THE AAO
JOURNAL
A Publication of the American Academy of Osteopathy

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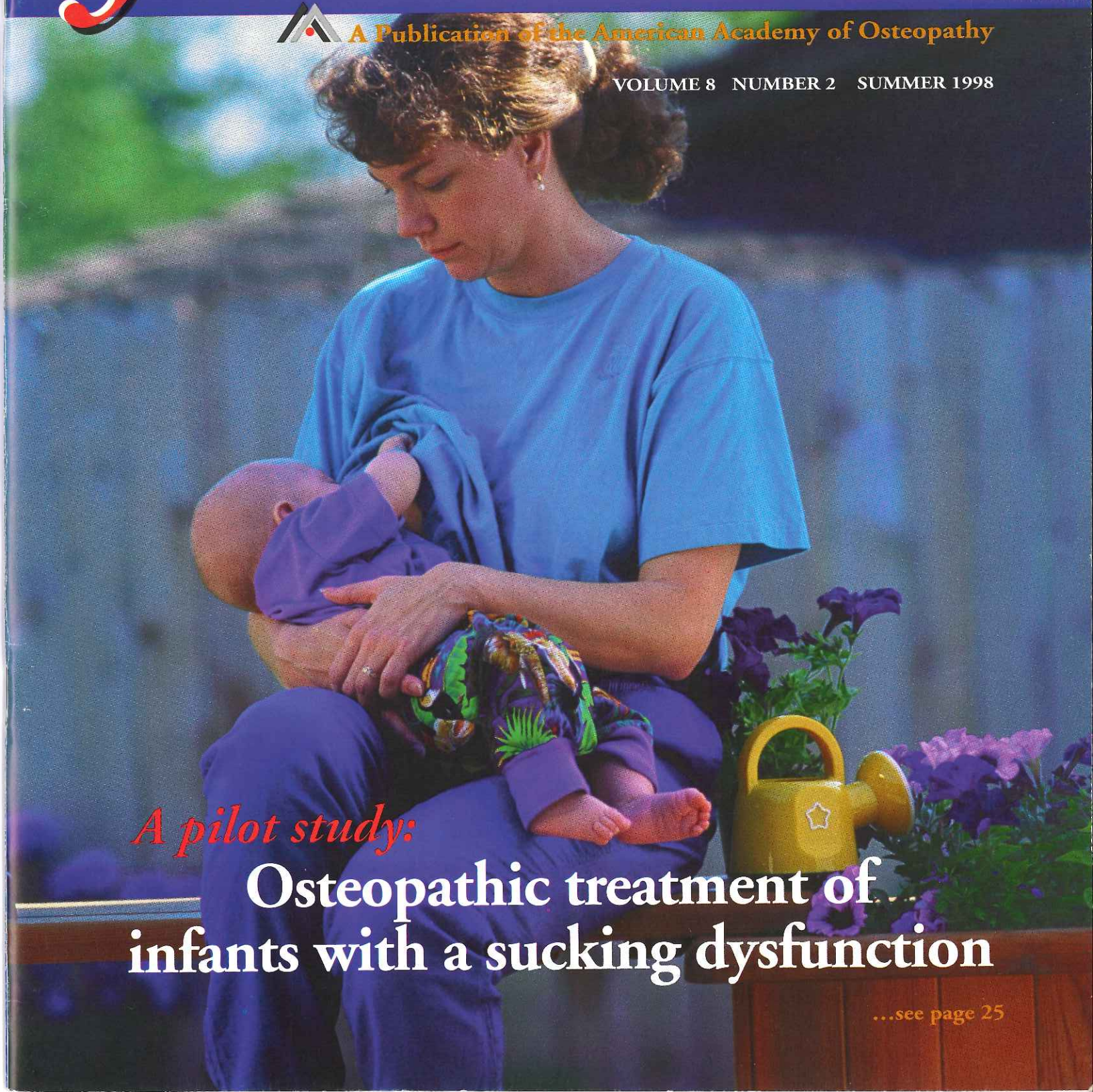

A pilot study:

Osteopathic treatment of infants with a sucking dysfunction

...see page 25

AAO's CME Calendar

American Academy of Osteopathy®
3500 DePauw Boulevard, Suite 1080
Indianapolis, IN 46268-1136
Phone: (317) 879-1881 or FAX: (317)879-0563

July

17-19
Counterstrain
John Glover, DO, Program Chair
Midwestern Univ/CCOM
Chicago, IL
Hours: 20 Category 1A

August

14-16
Levitor
Michael Kuchera, DO, FAAO
Program Chairperson
St. Paul. MN
Hours: 20 Category 1A

14-16
Visceral Manipulation
John Glover, DO, Program Chair
St. Paul, MN
Hours: 24 Category 1A

September

17-20
Fall OMT Update
Ann Habenicht, DO, FAAO,
Program Chairperson
The Coronado Springs Resort Hotel
Orlando, FL
Hours: 23 Category 1A

October

5-8
AOA/AAO Convention
Elaine Wallace, DO, Program Chair
New Orleans, LA

22-23
Introduction to OMT
John M. Jones, DO, Program Chair
Virginia Beach, VA
Hours: 16 Category 1A

24-25
Basic Muscle Energy
Walter Ehrenfeuchter, DO, FAAO
Program Chairperson
Virginia Beach, VA
Hours: 16 Category 1A

November

6-8
Visceral Manipulation (Thoracic)
John Glover, DO, Program Chair
SFCOM
San Francisco, CA
Hours: 24 Category 1A

December

4-6
Myofascial Release
Judith O'Connell, DO, FAAO
Program Chairperson
UOMHS
Des Moines, IA
Hours: 20 Category 1A

5-6
Basic Percussion 'Vibrator
Richard Koss, DO, Program Chair
UOMHS
Des Moines, IA
Hours: 15 Category 1A

January 1999

14-17
Introduction to OMT
Boyd Buser, DO, Program Chair
Turtle Bay Hilton
Kahuku, Oahu, Hawaii
Hours: 20 Category 1A

TRUST 2000:
A Legacy to the Osteopathic Profession

*Chairman **Ross Pope** and members of the AAO Finance Committee invite all AAO members to consider a "planned gift" to the Academy as part of the endowment program TRUST 2000: A Legacy to the Osteopathic Profession. In 1992, **Alan R. Becker**, DO, FAAO created a charitable remainder unitrust in memory of his spouse, **Catherine S. Becker**. In an ongoing feature to promote the endowment campaign, the Academy is pleased to reprint the following abridged article which first appeared in the Fall 1992 issue of The AAO Journal.*

Winston Churchill stated that "We make a living by what we get, but we make a life by what we give."

Alan R. Becker, DO, FAAO, medical director of the Becker Osteopathic & Metrecom Clinic in Kailua, Hawaii, has established the Catherine S. Becker Memorial Charitable Remainder Unitrust with the Academy as the beneficiary.

Dr. Becker established the fund to honor his late wife, Cay, who for many years assisted the Academy, serving as structural consultation assistant during the annual conventions and convocations. "Cay was always willing to take on tasks that would help the Academy, and in so doing helped the staff, the students and the physicians during a very busy time."

By setting up the charitable remainder unitrust, Dr. Becker received an immediate tax deduction for his gift, and will be able to receive a portion of the income from the trust during his lifetime. Upon his passing, the Academy will receive the balance of the funds.

Dr. Becker has contributed greatly to the Academy through his generosity and vision, and hopes that his gift to the Academy will inspire others to do the same.

There are a number of planned giving techniques which can be individually tailored to meet your personal needs. Many provide significant tax savings, professional management and lifetime income for individuals and members of their families.

Gifts can be funded by gifts of cash or property such as land, jewelry, silver, coin collections, interests in limited partnerships, corporations and so forth. In your estate planning, please remember the Academy. Likewise, if you have grateful patients who wish to contribute to the profession as a thank you for your services to them, why not recommend the Academy as a beneficiary.❐

American® Academy of Osteopathy

3500 DePauw Boulevard
Suite 1080
Indianapolis, IN 46268-1136
(317) 879-1881
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THE AAO
JOURNAL
A Publication of the American Academy of Osteopathy

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

Editorial Section

Peer-Reviewed Section

Instructions for Authors

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

The AAO Journal welcomes contributions in the following categories:

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

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

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

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

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

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

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

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

Submission
Submit all papers to Raymond J. Hruby, DO, FAAO, Editor-in-Chief, MSU-COM, Dept. of Osteopathic Manipulative Medicine, A-439 E. Fee Hall, East Lansing, MI 48824.

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

Requirements for manuscript submission:

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

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

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

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

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

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

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

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

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

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

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

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

3. Include a caption for each figure.

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

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

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

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

From the Editor

by Raymond J. Hruby, DO, FAAO

The growing international scene in osteopathy

I just returned from a trip to the recent Congress of the Federation Internationale de Medicine Manuelle, held in Brisbane, Australia. The AAO was well represented at the meeting. Other than myself, other Academy members in attendance were Herb Yates, Michael Kuchera, Robert Ward, Walt Mill, Harry Friedman, Wolfgang Gilliar and Craig Wax. Elsewhere in this issue of the Journal you can read a brief report about the Congress written by Dr. Wax.

The meeting got me thinking more about the international status of osteopathic medicine. A lot of things have changed since I first became active with the AAO in the 1970s. This can readily be seen by the increasing number of overseas osteopaths attending Convocation each year. We are also receiving more manuscripts for our Journal from our friends from other countries.

There is increasing interest on the part of the AOA regarding international osteopathy. The AOA leadership has formulated a Council on International Osteopathic Medical Education and Affairs to learn what is happening internationally with osteopathic medicine.

Why all the fuss? Well, the political nature of osteopathy becomes quite complex once we go outside the borders of the United States. There are few countries where DOs can practice as fully-licensed physicians. If you are interested you might want to peruse the section of foreign licensing requirements in the *AOA Yearbook and Directory*.

In some countries, there are groups of allopathic physicians who have become intensely interested in osteopathic medicine and wish to incorporate it into their training and practice. There are some places like England and Australia where non-physician osteopaths are allowed to practice osteopathy, but do not have full-scope practice rights as we do here. There are many countries where non-physician osteopaths have no governmental recognition or practice rights, although most are fighting hard to get recognition by their respective governments.

So you see, the situation is quite complicated in many ways. I have served on the AAO International Committee for some years, and believe me, I can hardly begin to explain osteopathy on an international level in a column as short as this one.

The situation is a challenging one for both the AOA and the AAO. The U.S. and international allopathic groups present a little less political difficulty. The AOA has traditionally allowed for the exchange of information with fully-licensed physicians. However, some AOA and AAO members feel that MDs may be able to learn osteopathic techniques but may not be able to fully absorb the osteopathic principles and philosophy that permeates what we do.

What about the non-physician osteopaths? Should we continue to have some formal relationship with these groups? And, what about the ones who have no licensing or practice rights in their countries? All of these groups look to Andrew Taylor Still and American osteopathic medicine as the roots of what they do. So, should we say no to them or should we try to work with them in some way? And, what about practice rights and licensing in other countries for American-trained DOs?

For me the issue is this: should we have a global osteopathic profession united by full licensure and practice rights as full-scope physicians? Or, should we have a global osteopathic profession united by a common philosophy and set of principles, with varying scopes of practice?

There is no simple solution to this situation. I certainly do not have one. All I know is that osteopath medicine is becoming increasingly known around the world. We would all do well to keep our eyes and ears open to the continuing developments in the international arena. ❐

Message from the President

by Melicien A. Tettambel, DO, FAAO

AAO maintains and develops osteopathic concepts

Editor's Note: *Melicien A. Tettambel, DO, FAAO, a 1978 graduate of the Kirksville College of Osteopathic Medicine took office as president of the American Academy of Osteopathy at its annual presidential banquet, March 28, 1998, in Colorado Springs. She is board certified by the American College of Osteopathic Obstetricians and Gynecologists and in osteopathic manipulative medicine by AOBSPOMM. Dr. Tettambel is a member of AAO, AOA, ACOOG, The Cranial Academy, Sutherland Cranial Teaching Foundation and the Illinois Association of Osteopathic Physicians and Surgeons.*

I thank you all for your greetings, best wishes, and support as I begin my term as President of the American Academy of Osteopathy. I look forward to representing the Academy and its mission of educating American Osteopathic Association practice affiliates, osteopathic medical students, and the public regarding those practices and principles of Dr. A.T. Still to promote a uniquely comprehensive health care system. Historically, the Academy has maintained and developed osteopathic concepts through educational programs that have resulted in development of teachers in our colleges of osteopathic medicine, improvement of physician's palpatory diagnosis and skills, and creation of practice opportunities to serve our patients. All of these opportunities would not be possible without your many avenues of support.

During the 1998 Convocation, Secretary-Treasurer Anthony Chila reported that this organization is approaching "budget neutral." Congratulations to the Board and committees for astute planning of programming. Congratulations are also due to the membership-at-large for continued support not only through payment of dues and charitable donations but also through efforts to increase AAO membership.

The Academy's educational programs have been recognized for their excellence locally, nationally and internationally. Our members, as well as all others within the osteopathic profession, have opportunities to improve skills and infuse osteopathic concepts into residency training programs and practice affiliate disciplines. It is through countless hours and tireless efforts of our committee members that osteopathy continues to be promoted to such an august audience. In addition to maintaining excellent practice skills standards, the Academy provides education and information regarding coding and reimbursement to benefit ALL DOs. Perhaps more of our colleagues would continue to prosper educationally and financially through their involvement as members of the Academy, or at least contributors to the Golden Ram Society's annual fund raising campaign.

What better way to unite and strengthen our profession in the promotion of osteopathic principles and practices than through pathways of certification and fellowship? These opportunities are offered to ALL osteopathic physicians who desire to focus their efforts in osteopathic manipulative medicine as a practice discipline. Those who have completed these challenges have been sought as educators, peer-reviewers to protect practice rights of osteopathic manipulative practitioners, advisors to panels on third-party reimbursement issues, and health care advisory policy-makers.

It is through all the above avenues of support for the Academy by its membership and other generous sponsors that the entire osteopathic profession derives educational and financial benefit. I am truly honored to represent this organization to continue promoting osteopathic ideals and, perhaps, to become a unifying factor for our profession to realize that Dr. Still's concepts pertain to all those who declare themselves "osteopathic" physicians.

Visit the
American Academy of Osteopathy's
Home on the Internet
http:/www.aao.medguide.net

Message from the Executive Director

by Stephen J. Noone, CAE

Academy CME: Great value or too expensive!

For the first time during my association with the osteopathic medical profession, I had the privilege to attend the annual Convention of the American College of Osteopathic Family Physicians and represent the Academy at the President's Banquet. Max Helman, DO, a long-time friend from my tenure with the Indiana Osteopathic Association, was installed as the 1998-1999 ACOFP President.

The ACOFP leadership also offered me the opportunity to staff a complimentary exhibit booth where I was able to talk with conventioneers about the Academy and its programs. I conducted a brief survey for the Education Committee to learn the interests of family physicians in the Academy's hands-on continuing medical education programs in osteopathic manipulative medicine.

A significant number of physicians expressed great interest in the Academy's programs but noted the "high tuition" compared to CME sponsored by AOA practice affiliates, state associations, and local health care institutions. I welcomed the chance to explain the reasons why the Academy must charge significant tuition for its educational programs and am pleased to repeat that rationale in this column.

For most medical societies, the honorarium and expenses for speakers are underwritten by unrestricted grants from pharmaceutical companies, foundations and other industry supporters. These third parties provide these grants as marketing tools for their products. However, despite continued solicitations, it is rare that the Academy's program chairpersons are successful in obtaining such support. For example, the AAO only received a total of $2,000 in outside support for the 1997 AOA Convention program and $3,500 in grants for its 1998 Convocation.

Another reason for higher tuition levels is the overall cost of faculty for the Academy's programs. First of all, the fact is that primary faculty at the Academy's CME programs are the most thoroughly trained and most sought-after teachers in palpatory diagnosis and manipulative medicine in the world. They represent the "cream of the crop" in OMM and deserve to receive an honorarium consistent with their professional expertise and standing within the profession. Hence, faculty costs are significant at Academy programs.

Furthermore, since virtually all CME is geared to hands-on workshop instruction, there is a need not only for the main presenter but also for sufficient table trainers to ensure the highest level of learning by the physicians in the workshop. The AAO Education Committee has set a standard of one faculty/table trainer for each 12 participants. While one faculty member can deliver a lecture to 800 attendees at Convocation, for example, the delivery of a hands-on workshop requires significantly more table trainers to meet the standard ratio of 1:12. This increased faculty expense, since it is not underwritten by educational grants, must be borne by tuition.

In addition to this loss of "economy of scale," other factors which enter the equation are extraordinary equipment costs and staff expenses to manage 18 courses annually. The most visible evidence is the need for treatment tables, usually being shipped from the Academy's central Indiana office or borrowed from a supplier. The labor for packing tables and the shipping costs are considerable. Some programs require other equipment and supplies, e.g. percussion vibrators, pillows, latex gloves, exhibit piping/draping for privacy, etc.

In sponsoring 18 courses annually, the Education Committee has scheduled these CME programs in various regions of the country to make them more accessible to AAO members and other interested DOs. Since the majority of these programs are held in hotels, one of the Academy staff members must travel to these sites to manage the event (delivery of tables, setup for meeting, registration, ongoing management, and return shipment of all materials.)

In summary, the Education Committee must ensure that the budget for CME programs will result in a net income over expense. The only feasible way to meet this goal is to establish tuition at a level which will sustain the program. I strongly believe that participants at AAO's educational programs receive great value for their tuition — world renowned faculty, low student/teacher ratios, and skills which they can employ immediately to demonstrate the unique aspects of osteopathic medicine!

Letter to A.T. Still

Dear Doctor Still,

When I read some of the older literature about osteopathic medicine, I know that these writers were taught osteopathic principles and practice either by you or someone taught by you. I think of them as the first generation of osteopathic physicians, and therefore, the best people to express what your teachings were.

One I found interesting was written by Carter Harrison Downing, DO. In 1923, he wrote a text entitled *Principles and Practice of Osteopathy.* I am sure you knew of him, but you should also know that his colleagues thought very highly of him. In the introduction to this book, J. H. Stiles, Jr., DO, called Dr. Downing "...indubitably the greatest osteopath since Andrew Taylor Still." That is quite a compliment, I would say!

Dr. Downing, like other early osteopathic writers, pointed out what he thought were your greatest contributions to the field of medicine. He listed your achievements and contributions as follows:

"1. The importance of structural states in disease; a better and broader outlook on disease, and its causes, particularly the latter.

2. The greatest contribution to therapeutics in the whole history of medicine, so stupendous and complete in character as to revolutionize the entire world.

3. The first, best and only complete theory of natural immunity to be formulated since proven by scientific investigation to be true. "The body itself contains within itself all the element and all the medicines necessary for the cure of disease." – (Dr. A. T. Still).

4. The fallacy of internal drug medication."

There is much interesting information in Dr. Downing's book. We can all learn a lot from these early writers of osteopathic textbooks.

Your ongoing student,
Raymond J. Hruby, DO, FAAO

[Editor's Note: *Principles and Practice of Osteopathy* by Dr. Downing is currently available at the AAO Book Store; call for your copy (317) 879-1881.]

Affiliated Organization's CME Calendar...

July 24-26
Annual Convention
Colorado Society of Osteopathic Medicine
Manor Vail Lodge
Vail, CO
Hours: 18 Category 1A
Contact: Patricia Ellis
(303) 322-1752

August 7-9, 1998
Counterstrain - Head & Extremities
Eastmoreland Hospital/
Northwest Osteopathic Medical Foundation
Hours: 20 Category 1A
Contact: Al Turner, DO, Director
OMM Department
(503) 230-2501

August 21-23
Osteopathic Integration in Working with Complicated Patients with Disbiosis
Indiana Academy of Osteopathy
Indianapolis, IN
Hours: 20 Hours Category 1A
Contact: Michael Claphan, CAE
(317) 926-3009

September 3-6
1998 Great Smoky Mountain Regional Osteopathic Scientific Conference
North Carolina Osteopathic Medical Assn
Grove Park Inn Resort
Asheville, NC
Hours: 25-30 Category 1A anticipated
Contact: Dawn K. Mirran
(800) 499-5751

September 11-13
Mid-Year Seminar
Florida Osteopathic Medical Association
Hyatt Regency Westshore
Tampa, FL
Hours: 20 Hours Category 1A
Contact: FOMA Executive Office
(850) 878-7364

October 9-11
The Three Diaphragms,
A continuing studies course
Melicien Tettambel, DO, FAAO, Director
Sutherland Cranial Teaching Foundation, Inc
UNECOM, Biddeford, ME
Contact: Judy Staser
(817) 735-2498

November 6-8
The Face
SCTF Intermediate Course
Douglas Vick, DO, Program Director
Eastmoreland Hospital
Portland, OR
Contact: Eastmoreland Hospital
OMM Department
(503) 230-2501

November 20-22
Annual Conference
The Osteopathic Wilderness Medical Society
Anapolis, CA
Hours: 18-20 Category 1A
Contact: Dickie Hill, DO
(707) 745-3785

The principles of osteopathic structural therapy – Part I

From: *Principles of Osteopathy*, by Leon E. Page, DO, Published by the AAO, 1952, Chapter II

Osteopathic structural therapy consists of manipulative and other physical techniques for the correction of faulty body structure. During half a century of practice, A. T. Still used and taught most of the basic osteopathic techniques which are in use today. The cumulative experience of osteopathic physicians has added refinements here and there, but, with the possible exception of cranial techniques, the basic osteopathic procedures were known and practiced at the beginning of the century.

Osteopathic technique consists of specific procedures intended for anatomical correction and for the stimulation of natural body functions by physiological means. It includes manual methods of adjusting articular structures throughout the body, correction of postural imbalances, and manipulation for the purpose of bringing about physiological responses. The practice of osteopathy (osteopathic medicine) as a system of therapeutics includes all measures which may be indicated in a particular case, but the practice of osteopathic structural therapy includes those distinctive methods which are the contribution of the osteopathic profession to the healing art. Thus, any form of treatment which conforms to the basic osteopathic philosophy may properly be called osteopathic therapeutics. In speaking of specific methods and techniques, the qualifying adjective "osteopathic" should be restricted to those methods which have been developed within the osteopathic profession. For instance, the administration of an enema is not osteopathic technique, although it is a useful procedure in the practice of osteopathic therapeutics.

Osteopathic technique consists in corrective passive movements applied to the body manually for the purpose of adjusting mechanical derangements in anatomical structure. The immediate objectives of such technique are to restore motion to immobilized articulations, realign bones which are in abnormal position, relax muscular contractures, release tension upon ligaments, tendons, and fascial layers which are subject to undue stress, facilitate the flow of fluids through vascular channels, and remove structural abnormalities which may interfere with the transmission of nerve impulses through established reflex pathways.

Because of the complexity of the mechanical principles involved, osteopathic technique cannot be standardized, Human bodies vary in dimension and consistency of tissue. Structural errors are infinite in their variety. It is possible only to point out in a general way which techniques are most likely to be useful in dealing with the more common types of structural perversion. Detailed discussion of methods of dealing with specific lesion problems may be found in the many excellent manuals on osteopathic technique and in articles appearing regularly in current osteopathic literature.

In general, it may be said that the forces used in corrective manipulations are leverage, traction, and pressure.

Leverage is used to secure motion along the plane of an articulation for the purpose of restoring mobility and securing accurate apposition of the articulating surfaces. In osteopathic spinal technique, leverage may be brought to beau upon a single articulation by utilizing the bony processes adjacent to the involved joint, muscular attachments, movements to the free parts of the body. The localization of leverage is obtained by the use of fulcrums properly placed for maximum leverage. Forces may be applied at either high or low velocity, depending upon the degree of locking in the articulation and upon the strength and sensitivity of the surrounding tissues. Leverage may be applied to spinal areas, including several segments as in the normalization of group lesions with scoliotic spinal compensation. In appendicular technique, the extremities serve as excellent levers, as in sacroiliac correction, and force may be applied accurately by utilizing

muscular attachments. Aids to leverage may be provided by mechanical appliances which materially assist the operator in handling heavy patients.

Leverage is the most potentially dangerous form of manipulative technique. The concentration of movement in a small area may result in injury to articular membranes, ligaments, and muscles with all of the phenomena of acute trauma. Leverage should be applied with full knowledge of the articular planes involved and the state of the tissues surrounding them. The amount of force should be carefully controlled by the operator as well as its speed and direction. Repeated efforts to secure correction of a lesion by leverage should be avoided.

Traction is a useful mechanical device used to relieve impacted and maladjusted articulations and to overcome muscular contracture. It is generally applied in the long axis of the body, although it may occasionally be employed at varying angles. The effect of traction is to separate articular surfaces and to stretch ligaments and muscles. To be effective, traction must be applied steadily and for a sufficient period to overcome the inertia of the tissues. Traction may be employed to overcome muscular contraction and to make correction by leverage and pressure easier.

Manual traction to the trunk may be accomplished by grasping the head or ankles and exerting a steady pull. Traction may also be applied to the extremities. Frequently, it is applied with leverage and pressure in making articular corrections. In the presence of severe traumatic lesions it may be necessary to apply traction over a long period of time with the aid of mechanical devices such as the Buck's extension. In chronic lumbar and pelvic articular lesions which are difficult to correct, the effect of prolonged traction may be enhanced by the employment of caudal or other forms of local anesthesia.

Pressure, as used in osteopathic structural technique, may be applied suddenly, as in the adjustive thrust, or it may be applied slowly or intermittently to articulations or soft tissues. The thrust is used to secure sudden release of pressure upon articulating surfaces, chiefly in the spine. It is usually applied by the palm or surface of the hand over one or more vertebrae involved in a structural lesion.❐

Many *Thanks* to the Supporters of the AAO's 1998 Convocation...

American Anatomical Corp.
BioMeridian, Inc.
Boehringer Ingelheim Pharm., Inc.
College Pharmacy
Colorado Springs Osteop. Foundation
Cranial Academy
Eli Lilly and Company (grant)
Gleason Orthotics
International Medical Electronics, Ltd.
Karemor
Lane Labs
Lippincott-Raven Publishers
Midwestern University/CCOM
Mosby
Mountain States Health Care Products
National Osteopathic Foundation
NYMOX
OMM Recruiting, Inc.
Osteopathic Center for Children
PCOM
Preferred Health Resources
Standard Homeopathic Company
Stretching, Inc.
Stronglite, Inc.
Sutherland Cranial Teaching Foundation
Thorne Research, Inc.
Total Health Enterprises/Nutrabalance
U.S. Army Heatlh Care Recruiting
W.B. Saunders Company
Wyeth-Ayerst Laboratories (grant)

Case History

by: Billy W. Strait, DO

Chief Complaint

Pain in the left hip, leg, and low back.

History of Chief Complaint

This is a 79-year-old white female who presented with complaint of left hip pain and some pain that goes down the back of her left leg, into her knee, and sometimes into her foot. She has had this for approximately two to three years. Patient denies any numbness or tingling. She denies any specific traumatic event to cause the pain. Patient does have diagnosis of arthritis and takes Relafen for this. This relieves the pain temporarily.

Past Medical History

Non-insulin dependent diabetes, hypertension, and heart arrhythmias.

Past Surgical History

Appendectomy, hysterectomy.

Social History

Patient is widowed. She denies the use of alcohol or tobacco. She is on a diabetic diet.

Allergies

No known medical allergies. Patient is allergic to dust and smoke.

Medications

Relafen, 500 mg, one p.o. t.i.d., Glucotrol, 5 mg, one p.o. b.i.d., Quinidex, 100 mg, q. day.

Physical Examination

On examination, patient's blood pressure was 134/82. Patient was examined in the standing, sitting, supine, and prone positions. HEENT was within normal limits. Heart was regular in rate and rhythm without murmur, click, or thrill. Lungs were clear to auscultation bilaterally. Abdomen revealed bowel sounds X 4 without tenderness. No organomegaly noted. Neurologic exam showed DTRs to be +2/4 in all areas. Great toe resistant plantar flexion test was negative bilaterally. Negative straight leg raising bilaterally. Patient was noted to be very flexible and was able to touch the floor with her finger tips with forward bending. Patient had a positive standing flexion test on the right. Sitting flexion test positive on the left. Compression test positive on the left. Patient was found to have a left sacral shear and left piriformis tender point. L3-5 were neutral, sidebent right, rotated left. T3-7 were neutral, sidebent right, rotated left. Left innominate was anterior. Right tibia rotated externally. Patient did have increased genu valgus on the right and positive valgus stress test of the right knee. Cruciate ligaments appeared to be intact. Patient does have increased thoracic kyphosis. The right longitudinal arch of the foot is flattened.

Initial Assessment

1) Probable degenerative joint disease of the lumbar spine and right knee.
2) Probable short leg syndrome.
3) Somatic dysfunction pelvis, lumbars, right lower extremity.
4) Non-insulin dependent diabetes mellitus by history.

Treatment Plan

It was recommended patient receive osteopathic manipulative treatment for the above mentioned somatic dysfunctions. This was performed using springing, respiratory cooperation technique for the sacral shear as well as airplane articulatory technique to the left SI joint. Muscle energy performed to the innominate, lumbars, and thoracics. Percussion hammer was performed to the ASIS while monitoring the sacrum. Counterstrain was performed to the left piriformis muscle. Pain was decreased in the low back and left piriformis area after treatment. It was recommended patient get a type A postural x-ray and x-ray of the right knee. Patient was rescheduled to see me in two weeks. She was also taught a low back/piriformis stretch to be done at least once daily.

Course of Treatment

Type A postural x-ray did show a sacral base unleveling of 10 mm short on the left. It also showed degenerative arthritis of the lumbar spine as well as spondylolisthesis of L3-4, L4-5 due to disc space thinning. Osteoporosis was also noted. X-ray of the right knee showed moderate tri-compartmental degenerative osteoarthritis. Patient did return in two weeks and stated she was feeling much better with less hip pain. Patient was evaluated and a return of the left sacral shear was found as well as a tender point over the piriformis muscle on the left and lumbar somatic dysfunction. These areas were treated with counterstrain, percussion hammer, and muscle energy techniques. A 1/8 inch lift was placed in the patient's left shoe. Patient was taught a pelvic tilt exercise to do each day. She was

→

scheduled to return in two weeks. At the next visit, patient stated she continued to improve and felt the heel lift improved her low back pain. She was also having minimal right knee pain at this time. Somatic dysfunction was found in the first left rib, pelvis, and lumbars. These were again treated using muscle energy, springing, and respiratory cooperation techniques. Patient again returned in approximately three weeks. At this time, she continued to do well in her low back and hip. She was having some pain in the right knee and because of this, I referred her to an orthopedic physician for evaluation of this. He felt the knee wasn't bad enough to perform knee replacement and that treatments should continue. I did treat the patient again using respiratory cooperation, muscle energy, and percussion hammer to pelvis and lumbar areas. Patient returned in approximately two months and stated she was doing much better. She was not having any radiation of pain to her legs and no pain in the right knee. I again treated her for some mild somatic dysfunction I found in the lumbar and pelvic areas. I placed her on a PRN follow up basis.

Discussion

This is the case of a 79-year-old white female suffering from left low back, hip, and leg pain secondary to osteoarthritis and structural mal-alignment secondary to a left short leg measured at the sacral base. This short leg most likely contributed to postural decompensation and increased stress in the low back and hip areas, as well as increased strain and stress on the right knee. Osteopathic manipulation and the introduction of heel lift therapy on the left did help to regain some structural stability and pain was diminished if not eradicated. Patient was able to perform gardening activities and other activities of daily living that she enjoyed without having discomfort.❐

Video Reviews

by Raymond J. Hruby, DO, FAAO

Editor's Note: In this issue we are offering reviews of two video tapes recently sent to the *AAO Journal*. They both come to us from the Walkinstown Osteopathic Clinic in Dublin, Ireland. The first video is called *An Introduction to Osteopathy in the Cranial Field* featuring Helene Emilie Jackson, DO. The second video is entitled *The Osteopathic Techniques of T. Edward Hall.*

In the first video, *An Introduction to Osteopathy in the Cranial Field,* we see a video featuring considerable discussion by Helene Emilie Jackson, DO. Dr. Jackson graduated from Kirksville in 1939 and was first introduced to the cranial concept by Beryl Arbuckle, DO. She also indicates that her grandmother was treated by A.T. Still and he apparently affected a remarkable cure of a cardiac problem that she had.

The video, itself, I found to be quite interesting in that there is a great deal of historical information to be learned from watching this tape. Dr. Jackson spends a great deal of time discussing a number of topics including approaches to treatment of children and some discussion of cranial mechanics and various treatment techniques that she had learned in her career. She particularly includes some interesting techniques useful for problems with respiratory infections and sinus infections.

I was particularly struck by the fact that such an interesting person would be preserved on video tape so that all of us could not only get insight into her techniques and the techniques that she was taught by her own teachers, but also into some further history about our osteopathic profession.

In the video entitled *The Osteopathic Technique of T. Edward Hall*, I found another tape of considerable historic interest as well as educational interest. Dr. Hall, like Dr. Jackson, was a very well known and revered osteopathic physician in England for many years. Of particular interest was the fact that he was a student of J. Martin Littlejohn who, as we know, was one of the founders of the Chicago College of Osteopathic Medicine and who later migrated to England to form the foundation for the osteopathic profession in that country. Dr. Hall presents a great deal of technique on the tape so that we have some insight not only to his techniques but also to the influences on him from Dr. Littlejohn. In addition, the tape provides a great deal of historical information about Dr. Littlejohn and it is one of the nicest presentations of this historical information that I have seen in a long time.

If there is anything negative to be said about both of these video tapes it is that the production is homemade, if you will. It is not done by the kind of professional video company as we would experience on many video tapes here in the United States. However the quality is still good and the tapes offer valuable information.

Putting aside the above remark regarding the video quality of the tape, these tapes are extremely full of information that is not only historical in nature but is quite valuable to the practicing clinician. Although there is a great deal of repetition in some parts of the tape and perhaps they are a little bit longer than they need to be, nevertheless they are extremely well worth viewing and I would dare say that any DO who would be able to view these tapes would gain a great deal of knowledge and insight from them. I would recommend these for anyone's library.

[Information regarding pricing and availability of these tapes, as well as other available videos, can be obtained from the Walkinstown Osteopathic Clinic, 18 Cromwells Fort Road, Walkinstown, Dublin 12 Ireland; phone: 01-450-4438.]❐

Role of the DOs in the "Spanish Flu" Pandemic of 1918-1920

by Martyn E. Richardson, DO

In Feb. 4, 1998 issue of the *USA Today,* there was an article about influenza with reference to the 1918 flu pandemic with observation that the doctors had little to offer.

They did not know about the osteopathic physician – for the DOs had something extra to offer. As a result, their survival rate appeared to be far greater than the other professions.

My father kept records of all his patients and submitted the final report to the Academy of Applied Osteopathy as did 2,500 DOs.

I have some of my father's records, had conversations with some other DOs in Norfolk, VA (where servicemen from Europe apparently brought the virus to the port), but the most interesting observations were from my mother and patients.

My father (PCO '08) had a subspecialty in Ear, Nose, and Throat Surgery, had a large number of patients for general practice and regular osteopathic manipulation treatments. But, during the epidemic he curtailed office hours because he made house calls daily or twice daily. A DO author, in 1919, indicated that the DO should cancel all office hours in order to "properly" care for flu patients in their homes.

The house calls were for manipulative treatments. My father usually began with "raising ribs" with the patient on their back, going up and down both sides. He might "spring" the ribs with patient on side. Attention was also paid to thoracolumbar area for kidney function. There was relaxation of the cervical and suboccipital area with pressure over the supraorbital area and the maxillary sinus. Another cervical relaxation was followed by shoulder motion to "free the clavicles" and mobilize "the scapula." The patient extended the arms over the head and took one or two deep breaths to aid in respirations. The patients felt better, coughed phlegm, sinuses drained, and temperature stabilized. The treatment took 5 to 10 minutes, then was repeated in 30 minutes to 1 hour. "Do not fatigue the patient." Of 18 DOs who reported their experiences in *JAOA*, 1919-1920, 5 indicated OMT for general relaxation, 8 emphasized thorax and ribs, 6 stressed cervical and shoulders, 3 mentioned anterior cervicals, and 4 said to "treat osteopathically." None mentioned "lympathic pump." Several mentioned about temperature decline and patient feeling better after the treatment.

Sometimes, specific corrections were made in the bed. Others said "never while fabrile."

He outlined "supportive therapy." This usually included a saline enema (perhaps daily), cool packs to wrists to control fever, use of various counter irritants and emplastums to the chest (antiphlogistine). (The article in *USA Today* mentioned using hot pancakes to the chest). Vaporizers, Benzoin, Cremulsion, Vicks, etc. were used at intervals, but open windows and fresh air were required. The caretakers were shown how to "raise ribs" every 1-2 hours.

Of 18 DOs, 10 used enemas (prn or daily); 6 advised carthatics, 4 specified using bedpans to avoid getting out of bed, all used warm applications, blankets, and counter irritants or emplastums to chest. All mentioned fresh air in rooms. All physicians urged "forcing fluids," lots of water. The diet was frequent small feedings of "natural" fruit juice, vegetable juices, lemonade, broth, or clear soups., rice, wheat toast or crackers, baked potatoes. Milk was prohibited (something about making the body too alkali) until recovery. Other DOs used dilated milk, even ice cream. After recovery, milk toast, chicken, and cereals were resumed. My father advised no fried food and no salt for everyone. He also used orange juice, yeast cakes (B) and cod liver oil for vitamins in illness and recovery. My father also insisted that cheerfulness and hope be in the sick room.

Footnotes: Medications were not used at all by DOs, even aspirin being condemned by some. One author claimed that patients treated at the same time by MDs with medicines and DOs with manipualtion did not have a survival rate as good as DOs alone. Smith and others quote Dr. Osler and McCrae who reported in 1919 about the medicine being used for flu and pneumonia. "Antimony, chloroform, iron, quinine, creosote, and a host of other drugs have proved valueless. Serum therapy, vaccines, and leucocytic extracts have not been proved to be of benefit."

That is how my father and 2500 other DOs helped the thousands of patients survive the ravages of one of the most terrifying and deadly pandemics of modern times. This was done without the medicines of our day, before antibiotics, steroids, and high tech drugs and procedures of today.

They did it by utilizing Stills teachings. They focused on removing the primary and reflex changes produced by the severe infections so that the body could most efficiently master its army to fight the invader. This was by improving respiratory function, by supporting the circulations, by providing for increased lymphatic flow, and elimination of "toxins" through kidneys, bowels, and lungs.

It worked in 1918 and it still has validity today.

Are we using this osteopathic manual medicine approach effectively in the respiratory problems today?

Would we devote the same energies and time to our patients if the avian virus H5 in Hong Kong becomes a pandemic?

The history:

Many epidemiologists continue to study the 1918 "Spanish Flu" epidemic, now using DNA identification plus viral classification of tissues from 1918 patients. This is particularly urgent in view of what has happened in Hong Kong in 1997. (This is an avian virus which adapted to human infection (reassortment, antigenic drift) which has the potential to become another pandemic for which humans have no immunity.)

In March of 1918, some flu cases were reported at an Army camp in Kansas and went around the glob but did not attract much attention during World War I. Four months later the virus changed, exploding at about the same time in France, Spain, and Boston, causing people to die "awash in blood and gore literally drowning as fluid filled their lungs." It was also frightening that unlike most respiratory illnesses, this killed patients in the prime of their life, 25-34 years old, as well as the young, the old, and the sick.

During the next few months it was killing people all over the world, from isolated villages in Central Africa and the mountains of India, to all the major metropolitan centers of the world.

The 1918 flu had disappeared by 1921, returning to the variable but less deadly flu of the past "La Grippe." At the same time, farmers were discovering that their pigs were sick with high fevers and cough, dying just like humans had. This continued every year until 1930 when the virus was identified. (This is significant in the Hong Kong avian flu problem.)

1. In 1919, the *JAOA.* compiled statistics, evaluated the reports and making certain of validity of cases:
   1362 osteopathic physicians reporting 47,197 cases of influenza.
2. In January 1920, the statistics:
   2445 osteopathic physicians reporting 110,000 cases of influenza;
   257 deaths 0.25%;
   6258 cases of pneumonia;
   635 deaths 10%.
3. Another study: Pediatrics in Los Angeles County Hospital: 1936-39 - 300 cases with OMT compared with several MD children's hospitals:
   Pneumonia: LA. County Hospital 10.66% mortality;
   Pneumonia: Major children's hospitals 29.6% mortality.

Statistics:

1. Flu epidemic 1918:
   20 million deaths world wide;
   20 million cases in the US (1 person in 4);
   500,000 died in US;
   1 of 4 American soldiers died of the flu in WWI.
2. April 1919-1920 statistics:
   Fatalities from influenza:
   Medical losses estimated 12 to 15%;
   Osteopathic (actual) 0.25%.
   Fatalities pneumonia:
   Medical civilian - estimated - 25%.
   U S Army camps - actual - 38.9%.
   Osteopathic civilian - actual -10%.

Since essentially the only difference between DO and MD management was OMT and no medicines, it must be considered to be the significant reason for the difference and of equal value for any respiratory problem.

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* Other articles also in these issues.

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Margaret Rich, AOA Publications Department

❐

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5:45- 6:15	"Visceral Manipulation"
6:15- 6:45	"Cranial Osteopathy" includes Q/A
6:45- 7:15	"Myofascial Release"
7:15- 7:45	"Counterstrain"
7:45-8:15	"Muscle Energy"
8:15- 8:45	"High Velocity/Low Amplitude"
8:45- 9:15	"Exercise"
9:15- 9:30	Closing Comments

Friday, September 18

7:00- 8:00 am	Breakfast Lecture Coding Update – Getting Paid for What You Do"
8:00-10:30	Lecture: "Thoracic Trouble-shooting" (to include modalities approach - HVLA, ME, counterstrain, indirect-MFR & cranial) Skills Session: Thoracic
10:30-11:00	Break
11:00- 1:30	Lecture: "Cervical/Suboccipital Troubleshooting" Skills Session: Cervical/Suboccipital Wrap-Up Session: (Summary) – Faculty
Friday PM	Free time for exploration

Saturday, September 20

7:00- 8:00 am	Breakfast Lecture Coding Update – Part II
8:00-10:30	Lecture: "Lumbar/Pelvis Troubleshooting" Skills Session: Lumbar/Pelvis
10:30-11:00	Break
11:00- 1:00pm	Lecture: "Upper & Lower Extremities Troubleshooting" Skills Session: Extremities
1:00- 1:30	Wrap-Up Session: (Summary) – Faculty
Saturday PM	Free time for exploration

Sunday, September 21

7:00- 8:00 am	Breakfast Lecture – Coding Update Part III
8:00-10:30	"Exercise"
10:30-11:00	Break
11:00- 1:30	Preparation for Manipulative Boards

Case Study Preparation – "How to write them"
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Letter to the Editor

Dear Ray:

I was pleased to read Dr. Fred Mitchell Jr.'s letter to the editor, published in the spring 1998 issue of the *AAO Journal* in which he discusses **"The Sacrum a Bone of Contention."** I want to take this opportunity to thank Dr. Mitchell for his comments and respond to them.

"The Sacrum a Bone of Contention" was written with the intent of comparing two systems currently in use for diagnosing and treating Lumbo-Sacro-Pelvic somatic dysfunction. I recognized that although these two approaches deal with somatic dysfunction from very similar perspectives, many clinicians who use one system have difficulty understanding descriptions of dysfunctional mechanics when presented in terms of the other system. For this reason, I reviewed the writings of the authors who originally proposed both systems and sought out as many research papers that studied sacral motion as I could find. After reviewing the literature, I was impressed by the marked similarity between the description of motion mechanics of the two systems. From my review, I drew the following conclusions:

1) A sacral forward torsion (neutral) identifies the same lumbo sacral mechanics as type I (neutral) lumbosacral group mechanics.

2) A sacral forward torsion identifies the same sacroiliac mechanics as anterior sacrum and posterior sacrum, recognizing that the sacral torsion model does not lateralize to the side of the dysfunctional sacroiliac articulation the way the anterior sacrum - posterior sacrum model does.

3) A sacral backward torsion (nonneutral) identifies the same lumbosacral mechanics as L5/S1 type II (non-neutral) mechanics.

4) A sacral backward torsion describes a relationship between the sacrum and ilia which has not been specifically described elsewhere.

I further concluded:

5) Articular somatic dysfunction is most appropriately diagnosed by assessing available articular motion. This may be augmented by findings of tissue texture change and boney landmark position.

6) Sacroiliac coupled sidebending rotation is not type I mechanics since the sacroiliac articulation is not, according to Fryette's criteria, a typical vertebral articulation.

7) Axes of sacral motion must be recognized as descriptive conveniences rather than kinesiologic reality. Research into sacral motion has clearly demonstrated that sacral motion upon fixed axes of rotation does not occur.

8) The terms "sacroiliac" and "iliosacral," when applied to primary articular dysfunction between the sacrum and ilium are redundant. Selecting a single term to describe dysfunction of this articulation is proposed to reduce confusion.

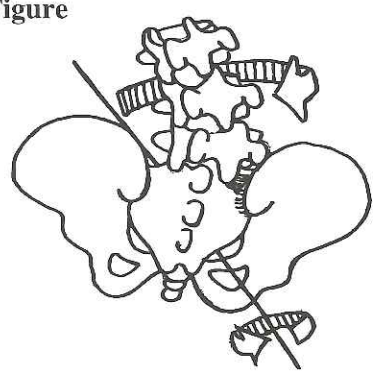
I made these statements, not as the absolute answer to the problem of the two systems, but rather as a challenge to the osteopathic profession to come to consensus. We discussed this subject at the "Conclave of Fellows" at the AAO convocation this past March. A consensus was not achieved, but a dialogue has been initiated. Dr. Mitchell's presence was greatly missed at the conclave but his letter contributes greatly to dialogue.

In specific I would, at this point, like to present the following responses to Dr. Mitchell's comments.

• Dr. Mitchell states he believes the anterior sacrum/posterior sacrum model should be replaced by the sacral torsion model because of the latter's "Greater Clinical Power."

The sacral torsion model has not yet replaced the anterior/posterior sacrum model. Both exist as contemporary diagnostic/therapeutic approaches as indicated by their inclusion in the recently published *Foundations for Osteopathic Medicine.* I believe it is inappropriate to discard one approach totally in favor of another, if both are, as they are, established and effective. Rather, I believe we should look intensely at both approaches. We should identify commonality and we should look particularly for individual uniqueness.

Figure



Sacral forward torsion: consisting of – sacrum rotated left on the left oblique axis and lumbar spine rotated right. Lumbar sidebending left results in engagement of the left oblique axis. Also consistent with – sacrum sidebent right/ rotated left (left oblique axis) resulting in anterior sacrum right and/or posterior sacrum left. The lumbar spine is sidebent left/rotated right (Fryette, Type I)

In my conclusions listed on the previous page, I underscored areas where one system offers insight that the other does not. Conclusions #1 and #2 compare sacral forward torsion with lumbar type I mechanics and anterior sacrum-posterior sacrum (see figure).

Sacral forward torsion gives a complete picture of the dysfunctional pattern of the entire lumbo-sacro-pelvic region. Anterior sacrum, posterior sacrum, and lumbar type I mechanics are descriptions of articular dysfunction. This articular approach considers dysfunctional mechanics which are consistant with but, (with the exception of the lumbo scacral pattern) may or may not be present within a forward torsion pattern.

Conclusion #4 recognizes that sacral backward torsion describes mechanics between the sacrum and ilium which are described nowhere else.

To replace one model with another, before they have both been thoroughly understood, risks the loss of those unique contributions.

It is impossible to definitively state that one of these models of sacral diagnosis is of "Greater Clinical Power" than the other because there are no clinical studies to unequivocally support such a statement.

• I must here apologize for misrepresenting Dr. Mitchell. My statement (Sacrum A Bone of Contention *AAO Journal,* Winter 1997, Pg. 19) was: "there are sufficient variations within normal anatomy to invalidate positional diagnosis alone." I referred to the following from *The Muscle Energy Manual, Vol. one*, pg 21, "The two cornua are often of different sizes and this may mislead the examiner to believe a sacral positional fault exists." Since this is the final sentence in the paragraph it must be considered to be a serious threat to positional diagnosis. While Dr. Mitchell does warn of the problem of anatomic asymmetry, he makes no reference to diagnosis by motion testing. He states that a definitive diagnosis may be made by systematically evaluating the symmetry of several additional pelvic landmarks. I readily acknowledge Dr. Mitchell's skill and have no doubt that this is a diagnostically effective approach. I do not dismiss the diagnostic usefulness of positional asymmetry.

• My conclusion that motion restriction is of greater diagnostic importance than positional asymmetry applies particularly to articular somatic dysfunction. It is based upon the following logic. The majority of osteopathic techniques are classified as "direct" or "indirect" according to how they address the "restrictive barrier." The "restrictive barrier" is defined in the *Glossary of Osteopathic Terminology* as "a functional limit within the anatomic range of motion..."

If techniques are employed to address a functional limitation of motion, it follows that identification of available motion is an extremely important parameter in the determination of the most effective therapy. Asymmetry of position, tissue texture change and tenderness are important diagnostic criteria; however, they do not specifically identify the circumstance (motion restriction) that OMT is most often intended to treat.

Visual assessment for positional asymmetry may be easier to master than the palpatory diagnostic skills necessary to identify available motion. Easier however, should not be equated with better. I have used motion restriction as my primary diagnostic criterion for somatic dysfunction for years. I also evaluate positional asymmetry and tissue texture change. I employ findings of tissue texture change to initially identify areas of significant dysfunction and to quantify the severity of the dysfunction. I identify positional asymmetry to give me an idea of the patients overall structural pattern. I employ the assessment of motion and identification of motion restriction to specifically define the dysfunctional mechanics and identify how I plan to initiate treatment. It is the reestablishment of unencumbered motion (function) that is my therapeutic goal in addressing the restrictive barrier.

The appreciation of motion restriction, as Dr. Mitchell points out, may be obscured by tissue texture change (including muscle spasm) around the dysfunctional articulation. Typically, the pattern of motion is not obliterated. The palpatory quality of the dysfunctional barrier is different than that of articular dysfunction in the absence of marked soft tissue involvement. In the presence of marked soft tissue involvement dysfunctional barrier is less distinct. Such findings are important. The quality of the restrictive barrier and tissue texture change not only allow the examiner to assess the acuity (or chronicity) of articular dysfunction but also to identify nonarticular dysfunctions (fascial problems or viscerosomatic reflexes) as well.

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The identification of a specific articular dysfunctional motion pattern allows one to select a specific articular therapeutic technique. Failure to identify a specific articular dysfunctional motion pattern means that a technique directed at soft tissue dysfunction or the primary respiratory mechanism is a more appropriate choice. Dr. Mitchell makes the point that "degrees of articular mobility can logically and reliably be inferred from scientific observation..." This is most probably true but I believe that whenever possible one should base diagnosis upon direct assessment (in this case of articular motion) rather than inferences.

It may well be that the description of dysfunction identified by the sacral torsion model is satisfactory when employing muscle energy technique. It lacks specificity, however, when attempting to address the problem using high velocity/low amplitude technique. Yet, there is little doubt that both technical approaches can be effective.

• In reference to my suggestion that "technical methods," be used wherever possible to delineate articular motion I did mean instrumental recording. The best way to assess the clinician's subjective interpretation of motion by palpation is through the use of objective reproducible methods. This does not mean that instrumental recording should be employed clinically to diagnose somatic dysfunction. Contemporary medicine has become too expensive, in part, because so many clinicians no longer trust their skills of physical diagnosis as first-line indicators of pathology, relying instead on more costly technology. Wherever possible, when scientifically studying osteopathic diagnosis and treatment, instrumental methods must be used to delineate unequivocally the functional mechanics that osteopathic techniques are used upon. Osteopathic medicine is predominantly based upon clinical empiricism. This is acceptable for an art. It is not acceptable for a science.

• The issue of "Ilioscral" versus "Sacroiliac" must be considered by answering the following questions:

1) Are these terms intended to describe somatic dysfunction of the articulation between the sacrum and Ilium? If they are, then they are redundant, and only one term is necessary.

2) Are these terms intended to describe the mechanics (or forces) that have resulted in somatic dysfunction of the articulation between the sacrum and ilium? If so, the following questions arise.

A) Have these causative mechanics or forces resolved? If they have resolved and the residual dysfunction is, as above, of the articulation between the sacrum and ilium, then terms are redundant.

B) Do the causative mechanics (or forces) remain, and as such are they responsible for maintaining the articular dysfunction between the sacrum and ilium? If so, the dysfunction between sacrum and ilium is secondary to the causative mechanics (or forces) and the dysfunction is most appropriately named in terms of those etiologic mechanics (or forces). The terms sacroiliac and iliosacral DO NOT communicate specific etiology.

Dr. Mitchell states the terms iliosacral and sacroiliac "refer to different physiologic mobility functions" produced by lower extremity and spinal movement. If respectively by this he means these terms do not designate articular dysfunction, but rather are descriptions of complex dysfunctional relationships between the pelvis and lower extremity and between the pelvis and spine, then I recognized the differentiation being made. In this instance, the muscle energy model of nomenclature appears to be more global than the high velocity/low amplitude model, which must of necessity designate specific articular dysfunctional motion patterns.

• The rotation of the sacrum upon an oblique axis, as both Drs. Magoun Sr. and Mitchell Sr. stated, is the motion that occurs as the sacrum moves through coupled sidebending and rotation. I do not believe that the sacral axes of motion, as descriptors, should be discarded. These descriptors facilitate understanding of sacral mechanics and are useful teaching tools; however, the actual motions appear to be complex combinations of rotation and glide. To state that the sacrum rotates upon any axis like a wheel upon an axle is a gross oversimplification.

Dr. Mitchell Jr.'s work with Dr. Pruzzo demonstrated a horizontal transverse axis of sacral respiratory motion at the level of S2. Other authors cited measured active sacroiliac flexion/extension with variable results. Only Strachan et al. (1938) and Figerio (1974) considered motions outside the midsagittal plane. No specific axes were identified by these authors. Strachan specifically stated that precise axes of motion were not demonstrated. Further, the demonstration of a horizontal transverse axis for motion in the coronal plane can in no way be construed as proof of the existence of any other axis of motion, particularly the oblique axes.

Be that as it may, I do not believe that Dr. Mitchell and I are that much in disagreement. I use the hypothetical axes of sacral motion when teaching. They are very convenient when attempting to explain the complexities of sacral motion. When reading Dr. Mitchell, I could not help but notice that when describing the various sacral axes on page 21 of *The Muscle Energy Manual, Vol. One* he makes the following qualified statements:

"Normal flexion and extension nutations of the sacrum appear to occur around more than one transverse axis."

"The axis for this respiratory movement is thought to be..."

"Under these circumstances the

sacrum probably rotates around a transverse axis located..."

I believe it is appropriate to use axes of-motion to describe sacral mechanics. If osteopathic medicine is to continue to gain in creditability within the scientific community, however, we must clearly differentiate between our conclusions that are based upon science and those based upon empiricism. The axes of sacral motion have not been thoroughly tested by rigorous scientific method. The few studies that appear to demonstrate sacral motion, as if occurring upon an axis, have not been reproduced independently. It is too soon to state that scientific proof of such motion exists. Rather, it is appropriate to qualify our statements with such praises as "appear to," "thought to be" and "probably."

A clinician who uses only high velocity/low amplitude technique may be satisfied with the anterior sacrum/posterior sacrum model. A clinician who uses only muscle energy may be satisfied with the sacral torsion model. However, the osteopathic profession embraces multiple systems of diagnosis and treatment. It is to our advantage to identify, wherever possible, redundance within these systems. The progress of osteopathic science depends upon precision of vocabulary. In my conclusions listed above, I have noted that the term "sacral forward torsion" fails to descriptively lateralize articular somatic dysfunction between the sacrum and ilium the way the terms "anterior sacrum" and "posterior sacrum" do. Further, the term "sacral backward torsion" describes a relationship between the sacrum and ilia which does not appear to have been described elsewhere. The two systems we are discussing have their strengths and weaknesses. I believe it would be a loss to replace one with the other, either way. Wherever possible it is desirable to at least be able to understand the diagnostics of one system in terms of a comparable approach. The challenge before us is not to defend the specific approach we employ. These diagnostic systems work. They do not need to be defended. They need to be understood, recognized when they are similar, and appreciated where they are different. The challenge to us all is to identify where they are similar and HOW they are different. This will allow us to formulate a single vocabulary that incorporates both systems.

Sincerely yours,
Ken Nelson, DO, FAAO

1998 FIMM Congress Review

by Craig M. Wax, DO

The 12th International Congress of FIMM - Federation Internationale de Medicine Manuelle convened 13-17 April 1998 in Gold Coast, Australia. The conference was hosted by FIMM and the Australian Association of Musculoskeletal Medicine (AAMM). The conference focussed on Musculoskeletal Science in Practice – Strategies for Tomorrow.

The conference was well attended by over 200 physicians and therapists. There were osteopathic physicians from the United States, Australia and Europe, medical doctors, chiropractors, and physiotherapists in attendance. The event was chaired by Dr. Philip Watson of New Zealand.

The site was Conrad Jupiters Casino/Hotel in Gold Coast, Australia. The accommodations were located 40 minutes drive from the Brisbane airport.

American-trained and AOA-certified DOs had a significant presence at the conference. **Herb Yates, DO, FAAO** of Kirksville College of Osteopathic Medicine led a workshop on counterstrain technique preceding the Congress opening. **Ray Hruby, DO, FAAO** served as one of the table trainers.

The conference conducted lecture series and paper presentations on various topics. The plenary sessions were organized into categories; anatomy and mechanisms, evaluation of assessment methods, manual therapy and promotion and education. The first session was entitled Aetiology of Musculoskeletal Pain.

Craig M. Wax, DO of Middletown, NJ presented his paper, "Chest Pain and the Role of Somatic Dysfunction"; *JAOA*, Vol 97, No 6, 347-355. A case of chest pain was presented and osteopathic manipulative methods of diagnosis and treatment were discussed.

Wolfgang Gilliar, DO, a physiatrist from California, presented a discussion on a Consensus Document on the "Role of Manual Therapy in the Treatment of Spinal Pain of Mechanical Origin." Dr. Gilliar began the next plenary session with "Overview of Conservative Treatment Management for Various Musculoskeletal Disorders."

Harry Friedman, DO of California presented a workshop on "Standardized Records for Musculoskeletal Examination."

Concurrent Evaluation of Treatment Methods was the next set of lectures. These included topics on sitting and low back pain, surgical treatments, midline thoracic pain and variations in lumbar intradiscal pressures during manipulation. Subsequent sessions addressed pain management and medicolegal issues. A workshop on epidural injection was offered.

Professor **Robert Ward, DO** of Michigan State University College of Osteopathic Medicine presented a lecture on "Integrating Cranial Nerve and Release Enhancing Exercise Assists Several Manipulative Methods." The presentation was interactive and informative.

Overall, the 1998 FIMM Congress succeeded in bringing together world approaches to musculoskeletal health. The next Congress, in 2001, will be a United States site yet to be determined. The chairperson will be **Michael Kuchera, DO, FAAO** of the Kirksville College of Osteopathic Medicine. ❐

AOA Convention

October 6-8, 1998

New Orleans, LA

AAO Program

Elaine Wallace, DO, Program Chair

"Helping the Body Heal"

Tuesday, October 6

9:30 am *History of lymphatic techniques*
Hugh Ettlinger, DO, FAAO

10:30 *A review of the immune system (basic and neuroimmunology)*
Bonnie Buxton, PhD

11:30 *Lymphatic function & dysfunction*
Brian Degenhardt, DO

3:00 pm *Lab: Balancing the diaphragms*
Boyd R. Buser, DO

4:00 *Lab : Self administered lymphatic pumps*
David Essig-Beatty, DO

Wednesday, October 7

8:15 am *Lab: Gateway to the upper extremity (Posterior axillary fold)*
Michael Kuchera, DO, FAAO

9:15 *Lab: Lower extremity techniques (Faciliated Positional Release)*
Eileen DiGiovanna, DO, FAAO

10:15 *Lab: Chest/Lung Disease (Inhibition Techniques)*
Dennis Dowling, DO

11:15 *Lab: GI Disease (Visceral Technqiues)*
John Glover, DO

2:00 pm *Lab: OMT and the OB patient*
Melicien Tettambel, DO, FAAO

3:00 *Lab: OMT in the pediatric patient*
Robert Kappler, DO

4:00 *Lab: HEENT (Techniques for the Office)*
Dave Boesler, DO

Thursday, October 8

(Co-sponsored by the American Osteopathic Academy of Sports Medicine)

8:15 am *Exercise and the lymphatics*
David Eland, DO

9:15 TBD

11:15 Northup Lecturer
Eileen DiGiovanna, DO, FAAO

2:00 pm *Open Forum – New techniques from the field*
Donald Hampton, DO, host

3:00 *Coding Update*
Judith O'Connell, DO, FAAO

Last two Visceral Courses in 1998. . . then . . .

Jean-Pierre Barral, DO of France will visit the AAO, once again!

Prepare for Dr. Barral's visit, March 1999
at the 1999 Annual Convocation in St. Louis, Missouri
by attending the . . .

G.I./Abdominal Visceral Manipulation Workshop

August 14-16, 1998
St. Paul, Minnesota

Thorax-Dura Visceral Manipulation Workshop

November 6-8, 1998
San Francisco, California

G.I./ Abdominal Workshop

Demo of visceral manipulation, general principles, general listening, local listening, liver, gall bladder, stomach, duodenum, small intestine, large intestine, sphincters.

This course provides a theoretical background for subsequent courses using the gastrointestinal tract as a model. Issues of induced motion (mobility), inherent motion (motility), ligamentous attachments, sympathetic and parasympathetic nerve associations, diagnostic techniques such as general listening, local listening, and inhibition, ligamentous tension, short and long lever arm.

In this course, we will cover the role of the visceral dysfunction involved in such diagnosis as gastroesophageal reflux disease, dyspepsia, hiatal hernias, peptic ulcers, acute and recurrent mid-thoracic pain, cholestasis, cholelithiasis, biliary congestion, sinusitis, hepatitis, irritable bowel, constipation, thoracolumbar pain, pelvic pain, low back pain, and recurrent SI dysfunction, to name a few.

By the end of the course, the participant should be able to manually diagnose, and manipulate associated structures in the above mentioned diagnosis. Prior course participants report using this knowledge daily in a wide variety of medical and musculoskeletal complaints.

Thorax-Dura Workshop

In this course, we will explore viscera concepts in the deeper visceral structures, the thorax. Since these are more protected by the thoracic cage, they can be more difficult to accurately diagnose and treat. Labs will emphasize evaluation of the participants palpatory diagnosis by the instructors. At this level, participants generally begin to find that they can more consistently predict patient's symptoms, based on physical diagnosis (palpation). We also explore a very fast and precise cranial evaluation and direct treatment approach including the sutures, membranes, dura up to and including the eyes, foramen magnum, upper cervical spine, lower thoracic spine, S2, and the coccyx.

Relevant diagnosis covered are recurrent respiratory infection, chronic bronchitis, prior respiratory infection with resultant pleural scarring, asthma, recurrent upper thoracic pain, sciatica, radiculopathy, compromised respiration, coronary tension, CHF, chronic pain syndrome, recurrent sternal restrictions, chronic chostochondritis.

For more information, contact:

American Academy of Osteopathy®
3500 DePauw Blvd.,Suite 1080
Indianapolis, IN 46268-1136
Phone: (317) 879-1881
Fax: (317) 879-0563

Osteopathic Clinical Research Communications

by Deborah M. Heath, DO and Albert F. Kelso, PhD

The goal of this series is to foster osteopathic clinical research to advance osteopathic practice and theory. Increasing communication with and between researchers who share this same goal insures the quality of osteopath practice and contributes to effective health care delivery. The first article in the series identifies a new federal clinical research initiative that uses quality health measures to support the benefits provided for patients and evaluates the satisfaction and benefits received. Health benefits and outcomes have become a benchmark for measuring health care delivery. Such data provides credible information for participating physicians; third-party payers; government regulars; and funding agencies. The Agency for Health Care Policy and Research (AHCPR) has added $25 million to their budget to support three priority clinical research programs that emphasize improvement in health care outcomes.

These federal initiatives provide the osteopathic profession and its physicians with a golden opportunity. The obvious immediate and long-term health benefits experienced by the patient has captured the interest of many involved in the health care delivery system. Additionally, osteopathic manipulative medicine consistently reduces costs and reduces unnecessary expenditures for health care but documented information on DO's practices is limited. How many costly diagnostic studies have failed to yield an accurate diagnosis of health problems related to somatic behavior? These failures lead to further procedures and specialty consults. Many times patients state with appreciation, "I wish I was able to get to you sooner, I cannot believe that nobody knew what my problem was and you are the only one that has helped me. Thank you. "This is patient satisfaction!

Systemic documentation is essential for effectively communicating these health benefits, cost savings and health outcomes. Information on outcomes require longitudinal plans for data collection and reporting. Cooperating physicians, research teams and a research center to manage profession wide data collection establish the system and provide for data collection, management and reporting. The professional center is essential; it provides the needed infrastructure and a permanent center to receive, analyze, store and retrieve data. In the absence of such an infra structure the system is unlikely to survive. The AOA, AAO or officially designated service is needed for managing the system. Equally important to reliable documentation of care are physician and research team cooperation and compliance in reporting patients' needs, the health care provided and the outcomes.

An indication of your interest in seeing such research conducted or a willingness to participate in such a program should be made known to your college, clinic, hospital, provider organization and the American Academy of Osteopathy. In additional to your other contacts, we would appreciate receiving a copy of your communications. Direct your copies to the Louisa Burns Clinical Research Committee. Also, for further information on the AHCPR programs, visit the AHCPR web site: http://www.ahcpr.gov/ and click on "funding opportunties."❐

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A pilot study: Osteopathic treatment of infants with a sucking dysfunction

by Maxwell M.P.R. Fraval DO, M. Osteo. Sc. (Paed.)

Editor's Note: *Dr. Maxwell is an affiliate member of the American Academy of Osteopathy as well as The Cranial Academy. He currently is in practice in Australia.*

Abstract

The estimation of the fat content of breastmilk, by simple centrifuge method, is a reliable measure that is easily obtained. Estimations from six infants who were feeding normally demonstrate that it provides a gold standard against which patient outcomes can be measured.

A pilot study of six infants is reported on. At the time of first measurement, the difference between pre- post feed fat estimations of breastmilk was small in infants with a dysfunctional suck. Following osteopathic treatment the difference between pre- post feed fat estimations were comparable with the fat estimations from the breastmilk of infants who were feeding normally. The results are encouraging enough to warrant extending this to an age- and sex-matched case-control study.

Key words

Breastmilk fat estimation, sucking dysfunction, lactation and osteopathic treatment.

Acknowledgments

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I particularly wish to acknowledge the early encouragement received from Dr. John Harakal (now deceased), former President of the Sutherland Cranial Teaching Foundation and Professor of Osteopathic Manipulative Medicine at the Texas College of Osteopathic Medicine.

Thanks are also due to Dr. Viola Frymann, Director of the Osteopathic Children's Centre in San Diego California and Dr. Edna Lay, Vice President of the Sutherland Cranial Teaching Foundation.

I am also much indebted to Professor Peter Hartmann of the Department of Biochemistry at the University of Western Australia who so freely provided me with information about his research. The estimation of fat concentration in breastmilk was one of the measurement techniques to which he drew my attention.

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* 51 Learmonth Drive, Kambah 2902 A.C.T. Australia;
Telephone +61 2 6231 9111; Facsimile +61 2 6231 9195; E-mail mfraval@effect.net.au
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Introduction

Osteopathy has been reported as effectively delivering paediatric care.[1,2,3,4,5,6,7,8,9] This study assumed that, as proposed by Sutherland,[10] there is a pattern of normal motion in the cranium that is present throughout life and that the birth process may produce strain patterns within the cranium which may alter normal physiology. The scientific basis for this was recently reviewed by Myers.[11] The study further assumed that these strain patterns can be recognised and accurately recorded by careful palpation.[12]

At parturition the tentorium cerebelli and falx cerebri can sustain tremendous strain*[13] with consequent deformation.[14,15] Initially, the head presents in the transverse diameter of the pelvic brim with the occiput to the left side of the maternal pelvis. Rotational forces affect the occiput which engages eccentrically with one part leading first (asynclitism).[16]

Particularly in the case of a prolonged second stage or other complication, the resulting micro- or macro-trauma has been postulated (within the osteopathic profession) to cause distortion of function in both the bony and soft tissues as well as related fluid (vascular, lymphatic and cerebrospinal) systems.[14,15,16,17]

Magoun states that a commonly occurring distortion resulting from the birth process is an approximation of the anterior ends of the condylar parts which "may have the effect of putting increased tension through the slip of dura mater that divides the jugular foramen and thereby irritating the cranial nerves (Glossopharyngeal, Vagus and Accessory nerves) passing through the area. In addition the petrosal and sigmoid sinuses are vulnerable to distorted function as they exit through the jugular foramina carrying 95 percent of the drainage from the head. Circulatory retardation may result in ischaemia" (Magoun 1975; p250).

The irritation of the vagus nerve as it passes through the jugular foramen or of the hypoglossal nerve as it passes through the hypoglossal canal[13] may affect the infant's ability to suck or swallow. Infants have been treated to resolve cranial strains in the United States of America since the 1940s where the clinical effectiveness of osteopathic treatment has been observed.[18]

Considerable interest in the fat content of milk has been seen in recent years. It has been postulated[19] that milk secretion is modulated by local chemical feedback inhibition, and that there is some mechanism operating, within the breast tissue, that is additional to hormonal control (via prolactin, growth hormone and oxytocin). The inhibitor is thought to be present in the milk itself and to have a regulatory effect.

In one study it was shown that if only one of the glands of goats were milked more than twice daily (three times daily or up to hourly), an increase in milk secretion occurred only in the gland that had been milked more frequently.[20]

It is accepted that fat content of human milk is greater in the hind milk than in the fore milk.[21,22,23] An infant that is feeding effectively will take both fore milk and hind milk at each feed. One way of measuring the extent to which the breast is emptied is by measuring the fat content before and after each feed. The assumption that an infant with a dysfunctional suck, will only effect a small variation in the pre- and post-feed breastmilk fat estimations was tested in this pilot study. The greater the difference between pre-post fat concentrations, the more the infant would demonstrate the ability to effectively empty the breast of milk and attain the fat-rich hind milk.

The aims of this pilot study were to establish the feasibility of utilising the estimation of the concentration of breastmilk fat as a standard for measuring performance outcomes in infants with a dysfunctional suck. The pre-post feed fat concentrations in the breastmilk of six mothers whose infants were feeding normally was measured. The results are described and analysed to assess whether a standard had been established against which the progress of infants with sucking problems could be compared.

Further, six infants who were feeding normally were assessed to establish the difference between pre- post feed fat concentrations. A pilot study of six treated babies (each of which was treated for 4 weeks) then assessed whether, when first measured, the difference between pre- post feed fat concentrations was less than normal babies. It was then considered whether osteopathic treatment of these infants improved their pre- post feed fat concentrations to a level that compared favorably with normal infants.

The literature

The current interest in autocrine control of lactation, and the interest in the fat component of milk, has highlighted the possibility of fat concentration as a marker for effective feeding. It has been noted that the fat concentration in breastmilk varies over 24 hours.[24,25] When milk that was stored in a goat's gland was not milked, but diluted with an inert solution, the secretory rate increased.[26,27,28] This suggests that it is not the mere emptying of the gland, but the concentration/dilution of substances within it, that determine milk production. This view was strengthened by the observation that the goat's gland that had been milked three or more times daily had a greater number of secretory cells than the gland that had only been milked twice daily.[30,31,32] More recently it has been shown that it is the extent to which the breast is emptied, rather than the frequency of feeding, that is important in the short-term control of the human milk supply.[33] Milk synthesis in the alveoli of the mammary glands is a com-

* Strain means sufficient force to cause deformation of tissue.

plex process involving at least four secretory mechanisms. These include exocytosis, fat synthesis and transfer, secretion of ions and water and immunoglobulin transfer from the extracellular space.[21] The average lipid content of human milk ranges from 3.2 - 3.5g/100ml[34] whereas colostrum, which is the initial fluid released during the first few days of lactation, contains about 2.9g fat/100ml.[35]

The fat in human milk occurs as microscopic globules 2-3 μm in diameter and coated with a membrane derived from the mammary epithelial cell that secreted it. Fat, which constitutes 50 percent of the total energy value of human milk, plays an essential role in infant nutrition since it is vital to normal brain development, the structure and function of cell membranes and for prostaglandin synthesis.[36]

Woodward has noted that a far more dramatic increase in fat content occurs if an infant feeds from only one breast at any one feed. The concentration in the hind milk is typically at least twice that in the fore milk.[37] As the volume of milk in the breast decreases, higher levels of fat, sodium and immunoglobulins, and lower levels of lactose are found.[38] The endocrine control of lactation involves control prolactin and growth hormone[39] which governs milk production and oxytocin which governs milk ejection. So for example, growth hormone has been shown to increase the breastmilk in the mothers of pre-term infants.[40] Insofar as milk ejection, it is now thought that the event described as "the milk coming in" which occurs 24-48 hours after birth marks the shift from lactation driven by endocrine to lactation under autocrine control.[41,42]

Serum prolactin concentration levels increase substantially during pregnancy from the nonpregnant level of about 10 nanograms/ml (1μm = 1000 nanograms). However its concentration decreases sharply after pregnancy and at 4 weeks postpartum it is about 20-30 nanograms/ml.[43] This reduction in the prolactin production occurs at a time when milk production has been established and there is a need for it to continue for many months. It suggests that another mechanism for regulation is in the ascendant and is associated with longitudinal changes in human milk yield and composition.[23]

A study of mothers who were breast-feeding infants older than 1 month,[44] found that whilst the concentration of prolactin in the mothers related to the degree of fullness in the breast, there was **no** relationship between the concentration of prolactin in the plasma and the rate of synthesis. So that whilst prolactin is important in the initial phase of lactation, autocrine regulation becomes more important thereafter.

Apart from the question of autocrine control it is important to refer briefly to a separate issue. This concerns the level of maternal pain associated with breast-feeding an infant with a sucking dysfunction. It is recognised that in the early stages of breast-feeding, even normal infants may cause their mothers to experience nipple pain.[45] It is important, where such pain persists, to exclude the possibility of Candida albicans as a cause,[46] rather than assume that the pain is caused by the infant's sucking. However, having excluded this possibility, it is certainly recognised that pain can result from an infant's dysfunctional suck (which invariably leads to exquisitely tender, cracked and/or bleeding nipples of the breast-feeding mother) and that this alters the milk composition or secretion.[47] This maternal nipple pain resulting from an infant's dysfunctional sucking can be severe.[48] The maternal record of pain on a visual analog scale can be a useful indicator of improvement in the infant's sucking.

Method

A simple technique for estimating fat concentration has been described[49,50] and has been the basis for estimations in this study. The estimation was made by the centrifugation of milk in a Sarvall Capspin haematocrit centrifuge for a period of 15 minutes.

The samples (of approximately 0.1ml) were centrifuged at 12,000rpm using micro-haematocrit tubes that were 75mm in length and an inner diameter of 1.1 - 1.2mm. The tubes were sealed with Seal-ease (Clay Adams) and after centrifugation, the length of the cream column for each tube was measured. This gave a so called creamatocrit reading.

In view of Woodward D.R. Boon J. and Rees B.'s study[36] the mothers of infants being measured were advised to feed from one breast only. Creamatocrit readings were made from breastmilk taken from the same breast. The samples were taken by expressing a few millilitres of breastmilk before and after each feed. All fat estimations were made between 5:00 pm - 7:00 pm as this appears to be the time of day when the fat concentration in breastmilk is high. This ensured the widest possible difference between pre- and post-feed fat concentration.

The inclusion criteria for this study are set out below.

1. Be of an appropriate weight for gestational age; and
2. Had an Apgar score of 7 or more after 5 minutes after birth; and
3. Be less than 6 months of age; and
4. Be attempting to breast-feed and are reported as having, a weak or dysfunctional suck; and
5. Be seen by a lactation consultant for at least two weeks prior to the inclusion in the study; and
6. Be seen 21 days or more postnatally; and
7. Consented through their parents prior to the palpatory assessments, sucking measurements and/ or breastmilk fat estimations.

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The exclusion criteria for the study were: 1. Infants less than 21 days old; (Note: the sucking measurements and breastmilk fat estimations form the base-line for infants with dysfunction to be measured in a later study. If infant under 21 days were not excluded, improvements in this latter group might otherwise be attributable to maturation only. An extension of this study, if carried out subsequently, will have to ensure that treated and control groups are matched for age and sex.) 2. Infants with a diagnosed dysphagia of which the sucking disorder forms a part.

All six of the treated infants had been seen by a lactation consultant for at least two weeks before osteopathic treatment commenced and some had been seen since birth. In all cases the mothers had received advice about posture, the method of attachment and the positioning of the infant. Where it was considered appropriate by the lactation consultant, the mother was taught manual oral/lingual exercises to normalise the infant's tongue movements. Each mother and infant had been supervised by a lactation consultant during at least one feed and most had been accompanied in this way several times. Infants were only referred for inclusion in the study when the lactation consultant was satisfied that no further advice could be offered regarding posture or technique. The greatest difference between pre- and post feed was seen in treated baby

continued on page 30

Results

Six samples of normal infants were taken in a pilot study. The results are set out in **Table 1** below.

	DOB.	Test date	Fat Estimation Feed @	Pre-feed	Post-feed
Baby A.P	6.10.96	18.1.97	5.30 - 6.00 p.m.	4mm	14mm
Baby R.E.	21.10.96	19.1.97	6.00 - 6.20 p.m.	3mm	10mm
Baby A.D.	26.6.96	21.1.97	5.45 - 5.55 p.m.	4mm	12mm
Baby L.M.	10.12.96	24.1.97	6.50 - 7.00 p.m.	2mm	11mm
Baby N.S.	12.9.96	28.1.97	6.00 - 6.15 p.m.	2mm	8mm
Baby J.F.	21.12.96	29.1.97	6.50 - 7.10 p.m.	2mm	9mm

Table 1. Pre- and postfeed breastmilk fat estimations of 6 normal infants.

In the normal group, the average fat estimation at the onset of a feed was 2.83 mm, and at completion was 10.67 mm. The variance associated with results for normal babies was:-

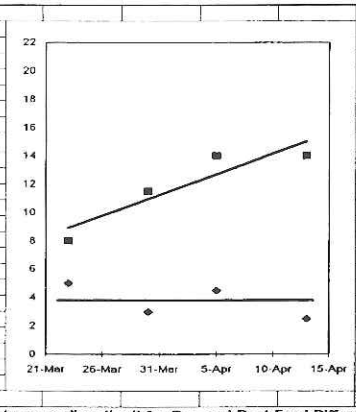
Pre-feed average	2.83	Standard deviation	0.98
Post-feed average	10.67	Standard deviation	2.16

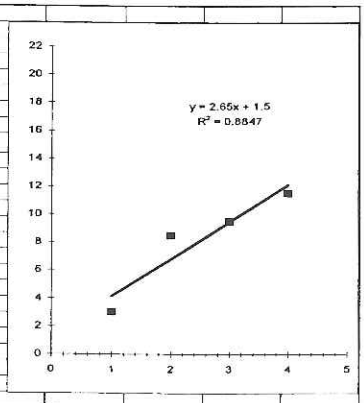
The average difference between pre-post feed fat estimation was 7.84 mm.

A small number of babies with a dysfunctional suck were treated and the results of each are included in **Tables 2-7.**

BABY #1

		Pre-feed	Post-feed	Post-f trend (linear)	Difference	Diff trend (linear)
1	23-Mar	5	8	8.9	3.0	4.2
2	30-Mar	3	11.5	10.9	8.5	6.9
3	5-Apr	4.5	14	12.7	9.5	9.2
4	13-Apr	2.5	14	15.0	11.5	12.2
	Mean	3.75				
	Std Dev	1.19			r^2 =	0.8847
					r =	0.9406





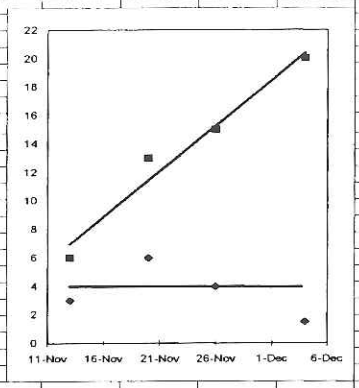
Linear regression analysis ("least squares" method) for Pre- and Post-Feed Differences

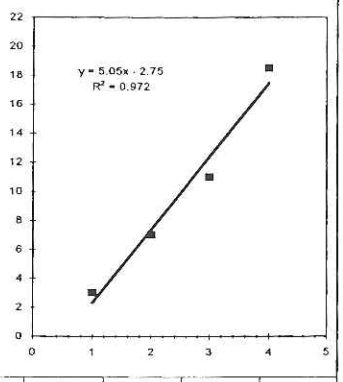
#	Differences			Equation for the line: y = mx + b		
1	3.0	m -slope	2.65	b - y intercept	1.5	
2	8.5	standard error value for m	0.676387	standard error value for b	1.852363	
3	9.5	r^2 - coefficient of determination	0.884724	The standard error for the y estimate	1.512448	
4	11.5	F - F statistic (F-observed value)	15.34973	df - degrees of freedom	2	
		ssreg - regression sum of squares	35.1125	ssresid - residual sum of squares	4.575	

Table 2. Analysis of treated baby #1

BABY #2

		Pre-feed	Post-feed	Post-f trend (linear)	Difference	Diff trend (linear)
1	13-Nov	3	6	7.0	3.0	2.3
2	20-Nov	6	13	11.4	7.0	7.5
3	26-Nov	4	15	15.3	11.0	11.9
4	4-Dec	1.5	20	20.4	18.5	17.8
	Mean	3.63			r^2 =	0.9720
	Std Dev	1.89			r =	0.9859





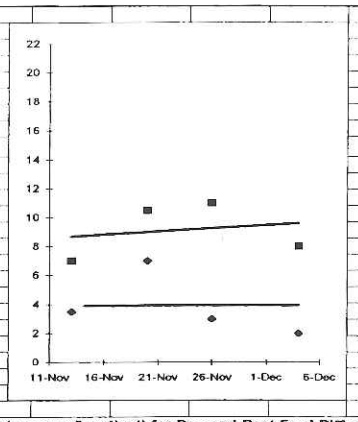
Linear regression analysis ("least squares" method) for Pre- and Post-Feed Differences

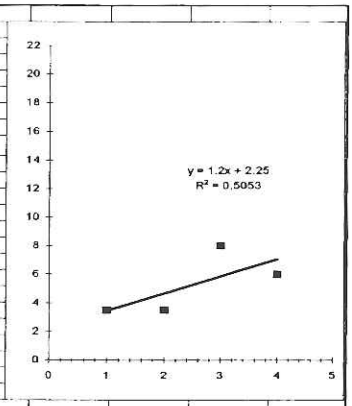
#	Differences			Equation for the line:	y = mx + b		
1	3	m -slope	5.05	b - y intercept	-2.75		
2	7	standard error value for m	0.606218	standard error value for b	1.660196		
3	11	r^2 - coefficient of determination	0.971987	The standard error for the y estimate	1.355544		
4	18.5	F - F statistic (F-observed value)	69.39456	df - degrees of freedom	2		
		ssreg - regression sum of squares	127.5125	ssresid - residual sum of squares	3.675		

Table 3. Analysis of treated baby #2

BABY #3

		Pre-feed	Post-feed	Post-f trend (linear)	Difference	Diff trend (linear)
1	13-Nov	3.5	7	8.7	3.5	3.6
2	20-Nov	7	10.5	9.0	3.5	4.7
3	26-Nov	3	11	9.2	8.0	5.7
4	4-Dec	2	8	9.6	6.0	7.0
	Mean	3.88			r^2 =	0.5053
	Std Dev	2.17			r =	0.7108





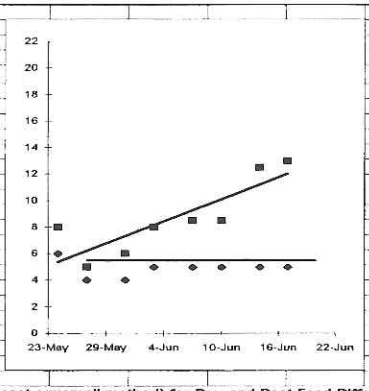
Linear regression analysis ("least squares" method) for Pre- and Post-Feed Differences

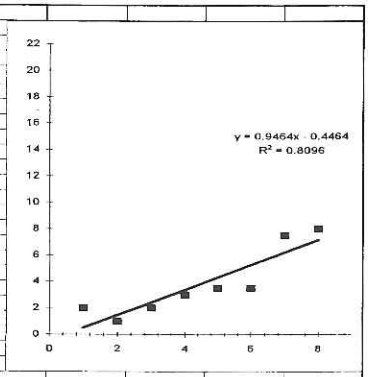
#	Differences			Equation for the line:	y = mx + b		
1	3.5	m -slope	1.2	b - y intercept	2.25		
2	3.5	standard error value for m	0.839643	standard error value for b	2.299456		
3	8	r^2 - coefficient of determination	0.505263	The standard error for the y estimate	1.877498		
4	6	F - F statistic (F-observed value)	2.042553	df - degrees of freedom	2		
		ssreg - regression sum of squares	7.2	ssresid - residual sum of squares	7.05		

Table 4. Analysis of treated baby #3

BABY #4

		Pre-feed	Post-feed	Post-f trend (linear)	Difference	Diff trend (linear)
1	24-May	6	8	5.4	2.0	0.5
2	27-May	4	5	6.2	1.0	1.3
3	31-May	4	6	7.3	2.0	2.4
4	3-Jun	5	8	8.1	3.0	3.3
5	7-Jun	5	8.5	9.2	3.5	4.4
6	10-Jun	5	8.5	10.1	3.5	5.2
7	14-Jun	5	12.5	11.2	7.5	6.3
8	17-Jun	5	13	12.0	8.0	7.1
	Mean	4.80			r^2 =	0.8096
	Std Dev	0.64			r =	0.8998

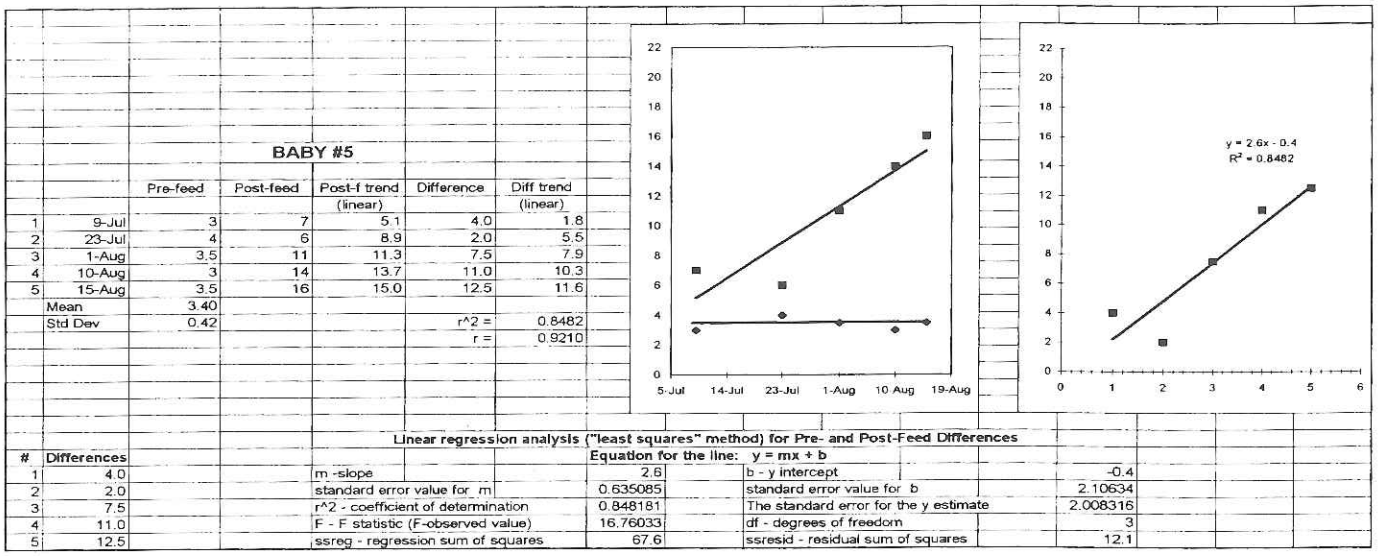




Linear regression analysis ("least squares" method) for Pre- and Post-Feed Differences

#	Differences			Equation for the line:	y = mx + b		
1	2.0	m -slope	0.946429	b - y intercept	-0.446429		
2	1.0	standard error value for m	0.187382	standard error value for b	0.946232		
3	2.0	r^2 - coefficient of determination	0.809588	The standard error for the y estimate	1.214373		
4	3.0	F - F statistic (F-observed value)	25.5106	df - degrees of freedom	6		
5	3.5	ssreg - regression sum of squares	37.62054	ssresid - residual sum of squares	8.848214		
6	3.5						
7	7.5						
8	8.0						

Table 5. Analysis of treated baby #4 →



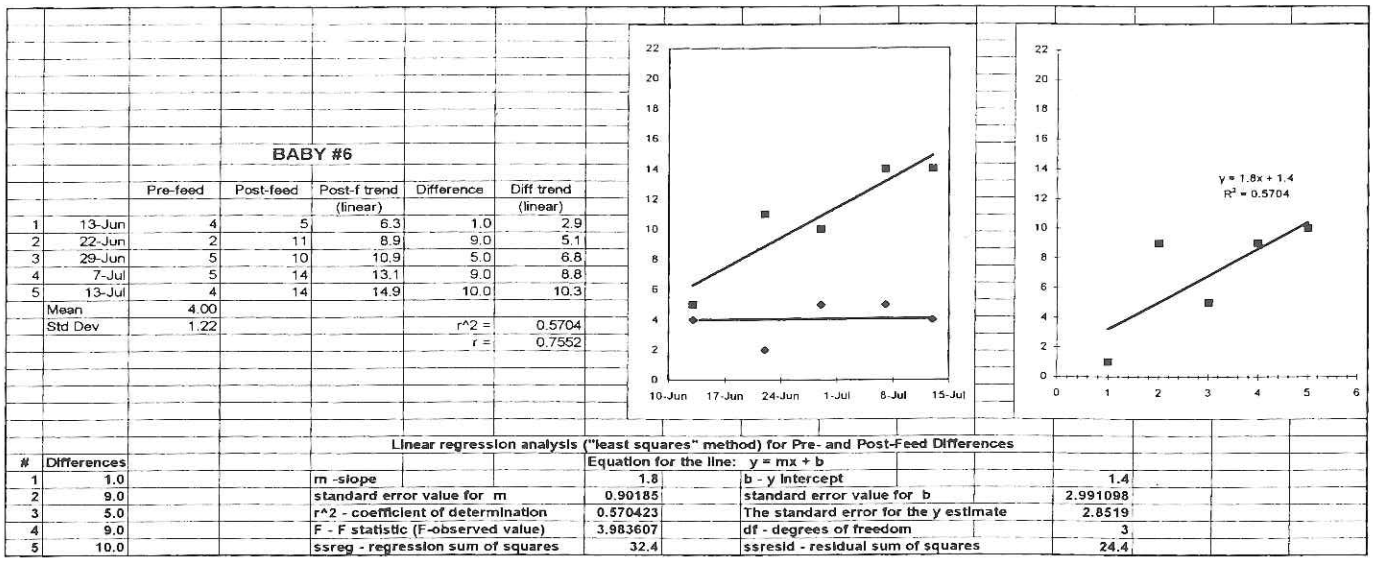
BABY #5

		Pre-feed	Post-feed	Post-f trend (linear)	Difference	Diff trend (linear)
1	9-Jul	3	7	5.1	4.0	1.8
2	23-Jul	4	6	8.9	2.0	5.5
3	1-Aug	3.5	11	11.3	7.5	7.9
4	10-Aug	3	14	13.7	11.0	10.3
5	15-Aug	3.5	16	15.0	12.5	11.6
	Mean	3.40			r^2 =	0.8482
	Std Dev	0.42			r =	0.9210

Linear regression analysis ("least squares" method) for Pre- and Post-Feed Differences

#	Differences				
1	4.0	m -slope	2.6	b - y intercept	-0.4
2	2.0	standard error value for m	0.635085	standard error value for b	2.10634
3	7.5	r^2 - coefficient of determination	0.848181	The standard error for the y estimate	2.008316
4	11.0	F - F statistic (F-observed value)	16.76033	df - degrees of freedom	3
5	12.5	ssreg - regression sum of squares	67.6	ssresid - residual sum of squares	12.1

Table 6. Analysis of treated baby #5



BABY #6

		Pre-feed	Post-feed	Post-f trend (linear)	Difference	Diff trend (linear)
1	13-Jun	4	5	6.3	1.0	2.9
2	22-Jun	2	11	8.9	9.0	5.1
3	29-Jun	5	10	10.9	5.0	6.8
4	7-Jul	5	14	13.1	9.0	8.8
5	13-Jul	4	14	14.9	10.0	10.3
	Mean	4.00			r^2 =	0.5704
	Std Dev	1.22			r =	0.7552

Linear regression analysis ("least squares" method) for Pre- and Post-Feed Differences

#	Differences				
1	1.0	m -slope	1.8	b - y intercept	1.4
2	9.0	standard error value for m	0.90185	standard error value for b	2.991098
3	5.0	r^2 - coefficient of determination	0.570423	The standard error for the y estimate	2.8519
4	9.0	F - F statistic (F-observed value)	3.983607	df - degrees of freedom	3
5	10.0	ssreg - regression sum of squares	32.4	ssresid - residual sum of squares	24.4

Table 7. Analysis of treated baby #6

#2. A detailed consideration of the clinical presentation of these babies and the rationale for treatment is beyond the scope of this article, which presents the outcomes in order to assess whether a more extensive study (which would include the above considerations as a part of any ensuing report) would be worthwhile. An analysis of the outcome for the pilot study appears in the next section. The average difference between pre- and post-feed fat estimation at the beginning of the month of treatment was 2.58mm. The average difference between pre- and post-feed fat estimation at the end of the month of treatment was 11 .08mm. This compares favorably with the 7.84mm average for the normal infants.

Analysis and Discussion

The pre- and post-feed creamatocrit reading provides a simple, non-invasive means of assessing the effectiveness of the infant's sucking ability; normal infants will feed for 10 to 30 minutes and empty the breast sufficiently to obtain the hind milk (which has a higher fat concentration). An infant with a dysfunctional suck (which is defined as a sucking pattern which is characterised by an excessive amount of up and down movements, marked variations in sucking rhythm, lack of characteristic rhythm or any pattern which more subtly affects the speed, skill or amount of milk taken at a feed) will either feed for a much shorter or longer time than infants who are feeding normally.

In the former case, the feed is discontinued for a variety of reasons which include maternal pain and tiredness/ pain of the infant. In the latter case, the infant may go on sucking intermittently and ineffectually for a long time. In both cases the infant's ineffectiveness will fail to obtain the hind milk because the breast has not been sufficiently drained. The data displayed in **Tables 2 - 7** give the results of fat estimation for the 6 babies with a dysfunctional suck. A linear regression analysis has been made using the 'least squares' method. The regression line provides the best fit for the data. In each case the slope of the line measures the differences in the pre- and post-feed breastmilk fat estimation per unit of time during which the baby was treated. The standard error term was low, varying between 0.95 and 2.99. In linear regression, an r value (the coefficient of determination) of 1 would indicate that there was no difference between the estimated difference between pre- and post-feed breastmilk fat estimation and the actual differences. At the other extreme, if the coefficient of determination is 0, the linear regression equation is of no value in predicting the differences. Each of the treated babies has an r value which exceeds 0.7. The r value for 4 of the treated babies was 0.85 or higher which would be regarded as a very good outcome. In each case the coefficient of determination is greater than 0.50 which shows a strong relationship between the duration of the treatment and increase in breastmilk fat estimation. In 4 of the babies this is greater than 0.85 which demonstrates a very strong correlation.

The results of the linear regression equation for the pre-post-fat differences is shown in **Table 8** below.

Baby #	Initial	Final	Standard error of estimator
1	4.2	12.2	1.5
2	2.3	17.8	1.4
3	3.6	7.0	1.9
4	0.5	7.1	1.0
5	1.8	11.6	2.1
6	2.9	10.3	3.0

Table 8. Linear regression equation for the pre-post fat concentrations

The crucial test of the effectiveness of the osteopathic treatments is whether the slope of the regression line, fitted to the differences in the pre- post-feed fat estimates, increases significantly. t-statistics can be used to determine the probability of the slope of the regression line being obtained by chance. **Table 8** presents the t-critical and t-observed values for each line.

Baby #	t-observed*	t-critical+ for Alpha=0.05	Alpha=0.10	Alpha=0.25
1	3.92	2.92	1.89	0.82
2	8.33	2.92	1.89	0.82
3	1.43	2.92	1.89	0.82
4	5.05	1.94	1.44	0.72
5	4.09	2.35	1.64	0.77
6	2.00	2.35	1.64	0.77

* t-observed is calculated by dividing the regression line slope by the standard error for the slope

\+ t-critical values (obtained from standard statistical tables) are presented for both Alpha = 0.05, 0.10, and 0.25 i.e. for the probabilities of 0.05, 0.10, and 0.25 that the slope was obtained by chance.

Table 9 The t-critical and t-observed values for six treated infants.

When t-observed values are greater than the t-critical values, then the probability of the slope being obtained by chance is less than the corresponding Alpha value. Therefore, there is less than a 5 percent probability that the slope of the regression line was obtained by chance for treated babies # 1, 2, 4 and 5, less than 10 percent probability for treated baby #6, and less than 25 percent probability for treated baby #3.

Treated baby #6 was clinically a very difficult case and particularly successful. However, the result is skewed by virtue of the second pre-feed reading being uncharacteristically low and the post-feed measurement being uncharacteristically high. It suggests that if a further study is done, that twice weekly measurements should be taken so that variations like this do not have such an impact upon the statistical outcome. The mother of treated baby #2 kept a daily record of her pain (on a scale of 0 to 10 where 0 = no pain and 10 = severe pain), without being asked to do so. It was noted that when she started to record a reduction in pain, the fat concentration in the milk increased. This tends to bear out the Barowicz' contention[46] that maternal pain can alter the milk composition. Any extension to this study should therefore include the keeping of a daily visual analog scale of maternal breast pain. The fact that the average pre-post fat difference for the dysfunctional group is higher than for the normal group may be explained by the longer feed time and increased focus on the whole process of lactation of mothers whose babies initially had breast-feeding problems. Further, normal babies need to be measured with an instruction to mothers to persist in feeding from one breast for as long as possible.

This pilot study demonstrates that as the infant's sucking becomes more effective following osteopathic treatment, a greater variance in the pre- and post-feed creamatocrit reading is seen. →

Conclusion

It appears that the milk fat estimation provides a simple, non-invasive and reliable standard measurement of the ability of an infant to feed effectively. The results with the small sample of treated babies are encouraging. The difference between pre- post-feed fat estimations of breastmilk were small in infants with a dysfunctional suck. In all but one of six cases, there was a substantial increase in the pre- post-feed fat estimation by the end of the month of osteopathic treatment. All of these infants had been reviewed by a lactation consultant and all were 4 weeks or older prior to the commencement of osteopathic treatment. All 6 of the infants continued to breast-feed for 5 months or more following treatment. At the time the infants commenced treatment each of the mothers was at the point of discontinuing feeding. It is believed that it would be worthwhile extending the study to a cohort of 40 infants. A control group would be required to ensure validity.

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S.T.A.R.: A more viable alternative descriptor system of somatic dysfunction

by Dennis J. Dowling, DO, CSPOMM, New York

Edtior's Note: *Dr. Dennis Dowling, is currently an Associate Professor and Chairman of The Stanley Schiowitz, DO, FAAO's Dept of OMM at NYCOM/NYIT. He is also the Director of Manipulation in the Dept. of Physical Medicine and Rehabilitation at the Nassau County Medical Center and Chairman of the OMM Committee of the Massapequa General Hospital.*

There are several references/definitions which appear in the *Glossary of Osteopathic Terminology* (1997) related to somatic dysfunction, such as:

somatic dysfunction: impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial, and myofascial structures, and related vascular, lymphatic, and neural elements. Somatic dysfunction is treatable using osteopathic manipulative treatment. The positional and motion aspects of somatic dysfunction are best described using at least one of three parameters: 1. the position of a body part as determined by palpation and referenced to its adjacent defined structure, 2. the directions in which motion is freer, and 3. the directions in which motion is restricted. see also osteopathic lesion (osteopathic lesion complex).

somatic dysfunction, acute: immediate or short-term impairment or altered function of related components of the somatic (body framework) system; characterized in early stages by vasodilation, edema, tenderness, pain, and contraction; identified by T.A.R.T. (qv); palpatorily diagnosed by assessment of tenderness, asymmetry of motion and relative position, restriction of motion and tissue texture change (T.A.R.T.). see T.A.R.T.

somatic dysfunction, chronic: impairment or altered function of related components of the somatic body framework) system, characterized by tenderness, itching, fibrosis, paresthesias, contracture; identified by T.A.R.T. (qv). see T.A.R.T.

somatic dysfunction, secondary: somatic dysfunction (qv) arising subsequent to or as a consequence of other etiologies.

somatic dysfunction, type I: a group of thoracic and/or lumbar vertebrae in which the freedoms of motion are in neural with sidebending and rotation in opposite directions (rotation occurs toward the convexity of the curve).

somatic dysfunction, type II: thoracic or lumbar somatic dysfunction of a single vertebral unit in which the vertebra is flexed or extended with sidebending and rotation in the same direction (rotation occurs into the concavity of the curve).

The *Glossary of Osteopathic Terminology* also lists the definition of T.A.R.T. as:

T.A.R.T.: a mnemonic for the four diagnostic criteria of somatic dysfunction- tissue texture abnormality, asymmetry, restriction of motion and tenderness-any one of which must be present for the diagnosis.

There are also references to the original mnemonic, "A.R.T." which in the glossary is reduced to "see T.A.R.T."

The difficulty with this basic mnemonic and its derivations is that there are three objective components (A.R.T.) which should be reproducible and one subjective finding which is limited in its scope in the currently used T.A.R.T. system. Recognition of the subjective factor allows for the patient to identify a pain reaction, tenderness, in response to palpatory examination by the osteopathic physician. The patients' complaints should be a part of the determination of somatic dysfunction but limiting their contribution to just tenderness, or even pain, limits the actual determination of somatic dysfunction. Tenderness is also open to interpretation by the patient. Subjective appreciation of pain varies widely depending on the individual's tolerance for pain, their expectation, and the depth of palpation. There are many other

sensory and motor changes which can be associated with the occurrence of a somatic dysfunction as even expressed in the various definitions listed ("tenderness, itching, fibrosis, paresthesias, contracture.") Anesthesias (numbness), temperature changes (hot/cold, circulatory changes (flushing), allodynia, pain reference patterns (triggerpoints), weakness, tightness, etc. all represent sensory changes on the part of the individual and may be additional signposts to the presence of somatic dysfunction.

Instead of the mnemonic T.A.R.T. the mnemonic "S.T.A.R." is suggested. This indicates:

S. Sensory changes (increase in tenderness, decrease in sensitivity, anesthetic quality, paresthesias, etc.) which result following palpation
T. Tissue tension changes (spasm, boggy muscle, ropy muscle, atrophy, edema, oiliness, dryness, hyperhidrosis, pigment changes, etc.)
A. Asymmetry (positional findings where one side does not compare to the other)
R. Restriction of Motion (A form of asymmetry where motion testing results in findings of relative limitation of motion in at least once direction and the components of other directions can be concluded or implied).

This allows for several clinical considerations as well as simplifications of the mnemonic. There would no longer be the confusion of two "Ts." As concerns the diagnosis of somatic dysfunction, there is also a sequential aspect to the "S.T.A.R." mnemonic. Sensory changes indicate the presence of somatic dysfunction but may not be definitive in the localization or even the side. The presence of these subjective complaints are usually what instigates the patient to present for treatment. Often, pain in a region indicates that there is a problem without completely localizing the etiology. Sometimes muscle pain does not occur in the contracted muscles themselves, The contralateral or antagonist muscles may actually become irritated due to the persistent stretching which occurs opposite to the contracted muscle. As an example, the classic patient with back pain found in a fetal position may actually have quite significant counterstrain tenderpoints located in the abdominal flexors (Jones, 1995). By beginning and directing attention to the sensory components in response to palpation, the osteopathic physician is validating and coordinating the patient's complaints.

Tissue tension may be present in either Type I or Type II dysfunctions. One of the theoretical and clinical considerations of either of these according to osteopathic practice, especially the muscle energy concept (Mitchell, 1995), is that the muscles are involved in either bringing about or maintaining Type II somatic dysfunction. The muscles which are involved are probably the short muscles which can be classified as "shunt & spurt" muscles. They typically involve the small muscles creating small amounts of motion)such as the intertranversii, short rotators, etc.). Because of their size, location, and more superficial structures, palpation alone may not determine the exact muscle. Due to the segmental neurological nature of the relationship of the particular involved muscle and those related other soft tissue (skin, other muscle, fascia) in the immediate region and their relative reactivity, the effect may be magnified. With Type II dysfunction, larger regional muscles are probably involved. These, along with their reflective other soft tissue changes, size and relative superficiality, are generally more susceptible to appreciation even by a beginning student of osteopathic manipulation, with some guidance. The findings of ropiness indicate a more chronic nature while bogginess tends to imply a more acute history. Experienced examiners should be limited to the presence of either ropy or boggy muscles. Sudomotor changes including the presence of pallor, erythema, perspiration, oil, hair distribution, and changes in pigmentation also represent deviations from soft tissue normalcy (DiGiovanna, 1997). A third likelihood, more often associated with Type II dysfunction, is the presence of a viscerosomatic reflex as the etiology.

Asymmetry indicates the possible presence of a somatic dysfunction when it indicates altered positioning of bilateral structures from the non-dysfunctional symmetrical appearance. Its presence allows a greater localization with indication of a possible involved side (DiGiovanna). However, bony and other anomalies may mislead the examiner Each finding leads to further investigation and may result in determination of benign or malignant pathology as well as the presence of single or multilevel dysfunction. Appreciation of asymmetry includes palpatory as well as visual and auditory alterations. Visual clues of asymmetry include higher or lower, anterior or inferior, or rotational alterations of various landmarks. Scanning the individual further may reveal more subtle findings such as trophic changes of the skin and other structures. Altered components such as occurs secondary to injury or disease does not necessarily equate with somatic dysfunctions but there is a frequent relationship. Fractures, arthritis, surgical fixation or removal change the function of the region and therefore predispose to somatic dysfunction. Auditory alterations can also be appreciated by percussing the region and evaluating the vibration as well as the produced sound.

The *sine qua non* criterion of somatic dysfunction is "restriction of motion." It allows for the final determination of the restrictions and relative freedoms of either the

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joint, segment, or region. Gross motion changes across a large joint, such as the shoulder, may indicate spasm of large muscles such as the latissimus dorsi or pectoralis major. Rotos-coliosis and intersegmental motion testing of the individual vertebral segment determine the barriers and Type II somatic dysfunction determinants in three planes of motion. Utilizing Fryette's principles, the examiner can introduce a testing motion and then imply the other motion freedoms. This sequence, S.T.A.R., represents a more natural thought sequence in the final determination of somatic dysfunction. The patient presents with a chief complaint which contains a sensory component the majority of the time. There may also be a complaint of decrease in function, but this is usually closely associated with complaints of pain or altered sensation. Next, the combination of tissue tension changes and asymmetry are evaluated over the involved and perhaps contiguous regions to the chief complaint. Finally, the examiner fine tunes the definition of the diagnosis by performing tests of the quality, symmetry and quantity of motion. The mnemonic may also be utilized to record components in a shorthand as follows:

Through a combination of these slashes, the examiner may record the components of the findings indicating somatic dysfunction as illustrated in the chart (Table #2) on the previous page. Such notation would be required to appear in the chart with an explanation of the meaning of individual and combination of markings. Recording of these would allow the physician to quickly make notes as to the findings and allow for comparison on follow-up visits. Findings which result in the formation of an eight-point star indicate the presence of some form of all four elements of somatic dysfunction. A dysfunction may be present but one or more of the elements may be missing. A somatic dysfunction which reappeared with only a restriction component may represent significant improvement if the other three elements were not found. Soft tissue changes accompanied by asymmetry may represent an underlying etiology or an improvement. Asymmetry, especially when occurring singularly, may represent a congenital anomaly or a structure change which has been induced by chronic regional dysfunction. Persistent soft tis-

STAR Article Table #1

S	
T	
A	
R	

STAR Article Table #2

otion	
	○
	S
	T
	A
	R

S	|	sensory changes only
T	–	tissue texture changes only
A	/	asymmetry only
R	\	restriction of motion
ST	:	sensory & tissue texture changes
SR	| \	sensory changes & restriction of motion
TA	– /	tissue texture changes & asymmetry
AR	/ \	asymmetry & restriction of motion
SA	| /	sensory changes and asymmetry
TR	– \	tissue texture changes and restriction of motion
STR	| – \	sensory, tissue texture changes & restriction of motion
STA	| – /	sensory, tissue texture changes & asymmetry
SAR	| / \	sensory changes, asymmetry, & restriction of motion
TAR	– / \	tissue texture changes, asymmetry & restriction of motion
STAR	| – / \	sensory, tissue texture, asymmetry, and restriction of motio n
NONE	○	

sue changes may reflect underlying pathology. The presence of sensory alterations without the occurrence of the other three may raise suspicion. Complaints of tenderness or symptom radiation, in the absence of tissue changes, asymmetry, and restriction may indicate symptom magnification, somatization, malingering or maintaining secondary gain. All of these raise the concern that the patient is manipulating the therapeutic relationship. However, the presence of symptoms in the absence of other evidence may actually indicate the need to perform further investigation. Complaints which are out of proportion to the findings may be indicative of a disease process which may be of an infectious, neoplastic, or hormonal nature.

A further consideration would utilize the recording of the components of somatic dysfunction beyond the relative ranges of relative freedoms for research. When a third thoracic vertebra is found to sidebend and rotate to the right to a greater extent than the left, and to be freer in flexion than extension we write either $T3FS_RR_R$ or $T3FRS_R$. Further notation which includes a ♠ would demonstrate that all four "STAR" components were present. A description of the examiner's opinion as to the temporal status, acute versus chronic, would round out the picture.

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Solar Plexus Injuries; An old injury with a new twist

by John M. Jones, III, DO, CSPOMM and Todd May, DO, LT MC USNR

Editor's Note: *John M. Jones, III, DO is the Assistant Dean of Osteopathc Development and Research and the Chair of the OMM Department at Western University/College of Osteopathic Medicine of the Pacific. Lt. Todd May, DO is currently stationed in Okinawa in the medical corps. He graduated from the College of Osteopathic Medicine of hte Pacific in 1994.*

Abstract

Diaphragmatic spasms due to a blow to the upper abdomen while not on guard have been troublesome to athletes because the athlete is momentarily unable to catch his breath and feels as if he is in danger of death.

An old technique used by osteopathic physicians for improving diaphragmatic efficiency and function has been employed by the authors to provide relief from the muscle spasm once the breathing resumed.

All responses were favorable and the technique warrants further trial and investigation.

A review of current sports medicine textbooks and a medline search reveals presently no new information, other than a variety of positioning techniques, coupled with reassurance and breathing observation are now commonly employed.

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Diaphragmatic spasm, or "having the wind knocked out" has annoyed athletes, perhaps because the athlete is momentarily unable to catch his breath and feels as if he is in danger of death.[1] Presently, no treatment other than time and reassurance is generally used. Consequently, athletes, their trainers and team physicians have limited resources in dealing with the injury. Present management consists of quietly reassuring the athlete that all will be well, and encouraging the athlete to relax, loosen the belt and clothing, bend the knees, take short inspirations and long expirations.[2]

An old technique used by osteopathic physicians for improving diaphragmatic efficiency and function can provide direct relief for this problem. Although the authors first applied the technique on football players, the treatment can be used with athletes who suffer this injury in many sports.

According to Roy and Irvin[3], a blow to the solar plexus causes a spasm of the diaphragm which reflexively results in a temporary paralysis of breathing.[3] While the exact mechanism for the paralysis, spasm and treatment are presently unknown, the underlying cause of the paralysis could be at the receptor level. Current theories of receptors and their interaction provide a working mechanism for the problem and solution. The diaphragm has both golgi tendon organs and muscle spindles, with a preponderance of golgi organs, as well as free nerve endings.[4] Transient paralysis can occur when a blow forces the diaphragm upward during the inspiratory phase of respiration, or it may be due to a forceful expiration of the residual volume of air normally present in the lung.[5] Both would result in a rapid overstretching of the diaphragm's muscle spindles. The length of the muscle spindles *can* be controlled, depending upon the degree of anticipated stretch.[6] This abrupt overstretching of the spindles may result in a reflex contracture of the extrafusal fibers, and the static *stretch* reflex causes a continuation of the contraction as long as the muscle is maintained at an excessive length,[7] causing a momentary cessation of breathing. With this reflex contraction, too much tension is generated by the diaphragm, and the golgi organs are stimulated and override the extrafusal contraction.[8] Another contributing factor may be the increase in $pC0_2$. Because of the spasm, the diaphragm decreases its range of motion, perhaps due to the spindles being reset ant functioning at a shorter length.[9] This strained muscle would not perform the act of breathing as efficiently as it did before the injury.

Dr. Richard Van Buskirk has proposed a nociceptive theory of musculoskeletal dysfunction, focusing on free nerve ending reporting of noxious stimuli.[10] Nociceptors could account for both the pain perception and maintained motion restriction. These receptors respond to strong mechanical forces, sending signals to the CNS, some of which reach the cortex and are perceived as pain. Within the muscle itself, signals sent via nociceptor axons are capable of stimulating other nociceptors. This recruitment causes the release of peptide transmitters which in turn cause vasodilation. Simultaneously, a protective reflex action (nocifensive reflex) attempts to minimize the pain by shortening the muscle, protecting tissue from further insult (splinting). A combination of muscle shortening, vasodilation, and pain cause a direct motion restriction. Any attempt to quickly stretch the muscle to its normal resting length restresses the receptors and triggers continued reflexive shortening.

Knowledge of both mechanoreceptive and nociceptive effects on reflex contraction of the diaphragm suggests that more efficient management of the problem can be achieved. A technique called the "diaphragm release" has been used for decades by osteopathic physicians to improve breathing efficiency as well as venous and lymphatic return to the heart. The rhythmic ascent and descent of the diaphragm during respiration creates/ enhances abdominothoracic pressure differentials which provide the impetus for fluid motion in a negative pressure fluid flow system. The great vessels and thoracic duct pass through the diaphragm. Increased tone at rest as a result of a strain of the diaphragm may adversely affect the motion of fluid through the structures.[11] In this system, if cardiac preload is decreased, less blood will be pumped through the lungs, leading to a decrease in $P0_2$ and an increase in $PC0_2$ of the blood entering the systemic circulation. With less 0_2 reaching the working tissues, a mild hypoxia develops, concurrent with a decrease in nutrient supply. Then the muscles fatigue much more easily.[12] This gives us a second reason for restoring proper diaphragmatic motion.

The treatment technique is simple. Since the athlete took a blow to the abdomen, first eliminate the possibility that a more serious injury has occurred. Then, have the athlete assume a seated posture either on the field or on a bench. Kneel or stand behind the athlete. Inform the athlete the treatment may be uncomfortable, but should not be painful, and tell him/ her to relax and remain passive throughout the treatment. Have the athlete lean back against you. Lift up the jersey, and with the fingers spread apart on both hands, make contact with the skin just below the inferior costal border. Your hands should be parallel to the costal border, with your little fingers and hypothenar region against the skin. Lean the patient forward, folding him around your hands, which will now contact the accessible region of the diaphragm. Now move the thorax slowly first to the right, then the left, testing for a motion restriction. This is not a *test* of how far you can rotate the patient; you should actually turn the patient minimally

while you palpate for increased diaphragmatic tension. Within one cycle of motion, determine the direction in which the player can be rotated more easily (with less diaphragmatic tension), and rotate the thorax toward that position of ease. Hold the thorax in this position and have the athlete inhale while you resist the natural tendency to come out of the rotated position. Then have him slowly exhale. As the muscular relaxation (called "unwinding") is felt, gently rotate slightly farther in the direction of ease. Continue this process until the motion of the diaphragm feels smooth, and rhythmic. The unwinding may feel dramatic, or be more subtle. Now slowly retest the bilateral range of motion. It should be more symmetrical when you compare rotation of the two sides. Return the thorax to the neutral position.[13] If a restriction still exists, repeat the technique.

While the mechanism for the release is not completely understood, current research on the neurophysiologic response to manipulation suggests the following explanation. As the thorax is rotated into its position of ease, two reflexes are employed to halt the muscle spasm. First, the golgi tendon reflex is stimulated sufficiently to cause complete relaxation of the muscle.[14] Secondly, the stretch reflex is utilized by doing a slow stretch. This allows the muscle spindles to be slowly stretched,[15] thus enabling the spindles to be reset at a longer resting length, and proper diaphragm range of motion is restored. The nociceptive input is also decreased by putting the diaphragm in a position of comfort. This shuts off the pain perception in the CNS, also contributing to the ease of breathing. Once nociceptive input is decreased or eliminated, a gradual stretch of the diaphragm, synergistic muscles, and connective tissues decreases the likelihood of restressing the receptors.[16] Both mechanoreceptor and nociceptor changes may, therefore, be induced by the treatment. The gradual relaxation of the diaphragm accounts for the "unwinding" sensation that is palpated, and the sensation of increased ease of respiration.

During the 1991 football season this technique was tried on several high school athletes and achieved positive results. All of the athletes reported the pain associated with the injury, as well as the breathing restrictions they felt immediately post injury, totally resolved upon completion of the treatment. All players, after being injured, were allowed to "catch their breath" before they left the field. While exiting, all showed varying degrees of splinting the abdomen and shallowed respirations. Once off the field, the treatment was begun, and once the diaphragm released, all demonstrated improved, relaxed pain-free respiration and had no further signs of splinting. The athletes subjectively reported a complete resolution of the symptoms. The treatment may not change the time required for the athlete to return to activity, but subjectively decreases symptoms and improves the recovery period. Using the technique will also give the athlete the assurance that something is being done to alleviate this painful, frightening injury. After returning to activity, none of the treated athletes showed any visible signs of breathing or performance impairment.

All responses were favorable and the treatment warrants further trial and investigation. A controlled clinical trial would be difficult to achieve, as stated in the opening the injury occurs due to an unexpected blow to the abdomen, also athletes would not want to have the "wind knocked out" of them, and would guard against or anticipate having it done if told they were part of a study. As stated before, the muscle spindles lengthen or shorten depending upon the degree of anticipated stretch.[17]

Early treatment can reduce the initial insult to the tissue, therefore decreasing lost time on the field. At the very least, the act of touching the patient while reassuring that patient promotes relaxation and a more rapid recovery.

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