply. They often do not use them because it hurts. The biggest advantage is that patients can be passive and receive OMT that facilitates the healing processes where they may have had difficulty in performing the incentive spirometry. It all takes a few minutes on the part of the osteopathic physician. Patients with chronic asthma demonstrated measurements of both upper thoracic and lower thoracic forced respiratory excursion statistically increased after osteopathic manipulative procedures compared with sham procedures.[12] A study done in the 1930s, the pre-antibiotic era, demonstrated that hospitalized children with bronchial pneumonia had one third of the mortality rate of children in a non-osteopathic hospital with the same condition. The hospitals were county facilities in similar types of communities in Los Angeles and New York City respectively. It is also interesting to note that the deaths from lobar pneumonia were equivalent. Think of the consequences: the children in Los Angeles who survived are quite possibly grandparents now all because they received osteopathic manipulation.[13] In a similar application of lymphatic pumping techniques, there was at least 1/20th and as low as 1/40th of the reported mortality in those who were treated by osteopathic physicians during the Spanish Flu pandemic of 1917-18, depending on which article one reads.[14,15] An estimated half a million Americans died directly or indirectly from the flu those years. How many of those could have been saved had osteopathic approaches been the standard of care? A few hundred thousand, at least, may have lived. I know from an analysis of my own family that my grandfather was one of thirteen children, including his twin brother. Only three survived the flu, not the twin. Think of the toll on a family to need to bury more than three quarters of their children within a year's time. Had there been 20 times as many

osteopathic physicians as there actually were, perhaps the outcome would have been very much different.

If you needed to receive a vaccination and were offered the opportunity to feel more confident that you had greater immunity earlier, wouldn't you choose to be in that group? It has been demonstrated that there was greater immunity to the majority of the pneumococcal sub-serotypes in the OMT treated group versus the untreated one[16]. Dr. Measel at the Texas College of Osteopathic Medicine performed this study in the early '80s. The researchers at the West Virginia School of Osteopathic Medicine showed that 50% of the OMT treated participants in their study had what were considered to be immune antibody levels after two inoculations for hepatitis B as opposed to 16% of the control subjects. The mean antibody level, the innate immuno-protecting armies of these individuals, remained higher for all 34 weeks of the followup but was statistically so for 25 of the weeks of the study.[17] Statistics is a strange tool. Years ago, my father-in-law was faced with the prospect of getting an angiogram. He did not have any real symptoms and he was not sure that he wanted to take the risk. When he asked them what were the chances of something going wrong, they answered 1/3000. His next question was, "What number are you up to"? He understood their thinking, but I wonder if they understood his. He wanted to be in the successful group or at least the group without complications. Would you not want to be in the group with the best chances.The current protocol for exposure to Hepatitis B in a non-immunized person is to give an immediate injection followed by another, one month later. Shouldn't we be treating all of our patients with OMT following vaccination for Hepatitis? Why isn't it a standard protocol to treat all patients presenting for routine inoculation? These concepts changed the way in which I practiced many years ago. Prior to this, I would have had the medical assistant give the injection and I barely needed to do anything with the patient. I subsequently offered treatment with OMT to all such patients. Years ago, I had to coordinate immunization to hepatitis, measles, mumps, and rubella for a whole class of osteopathic medical students. There was a station where we checked their records, another where we gave injections, and a third where they performed lymphatic pumping techniques to each other. I hope that in some small way, their chances of immunity were assisted.

Eight years ago, my family bought a dog, a Shetland sheepherder that we named Amy. As part of the purchase, we were given two free visits to a veterinarian. It turned out that Amy had otitis media, an inner ear infection. After showing me how to clean her ears, administer her pills, and put in eardrops, Dr. Z., the veterinarian, then proceeded to show me a procedure of mandible and cervical stroking that was very familiar to me. He was performing the Galbreath technique for Eustachian tube and cervical lymphatic drainage. He informed me that he was taught to do so in veterinarian school many years before. Perhaps, they evolved

"What else do you have to offer the patient, or rather the anxious patient's mother and father? Quite simply, you could use those instruments at the end of your arms to facilitate the patient in finding health."

the technique independently or someone years ago learned it from an osteopathic physician. Is it not strange that our pets receive a standard treatment but our typical patients do not? Miriam Mills, MD studied osteopathic manipulative treatment in children with otitis media. She and her colleagues demonstrated that the treated group had significantly lower incidence in almost all components: fewer episodes of acute otitis media, fewer surgical procedures, great number of mean surgery-free months, and the relative absence of hearing changes. The results were published in the *Archives of Pediatrics and Adolescent Medicine*[18]. Other osteopathic sources have discussed these advantages for decades. You also should consider that the American Pediatric Association, the New York Health Department, and many other entities are highly recommending that antibiotics not be used in children who have otitis media. The indications for use of antibiotics have narrowed tremendously. The concern is that frequent antibiotic exposure results in resistance and super-infections. For decades, doctors would try to mollify parents and children by giving antibiotics. The most dangerous and destructive bacteria have been quite effectively prevented by the HIB vaccine. The prescription pad will more likely do harm and not good by indiscriminate prescribing. What are the other alternatives? Children are uncomfortable and parents are distressed by having to watch helplessly as they suffer. The placement of myringotomy tubes to drain fluids has been unsuccessful in so many cases. What else do you have to offer the patient, or rather the anxious patient's mother and father? Quite simply, you could use those instruments at the end of your arms to facilitate the patient in finding health. You will want studies to back up your use and they will be coming. The Osteopathic Research Center is in the process of securing funding for larger studies in this and other areas. However, you do not need to and should not wait for studies when clinical practice has been demonstrated to help. Because of this, the use of osteopathic manipulative medicine should be the standard of care.

We can go on and on with "evidence". The literature is available. Standard medical care combined with OMT was found to be more efficacious in patients with fibromyalgia.[19] It has enhanced the ability of patients with Parkinson's disease to move more easily as demonstrated by our study and work at the New York College of Osteopathic Medicine years ago.[20] There are some clear and simple studies indicating the effectiveness with low back pain.[21,22] For any more proof, you have only to walk through the halls outside this room and read the poster presentations of osteopathic research conducted here at KCOM.

There will be people who say that there is no evidence that OMT is beneficial yet they will not have evidence that it is not. By listening to them and withholding treatment, you will be robbing the patients who trust you of a chance to benefit. As a graduate of an osteopathic

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medical school your training is equivalent to that of the other nearly three thousand new osteopathic physicians graduating every year. To not incorporate osteopathic principles and OMT into the common practice is to leave the door open for the "loss of chance" doctrine to be employed when a patient could have been helped. In your future careers, you, and all DOs should be held to a standard of care in which OMT is the norm, the expected, and the necessary. Will failure to use OMT be grounds for malpractice as some have suggested? [23] I do not know that, but I do feel that we need to meet the needs of our patients. The excuse that there is not enough time, desire, or recall of how to do treatment with osteopathic manipulation is downright lame. It is not only that, it is negligent. I find it incredible that students would admit to an attending physician that they forgot how to do OMT, when there is practically no other diagnostic or therapeutic skill that they had performed even once where they would admit to a lack of ability. If they have seen and helped with doing a procedure, you can be certain that they would want the opportunity to do it themselves. The fact that osteopathic students and physicians claim to forget gives an indication that they had not put enough emphasis on these aspects in the first place. It indicates that the individuals maintained the practices that they used as college students of earlier times; binge and purge. One would learn what was required for an exam and forget it soon afterwards. Despite the overwhelming substance of what you must learn to be a graduate of an osteopathic medical school, you must continue learning to learn, retain any skills and knowledge that will help your patients survive and thrive, and be the best osteopathic physician that you can be. In order to diagnose the patient correctly give the correct and thorough treatment then the most logical and appropriate approach would be the osteopathic one, including osteopathic manipulation. That should be the standard of care. There may come a time when those who do oversight might enforce it. Manipulation is already considered a first line intervention for low back pain. There may come a time when the literature that we as a profession have been producing to document and substantiate our uniqueness will be used to also demonstrate that we have not been holding to our own standard of care.

"Despite the overwhelming substance of what you must learn to be a graduate of an osteopathic medical school, you must continue learning to learn, retain any skills and knowledge that will help your patients survive and thrive, and be the best osteopathic physician that you can be."

From personal experience, I have never been disappointed in the advantages of my training. On my first rotation in a hospital as a third-year osteopathic medical student, I rounded the floors with five MDs: an attending physician, a third-year resident, a second-year resident, and two first-year internal medicine residents. Even though they had never had experience with an osteopath of any type, they accepted me. I worked hard to make their jobs easier by doing everything that was asked of me and more. One day, we came to one of the inpatient service patients who had been admitted the day before for intensive workup and treatment of an intractable headache. He had had this headache for eight weeks and they had done all possible lab work, angiograms, EEGs, and CT scans. They had even shipped him by ambulance to the one hospital in New York City that had a new MRI machine. Yes, those were days of archaic practice; there was only one MRI machine in all of New York City. Now, they are almost as common as pizza parlors. The physicians at our hospital were even going to do a temporal artery biopsy despite the fact that there was no other evidence to indicate doing this procedure. After examining the patient, the others had expressed their frustration in dealing with the patient's condition. I asked if I could examine the patient and treat him. The residents were shocked but the attending physician stated that he did not know what it was that I would do differently. I explained it to Dr. R., the attending, and he agreed that I could do so after rounds. An hour later, I pulled the curtain around Charlie's bed and examined him. With no more training than one would have after two years of education here at KCOM or any other osteopathic medical school, I treated him with osteopathic manipulative medicine. He gave a gasp and then a sigh 30 minutes into the treatment. I asked him to describe what it was that he had felt. He stated that his headache was gone for the first time in two months. It was all that I could do to keep him from jumping out of bed. He got out anyway and went to the nurses' station where he told the residents and the nurses that I had "cured" his headache. When the attending heard, he called me to his office. The residents accused me of having some brass body parts and advised that I should be afraid since Dr. R. was the most feared attending in the hospital. When I subsequently met with him, Dr. R. asked what I had actually done. We talked for awhile and he asked if I could treat the patients in the Respiratory Intensive Care Unit. He was so pleased with the results that I was to go to the RICU every day and he would include an order on all patients. Two years later, I rotated through the hospital again, this time on a Pulmonary rotation, Dr. R.'s specialty. I met up with Dr. R. the first day while he was doing RICU rounds. He did not immediately acknowledge my presence. As we finished, he told the crew that he was taking his geriatric boards the next day. This was a physician who was already board certified in internal medicine, pulmonary medicine, critical care medicine, and emergency medicine. I asked him several questions about geriatrics and everyone looked at me strangely. Remember, he was still the "most feared Attending in the hospital." He said that I was to meet

with him personally when he came back two days later. When we met, he told me that 8 out of 10 questions that I asked him were on the geriatric boards. I was to continue to treat all of the RICU patients and anyone else whom he referred. Not only that, I met Charlie again in the outpatient clinic when he brought his son in for treatment of asthma. He was so thrilled and told everyone about our experience and insisted that I alone treat his son. This story does not end there. Years later when I was on the faculty at the New York College of Osteopathic Medicine, one of the students was in the clinic and asked if I remembered Dr. R. because my name came up one time when they were on rounds. I asked the student how that happened. He said that Dr. R. had asked the osteopathic students to treat his patients. When they informed him that they did not know how, he proceeded to yell at them at length and stated that they were stupid and that he knew that at least I had been able to treat his patients with OMT. I do not know if you will necessarily have a Dr. R. to berate you into doing OMT, but maybe you should. Perhaps, like Jiminy Cricket, there should be some voice in your head urging you to do the right thing for your patients.

Osteopathy is based on the principles first presented by Andrew Taylor Still, MD and refined by others: The person is a whole; structure and function are interrelated as are abnormal structure and abnormal function; disease occurs when the individual is overwhelmed or under-prepared; the person has a unique ability to repair, defend, compensate and adapt; and the physician is a facilitator who utilizes these principles to assist the patients in finding health. Any physician should be practicing in this manner. However, you have and will be given an advantage; the ability to use your hands and mind to diagnose and treat. Many medical textbooks decry the fact that palpation as a tool is a lost art. Secondly, the directed use of one's hands to effect a change in a patient's condition is a method that goes back thousands of years and throughout many cultures. In today's medical practice, it too has fallen by the wayside, with the apparent exception of the osteopathic profession. The tradition has been passed down for 11 decades to us and is in danger of dying despite the fact that medical science is catching up. Too many of our graduates take what they feel is either the quick or "approved way" to patient care. With the acceptance into the non-osteopathic theater has come mimicry. Many graduates of osteopathic medical schools claim more and more that there is no difference in the medicine that they practice and that practiced by MDs. I do not doubt that one can be a good osteopath and not do manipulation if one applies the principles. I also know that many MDs are excellent physicians giving the best care possible for their patients. However, I feel that one can not be a whole osteopathic physician without integrating the knowledge and working to maximize the patient's abilities. Osteopathic manipulative medicine allows us to do this. Yet, in a survey, somewhere in the neighborhood of 50% of the responders stated that they treated less than 5% of there patients with OMT.[24] Between 64% and 73% of osteopathic medical students have responded to surveys and reported they had few opportunities to use these skills during clinical rotations. Most held some belief that they would use palpatory diagnosis and OMT for fewer than 25% of their future patients and then primarily for patients with musculoskeletal complaints.[25] Why are the numbers not higher? Again, there is little opportunity and unless you and they make the commitment to use it regularly or even look for instances that you would not otherwise think of doing so, you will be giving your patients less than optimal treatment. You and the people you will swear to protect will both have lost chances. For them it could be a matter of life and death. There may consequences for you as well.

When an Olympic skater is able to compete after a severe laceration to his thigh without the use of medication, when patients can leave the hospital sooner than their age-matched peers, when students are 30% more likely to be immune following vaccination for hepatitis sooner than their peers, when children with otitis media suffer fewer instances of hearing loss and need less antibiotics, and when patients with Parkinson's disease can move their arms, legs, and head significantly better following even just a single treatment with OMT, then there is more than proof enough that osteopathy works. In fact, many of the interventions used are those taught within the first two years of the curriculum you as students are about to receive. Also, the research studies used protocols that were standardized. The actual clinical application of osteopathic manipulation is individualized to the patient, as should all medical interventions. That is part of the "Art of Medicine."

When people tell me that the use of OMT is unnecessary and unproven, I frequently will reverse the situation and ask them to prove some aspect of their practice that is commonplace. Over the years, I have heard so many directives that did or did not make sense. There are a million factoids and medical practice has evolved. In my relatively short time of practice, I have seen the ACLS protocols go through several changes and it has not been all due to the development of newer medications. I have seen rapid cooling of patients with fever by alcohol and ice baths be replaced with tepid sponge baths. One of my professors, an MD specializing in pediatrics has often said that 75% of what he had learned in medical school was wrong. My mentor, Dr. Schiowitz, replied that he might not know which of the 75% was incorrect. Sometimes other physicians have realized that in many instances the body's own temperature variances were a good thing and impaired the invaders' own replication system. I have seen physicians write "what if" orders to keep them, the physician, from being disturbed. One of the most typical is the acetaminophen or ibuprofen order for temperatures of a certain level. Rather than calling the physician, the nurse gives the medication when the criterion is reached. Doctors as well as moms fed colds and starved fevers. This may have mixed meaning, but there is a physician in New England who has "I fed fevers" on his cemetery headstone. He was supporting his patients by doing so in the face of overwhelming "typical" medical practice for the nineteenth century. He rebelled and did what he felt was right. He showed a little piece of artful practice in the face the incorrect majority. Yet, a country physician from the backwoods described that the physician should not get in the way of the patient's own healing processes. We have not gotten too far from the personal practices of Dr.

Still by coming around full circle to realize how correct he was in his thinking.

This lecture is named and is in memory of John Herbert Bryce Scott, DO and Katherine Fraser MacLeod Scott, DO, graduates of this school in 1906 and 1905 respectively. They practiced for 50 years in Columbus, Ohio and taught in the tradition of Andrew Taylor Still.[26] When these doctors had practiced, there were no specialists to speak of. Psychiatry was a branch of Neurology, the x-ray machine was barely a decade old, and the electrocardiogram was a theory. Nowadays, the numbers of specialties and subspecialties have proliferated. We do need specialists; those practitioners who are smarter and better in some areas of knowledge and who focus more intently and intensively upon the details. However, I think that when we have a cardiologist treating chest wall pain with counterstrain; when a pulmonologist requires that diaphragmatic and rib motion be addressed in her patients with respiratory difficulties, when the internist reduces edema in patients with lymphatic techniques, and the neurologist sees to it that patients with parkinsonism or multiple sclerosis are treated to maximize their musculoskeletal activities, then we will be approaching the ideal of the practice of osteopathic medicine. All graduates of osteopathic medical schools must direct their attention to the full practice of osteopathy. There is a mistaken belief that this requires a great deal of time. Actually, each treatment could last a few minutes. According to Dr. Still, "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."[27] We can all honor him and the Drs. Scott by continuing their work. The osteopathic approach should be the standard of care whereby all patients are given the optimal chance to heal, live, and return to their optimal conditions. We have to discard the choice that we can not afford to spend the time and energy to perform osteopathic manipulative medicine. Those who state that they do not have time are not practicing a vocation but are practicing business. Truthfully, how can we afford not to use it? That is patient centered and necessary. A few minutes spent assisting the patient can have exponential benefits.

What can you do? You are being given an exceptional opportunity to learn unique skills. Pay attention. Practice. Require that your educational experiences be expanded in opportunities, time, efforts, and assistance. The more that you have, the more that you will be able to contribute. Ask for opportunities. Learn as many skills as you possibly can and practice so that you will be prepared. There has to be a paradigm switch; the determination that osteopathic graduates will only become interested if they are at an institution that encourages them is not working. Almost all of the colleges of osteopathic medicine do a fine job in teaching osteopathic manipulative medicine in the first few years. However, they have less control over what occurs in the clinical rotations and the post-graduate training. Many of the schools have tried and there has been disappointing failure in doing so. Osteopathic students, interns, and residents must look for the opportunity and demonstrate osteopathic manipulative medicine. You must take the responsibility. By being determined and making your own attempts, you will be able to give your patients a better chance rather than depriving them of such. Put your hands on the patient and try. You may be surprised. You most certainly will be reinforced by the results. That is an experiment worth trying.

I would like to thank President McGovern, Dean Slocum, the board of trustees of Andrew Taylor Still University of Health Science, the faculty of the Kirksville College of Osteopathic Medicine, the members of the American Academy of Osteopathy for their selection of me for the honor and privilege of this lecture opportunity. Thank you for your attention and consideration as well. It has been an honor to have this opportunity to address you.

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Errata: The references for the article, "Lymphatic Manipulative Pump Research: A Brief Review of Literature" on pages 32-33 of the September 2004 issue of the *AAOJ* by Sean McMillan, William T. Crow, Charlotte H. Greene was published without its references. Our apologies to the authors. Please find them listed below:

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A Case Study of Left Adhesive Capsulitis Presumably Resulting from Previous Treatment with Protease Inhibitors*

James A. Lipton** and Michele Neil***

Abstract

Adhesive capsulitis may be linked to the administration of protease inhibitors used to treat the human immunodeficiency virus. One such case is presented where left adhesive capsulitis was diagnosed following an exhaustive workup. The etiology was presumed to stem from administration of protease inhibitors over one year prior to protect the patient following a needle stick from an HIV-infected source. The adhesive capsulitis, which presumably followed, was successfully treated through the use of osteopathic manipulative medicine.

Presented is a case illustrating the treatment of left adhesive capsulitis presumably from unknown causes until a literature search revealed a correlation between the use of protease inhibitors and adhesive capsulitis.[1-6] The case is remarkable for the lack of anatomic evidence on imaging combined with the absolute reliability of the patient.

On 10 October 2000, the patient presented in this case was seen by a physiatrist. She was a 42-year-old active duty female orthodontist complaining of left shoulder adhesive capsulitis since May 2000. She had been given medications including celecoxib, ibubrofen, indomethacin, and piroxicam without relief. Her worst pain was described as being an 85 on a scale of 0-100. After prescribed treatment with medications her pain remained at a 15 out of 100 and at the time of presentation 15 out of 100. The patient was searching for some modality that would assist her in moving her shoulder. Patient was assessed and given directions to continue indomethacin, to obtain an MRI of the left shoulder to rule out a rotator cuff tear and was sent to the lab to obtain a CBC, ESR, and chemistry panel.

An MRI (taken 9 November 2000), showed that the muscles and tendons of the rotator cuff demonstrated no evidence of focal signal intensity abnormalities or evidence of rotator cuff rupture or tear. The subacromial subdeltoid bursa revealed no evidence of increased fluid accumulation. The glenoid labrum and osseous glenoid were normal in appearance without evidence of tear or fracture respectfully. There was no evidence of intraarticular loose bodies or fracture fragments. The humeral head maintained normal signal intensities and articular uniformity. The deltoid muscle in its anatomical acromial insertion appeared normal. Acromioclavicular joint demonstrated no evidence of abnormalities. The long head biceps tendon and its tendon sheath appeared normal. The impression reported by the interpreting radiologist was magnetic resonance imaging revealed an intact rotator cuff without tear. Normal appearance of the joint capsule and synovial structures were also demonstrated.

The patient was followed up on 28 November 2000 by the same physiatrist and assessed as having an adhesive capsulitis. She was prescribed rofecoxib 25 mg taken by mouth once daily. The natural history of the disorder was discussed and the patient was told to follow up in two to three months.

On 1 February 2001, the patient had her first visit with the author (also a physiatrist). The 42-year-old active duty female was complaining of left shoulder pain with decreased range of motion since June 2000, without injury. She had been worked up and treated by a previous physiatrist as well as a general medical officer and physician assistant. The working diagnosis was adhesive capsulitis. The MRI of the left shoulder was negative and ESR, Chem 17, and CBC were normal. The past surgical history of the patient was a C-section, tonsillectomy, and adenoidectomy. Past medical history

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was allegedly unremarkable according to the patient. She was a gravida 4, para 3, ab 1. Her last menstrual period was a week prior to her visit and normal. Her breasts were normal and pap smears were normal. Patient reported a history of migraine headaches. She was negative for hypertension, cancer, diabetes, and TB. She never smoked nor chewed tobacco. She was in bed by 2100, asleep by 2110 and awake at 0500 for the day after having slept throughout the night. Her worst pain was assessed at 80 out of 100, after medication 50 out of 100, and at the time of the interview 20 out of 100. Review of her family history revealed some question of arthritis in her mother and there was also some question about whether or not this was rheumatoid arthritis. She had been to a chiropractor twice without relief. She had also undergone a left shoulder arthrogram, which similarly was negative.

Physical exam revealed an extended occiput, an atlantoaxial joint rotated left and sidebent right, an elevated first rib on the right, a restricted left sternoclavicular joint with decreased sternal motion and rib restrictions bilaterally (T-3 through T-6). Pre-treatment (with 0MM) she was only able to achieve 90 degrees of abduction and post-treatment using a variety of soft tissue muscle energy, myofascial release, high velocity, low amplitude, cranial sacral, facilitated positional release, and lymphatic pump techniques. Her post-treatment ability to abduct the left shoulder was 120 degrees. Adhesive capsulitis of the left shoulder with multiple somatic dysfunctions were assessed. Obtained was an anti-nuclear antibody, rheumatoid factor, Denver panel, CK, a bone scan, and an EMG (performed by the author), as well as a followup reevaluation. She was prescribed 0MM treatments approximately two to three times per week until the end goal of full range of motion was achieved.

On 6 February 2001, the patient returned for her second OMM treatment. She had increased range of motion since the last visit, though she still felt tight. She had not received the results of her labs or her bone scan as of yet. The pre-treatment abduction was 90 degrees, versus post-treatment of 150 degrees with active range of motion. Pre-treatment with passive range of motion, in abduction was 115 degrees. External rotation was 0 degrees and post-treatment was 30 degrees. It was noted that her left deltoid and supraspinatus were somewhat contracted and the assessment was that she had improved, but it was necessary to rule out a C5-6 involvement, perhaps with a possible herniated nucleus pulposus or mass. In addition, the rest of the differential diagnosis was considered. The plan was to obtain her labs, bone scan, and EMG as directed. Also, an MRI of the neck and a chest x-ray (PA and lateral) were ordered.

On 8 February 2001, the patient followed up for Treatment #3. She said that she felt looser, less stiff. The pre-treatment exam revealed she abducted to 130 degrees, posttreatment 150 degrees. She had improved and was directed to followup with her MRI, bone scan, and EMG.

On 13 February 2001, the patient underwent Treatment #4. She had abducted up to 160 degrees. She felt tight in her neck muscles and thought that she needed more treatment. It agreed that treatment frequency should increase.

On 15 February 2001, after Treatment #5, she was able to abduct to 180 degrees. Her left biceps touched her left ear with her head in neutral position.

On 22 February 2001, treatment #6 was administered. Patient was able to abduct her arm beyond 180 degrees.

On 27 February 2001, the patient returned for Treatment #7. Her bone scan showed mild degenerative joint disease in the left shoulder and mild degenerative joint disease in the thoracic spine. She was able to abduct to 180 degrees and the only remaining problem was flexion, which was only to 150 degrees when lying on her back. Some tightness was felt in the pectoralis minor, and the coracoclavicular ligament. Physical therapy was enlisted to assist with these areas.

On 1 March 2001, the patient returned for Treatment #8. She was able to abduct to 180 degrees, and was 160 degrees in flexion. The teres minor insertion was tender on palpation as well as the deltoid insertion. She was directed to keep working and to obtain her c-spine MRI and EMG results.

On 5 March 2001, the patient returned for Treatment #9. She was able to abduct to 180 degrees and flex to 170 degrees with good release.

On 8 March 2001, the patient returned for Treatment #10. She was able to abduct 180 degrees and flex to 170 degrees. We again discussed with physical therapy the targeting of the areas of the pectoralis minor tendon and coracoclavicular ligament to maintain increased range of motion.

On 12 March 2001, the patient presented for an EMG. The EMG of the upper extremity was performed by the author. The results were a normal study including examination of the biceps, deltoid, triceps, trapezius, infraspinatus, supraspinatus, teres major, serratus anterior, paraspinal musculature, abductor pollicis brevis as well as additional muscles. Nerve conduction studies and EMG were normal.

On 14 March 2001, the patient returned and informed the author (jal) that she had been stuck by an HIV-infected needle and treated with protease inhibitors (stavudine, lamivudine, as nucleoside analogs, and nelfinavir as a protease inhibitor) over one year prior. She had forgotten to mention this and wondered if it might be significant. The literature was searched and a cause and effect relationship was postulated.[1-6] On 3 April 2001, the patient returns for Treatment #11. Pre-treatment abduction was 160 degrees, post-treatment was 180 degrees. Pre-treatment flexion was 110 and post-treatment was 170 degrees.

On 17 April 2001, Patient underwent Treatment #12. The use of facilitated position release resulted in progress. At the time, there was a deficit of less than 15 degrees of flexion in the supine position with arms over the head. The patient was instructed to return for her final treatment.

On 26 April 2001, the patient underwent Treatment #13. She was able to flex to 190 degrees and abduct to 190 degrees. At this point she was discharged from Physical Medicine and Rehabilitation with directions to maintain range of motion by using a pool for range of motion and flexibility.

Discussion

The paucity of anatomic findings and clues in the diagnostic workup were striking compared to the patient's frank mobility deficits. The clinical course of this type of adhesive capsulitis is generally self-limiting with appropriate treatment.

In this case, the etiology may have been secondary to the use of protease inhibitor. Protease inhibitors had previously been administered to the patient as treatment for an accidental needlestick. The patient was stuck with a needle from an HIV-infected host. She had been treating over one year prior to the development of her adhesive capsulitis. The relationship, though circumstantial, is of some interest in the literature, and as a result, was reported.

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From the Archives – continued from page 9

Subjective Symptoms of the Spinal Lesion

Traumatic	Reflex
Sensitive points: Present in deep muscles and spreading to superficial muscles if lesion is acute. Present in deep tissues only if lesion is chronic.	Sensitive points: Present if lesion is acute. May spread into superficial muscles. Always present in deep spinal muscles if the lesion is chronic.
Fatigue in back: Not present when there is pain.	Fatigue in back: Not present when there is pain.
Functional capacity impaired: Seriously if lesion is acute. Insidiously if lesion is chronic.	Functional capacity impaired: Probably is if lesion is acute. Probably note if lesion is chronic.
History: Injury to back.	History: No injury to back.

Objective Symptoms of the Spinal Lesion

Rigidity in joint tissues: Very pronounced if lesion is acute. Pronounced if lesion is chronic.	Rigidity in joint tissues: Noticeable in all muscles if lesion is acute. Noticeable in deep muscle only if lesion is chronic.
Malposition: Usually present.	Malposition: Usually none.
Lesion: Usually unilateral.	Lesion: Usually bilateral.
Oedema: Pronounced if acute. Slight if chronic	Oedema: Present if acute. Slight if chronic.
Perversion of movement: Cause by malposition and tonic muscle shortening.	Perversion of movement: Caused by tonic muscle shortening.
Postural changes: Caused by bony maladjustment.	Postural changes: Caused by muscular tension.
Gait changes: Apparent	Gait Changes: Slight.

Subjective Symptoms of the Spinal Lesion

Acute	Chronic
Tender points on palpation: Acute and easily elicited.	Tender points on palpation: Present but not easily elicited.
Pain in joint: Acute.	Pain in joint: Present but not acute.
Fatigue in back: Present (not present when there is pain).	Fatigue in back: Present (not present if pain is acute).
Functional capacity: Openly impaired.	Functional capacity: Insidiously impaired.
History of injury: Usually easily discovered.	History of injury: Often not easily discovered.

Objective Symptoms of the Spinal Lesion

Oedema over facets: Considerable	Oedema over facets: Slight or absent.
Perversion of joint movement: Present by test of all tissues.	Perversion of joint movement: Present by test of deep tissues.
Malposition of bony parts: Difficult to test for on account of inflammation.	Malposition of bony parts: Present and easy to test for, (not present in reflex lesion).
Thickening of deep short muscles: Present and oedematous-like.	Thickening of deep short muscles: Present and rope-like.
Postural stress: Apparent.	Postural stress: Insidious
Gait changes: Apparent.	Gait changes: Insidious

Lesion Causes Disease Symptoms in Two Places

The Spinal lesion may cause disease either
(1) in the spine itself or (2) in tissue distant from the spine.

(1) In the *spine itself*. Patients present themselves with the complaint in the spine. The spinal lesion itself is the disability.

These patients have a disturbance in tissues remote from the spine, but a disease has not as yet made itself apparent there.

The spinal lesion may be *chronic*, but usually it is acute – very often it is a chronic lesion with pronounced acute symptoms.

The lesion usually has been produced by *trauma*, but reflex irritations may complicate it. It may be a purely reflex lesion.

(2) In *tissue remote from the spine*. Patients present themselves with the complaint in tissue remote from the spine.

These patients have spinal lesions, but they may not be aware of them.

In chronic organic diseases the spinal lesion found is chronic, but it may also show acute, *reflex* symptoms in addition.

In fevers, acute, reflexly formed spinal lesions are present. They may be at the site of chronic lesions.

A reflexly formed spinal lesion is always present. A traumatic causative lesion is also frequently present.❐

Case Study: An Osteopathic Resolution of a Neurocardiogenic Syncope

Yvette Somoano and Stefan Hagopian

Abstract

Unexplained intermittent neurocardiogenic syncope in a 24-year-old patient, managed, but unresolved by chemical treatment, is treated osteopathically and resolved. Description of this case and a brief discussion of questions raised addresses a deficit in the osteopathic literature.

Neurocardiogenic syncope is a condition in which cerebral metabolism is temporarily impaired by a reflex reduction in blood pressure causing a decrease in cerebral blood flow and loss of consciousness.[1] Recent investigations show this to be a broad spectrum of autonomic disorders variously triggered, all manifesting with syncope, hypotension, and orthostatic intolerance.[2] In cases of unexplained syncope, this diagnosis is made with a positive tilt-table test.[3] It is known that neurogenic syncope involves interplay between an imbalanced autonomic nervous system and cardiovascular regulation.[4] What is still unclear, is the exact mechanism causing this disorder.

Case Report

The patient first presented in February 2001, age 24, with a seven-year history of neurocardiogenic syncope, for which she was taking Disopyramide, as she had since shortly after diagnosis.

A healthy-looking woman, she related the beginnings of her problem in 1994, at age 18. At a roller-coaster ride at an amusement park, she would repeatedly hit her head on the back of the seat and had briefly blacked out, with residual loss of memory for parts of the ride. A week later while rollerblading, she would become lightheaded and dizzy; the first time this had happened in years of active daily exercise. Diagnosis resulted from a frank syncopal episode during the subsequent exam by her primary physician. While taking deep inspirations during lung auscultation, she blacked out. On waking, confused, after a few seconds, she was rushed to the hospital for observation.

Findings were not significant, with a normal CT, EKG, and EEG. While being monitored on telemetry, awaiting a lumbar puncture, the patient experienced yet another syncopal episode during which she went into asystole for a few seconds. A tilt-table test was done; results were positive, with decreases in blood pressure and pulse leading to asystole (HR 86 to 113, BP 101-118/62-70, until syncope, when neither HR nor BP could be monitored).

Started on Lopressor, a beta-blocker, which after two weeks eventuated a similar positive result on the tilt table (HR 75-86; BP 85-111/57-70), the patient was switched to Disopyramide, a sodium channel blocker. After two more weeks, a tilt-table test proved negative for 15 minutes (HR 61-120; BP 62-136/32-81); an Isoproterenol challenge produced negative results (HR 120-152, BP 73-146/40-89). Within a few months, the patient's Disopyramide dosage was reduced from 300mg BID to 300mg QAM and 150mg QHS, and the patient remained stable on this dose for seven years during which only three events suggestive of syncope occurred. Twice she experienced tachycardia secondary to dehydration – quickly resolved with intravenous fluids – and once, prodrome to syncope during a neck massage. In one episode the patient had been supine, not upright as is commonly described in the literature.

On this day, in addition to her history of syncope, the patient complained of chronic upper back pain on the left side.

Birth/Childhood History

The patient had been born at 42 weeks of gestation after being "engaged" for over a month. As an infant, she would had emesis with every meal.

Past Medical/Surgical/ Trauma History

Nine months prior to first noted syncopal episode, the patient had sustained thoracic strain injuries in an MVA; no surgeries. Medical history insignificant, except as above noted.

Initial Physical Exam/ Osteopathic Findings

The physical exam was grossly normal except for the following: an imbalance in the autonomic nervous system as indicated by diaphragm and thorax restricted in inhalation position; emotional distress regarding even the memory of an aborted lumbar puncture; and a slight but palpable muscular hypertonicity. The patient was also found to have compression through the cranio-vertebral junction and markedly decreased thoracic kyphosis. She had restrictions in movement in segments C7-T3 and T11-L2 and at the sacroiliac joint, and a decrease in the excursion of her thoracic and pelvic diaphragms.

Treatment and Results

Osteopathic treatment was begun on the initial visit by performing an EV4 (phasic inverse of a cranial CV4); performing condylar decompression; and balancing ligamentous tension throughout the lower cervical and upper thoracic regions, as well as the thoraco-lumbar junction and sacroiliac joints. Post-treatment, over the next month the patient experienced no syncopal episodes and felt fine.

On a second visit, the strain pattern in the patient's upper thoracic region was still present, but this time resolved with the use of respiratory cooperation during osteopathic treatment. A compression of the right occipital-mastoid suture was found and treated by balancing the dural membranous tension in the region.

During a third visit, the patient was treated similarly, and was newly found to be expressing a marked restriction in the region of the right middle face. By now, the strain patterns in the upper thoracic region had improved, but side effects of medication were increasing. The patient's cardiologist suggested she stop taking Disopyramide and return for testing in a week.

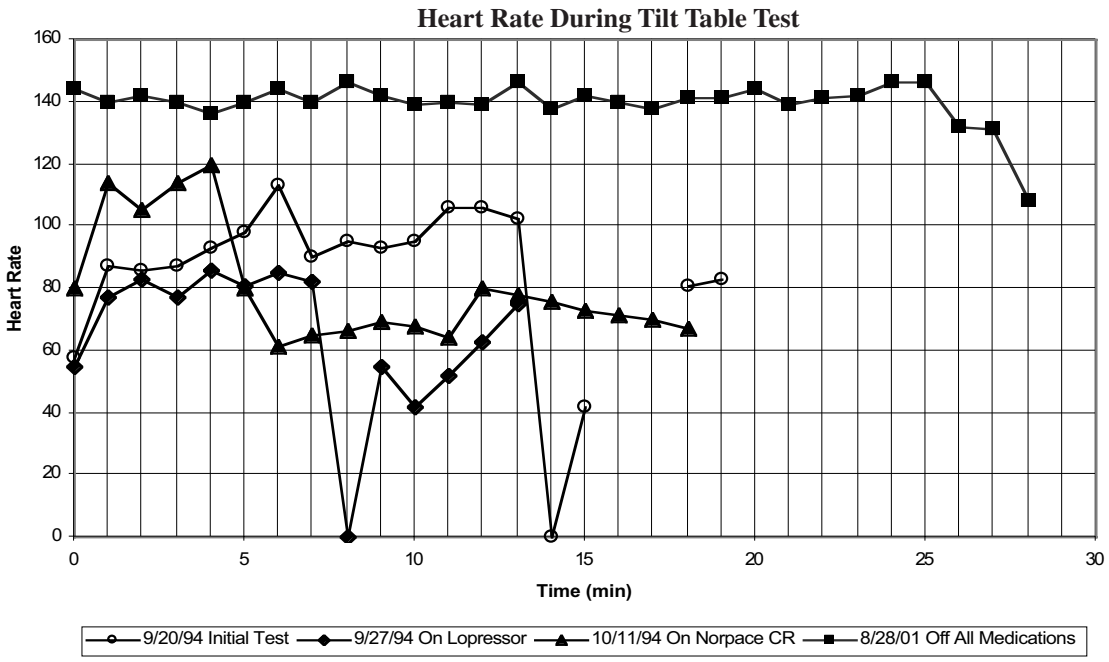

Figure 1. Heart Rate during tilt-table test without Isoproterenol Challenge.

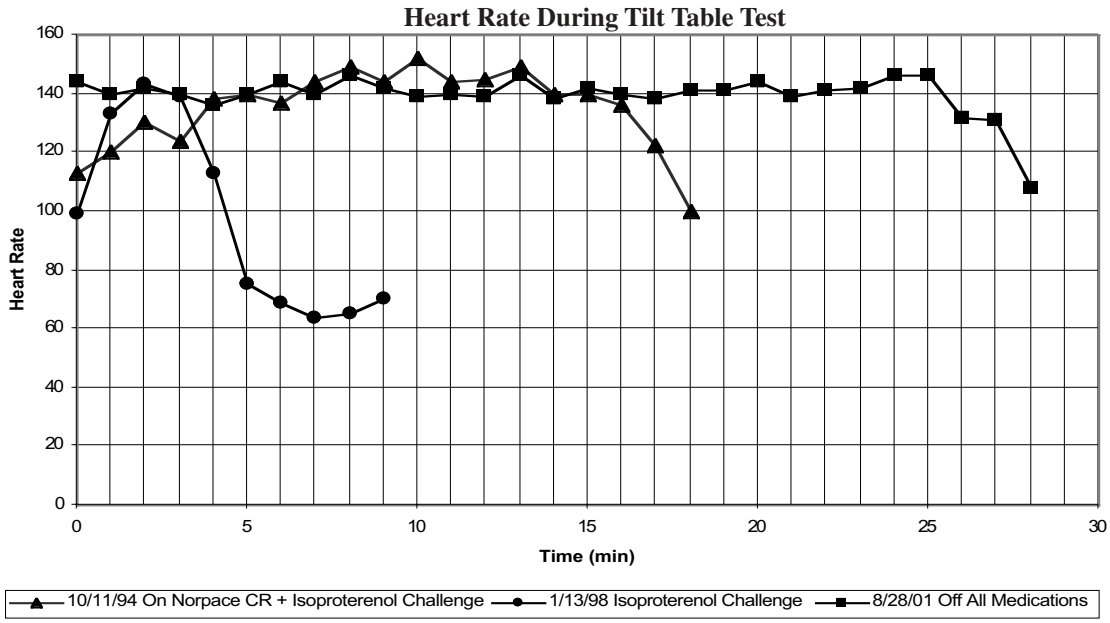

Figure 2. Heart rate during tilt-table with Isoproterenol Challenge on 10/11/94 and 1/13/98, and without Isoproterenol Challenge on 8/28/01

Eight days later, tilted for 30 minutes at 65 degrees [HR 103-146, BP 112-155/46-103], the patient experienced no syncope nor near syncopal episodes. The test was negative, even though she was anxious, even tachycardic (figure 1). Her heart rate reached levels comparable to those previously experienced only when stimulated by Isoproterenol (figure 2). The patient was symptom-free. The cardiologist discontinued the medication. To this day, 26 months later, the patient has experienced no further episodes. She tolerates a full range of activities and continues to be monitored and supported with osteopathic treatment approximately quarterly.

Discussion

What caused this patient's neurocardiogenic syncope, what triggered the episodes and how was the condition resolved?

A complex series of imbalances occurring over a period of many years, probably resulted from or compensated for injuries to the cranial base and upper thorax. Such injuries can have occurred in the process of birth and in early childhood through falls and maladaptive postural patterns. These imbalances can create a chronic neurocardiologic instability. Several texts note that syncope can be a response to strong emotions, stress, pain, fear, fatigue, hunger or thirst, overcrowded or humid environments, $CO_2/0_2$ imbalance, medications, exercise or even prolonged standing.[5,6] This patient had indicated deep breathing during lung auscultation, exercise, stress, and dehydration as triggers at various points in her history.

In this case, autonomic imbalance resolved with the decompression of the cranial base and a release with restoration of the originally flattened kyphotic curve in the upper thoracic region. Treatment results suggest as cause, some physical interference with the nerves in these areas, which may have developed secondary to birth trauma. Support for this theory is found in the patient's history of infantile emesis. Such emesis is documented in infants with strain patterns at the cranial base,[7] where the vagus nerve exits the cranium through the jugular foramen, which lies between the occiput and the temporal bone.

As for the thoracic restrictions, although they may have originated from postural adaptations to emotional stress (decreased kyphosis), they may have been exacerbated by the patient's injuries, such as the unresolved upper thoracic injury from an MVA. And, though relatively minor, the multiple blows to the head during the amusement park ride cannot be discounted.

An exact mechanism for neuro-cardiogenic syncope is still unknown. The current literature suggests that central hypovolemia sparked by venous pooling while in an upright posture, causes a decrease in atrial pressure.[8] A reflex tachycardia stimulates C-fibers (atrial and ventricular afferent stretch receptors) within the myocardium. A consequent afferent transmission is sent to the vagal nuclei of the brainstem, mimicking hypertension, causing a decrease in sympathetic activity, bradycardia and hypotension, classically known as Bezold-Jarisch reflex.[9] This theory is controversial: studies show that syncope can occur even with sudden volume depletion in animals who have surgically denervated hearts.[9]

Osteopathic and other research shows that the autonomic imbalance in syncope can be caused by a mechanical impingement or irritation of the nervous system, either peripherally or centrally. Such disruption may occur with inflammation, nerve or vaso-neural compression from surrounding anatomical structures, or as a response to chemical stimulation.[10] Animal studies have shown that long-term impingement of nerves at the third or fourth thoracic level destabilizes the nervous system, leaving the subject unable to compensate under the stress of, for example, central hypovolemia.[11] And a lesion of the atlas in rabbits produces vasodilation of the extremities and variation of heart rate and rhythm—responses seen with autonomic imbalance.[12] Vasodilation has long been thought to influence the Bezold-Jarisch reflex.[9] The structural impingement on these nerves is thought to cause a magnification of efferent impulses that pass through these segments to the viscera, strongly affecting the tissue.[13] The facilitation of the nervous system due to this structural impingement could be a cause of neurocardiogenic syncope secondary to stress placed anywhere along the autonomic nervous system.

Other than osteopathic treatment, therapeutic approaches for neuro-cardiogenic syncope include general measures such as volume expansion; pharmacologic approaches; and even invasive methods such as placement of a dual-chamber cardiac pacemaker.[14] Experimentally, the simple measures of orthostatic training (standing against a wall)[15] and tilt-table training,[16] and maintaining proper hydration[17] and CO_2/O_2 balance,[18] show promise in improving orthostatic tolerance and diminishing probability of some syncopal episodes. By far the most common treatment choice, though, is pharmacologic.

Further research is essential to determine a more precise mechanism, and range of treatments, for neurocardiogenic syncope. The theories noted in this case report piece together a complex mechanism with both a cause and an effect, in cases where autonomic imbalance is secondary to somatic dysfunction.

A large portion of the population – 28% in one study of 400 healthy young adults[19] – suffers from syncopal episodes of unknown causes. Surely many of these people can be helped without the need for chemical treatment, if the biophysical components are properly identified and addressed, as they can with complete osteopathic diagnosis and treatment.

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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, *An Osteopathic Resolution of Neurocardiogenic Syncope* by Yvette Somoano, DO and Stefan Hagopian, DO.

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1. In this case study, neurocardiogenic instability was thought to be caused by:

___A. Compression of the lower cervical region from multiple whiplash injuries.
___B. Multiple falls during childhood causing compensation of L5 and the Sacrum.
___C. Birth trauma and maladaptive postural patterns causing compensation of the cranial base and upper thorax.
___D. Birth trauma and multiple falls causing compression of the sacrum.

2. Autonomic imbalance triggering syncopy may be initiated by:

___A. Inflammation
___B. Nerve or vaso-neural compression from surrounding anatomical structures.
___C. A response to chemical stimulation.
___D. All of the Above

3. A lesion of the atlas in animal models has been shown to cause:

___A. Vasoconstriction of the extremities.
___B. Variation of heart rate with vasodilation of the extremities.
___C. Hypertension with vasoconstriction of the extremities.
___D. Autonomic balance with vasodilation of the extremities.

4. Infantile emesis, noted in this patient, has been previously documented in the literature to be caused by strain patterns at the:

___A. Upper thorax.
___B. Lower thorax involving the diaphragm
___C. Lower cervical region.
___D. Cranial Base.

5. It is thought that in a patient with structural impingement of the nerves at the third or fourth thoracic levels and/or the vagus nerve, neurocardiogenic syncopy could be facilitated.

___A. Only if stress is placed directly on the point of impingement.
___B. If stress is placed anywhere affecting the autonomic nervous system.
___C. Only if multiple stressors occur at once.
___D. None of the above.

Answer sheet to December 2004 AAOJ CME quiz will appear in the March 2005 issue.

September 2004 AAOJ CME quiz answers:
1. B
2. D
3. B
4. A
5. A

Could Joint Hypomobility Alter Optimal Proprioceptive Information?

Rafael Zegarra-Parodi

Key Words:

joint complex dysfunction
proprioception
dysafferentation
hypomobility
adjustment

Abstract

It has long been thought that "joint complex dysfunctions" (JCD) such as those treated by osteopaths only had detrimental effects on local joints and surrounding soft tissues due to the focus on the kinesiopathological component of JCD. More recent theories emphasize on the neurophysiological component, involving afferent inputs to the spinal cord By reviewing recent papers, it has been shown that joint hypomobility is associated with altered reflex responses involving mechanoreceptive and nociceptive pathways. As these pathways are crucial for an optimal proprioceptive function, alteration of these inputs to the spinal cord created by a JCD could decrease appropriate proprioceptive informations. In this light, osteopathic treatment should be seen as more than treatment for musculoskeletal conditions. While improving mobility to restricted joints, it could have a favourable influence on several neurological reflex responses: by reducing abnormal inputs to the spinal cord, it could improve the body's ability to recover an optimal proprioceptive function.

Introduction

Littlejohn stated in 1901. *"Osteopathy may be defined as a system, or science, of healing that uses the natural resources of the body for the adjustment of its structure, to stimulate the preparation, and distribution of the fluids and forces of the body, and to promote cooperation and harmony in the body mechanism."*[1] Osteopathy then is a system of clinical practice that looks at a person from a mechanical point of view and recognizes the musculoskeletal system as crucially important to the overall function and health of a person.[2] The musculoskeletal system is intimately connected with all other systems of the body through both the somatic and the autonomic nervous system and is, therefore, considered as a mirror of both health and disease, responding as it does to inflammation and pain from disorder in other body systems.[3]

A host of interacting factors have the ability to produce dysfunction of the musculoskeletal system, including stress response, postural anomalies and overload, repetitive physical actions (sport, occupation, hobbies, and so on), emotional distress, trauma, structural factors (congenital short leg, cranial distortion at birth), visceral, and other reflex activity. These can be summarised as overuse, misuse, and abuse of the musculoskeletal system.[4]

The purpose of adjustment is to restore joint mobility by manually applying a controlled force into joints, which have become hypomobile. When a joint is adjusted, compression of corpuscular endings of somatic receptors will create depolarization of afferent neurons. These afferent inputs have a variety of effects in the central nervous system (CNS) that affect the amount of pain, different primary perceptual experiences, the integrity of the motor system, as well as the function of autonomic nervous system for the integrity of life itself. Since biomechanical integrity will give us the highest population of receptor afferents, it is very simple to understand that a decrease in biomechanical integrity or aberrant biomechanical relationships will have a very high probability of decreasing the population of receptor afferents. This, in turn, will modify our primary perceptual experiences.[5]

Proprioception refers to kinaesthetic awareness. Proprioception occurs as a consequence of the integration of vestibular input, visual input, and tissue mechanoreceptor input to the cerebral cortex and cerebellum. It is thought that mechanoreceptor input is of the utmost importance for proprioception. Neurons of the motor cortex respond to activation of peripheral somatosensory and proprioceptive afferents. These input pathways may be involved in the control of the output of the motor cortex. Thus, mechanoreceptors give rise to local segmental reflexes and suprasegmental proprioceptive reflex effects.[6] Various areas of the musculoskeletal system are extensively and richly innervated with mechanoreceptors, especially around the spinal column, which seems to operate as one vast proprioceptive organ.[7]

We will use in this paper the zygapophyseal joint (ZJ) as a model for description. Our purpose is to review recent scientific literature about physiological relations between a biomechanical dysfunction such as hypomobility (structure) and proprioception (function). Since such biomechanical dysfunction is reversible in nature, we will discuss the opportunity and the aim of osteopathic care for patient-specific conditions.

Discussion

The adjustable spinal disorder

Several terms have been coined to describe the adjustable spinal disorder from a mechanical point of view: "subluxation" in chiropractic; "somatic dysfunction" in osteopathy; and "fixation" or "functional blockage" in manual medicine. For a better comprehension of the following paper, a single term will be used: "joint complex dysfunction (JCD)".

This is characterized as a ZJ strain/sprain with associated local and referred pain and muscle spasm. The function of the ZJ is deranged by virtue of static misalignment and/or reduction of motion (i.e., "fixation," "blockage," or the more generic term "hypomobility").[8] The hallmarks of diagnostic criteria for segmental JCD are asymmetry, range of motion abnormality, and segmental tissue texture change. Some authors include tenderness among these criteria.[9]

Mechanisms that have been proposed for this dysfunction, particularly the hypomobility, include:

1. Entrapment of a ZJ inclusion or meniscoid, which have been shown to be heavily innervated by nociceptors.
2. Entrapment of a fragment of posterior annular material from the intervertebral disc, again, innervated by nociceptors.
3. Stiffness induced by adhesions and scar tissue from previous injury and/or degenerative changes and adaptive shortening of myofascial tissues.
4. Excessive activity (spasm, hypertonicity) of the deep intrinsic spinal musculature, particularly in unilateral, asymmetric patterns.[8]

Common to all concepts of JCD are some form of kinesiologic dysfunction and some form of neurologic involvement, let's onto the anatomy of receptors affected by JCD.

Zygapophyseal joint receptors

Zygapophyseal joint (ZJ) receptors are innervated by a variety of neuroreceptors all derived from the dorsal and ventral rami as well as the recurrent meningeal nerve of each segmental spinal nerve. Information from these receptors crosses many segmental levels because of multilevel ascending and descending primary afferents. There are two categories of somatic receptors: nociceptors and mechanoreceptors. McLain stated, *"The presence of mechanoreceptive and nocicepttve nerve endings in cervical facet capsules proves that these tissues are monitored by the CNS and implies that neural input from the facets is important to p roprioception and pain sensation in the spine."* McLain's statement reinforces the concept that the very presence of neural structures in spinal tissues provides the evidence of the integration between the spinal joints and the nervous system.[10] More than half of all nerve fibers ascending and descending in the spinal cord are propriospinal fibers, which run from one segment to another, providing pathways for multisegmental reflexes.

There are four types of somatic receptors: nociceptors and three types of mechanoreceptors. D'Astolfo[8] gives us an overview of their location and function.

Type I mechanoreceptors are confined to the outer layers of the joint capsule and are stimulated by active or passive joint movements. Stimulation of type I receptors is involved with:

1. Reflex modulation of posture and movement.
2. Perception of posture and movement.
3. Tonic effects on neck, eye, limbs, jaw, and eye muscles.
4. Inhibition of pain from receptors via an enkephalin interneuron transmitter.

Type II mechanoreceptors are found within the deeper layers of the joint capsule. They are stimulated when minor changes in tension within the inner joint occur. Type II receptors are likely to achieve the following:

1. Inhibition of pain from receptors via an enkephalin transmitter.
2. Monitoring for reflex actions.
3. Phasic effects on neck, eye, limbs, jaw, and eye.

Type III mechanoreceptors are located only in ligaments of the peripheral joints and like Golgi tendon organs, impose an inhibitory effect on motoneurons. These receptors:

1. Monitor direction of movement.
2. Create reflex effects on segmental muscle tone.
3. Recognize potentially harmful joint movements.

Mechanoreception refers to the process by which tissue mechanoreceptors are stimulated by mechanical input such as touch, muscle stretching, and joint motion. A-alpha and A-beta fibers carry mechanoreceptive information into the CNS. Segmental reflex effects of mechanoreceptor input can be both excitatory and inhibitory. An important inhibitory effect is the presynaptic and postsynaptic inhibition of the nociceptive pathways. There are also suprasegmental reflex effects of mechanoreceptor stimulation, which are proprioceptive in nature.[11] Human facets contain mechanoreceptors to detect motion and distortion. Spinal proprioceptors may play a role in modulating protective proprioceptive mechanoreceptors. The densities of mechanoreceptors are greater in areas related to extreme movements, explaining why cervical facets have more receptors.[12] Mechanoreceptors are the first line of defense in sensing the safe limit of range of motion of a joint. This input activates reflex mechanisms that act to prevent joint injury.[13]

Type IV receptors or nociceptors are free nerve endings located throughout the fibrous portion of the joint capsule and ligaments. Type IV receptors are absent from articular cartilage and synovial linings but have been found in synovial folds and within the annulus fibrosus of the disc. These receptors are associated with the following:

1. Evoke pain.
2. Tonic effects on neck, limb, jaw, and eyes muscles.
3. Central reflex connections for pain inhibition and autonomic effects.[8]

Nociception is the process by which nociceptive receptors receive tissue-damaging stimuli which are then carried into the CNS by nociceptive axons (A-delta and C fibers). The receptors involved in pain detection are referred to as nociceptors (receptors for noxious stimuli). It is important to note that almost all body tissue is equipped with nociceptors as pain has primary warning functions. If we did not feel pain and if pain did not impinge on our well-being,

we would not seek help when our body aches. Nociceptors are free nerve endings that terminate just below the skin so as to detect cutaneous pain. Nociceptors are also located in tendons and joints for detection of somatic pain and in body organs to detect visceral pain. Pain receptors are very numerous in the skin, hence pain detection here is well defined and the source of pain can be easily localized. In tendons, joints, and body organs the pain receptors are fewer.[14]

We must remember that nociception and pain are completely different: pain is a combination of sensory (discriminative) and affective (emotional) components. The sensory component of pain is defined as nociception.[15] Potential outcomes of nociceptive input to the cord include pain, autonomic symptoms, vasoconstriction, and muscle spasm. A devastating consequence of both pain and nociceptive stimulation of the hypothalamus is the release of cortisol by the adrenal glands. Over time, elevated levels of cortisol will promote glucose intolerance, inhibit collagen formation, increase protein breakdown, inhibit secretory IgA output, and inhibit white blood cell function. Clearly, the clinical importance of pain and nociception should not be minimized.[11]

Joint receptors contribute both to the coordination of muscle tone around joints and provide neurologic feedback to enhance joint stability. Spinal ligaments, like those around knee joints, are richly innervated, responsive to mechanical stimulation, and provide proprioceptive feedback that mediates reflex muscular stabilization about the joint. This provides dynamic joint stability which is part of a neurologic protective mechanism.[16] Joint and muscle receptors detect limits of movement of a joint and play a role in synchronizing mechanisms involved with control of movement. While the kinesiologic component of the JCD is largely accepted by the medical community, the neurologic involvement is poorly accepted, even rejected. The description given below of somatic receptors involved during normal function could help provide a better comprehension of neurologic involvement associated with JCD when dysfunction occurs.

Joint Complex Dysfunction

The osteopathic profession gives the following definition: *"Somatic dysfunction is impaired or altered function of related components of the somatic system (body framework): skeletal, arthrodial, and myofascial structures, and related vascular, lymphatic, and neural elements";* while the chiropractic profession gives this one: *"A subluxation is a complex of functional and/or structural and/or pathological articular changes that compromise neural integrity and may influence organ system function and general health".*

JCD is now described by Seaman as a kinesiopathogical joint lesion (usually hypomobility) that develops as a consequence of:

1. Micro and/or macrotraumatic tissue injury and the associated inflammatory response.
2. Inflammatory damage that may be perpetuated by a nutritional status that is pro-inflammatory in nature.
3. Degenerative changes in muscular and connective tissues due to sedentary living.
4. Decreased descending inhibitory pathway activity due to aberrant psychological states.
5. "Dysafferentation" (This term refers to an imbalance in afferent input such that there is an increase in nociceptor input and a reduction in mechanoreceptor input.[17]

Korr, et al. have provided experimental evidence to lend support to a neurological explanation for JCD.[18,19] The facilitated segment is a concept that is proposed to explain the behaviour of JCD: an injured somatic or visceral structure produces a barrage of discordant afferent impulses into the dorsal horn of the spinal cord, which "sensitises" that segment. It is proposed the spinal interneuron thresholds are lowered, allowing an exaggerated response to pathways synapsing at that level, thus contributing to increased pain perception, sympathetic outflow, and segmentally supplied muscle tone.[9]

Van Buskirk (1990) proposed a model of JCD where nociceptive input to the cord appeared to be the driving force behind the pathogenesis of JCD. He argued that nociceptors were the only receptors capable of producing reflex muscle contraction and sympathetic discharge, and proposed a cascade of events that produce JCD. Noxious stimuli (from viscera or soma) produce reflex axon effects promoting inflammation at all the terminal branches of that axon, which further sensitises other nociceptors. Afferents reaching the dorsal horn produce reflex muscle contraction and sympathetic discharge (producing visceral and immune effects). Over time the muscle becomes fibrotic and, if stretched or restrained, activates nociceptors once again.[20] Nociceptor activation produces further segmental tissue inflammation and sympathetic stimulation and the cycle becomes self-sustaining. Pain perception need not be involved. However, nociceptive processing in the dorsal horn may become disturbed, producing what has been described as "central sensitization" by Woolf leading to hyperalgesia and chronic pain.[9]

Subsequently, Fryer refined this concept of JCD. Same neurological events were described but the origin of JCD was much more a mechanical one, focusing on joint strain as a primary source of aberrant inputs to the CNS.

Strain to the ZJ capsule and ligaments creates inflammation, synovitis, synovial effusion, and activates nociceptors. Axon reflexes produce vasodilatation and inflammation at the terminal ends of all the axon branches, producing segmental tissue texture change and tenderness (possibly even segmental muscle inflammation and engorgement). Range of movement and end feel is altered due to tissue engorgement and joint effusion.

The research work of Lewit, Janda, and others has shown that postural muscles, when chronically abused, misused, overused, will tend to shorten and eventually to contract. Phasic muscles, however, when faced with the same insult, will tend to weaken but will not shorten.[4] These conclusions were applied for this model. Deep segmental "stabilising" muscles, like multifidus, are reflexly inhibited; excitability of longer polysegmental muscles like the erector spinae increase, making the joint less stable and vulnerable to further strain. Over time connective tissue changes in the strained capsule occur, producing long-term joint range of movement

➧

asymmetry. The multifidus atrophies, functional stability, and control are impaired and the joint undergoes continuing strain.

JCD may produce a variety of patient-specific symptoms depending upon specific end organ changes initiated by nociceptive reflex activity and, which central autonomic, endocrine, limbic, motor and/or sensory nuclei are most affected by "dysafferent" input.[17]

Common to these theories is the focus on aberrant inputs to CNS coming from a joint. This phenomenon sometimes becomes self-sustaining due to connective tissue changes and could alter other neurological pathways due to central sensitization. Nociception, mechanoreception, and proprioception are all intimately associated with the normal and abnormal function of the ZJ: nociception induces JCD (whether the source is located in somatic or visceral structures), which subsequently reduces mechanoreception and proprioception.[11]

Dysafferentation

Seaman, a chiropractor highly involved in neurophysiologic theories for JCD, concluded that an emerging body of research indicates that abnormal joint complex function can alter the activity of nociceptors mechanoreceptors, such that nociceptive activity increases and mechanoreceptive activity decreases.

Many authors and researchers involved in joint adjusting and manipulation realize this and use the terms "altered afferent input", "abnormal afferent input", or similar terms when discussing the neuropathophysiological component of JCD. Seaman cites several authors in his paper:[21]

"1.Peterson stated that, "somatic dysfunction and/or joint dysfunction induce persistent nociceptive input and altered proprioceptive input".
2. Peterson and Bergmann described vertebral joint dysfunctions (and their associated mechanical alterations, pain, and potential local inflammation) as "lesions capable of inducing chronically altered nociceptive and proprioceptive input".
3. Hooshmand illustrated how restricted joint mobility results in decreased firing of large diameter mechanoreceptor axons (A-beta fibers) and increased firing of nociceptive axons (A-delta and C fibers).
4. Henderson used the term "altered somatic afferent input theory" to classify a neurophysiologic theory of chiropractic subluxation."

Because researchers in different professions have acknowledged the fact that compromised joint function will alter afferent input such that nociception is enhanced and mechanoreception is reduced, Seaman proposed that the chiropractic profession adopt the word "dysafferentation" to describe the abnormal afferent input associated with JCD.

Aim of the Osteopathic Treatment

Where possible, the aim is to understand the anatomical and physiological breakdown and the resulting dysfunction in the context of the whole person. The osteopath should attempt to discover where and what the cause of the symptoms are, and also why the dysfunction has occurred, considering aetiological, predisposing, and maintaining factors. The purpose of osteopathic treatment and management is then to enhance the body's response by encouraging the restoration of normal function and also to remove or reduce the person's predisposition to the problem.[2]

The hypothesized effects of adjustment commonly accepted today can categorized as either mechanical or neurological. Some authors describe adjustment as mechanical treatment with reflex effects.

Mechanisms of action of adjustment, which have been proposed to affect mechanical issues, include:

1. Release of entrapped synovial or disc tissues, thus reducing pain restoring mobility.
2. Stretching and breaking of adhesions.
3. Dynamic stretching of musculature and myofascial tissues.[22]

Spinal adjustments produce a short-lasting (100-300 milliseconds), high velocity impulse into the body. Triano, et al. have quantified the applied forces of an adjustment and correlated them with physiologic responses (changes in leukocyte function) such that a threshold of approximately 500 N distinguishes potentially effective from "noneffective" procedures.[22] An audible release (cavitation) was not necessary for the burst of muscle activity to be stimulated, but the adjustment did have to be fast to produce this response (it is important to remember that these findings relate to immediate responses, not those measured a period of time after the treatment).

Proposed mechanisms of action of adjustment on neurological phenomena can be divided into two categories: reduction of compressive insult to neural tissues, and creation of stimulus-induced reflex changes.

The first mechanism is relatively straightforward in that adjustment is hypothesized to relieve the compressive insult on nerve roots and autonomic fibers within the intervertebral foraminae, or affect disc/facet athropathy.[22] As an example, intersegmental tractions produce their attenuating effects on peripheral structures via pumping action which increases the removal of inflammatory byproducts and reduces swelling.[23] Adjustment might exert its effect by dynamic stretching of the muscle spindles and Goigi tendon organs, copiously located in the deep spinal muscles, thereby resetting the length/tension ratio in these muscles. This would help break the pain/spasm/pain cycle by alleviating signals emanating from spastic muscle and irritated joint. Similarly, massage has the effect of sedation of pain and relaxation of muscle spasms via the attenuation of trigger points and other foci of irritation within the muscle.[24] Spinal adjustment has been proposed to reduce motor neuron excitability and produce reflex muscle relaxation, but results of studies testing this proposal have been conflicting. A recent study has demonstrated reflex electromyographic responses in spinal and limb muscles follow high velocity technique, but the responses were short lived (100 - 400msec) and no study has examined the effects in symptomatic patients.[9]

In the second mechanism it is proposed that the dynamic stretching produced by adjustment induces a barrage of activity in joint and muscular mecha-

noreceptors that is transmitted along large diameter afferents and, which produces inhibitory effects within the nervous system. These effects are proposed to be both local (at spinal level) and "central," in that they may involve so-called descending inhibitory pathways.[22]

Adjustment might inhibit incoming messages of pain according to the "gate control" theory of Melzac and Wall.[9] Action potentials from joint mechanoreceptors are conducted by fast large diameter axons, which reach the dorsal horn of the spinal cord before the nociceptor potentials, and can "close the gate" on the incoming pain messages. The spinal tissues appear to be particularly amenable to this process, probably because of their unique patterns of afferent input into the CNS, with a high level of convergence existing with other somatic and visceral inputs onto the same spinal tract projection cells.[21] Whether manual techniques produce a significant analgesic effect that lasts longer than the manual event is yet to be determined.[9]

There is a third target site for interaction between nervous system and adjustment, the enkephalin mediated pain control. It involves activating brainstem structures responsible for enkephalin production. Experimentally, analgesia has been produced by electrical stimulation of the skin, peripheral nerve trunks, dorsal and ventral columns of the spinal cord, thalamus, cortex, periaqueductal gray matter of the midbrain, and the nucleus raphe magnus of the medulla.[24] There are other effects of physiological therapeutic modalities, such as psychosomatic responses, which should also be considered, but they are beyond the scope of this paper.[23]

Joint Complex Dysfunction and Proprioception

Potential symptoms may develop as a consequence of JCD and may produce a variety of patient-specific symptoms. We will just focus on possible altered proprioception. Alteration in mechanoreceptor function may affect postural tone. Murphy summarized the neurological pathways associated with the maintenance of background postural tone: "Weight-bearing disc and mechanoreceptor functional integrity regulates and drives background postural neurologic information and function (muscular) through the unconscious mechanoreception anterior and posterior spinocerebellar tract, cerebellum, vestibular nuclei, descending medial longitudinal fusciculus (medial and lateral vestibulospinal tracts), regulatory anterior horn cell pathway." The anterior horn cells provide motor output, which travels via motor nerves to muscle fibres.[25]

The evidence that the neck plays a critical role in posture is overwhelming. Muscle receptors may be of great importance in sensing joint position. A characteristic of neck muscles is an abundance of muscle spindles; spindle density in large muscle of the neck range from 46-106 per gram, among the highest of anywhere. High spindle density is characteristic of muscle executing fine muscle control. The abundance of afferent information may not only be due to fine motor control. Polysynaptic pathways from neck muscles afferents to neck motoneurons are powerful. Afferents leaving neck muscles can exert profound effects on hind-limb motoneuron excitability. The neck structures are unusually rich in receptors. Small muscles close to the cervical vertebrae may have up to 500 muscle spindles per gram, a density almost 100 times as great as some muscles, of locomotion and 5 times greater than the large dorsal neck muscles, which are regarded as spindle rich. These deep structures play an important role in reflexes and maintenance of posture and provide precise information with respect to position.[26] Articular structure, like ligaments, not only provides mechanical restraint but also provide neurological feedback that directly mediates reflex muscular stabilization about the joint, providing dynamic joint stability.

According to these anatomical and physiological particulars, some studies were conducted to access relationships between JCD and proprioception. Seaman reviewed some of these:

"In a study lead by Rogers in 1997, 20 patients with chronic neck pain were evaluated at the beginning of the study for pain levels and proprioceptive functioning. The patients were then divided into two groups: Group A, who received 6 sessions of spinal adjustment; Group B: who were instructed to perform stretching exercises twice daily for 3-4 weeks. After the study period, the spinal adjustment patients showed a 44% improvement in pain symptoms on average, while the stretching patients showed just a 9% improvement. In regard to proprioceptive functioning, similar results were found: a 41% improvement in the adjustment group, but only an 11% improvement in the stretching group. How spinal adjustment affects proprioception is not yet known, but the authors speculate that chiropractic treatment somehow stimulates the deep articular mechanoreceptors in the spine, in turn leading to improved functioning.

In 1991, Revel, et al. demonstrated that patients with neck pain have an alteration in neck proprioception. The authors developed a proprioceptive test, which involved head and neck repositioning after an active head movement. Patients with neck pain consistently performed this test significantly worse than pain-free controls. The authors concluded: "The test may also permit a completion of post-trauma cervical pain investigation by studying the responsibility of neck proprioceptors in dizziness and unclear 'pseudovestibular' disorders."

In 1994, Revel et al. performed a study, which sought, in part, to determine if an exercise program based on eye-head coordination can improve cervicocephalic kinesthesia. The results demonstrated that such a rehabilitation program was successful. The authors concluded: "The rehabilitation of cervico-cephalic kinesthesia could be particularly appropriate for patients with neck pain and dizziness after neck trauma, because it has been postulated that this syndrome sometimes called 'cervical vertigo', could be the result of damage to cervical proprioceptors."[27]

McPartland, et al, studied the relationship between chronic neck pain, standing balance, and suboccipital muscle atrophy. They hypothesize that patients with chronic neck pain have more JCD in the cervical spine than control subjects without neck pain. They also hypothesize that patients with chronic neck pain and JCD exhibit more atrophy of suboccipital muscles than control subjects. Lastly, because suboccipital muscles have a high density of proprioceptors, they hypoth-

esize that chronic pain patients with atrophied suboccipital muscles demonstrate a loss in standing balance. This preliminary study suggests that there is a relationship between chronic pain, JCD, muscle atrophy and standing balance. They hypothesize a cycle initiated by chronic JCD, which may result in muscle atrophy, which can be expected to reduce proprioceptive output from atrophied muscles. The lack of proprioceptive inhibition of nociceptors at the dorsal horn of the spinal cord would result in chronic pain and a loss of standing balance.[28]

It appears that JCD (abnormal joint motion and abnormal afferent inputs) could decrease optimal proprioceptive information, and that alteration could be either symptomatic or not. Several hypotheses were formulated to explain this clinical phenomenon: muscle atrophy decreasing proprioceptive inputs, hypomobility decreasing afferent inputs, JCD resulting in dysafferentation. It is important to remember that this phenomenon could become chronic as soon as JCD has become self-sustaining due to connective tissue reorganization throughout surrounding soft tissues. Once again, this could be asymptomatic and predispose the affected cord segments to further dysfunctioning due to central sensitization.

Conclusion

JCD of the ZJ is truly based upon two events with a cascade of effects: segmental kinesiopathogical spinal lesion and dysafferentation. Since the nervous system controls and coordinates every function in the body, all the other components are merely effects of the JCD. It appears that adjustment might normalize articular afferent input to the CNS system, which reestablishes normal nociceptive and kinaesthetic reflex thresholds. *"Structure governs function"*, one of the traditional osteopathic principle, finds here a major clinical application through physiological relationship between hypomobility and proprioception. The pathways explaining both physiopathology and improvement with adjustment are not totally known.

This concept can be further extended to other functions. The osteopath should keep in mind the crucial relationships between biomechanical integrity and optimal afferent inputs and consider these inputs as a major component of an optimal health status. Afferent inputs are informing the CNS about self-awareness, which in turn will respond adaptively through efferent inputs (self-regulation). Alteration of these neurological reflex pathways could decrease the optimal state of health. It is fundamental to understand that while JCD could implicate mechanoreceptive and nociceptive pathways, JCD does not systematically induce pain and these aberrant inputs to CNS could exist without any symptomatology. This unique concept in health care explains the major focus we place in osteopathic care for the musculoskeletal system and why it is so important for the health of each individual.

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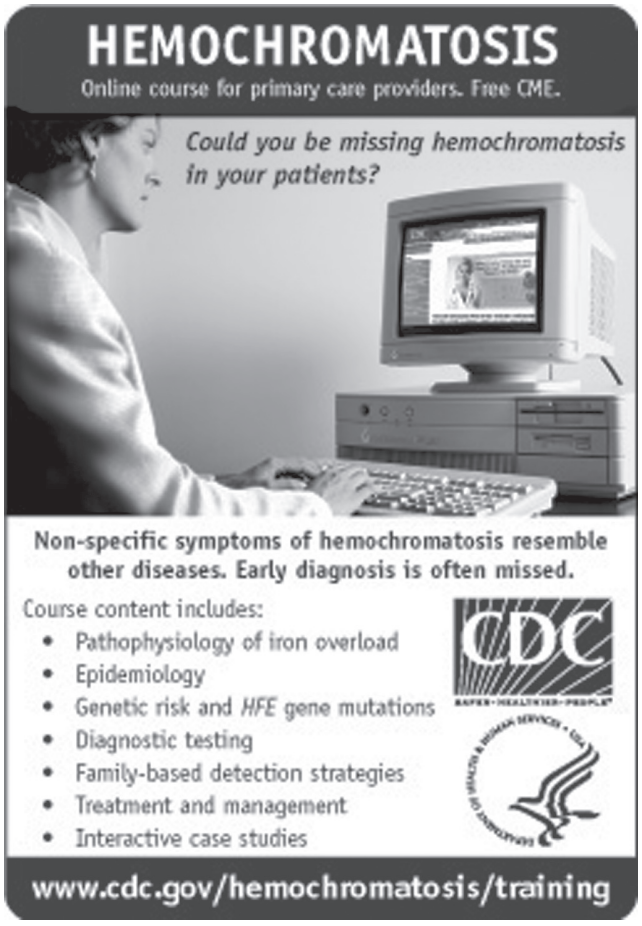


A Biodynamic View of Osteopathy in the Cranial Field

Course offerings by Tom Shaver, DO

Phase I – An introduction to a biodynamic model of O.C.F. C. N. S. motion and development. Balanced fluid tension.

Date: March 5-8, 2005 Course Fee: $650 (US)
Location: Kona, Hawaii CME: 21.5 Category 1A
Registration Deadline: January 30, 2005

Phase II – An in-depth perception of fluid dynamics. Therapeutic/diagnostic use of balanced fluid tension around the fulcrum incited by the tidal forces. Rates/ CV-4/EV-4 techniques. Automatic shifting.

Date: September 3-6, 2005 Course Fee: $650 (US)
Location: Kona, Hawaii CME: 23 Category 1A
Registration deadline: July 8, 2005

("The AAO designates these programs for AAO co-sponsorship as meeting the criteria for CME credits awarded through the AOA.")

Questions/Registration information, contact:
Tari Sargent
196 Weeks Mills Road, Farmington, ME 04938
Phone: 207/778-9847; E-mail: tsargent@tdstelme.net

Energetically Integrated Osteopathic Medicine

The life, thought, and work of Robert Fulford, DO as interpreted by Zachary Comeaux, DO, FAAO

CME: 18 Category 1A (anticipated)

February 11-13, 2005
WVSOM Campus • Lewisburg, WV

Course Fee: $475.00

Course Description: Level II
This course serves as an introduction or enhancement to the practitioner's use of subtle energy to complement other aspects of osteopathic diagnosis and treatment.

Learning Objectives:
- Review of the philosophical background bioenergetic work
- Reconciliation of energetic and biomechanical models
- Introduction to energetic palpation
- Discussion/demo and practice of Dr. Fulford's methods of diagnosis and treatment
- Introduction of percussion vibrator, Vogel crystal, and magnets in context
- Practical integration of these methods into primary care medicine/osteopathic practice

Contact: Zachary Comeaux, DO, FAAO, Program Chairperson and author of *Robert Fulford, DO and the Philosopher Physician*
Phone: 304/647-6270 or E-mail: zcomeaux@wvsom.edu

• This approach is especially helpful in cases resistant to traditional methods. Often the symptoms are due to unrecognized or unresolved latent effects of trauma.

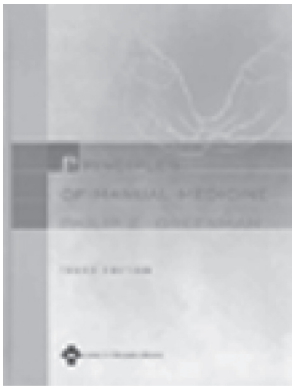
• This course is based on material presented and well received overseas over the last five years. This material draws on Dr. Fulford's sources including some not disclosed by himself in his courses. Although it includes work with the percussion vibrator, it complements and does not reduplicate the Basic and Advanced Percussion Vibrator courses taught through the American Academy of Osteopathy.

• Dr. Comeaux has practiced Family Medicine since 1988 and had provided care to both Dr. and Mrs. Fulford. In this capacity he spent significant time over the last years of Dr. Fulford's life discussing and co-treating patients with the Doctor as well discussing concepts and cooperating in the writing of *Dr. Fulford's Touch of Life*. He currently is an Associate Professor at the West Virginia School of Osteopathic Medicine.

• Early enrollees will receive a bibliography for pre-course study if desired.

Book Review

Reviewer: Anthony G. Chila



PRINCIPLES OF MANUAL MEDICINE

Philip E. Greenman, DO, FAAO, Emeritus Professor
Departments of Osteopathic Manipulative Medicine & Physical Medicine & Rehabilitation; College of Osteopathic Medicine; Michigan State University; East Lansing, Michigan

pp. 613, incl. Index. Third Edition, ©2003 by Lippincott Williams & Wilkins: 530 Walnut Street; Philadelphia, PA 19106, USA. LWW.com; $99.00

The author, a nationally and internationally respected osteopathic physician, conveys 50 years' experience in the use of manual medicine in a single author attempt. Previous editions were released in 1989 and 1996 by Williams & Wilkins.

Three sections of this text utilize 23 chapters to address Principles and Concepts, Technique Procedures, Clinical Integration, and Correlation. Adjunct disciplinary procedures including the use of exercise are provided. Each chapter begins with a detailed review of appropriate anatomy. This serves to build understanding and focus therapy. Complete disciplinary coverage is assured through the provision of information on both diagnostic testing and treatment techniques. Practical visual guidance is enhanced through the use of more than 1000 clinical photographs. Clear and thorough instructions provide the procedural detail necessary to assure proper performance and best results.

In this third edition, extensive new material in soft tissue considerations includes Chapman's Reflexes, tender points of the Counterstrain System, and visceral techniques. Chapter 12, which addresses Osteopathy in the Cranial Field, has been completely revised. New information is provided regarding the use of cranial approaches in the management of brain injury patients. Chapters 18 (Upper Extremity) and 19 (Lower Extremity) have been expanded to include new treatment options such as neural and dural mobilization of the extremities.

Doctor Greenman's assessment of his single author evolution in writing is worth noting, given in his own words:

"The first edition of this book was originally designed to support the Continuing Medical Education's courses offered through Michigan State University and its Colleges of Osteopathic Medicine and Human Medicine. Since that time it has been used in a number of colleges of osteopathic medicine, chiropractic colleges, schools of physical therapy, and schools of massage therapy. The second edition has been translated into seven foreign languages.

This third edition attempts to bring current new information, as well as answers to many of the questions I have received when teaching this material. This edition also adds newer techniques and visuals and clarification to some of the concepts presented. As an art form, manual medicine cannot be mastered by reading a text and requires considerable practice using the hands as a diagnostic and therapeutic instrument. For the beginner, be diligent and not discouraged. For the experienced, continue to explore this fascinating field. Our patients are always grateful for the personalized nature of manual medicine. Happy voyage."

Elsewhere in Print

Do CAM therapies work for pain management?
Patient Care: September 2004, 52-58
Robert Bonakdar, MD: Director of Pain Management; Scripps Center for Integrative Medicine, LaJolla, CA

David E. Bresler, PhD: Health psychologist and board-certified acupuncturist, founder and former director, UCLA Pain Control Unit; President, Academy for Guided Imagery, Malibu, CA

The authors offer a guide to making informed choices of CAM treatments for pain. As a consensus article, evidence supporting the use of CAM treatments and suggestions regarding incorporation into daily practice are provided.

Acupuncture
"Efficacy is seen with treatment of headache, joint pain, cancer pain, and low back pain (LBP). Transcutaneous electrical nerve stimulation (TENS) has shown efficacy for osteoarthritis pain in elderly patients. Percutaneous electrical nerve stimulation (PENS) and electroacupuncture have shown efficacy for fibromyalgia, headache, LBP, diabetic neuropathy, and cancer pain."

Mind-Body Therapy
"Mindfulness-based Stress Reduction (MBSR) incorporates meditation, yoga, and exercise to reduce pain. Studies show that treatment of cancer pain, tension headache, fibromyalgia, and acute pain with guided imagery is particularly efficacious."

Massage
"A reduction in blood pressure (BP) and an improvement in heart rate variability have been associated with massage. Efficacy is seen in soft tissue pain and LBP."

Manipulation
"Ideally suited to patients with recent emergence of pain when first-line medications have failed. Efficacy is seen in LBP and headaches."

The authors acknowledge that distinctions are made between chiropractic treatments and osteopathy, but consider that both practices can be reasonably discussed under the larger rubric of manipulation. Having said this, the remainder of their comments simply compare chiropractic care and medical treatment. Such reporting unfortunately completely omits the significance of structural diagnosis as evolved by osteopathic medicine in its contribution to the history of manipulative medicine. Had the differentiation been more thorough, the concepts of holism, neurologic control, circulatory function, energy expenditure and self-regulation would have considerably broadened the assessment.

Special Technic: Hypermobile Joint
The Pain Clinic: Volume 6, Number 6; September 2004, 14-16

This publication is the official journal of **The Florida Academy of Pain Medicine** and **The Ohio Pain Initiative**. *Special Technic: Hypermobile Joint (A Preliminary Report)* is the first in a series of historical articles of interest. Introduction and editorial comments are provided by Felix S. Linetsky, MD. The contributions of Earl H. Gedney, DO (1901-1976) are appropriately recognized: First described application of sclerotherapy for treatment of painful knee and sacroiliac instabilities; First described application of sclerotherapy and developed the injection technique for painful lumbar discs; Emphasized the pathology of sacroiliac joint and ligaments. Doctor Gedney's article was published in *Osteopathic Profession*, 4:9:30-31, June 1937.

2004 Journal Index

BY AUTHOR

BY SUBJECT

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Winter OMT Update

"Application of Osteopathic Concepts in Clinical Medicine"

PLUS PREPARATION FOR CERTIFYING BOARDS

Henderson, Nevada • January 28-30, 2005

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.)

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

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.hotels.com or www.expedia.com

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.

For Registration Information, contact:
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American Academy of Osteopathy®
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