JOURNAL

A Publication of the American Academy of Osteopathy

VOLUME 4 NUMBER 4 WINTER 1994

Osteopathic Medicine and Research

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Winter OMT Update

(Intermediate Course)

February 11-12, 1995

Adams Mark Hotel
Indianapolis, Indiana

Program

Application of Osteopathic Concepts in Clinical Medicine and Preparation for OMM Boards

Saturday, February 11

8:00 am *Introduction/Course Overview*
Melicien Tettambel, DO

8:20 am *High Velocity, Low Amplitude, with questions and answers*
Elaine Wallace, DO

9:00 am *Muscle Energy*
Harriett Shaw, DO

9:40 am Small Group Discussion

9:50 am *Myofascial Release*
Melicien Tettambel, DO, FAAO

10:30 am *Counterstrain*
Ann Habenicht, DO

11:10 am *Cranial Osteopathy*
Theresa Cisler, DO

12:00 nn Lunch

1:00 pm *Cervical-Suboccipital Trouble-shooting (with Case Histories & Treatment Modalities)*
Ann Habenicht, DO

3:00 pm *Thoracic Spine*
Melicien Tettambel, DO

5:00 pm *OMM Board Applications & Case Studies*
Faculty

Sunday, February 12

8:00 am *The Extremities with Skill Session*
Harriett Shaw, DO

10:00 am *Lumbar Spine and Pelvis*
Theresa Cisler, DO

12:00 nn Lunch

1:00 pm *Selecting a Treatment Modality*
Melicien Tettambel, DO, FAAO

2:00 pm *Dosage of OMT*
Ann Habenicht, DO

3:00 pm *Written Exam Preparation "What to Expect"*
Faculty

4:00 pm *Oral Preparation*
Faculty

5:00 pm *Questions & Answers Individually Troubleshooting*
Faculty

Program Chairperson
Melicien Tettambel, DO, FAAO
Certified AOBSPOMM
Certified AOBOGS

Faculty:
Ann Habenicht, DO
Certified AOBSPOMM

Harriett Shaw, DO
Certified AOBSPOMM

Elaine Wallace, DO
Certified AOBSPOMM

Theresa Cisler, DO
Certified AOBSPOMM

Course Objective:
This Academy program is designed for the physician desiring the following:

- OMT Review: Hands-on experience and troubleshooting
- Integration of OMT in treatment of various cases
- Preparation for OMT practical portions of certifying boards
- Preparation for AOBSPOMM (American Osteopathic Board of Special Proficiency in Osteopathic Manipulative Medicine)

Course Fees:

Prior to January 15, 1995

$475	AAO Members
$575	AAO Non-Members
$200	AAO Member Interns/Residents
$300	AAO Non-Mbr. Interns/Residents

Who May Attend:
Educational objectives for AAO are to provide programs aimed to improve understanding of philosophy and diagnostic and manipulative skills of osteopathic physicians and foreign DOs with a full license or a registration, medical, podiatric and dental professions within their licensed privileges of practice and for those in programs leading to such license.

American Academy of Osteopathy

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The AAO Journal is the official quarterly publication of the American Academy of Osteopathy (3500 DePauw Blvd., Suite 1080, Indianapolis, Indiana, 46268-1136). Third-class postage paid at Carmel, IN. Postmaster: Send address changes to American Academy of Osteopathy 3500 DePauw Blvd., Suite 1080, Indianapolis, IN., 46268-1136

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 palpatory diagnosis and osteopathic manipulative treatment.

From the Editor

The Uniqueness of Osteopathic Medicine: Do We Know What it is?

If you have been keeping up with the literature over the past year or so, you know that there has been a great deal written about how the osteopathic profession should go about preserving its identity for the future. Everyone seems to agree that in order to remain a viable profession we need to demonstrate our unique aspects — those things that justify our existence as a separate profession in the world of medicine. I. M. Korr, one of the most prolific researchers and writers our profession has had, feels that we need to define our role as a profession with respect to the needs of society.[1] Another astute writer, Norman Gevitz, has studied the osteopathic profession for many years. He feels that we need to clearly define what is unique about osteopathic medicine and establish ourselves once and for all as a parallel and distinctive profession.[2]

One thing everyone seems to agree on is that osteopathic manipulation is not the only unique aspect of our profession. The simple application of manipulative techniques to standard medical practice does not make us different. The principles of body unity, self-regulation and self-healing must all be integrated into the total care of the patient. How do we do this? And how do we demonstrate this integration of principles so that people clearly see that osteopathic medicine is indeed unique?

We often say the osteopathic medicine is both science and art. In times like this, when we want answers about the nature of osteopathic medicine, I find that the best source of information is A. T. Still himself. If we examine Dr. Still's writings, we see that for him the "science" of osteopathy was based on a thorough knowledge of anatomy. The "art" of osteopathy seemed to be embodied in his patient-centered approach. We all know that Dr. Still did not leave us with technique manuals on osteopathic approaches. He felt that each patient was unique, and therefore felt that an overall philosophy of health care was more important than descriptions of techniques. As he put it, "Then I will not have the worry of writing details of how to treat any organ of the human body, because he (i.e., the osteopath) is qualified to the degree of knowing what has produced variations of all kinds in form and motion. I want to establish in his mind the compass and searchlight by which to travel from the effect to the cause of all abnormality of the body."[3]

Another distinctive aspect of osteopathic medicine was Dr. Still's thoughts on where to look for the source of disease, and where to find the cure. Carol Trowbridge, in her book, *Andrew Taylor Still: 1828 - 1917*, says, "The osteopaths and the MDs studied the same anatomy, the same nervous system, muscles, ligaments, organs and lymphatic system; they faced the same diseases and conditions. Whereas the regular physician looked outside the body for cures, the osteopath looked within the body for cures, making Still's manipulative therapy, rather than internal drugs, an integral part of osteopathic practice."[4]

What are your thoughts about the distinctiveness of osteopathic medicine? Now more than ever it is important that we have a clear description of what is unique about our profession. Perhaps you would like to share your thoughts with the rest of your colleagues. If so, send us your ideas about what makes osteopathic medicine different. Maybe you will see them in print right here in the Journal!

1 Korr, I. M. Osteopathic medicine: the profession's role in society. JAOA, Vol. 90, No. 9, Sept. 1990.

2 Gevitz, N. Parallel and distinctive: the philosophic pathway to reform in osteopathic medical education. JAOA, Vol. 94, No. 4, Apr. 1994.

3 Still, A. T. Osteopathy: Research and Practice. Kirksville, MO. By the author, 1910, p. 38.

4 Trowbridge C. Andrew Taylor Still: 1828-1917. Kirksville, MO. The Thomas Jefferson University Press, 1991, p. 165. ❑

A.T. Still Medallion Deadline Nears

The deadline to submit the name of a candidate for the 1996 A.T. Still Medallion of Honor Award is April 15, 1995.

Deserving members of the Academy who shall have exhibited among other accomplishments in scientific or professional affairs an exceptional understanding and application of osteopathic principles, and of the concepts which are the outgrowth of those principles, may be awarded the Andrew Taylor Still Medallion of Honor. The Academy cherishes this award as its highest honor, and all petitions are considered confidential.

If you have any questions or need any additional information about this procedure, please contact the Academy office or refer to page 125 of your AAO 1994 Directory.

Instructions for Authors

The American Academy of Osteopathy (AAO) Journal is intended as a forum 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.

Professional News

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, University of New England, 11 Hills Beach Road, Biddeford, ME 04005.

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 one copy. Please 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.

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.

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).

Message from the Executive Director

Stephen J. Noone, CAE

Having just returned from the 1994 AOA Convention in San Francisco, I am pleased to report on the many successes of the Academy and its leadership throughout that week's experience. First of all, I received many compliments on the Academy's Program coordinated by Program Chairman Mark Cantieri around the theme "The Objective Documentation of Somatic Dysfunction." The competition for physician attendance was formidable, with programs from 13 of the AOA's practice affiliates and the many attractions of San Francisco and Northern California; however, the AAO program held its own. One special highlight was the T. L. Northup Lecture delivered by Gary L. Ostrow, DO of New York. Convention preregistration of AAO members totaled 126, a 10.5 percent increase over the 1993 AOA Convention in Boston.

This annual event offers the Academy the invaluable opportunity to interact with other leaders in the osteopathic profession in a variety of settings. The following represents a short commentary on these contacts; you will read more detailed reports on these matters in future AAO publications:

** President Eileen DiGiovanna and I attended the Leadership Conference of AOA Practice Affiliates where we had the opportunity to engage the AOA's Board of Trustees in a discussion of major issues, including the impact of managed care on osteopathic physicians.

** Dr. DiGiovanna and I also represented the Academy at the Federation of Osteopathic Specialty Organizations, an independent coalition of the AOA's specialty colleges and affiliated organizations. The agenda focused on the need to clarify the future direction of the profession and unify all osteopathic organizations to that direction.

** Dr. DiGiovanna, Drs. Boyd Buser, Michael Kuchera, Herb Yates and I met with the Board of Governors of the American College of Osteopathic Family Practitioners. The AAO leadership appealed to the ACOFP to work with the Academy on a variety of issues of mutual interest: establishment of a combined family practice and OMM residency; coordination of an OMT workshop and osteopathic diagnosis and treatment service at ACOFP Annual Convention; and facilitation of a mechanism to permit the enrollment of allopathic physicians in osteopathic graduate medical education programs in OMM.

** Dr. Mark Cantieri and I attended the Convention's Joint Session entitled "Osteopathic Medicine and Managed Care — A Look at the Evolving Healthcare Delivery System." The session included presentations from two osteopathic physicians who are CEOs of successful managed care organizations. Dr. Cantieri commented strongly from the floor that the profession must work to include osteopathic manipulative medicine within the services covered under all managed care plans.

** Osteopathic Medical Economics Chairperson Judith Lewis and a number of AAO members attended the AOA's Train the Trainers Workshop, sponsored by the AOA Work Group on Coding/Reimbursement. The presentation introduced the Health Care Financing Administration's new Documentation Guidelines for Evaluation and Management Services. Dr. Lewis has arranged for the Train the Trainers Workshop to be presented at the 1995 Convocation in Nashville.

** Dr. Lewis and I met with the Osteopathic Physicians and Surgeons of California's Task Force on Documentation of Osteopathic Manipulative Treatment. Also present at this meeting were Raymond Stowers, DO, Chairman of the AOA Work Group on Coding/Reimbursement and Nancy Edwards, AOA's Manager of Payer Relations. The focus of the discussion centered on the creation of educational materials to enable DOs to implement HCFA's new Documentation Guidelines for Evaluation and Management, especially as they relate to osteopathic manipulative medicine.

** Finally, I was able to attend the semiannual meeting of the Association of Osteopathic State Executive Directors (AOSED) where I was able to ensure the state societies of the Academy's continued attention to coding/reimbursement of osteopathic manipulative treatment. ❑

Call for Papers

The AAO's Education Committee has supported Chairperson Ann Habenicht's inclusion of a New Ideas Forum as part of her 1995 program at the Opryland Hotel in Nashville, Tennessee. The Committee directed me to issue a "Call for Papers" to be submitted to the Education Committee for consideration at their February 1995 meeting.

Presentations by leading clinicians and researchers are planned for the main sessions as at the 1995 AAO Convocation. Additional ideas are welcome in the form of posters and presentations. It is the policy of the Education Committee to invite additional contributions for possible consideration at the 1995 Convocation from all interested parties. Abstracts of ideas/papers should be directed to AAO Education Committee, 3500 DePauw Boulevard, Suite 1080, Indianapolis, Indiana 46268-1136. Phone (317) 879-1881.

Deadline for receipt of abstracts is

January 13, 1995.

The Cranial Academy

presents its

Basic Course in Osteopathy in the Cranial Field
June 17-21, 1995

and

Cranial Academy Annual Conference
"Tour de Force"
June 23-25, 1995

Arizona Biltmore
Phoenix, Arizona

For more information, contact:
The Cranial Academy
(317) 879-0713

To The Editor

Dear Dr. Hruby,

I enjoyed Dr. Lipton's article on the Cranial Rhythmic Impulse. His grasp of the literature is certainly comprehensive. But I'd like to debate one of Dr. Lipton's suppositions — that widespread use of Sutherland's techniques has not caused any conceivable side effects. Most cranial practitioners may recall a patient or two who complained of minor headaches or vertigo after treatment. These are side effects. In an upcoming JAOA paper, Philip Greenman and I report significant iatrogenesis from Sutherland's techniques, including emotional swings, psychiatric disturbances, nausea, vomiting, diarrhea, cardiac palpitations, and opisthotonos. These occurred in a population of patients with traumatic brain injuries.

Furthermore, most cranial practitioners have faced the clinical challenge of "undoing" a poor craniosacral treatment induced by others. "Others" includes osteopathic students, chiropractors, physical therapists, MDs, and lay practitioners. I've collected several case histories. Untoward effects include nystagmus, double vision, trochlear nerve palsy, trigeminal neuralgia, static labyrinthine reflex, convulsions, loss of consciousness, and hypo pituitary syndrome. Some Sutherland techniques seem particularly dangerous in the hands of ill-trained operators. I feel that a few illustrative cases should be documented in the literature. I'm putting a paper together and welcome anecdotes or case histories.

J. M. McPartland, DO, MS
53 Washington Street
Middlebury, VT 05753

Message from the President

Eileen DiGiovanna, DO, FAAO

The response from people wanting to become certified in osteopathic manipulative medicine has been overwhelming, deluging the examiners almost beyond their capacity. They have had to call for volunteers at the AOA Convention to assist with the examinations. Over 119 people are already certified. This is wonderful news. Osteopathic physicians and, most importantly, osteopathic medical students are reaching back to the roots of the profession. The number of Academy members swells every year. This is great.

Now we need to establish the worth of this certification. All who have become certified need to seek to have the government, insurance carriers and HMOs accept this as both primary care and specialty certification.

I recently received word that New York Medicaid has recognized our certification as a specialty ranking. I was accepted onto one of the smaller HMOs as a specialist in manipulative medicine. Each step we take is an important one.

We must continue to urge the AOA to try to get OMM listed as one of the primary care areas. They need to ensure that our certification will also be recognized as a specialty field.

Since there is strength in numbers, I urge all Academy members to seek certification and urge your friends in all other specialty fields to seek OMM certification as well. Keep the applications coming – we want you and we need you.

Now is the time!

New Name?
New Address?
New Phone?
New Fax?

Let us know, so we can keep you informed!

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Request for Proposals

The Academy's Louisa Burns Osteopathic Research Committee (LBORC) announces a request for proposals on clinical studies in osteopathic diagosis and treatment in patient management. Proposals must be submitted no later than January 1, 1995, to the LBORC, c/o The American Academy of Osteopathy, 3500 DePauw Boulevard, Suite 1080, Indianapolis, IN 46268-1136.

Research Committee Seeks Researchers

The Academy's Louisa Burns Osteopathic Research Committee (LBORC) is seeking physicians interested in participating in designing a protocol using osteopathic diagnosis and treatment in outcomes research. Interested physicians should write LBORC Chairperson Deborah Heath, DO, c/o The American Academy of Osteopathy, 3500 DePauw Boulevard, Suite 1080, Indianapolis, IN 46268-1136.

LBORC is also seeking physicians interested in participating in a standard protocol for collecting data from the use of osteopath diagnosis and treatment in their practices. Interested physicians should write LBORC Chairperson Deborah Heath, DO, c/o The American Academy of Osteopathy, 3500 DePauw Boulevard, Suite 1080, Indianapolis, IN 46268-1136.

A Hypothesis for the Facilitated Segment Based Upon Biological Principles Associated with Tumorigenesis

by G. Yonuschot, PhD, D. J. Mokler, PhD and B. J. Winterson, PhD
University of New England, College of Osteopathic Medicine
Biddeford, Maine

Introduction

Osteopathic clinical practice recognizes the important role played by the musculoskeletal system in health and disease. More specifically, osteopathic manipulative diagnosis and therapy focus on somatic dysfunction and its alleviation. In the 1940s, studies by Denslow and Denslow et al. provided evidence that chronic hyperactivity of the spinal cord (segmental facilitation) was responsible for producing some of the features of the somatic dysfunction, i.e., restricted range of motion, tissue texture changes and changes in galvanic skin response.[1,2,3,4,5] These authors suggested that segmental facilitation was caused by sensitized neurons restricted to a segment or section of the spinal cord controlling functionally related parts of the body. More recently, Van Buskirk has postulated that facilitation is induced by activation of nociceptors.[6] The hypothesis presented in this paper expands the concept of the facilitated segment beyond the functional and cellular to the molecular level.

Key Words
Facilitated segment
Neoplasia
Oncogenes
Growth factors
Genetic susceptibility
Astrocytes

Tremendous progress in cell and molecular biology over the last two decades has provided new insights about cancer and tumorigenesis. It is our view that these new biological concepts may be applicable when considering the facilitated segment. Two concepts are particularly illuminating.

First, cancer is now conceptualized in terms of oncogenes, tumor suppressor genes and growth factors (cytokines)[7] (Table 1). Oncogenes and tumor suppressor genes are intracellular and can control cell growth and differentiation. However, many growth factors are extracellular. Thus, other (noncan-cerous) cells contribute to the initiation and maintenance of neoplasms.[8] Similarly, modifications in gene expression and growth factors may contribute to the development and maintenance of the facilitated segment (Table 2).

The second concept is that several mutations, not just one, are necessary for development of neoplasia. Both somatic and germ cell mutations may be involved (Table 1).[9] Thus, some individuals are predisposed to develop neoplasms, whereas others are not. Further, some regions of the soma may develop neoplasms and some not. The following is a formal statement of our hypothesis for the facilitated segment that incorporates these concepts.

Hypothesis

A facilitated segment is the result of facilitated cells that may act alone or in concert to change the function of the spinal cord segment from normal to abnormal. Facilitated cells may be neuronal or non-neuronal and both types may arise from normal cells. Facilitated cells are transformed (Table 1) as a result of facilitated genes (abnormally expressed genes). Expression of facilitated genes may be permanent or reversible reflecting whether the change in the gene is permanent or temporary.

Rationale

The hypothesis developed from the idea that facilitation of a spinal cord segment is analogous to very early stages of neoplasia. All cells, including those destined to develop into tumors, go through phases of cell division and differentiation **(Table 1)**. This process is controlled by both the concentration of extracellular growth factors and the intrinsic ability of the cells to respond to growth factors.[10] A neoplasm occurs when the normal control mechanisms for cell division and differentiation go

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Table 1. Concepts of Neoplasia

Differentiation:	Acquiring a character or function different from that of the original type. Differentiation can be reversible.
Growth factors:	Mediators of intercellular communication that influence differentiation and proliferation.
Oncogene:	A gene whose activation, by increased expression or by alteration of protein product, contributes to the neoplastic state.
Proto-oncogene:	A normal gene involved in normal control of cell differentiation and proliferation. Oncogenes are mutated ("activated") forms of proto-oncogenes.
Neoplasia:	The pathological process causing neoplasm.
Transformation:	The conversion of one form of cell into another.
Tumor suppressor:	Genes that prevent abnormal cell proliferation.

Table 1

awry. Tumorigenesis is thought to result from a combination of mutations and extracellular growth signals, i.e., it is the result of changes in the immediate environment of the cell, the cellular mechanisms for responding to the environment, and/or other cellular mechanisms for controlling growth and differentiation.[11,12]

As with potential tumor cells, the cells responsible for the correct functioning of a region of the spinal cord are controlled by their intrinsic genetic expression and by extracellular factors modulating genetic expression. Under normal circumstances, almost all cells function within normal physiological ranges. Occasionally, however, the function of cells is changed due to mutations or to exposure to extreme concentrations of extracellular factors so that the cells respond abnormally. The abnormally responding cells are much more likely to initiate and maintain a facilitated segment.

Facilitated cells

The cells that comprise a facilitated segment are responsible for the heightened activities. A single cell type (i.e. gamma motoneurons) may be the sole contributor, but other cell types (i.e. astrocytes) may also have altered function. However, one or more facilitated cell types may not necessarily produce a facilitated segment. Other factors might be necessary for the facilitated segment to manifest as somatic dysfunction. We would like to distinguish facilitated cells, which arise from normal cells, as facilitation cells. Facilitation cells, due to their genetic makeup, are capable of becoming facilitated. Also, facilitated genes arise from normal genes called facilitation genes (**Table 2**).

Facilitated genes are genes that have been changed so that either an abnormal gene product or an abnormal amount of gene product is produced (**Table 2**). They are analogous to oncogenes, tumor suppressor genes and genes controlling growth factors that help to promote tumorigenesis. These genes are thought to be responsible for the initial stage of tumorigenesis.[13]

Oncogenes are derived by mutation from proto-oncogenes.[1] Proto-oncogenes are normal genes that are involved with normal cell division. Proto-oncogenes produce protein products that control normal cell division such as growth factors, growth factor receptors, G-proteins, proteins involved with second messenger systems and nuclear regulatory proteins (**Figure 1**).

Oncogenes are derived from proto-oncogenes by several types of mutations. One major category of mutations produces an abnormal protein that functions in the cellular response mechanism to extracellular growth factors. In this case, the abnormal protein results in an abnormal cell division signal despite the fact that no such signal is received at the cell membrane. A second major category of mutation results in the loss of the normal mechanisms controlling the rate of gene expression. This causes the overproduction of proteins controlling cell division. For example, the c-fos oncogene protein product is known to be increased or mutated in many forms of cancer. It is thought to function by helping to activate the transcription of many genes involved in cell division.

Segment cells may have genes that modulate facilitation in a manner similar to the way that proto-oncogenes modulate cell division and differentiation. For example, some genes might regulate the behavior or number of membrane channels necessary for normal facilitation. Changes in the expression of these genes would produce facilitated genes. It is extremely interesting that the c-fos protein product, mentioned above, is increased in the spinal cord by painful stimuli.[14,15] Thus, an oncogene might well be a facilitated gene.

Table 2. Terms used in the Hypothesis.

Facilitation Gene:	Any gene that has the capacity to produce a product that influences facilitation.
Facilitated Gene:	A prefacilitated gene that has been changed so that either an abnormal gene product or an abnormal amount of gene product is produced. A facilitated gene is necessary but may not be sufficient to produce a facilitated segment,
Temporarily Facilitated Gene:	A facilitated gene that has been abnormally activated or inactivated but that, after a time, will return to a normal state.
Permanently Facilitated Gene:	A facilitated gene that is the result of mutation.
Facilitation Cell:	Any cell that can influence facilitation. These cells contain facilitation genes and can be neuronal cells or cells that have the capacity to influence excitation and conduction.
Facilitation Factor:	An extracellular factor that modulates facilitation by interacting with neuronal or nonneuronal cells.
Facilitated Cell:	Any cell that contains a facilitated gene and thus may influence facilitation. Facilitated cells may be neuronal or nonneuronal and are necessary, but may not be sufficient, to produce a facilitated segment. More than one type of facilitated cell may cooperate to produce a facilitated segment.
Segment:	Interacting set of neuronal and nonneuronal cells serving a region of the body restricted in a rostro-caudal direction.
Facilitated Segment:	The abnormal facilitation of a segment.

Table 2

Tumor suppressor genes are recessive genes that regulate cell differentiation and division independent of extracellular factors.[16] Before tumorigenesis, both alleles must be inactivated by mutations. For example, several types of tumor suppressor genes are deleted during the development of colon cancer.[17] It appears that most oncogenes cannot be expressed in the presence of specific tumor suppressor genes. Thus, even though cells contain oncogenes, they will not become neoplastic until the tumor suppressor genes are deleted. It may be that segmental cells have genes that protect the segment against the abnormal expression of other genes. Thus, some facilitation genes would be similar to tumor suppressor genes. For example, even if a mutation occurred that produced an abnormal amount of a membrane channel, it might be that facilitation would not develop because another gene product would protect against this defect. However, loss of this second type of facilitation gene would allow the defect of the first facilitated gene to be manifested as a facilitated gene.

Finally, growth factors are involved in some neoplasias. For example, the *sis* oncogene product is Platelet Derived Growth Factor expressed at an abnormally high level.[18] In addition, much data has shown a relationship between hormones and cancers of the endometrium, breast, ovary, prostate, thyroid and bone.[19,20] Since it does not seem plausible that the tumors always develop a need for growth factors only after a neoplasm has developed, it is assumed that the need is present during the earliest stages of neoplasia. Finally, there is speculation and evidence that growth hormones act as tumor promoters.[10] Tumor promoters do not directly cause mutations but do cause increases in cell division and other cellular changes that increase the probability of mutations.[21] The genes that control growth factors may be found in both the cell being transformed into a tumor cell and in the other cells in the environment supporting tumorigenesis (**Table 1**).

In a similar fashion, genes producing growth factors may influence facilitation. Current hypotheses regarding facilitation suggest that the excitatory amino acids released in response to noxious stimuli may act to facilitate a segment of the spinal cord.[22]

Studies have shown that epidermal growth factor enhances excitatory amino acid effects in the central nervous system.[23] Thus, growth factors may also influence facilitation in the spinal cord. A general term we suggest to cover extracellular factors that influence facilitation is **facilitation factors (Table 2)**.

The fact that a cell contains an oncogene, or is lacking a tumor suppressor gene, or is exposed to abnormal concentrations of one or several growth factors, is not normally

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sufficient to cause tumor development. However, such cells are predisposed to do so. Cancer cells often contain several oncogenes, lack tumor suppressor genes and require certain extracellular factors to proliferate. Since all these mutations do not occur at once, certain cells are poised to develop neoplasms and are said to be 'initiated'.[24] Initiated cells are sufficiently transformed so that the addition of another oncogene or the loss of a tumor suppressor gene or the change in concentration of one growth factor may result in neoplasia. By analogy, the fact that one or more of the genes of a spinal cord segment have become facilitated may not be sufficient to produce a facilitated segment, but it may increase the probability that the segment will be facilitated. Thus, facilitated genes predispose the segment to become facilitated.

The concept developed here for an explanation for facilitation is different in at least two important ways from the conception of tumorigenesis. First, neurons and muscle fibers are key elements in the facilitated segment and neither cell type is prone to cell division. However, the non-neuronal cellular matrix could become hyperplastic. Second, tumors develop precisely because mechanisms controlling cell division are functioning inappropriately. Although facilitation may result from changes of such growth controlling genes, hyperexcitability is postulated to be a key element of facilitation. Therefore, alteration of genetic expression that influences cell excitability would produce facilitation.

Temporarily or permanently facilitated genes

Facilitated genes can be either temporarily or permanently facilitated **(Table 2)**. Temporarily facilitated genes would be the result of the up or down regulation of facilitation genes such that abnormal levels of the gene product would be expressed. With time, the signals causing abnormal expression would return to normal and, as a result, the facilitated gene would return to being a facilitation gene. Permanently facilitated genes would result from mutations. Some mutations would cause underexpression or eliminate expression of the gene. Other mutations would cause overexpression of the gene. Still other mutations would cause the production of abnormal proteins.

Non-neuronal Cells and Facilitation

Our hypothesis brings into focus the possible importance of non-neuronal cells and warrants further discussion. The non-neuronal cells we consider important compose the entire tissue matrix that encapsulates the reflex arc. The non-neuronal cells have the potential to produce extracellular factors that could influence sensory fibers, interneurons, motor neurons and muscle fibers themselves. Thus, cells reside in a matrix, receiving signals from neighboring cells. As an example from the renal system, renal glomerular mesangial cells in culture respond to 28 different naturally occurring factors (some associated with the immune system).[25] In addition, 20 separate factors are produced by mesangial cells. Furthermore, many of the factors made by mesangial cells also exert autocrine effects; these include: three eicosanoids, interleukin-la/B, interleukin-6, tumor necrosis factor-a, colony stimulating factor-1, insulin-like growth factor-1, insulin, and transforming growth factor-B.

CNS neurons are surrounded by a matrix of glial cells. How might surrounding non-neuronal cells interact with neurons? Consider the factors produced by astrocytes and astrocytoma cells (derived from mature astrocytes or a common stem cell). Astrocytes, like all other cells, produce eicosanoids[26], such as prostaglandins, and are influenced by them.[27] Furthermore, receptors for some of the eicosanoids produced by astrocytes are found on neurons of the CNS.[28] The several means by which eicosanoids modulate neuronal activity are reviewed by Murphy.[29]

Interleukin-1 (IL-l) is traditionally thought of as being a factor unique to the immune response; however, it is now apparent that IL-1 is produced by most cell types, and IL-l receptors are found on a wide variety of cells.[30] Indeed, astrocytes produce mRNA for IL-l and presumably IL-1,[31,32] and there is suggestive evidence for neuronal IL-l receptors.[33] When produced in abnormally high concentrations, IL-l contributes to neuronal damage.[34]

Other factors are likely produced by astrocytes. About 30 percent of astrocytoma cells express oncogenes for the epidermal growth factor. Some astrocytomas express both platelet-derived growth factor and platelet-derived growth factor receptor, suggestive of an autocrine loop. In human gliomas, transforming growth factor-alpha, insulin-like growth factors I and II and fibroblastic growth factor have been found.[35] Receptors for all of these factors have been found on neurons of the CNS in mammals.[36,37,38,39]

The Hypothesis Generates Corollaries of Clinical Importance

The hypothesis we propose can generate a number of statements which are logically consistent with it (corollaries). They are for the most part counterintuitive when considered in juxtaposition with prevailing neurophysiological explanations of somatic dysfunction. However, the practitioner of manipulative diagnosis and therapy may find much of the following consistent with his or her clinical experience:

continued on page 26

Review of the 1993 Journal of the *New Zealand Register of Osteopaths*

by Robert C. Clark, DO

Earlier this year I received a copy of the 1993 Journal of the New Zealand Register of Osteopaths (JNZRO) from our colleague Richard Carruthers, DO. Dr. Carruthers has graciously provided copies of prior issues of this annual journal for review. The 1993 JNZRO is different in that Dr. Carruthers is now the assistant editor having yielded the editor's chair to Tracy Livingston, DO. Be assured that with a change in editors there is no change in the high quality of articles and papers published.

The JNZRO continues its fine osteopathic tradition with a blend of major articles, original research, discussion papers, case studies and reports and book reviews. It is worthwhile reading for all DOs whose interests lie in the traditional osteopathic way of treating patients. Most articles are brief so that a busy practitioner can read as time allows. In fact in 50 pages there are over 30 items listed in the table contents. Do not let brevity imply incompleteness - far from it. In several of the articles brevity implies precision of thought on the part of the author.

Since there are too many articles to review only a few will be presented to whet the reader's appetite. The lead article is "The Osteopathic Treatment of Asthma" by Richard Carruthers, DO. This article reviews the literature regarding osteopathic manipulative treatment of asthma. It shows the areas of the body where treatment is reasoned to be beneficial and cites the few clinical trials of osteopathic treatment of patients with asthma. The trials showed improvement in patient symptoms from 55-70% from manipulative treatment.

In both the medical literature and the public press, one medical topic seems to garner much attention. That is the disease of osteoporosis. Jennifer Steinmetz reviews the prevention and current treatments of osteoporosis and concludes that the "best treatment is prevention". Although there are excellent hormonal treatments for the disease, the simple prevention includes proper diet with calcium rich foods, calcium supplements, regular exercise and avoidance of alcohol, smoking and other calcium depleting substances. These is the easiest and most cost effective treatment of preventing the effects of osteoporosis.

"Biomechanics of Sporting Injuries" by Belinda Fellowes, DO, analyzes the problems with each of the three component events of the triathlon. Along with the analysis of injury potential in swimming, cycling and running the author gives several pointers to the reader to aid in locating the source of the athlete's problems and addressing corrective efforts. Since many other sports involve running, many of the author's insights can be applied to other athletes.

M. C. McGrath, DO, reminds us that when a patient with lumbar dysfunction, buttock pain and thigh pain presents seeking relief, it is advisable to evaluate the patient for underlying etiology in the article, "Osteopathic Management of the Degenerative Hip Joint".

"A Pilot Study on the Value of Applied Kinesiology in Helping Children with Learning Disabilities", by Mark O. Matthews, Bsc, DO, et. al. was less than satisfying. The preliminary results presented are very interesting and suggest a valuable means of helping a disadvantaged portion of our population. But the article failed to give any real description of what the procedures were that produced the results reported. At the end of the article was the address of the author from whom the reader could purchase a more complete article with references and full treatment protocol.

In the United States the political debate is over health care or, more precisely, who is going to pay for what and who is going to be in control? It is no surprise to find that similar political debates occur elsewhere. Mark Franken, DO, the president of the *New Zealand Register of Osteopaths*, reports that the changes in the New Zealand government's handling of health services and the payments for same offer the DOs opportunity to advance the role of Osteopathy in the care of the people of New Zealand. In another article Richard Carruthers, DO, concludes that "... Osteopathy is an extremely cost-effective form of

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treatment for musculoskeletal complaints..."

The last article I wish to mention is "Sitting", by John Cullen, DO, who observes that, "Sitting in chairs has become the most common non-sleeping postural position of humans in the developed world." Dr. Cullen examines different types of sitting postures and analyzes the effect upon the person. He also gives suggestions for the ideal posture to prevent stress to the sacroiliac joints and other dysfunctions.

There are many other articles covering topics from cranial treatment to yoga as a form of self-treatment to short leg and the common compensatory pattern. Those who are interested in getting a copy of the 1993 JNZRO for their own benefit should contact Richard Carruthers, DO, Nelson Osteopathic Clinic, 300 Hardy St., Nelson, New Zealand. The cost in New Zealand currency is $18.00 plus overseas shipping. It is worth the purchase for this unique osteopathic journal.❑

Letter to A. T. Still

Dear Doctor Still,

I think most people would agree that there have been only a very few original thinkers in the history of the world. I'm referring, of course, to those people who were able to develop new, different and unique ideas that had a profound effect upon all the rest of us. We could argue about who would be considered an original thinker, but names like Socrates, Rene Descartes, Charles Darwin and Thomas Jefferson come to mind.

You are certainly amongst those considered to be original thinkers, as was one of your more notable students, William G. Sutherland, DO. I never cease to be impressed by the fact that you were able to develop the unique concepts of osteopathic medicine, and that Doctor Sutherland was able to extend these concepts into the cranial area.

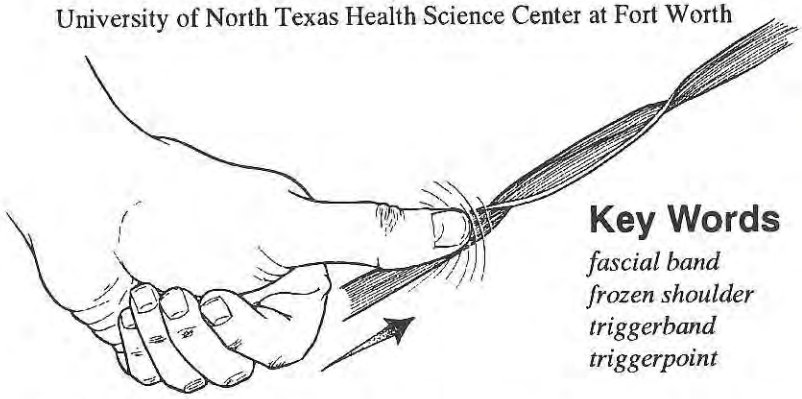
But there is an even more impressive point to think about here. You were able to put aside all of your previous training and experience in the standard medical world and allow yourself to completely rethink your approach to health and disease in the human body. Most of us tend to be so affected by our previous experiences and our knowledge of standard medical models that we have a very diffficult time allowing ourselves to look at things from fresh viewpoints.

You expressed your thoughts on this matter very elegantly in your book, *Philosophy of Osteopathy* (pp. 66-67), when you said, "As our investigations are without the assistance of ancient or modern writers, we will have to reason that man is a machine of form and power, forming its own parts and generating its own powers as it has use for them. At this time we begin to reason thus, that all powers are invisible and we see effect only. We know such forces to be abundant in nature, and life is sustained by them. To find the substances in the body that cause them to act and how to act, has been the object of my journey as an explorer. If they give us health when normal action prevails and disease only when abnormal, then we are admonished to form a more intimate acquaintance with the qualities, and with all the products, when formed in this great laboratory which compounds and qualifies each substance to fill its mission of force, construction, purity and action." You spent a lifetime developing a new and different approach to healing. We would all do well to contemplate the depth and breadth of knowledge contained in the simple principles you discovered.

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

Triggerband Technique

by Stephen Typaldos, DO, Fort Worth, Texas
The Manual Medical Center of Fort Worth
Illustrations by Gina Belsito,
University of North Texas Health Science Center at Fort Worth



Key Words
fascial band
frozen shoulder
triggerband
triggerpoint

Abstract

Triggerband Technique is a soft tissue manipulative approach that is used in the treatment of acute and chronic musculoskeletal pain and dysfunction. It is based on the premise that distorted or injured fascial bands are the cause of many types of musculoskeletal discomfort and that correction of these distortions will result in a reduction or elimination of both the pain and the somatic dysfunction.

Trigger-band Technique is a treatment for acute syndromes, such as lumbar sprain, whiplash injuries, headaches of a nonorganic nature, 'pulled muscles' and other athletic and nonathletic musculoskeletal problems. In addition, many chronic pain syndromes such as failed back surgery, frozen shoulders, 'arthritic-like' pain, 'pseudo-sciatica' and fibromyalgia often respond to this therapy.

Fascial Band Distortions in Musculoskeletal Pain

There is an extensive network of fascial bands in the human body. Except for the iliotibial tract, few fascial bands have been named or described previously. In 1990 Gerlach and Lierse[1] documented the existence of fibrous fascial bands in the lower extremity. See **Figure 1**.

From their drawings it can be seen that the fascial bands are interconnecting and interwoven. Because of this, fascial distortions can travel long distances and have what seem to be bizarre patterns of pain that do not follow known neurological, muscular or dermatome pathways.

Fascial band distortions can occur in different varieties. Some effect the fascial 'plane', others result in 'triggerpoints', and still others are predominantly bandular. However, I shall limit my discussion here to those that are most important for the physician learning Triggerband Technique. The most common of these is a 'triggerband' which is defined as a distorted fascial band that has become twisted, torn and shortened. This occurs during injury when some or all of the fibers become altered. See **Figure 2**.

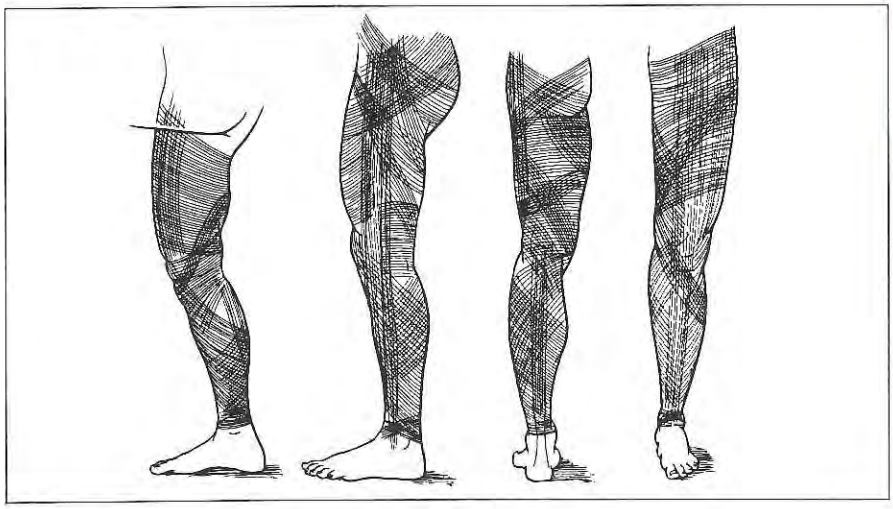


Figure 1

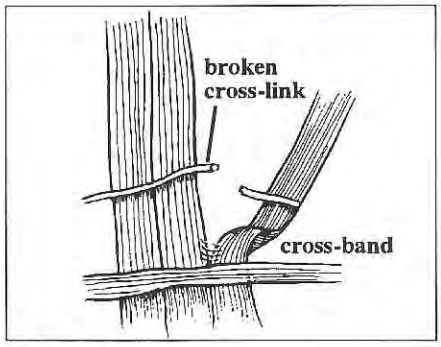


Figure 2

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Normal Fasical Band

Acutely Injuried Fascial Band

Chronically Injured Fascial Band

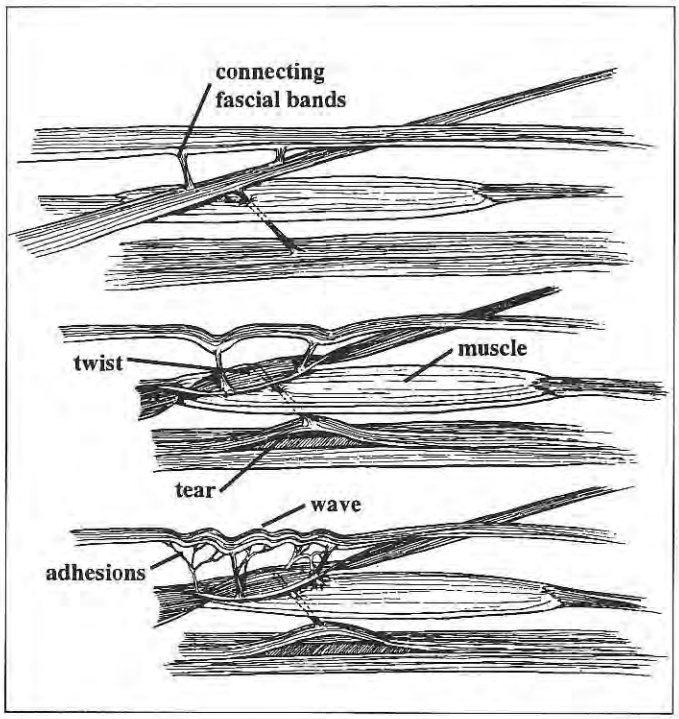

Figure 3

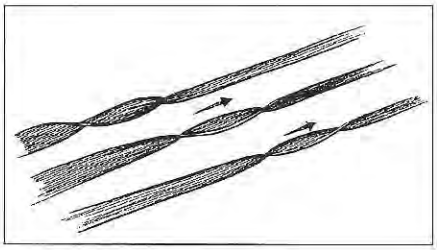

Figure 4

Both acute and chronic triggerbands are treated the same way, by manually untwisting the twist, straightening the band, reapproximating the tear and smoothing out the distortion. The difference is that in chronic pain the adhesions also must be broken making it much more painful. Please refer to **Figure 5**.

To be able to understand this concept better, it is beneficial to know the 'anatomy' of a triggerband. All triggerbands have certain components which include a 'tear', a 'twist'* and, in many cases, a 'wave'. In chronic pain 'adhesions' also occur. Examples of an 'acute pain' triggerband and a 'chronic pain' triggerband are shown in **Figure 3**.

Note that in acute pain, the twist can move up and down the entire band as shown in **Figure 4** and at times seems to 'jump' from one area to another.

This does not occur in chronic pain because the adhesions are holding the twisted band firmly in place. In chronic pain the number of adhesions gradually increases. As this occurs the patient will feel "tightness" and experience a loss of flexibility.

*or other triggerband subtype as shown in Figure 6.

Correct the twist by pushing until it is completely untwisted

Acutely Injured Fascial Band

Chronically Injured Fascial Band

Corrected Fascial Band

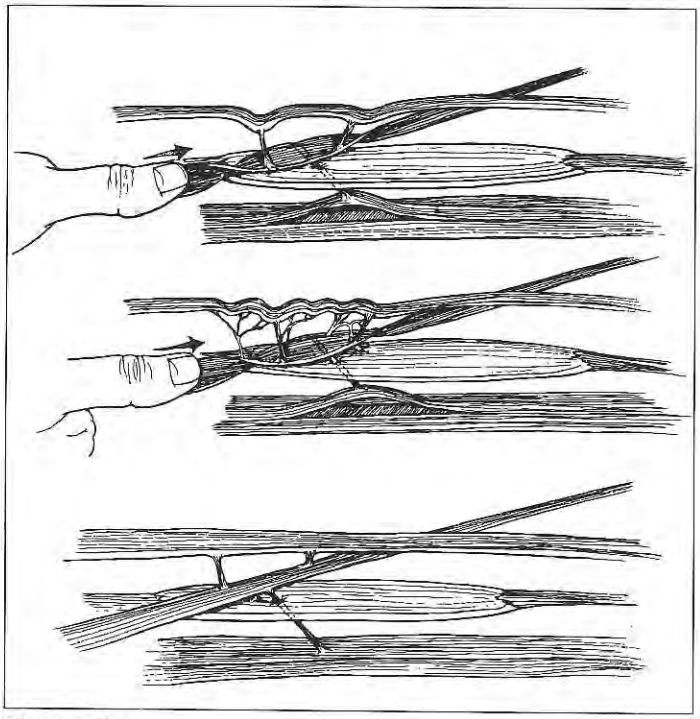

Figure 5

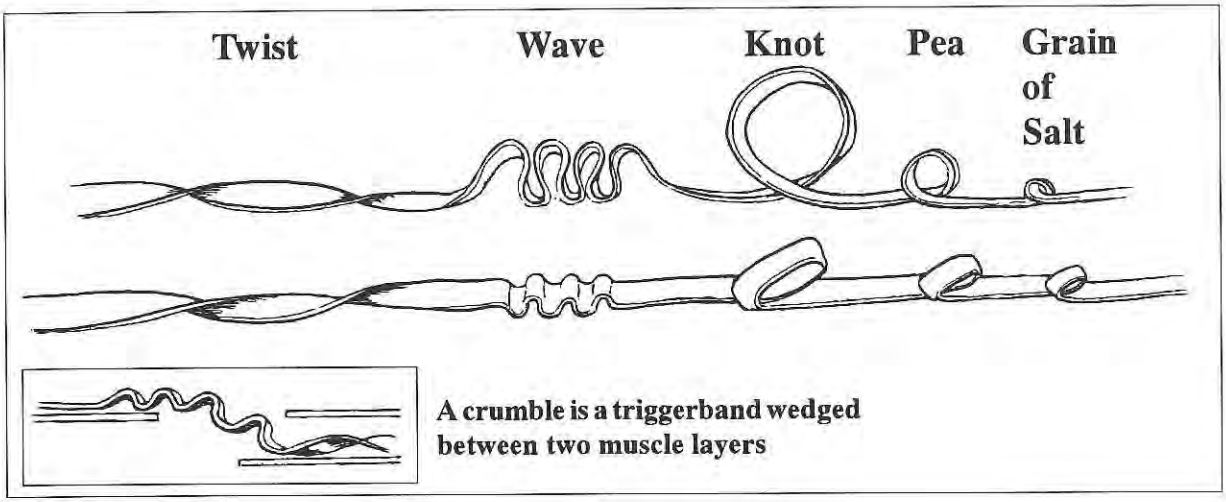

Figure 6

Triggerbands and Triggerpoints

To use Triggerband Technique, the physician first must be able to palpate a triggerband. When it is encountered, a triggerband may feel like any of those shown in **Figure 6.***

There are many other types of fascial distortions, some of which are discussed in detail in the accompanying paper *Introducing the Fascial Distortion Model.* Two of the most common are 'Banded Pseudo-Triggerpoints' and 'Herniated Triggerpoints' which are illustrated in **Figure 7**. Banded Pseudo-Triggerpoints occur when two or more triggerbands overlap. Correcting them requires following first one of the distortions and then the other. They are not actually triggerpoints per se but are an overlap of two distorted fascial bands. Herniated Triggerpoints are most common in the abdomen and are corrected by forcing the underlying material that has become 'trapped' in the distortion down below the fascial plane. Herniated Triggerpoints occur in two varieties and are described and compared in the fascial distortion model paper.

Triggerband Technique and the Physician

Triggerband Technique is a potentially painful modality for the patient. This is especially true in chronic pain. Fortunately, it is rare that patients refuse the treatment because of this. Once the treatment begins almost all patients will sense intuitively that the treatment is both appropriate and necessary. It is important that the patient and the physician realize that Triggerband Technique is normally a painful procedure and that generally the more subjective the patient's severity of pain is, the more helpful the treatment may be.

Another point to consider is that Triggerband Technique can be painful for the physician as well. The physician's thumbs may become tired and sore. Therefore, it is the advisable that the physician not attempt too many treatments in the early stages until the hand and thumb muscles have had a chance to strengthen.

When using Triggerband Technique no lotion or gel should be used on the patient's skin. Lotions or gels decrease friction and allow the fingers to glide over the skin. In Triggerband Technique it is necessary to use that friction to move and correct the underlying structure.

Some patients may complain of having their hair pulled during the treatments, and it may be necessary to shave the affected area to reduce their discomfort. This occurs normally in only particularly 'hairy' men, with the thighs and legs being the biggest problem.

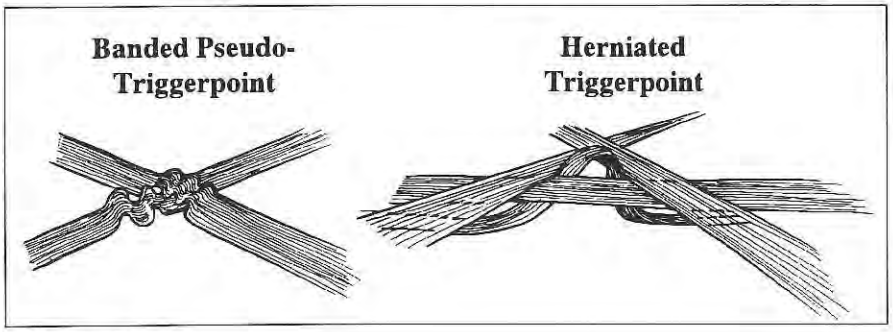

Figure 7

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*For comparison of triggerband subtypes see *Introducing the Fascial Distortion Model.* (JAAO; Summer, 1994)

Indications and Contraindications

The indications for Triggerband Technique are multiple and include most types of chronic and acute pain, back pain, neck pain, headaches, frozen shoulders, 'arthritis-like' syndromes, abdominal pain of a somatic origin and a host of other musculoskeletal dysfunctions of a nonorganic nature. As with all patients that are treated, the proper diagnosis is paramount. Metastatic cancer and multiple myeloma are two of the conditions that I have seen more than once on so-called nonorganic chronic pain patients. The fact that they may have seen many other doctors does not mean that the correct diagnosis was made. In particular, any patient who shows **no** response to Triggerband Technique should be carefully reexamined for an organic cause.

A typical type of chronic pain that may respond to Triggerband Technique is illustrated in a patient that has some or all of the 'Rule of Fours'.

Rule of Fours

- 4 or more years of pain
- 4 or more locations of pain
- 4 or more physicians previously seen
- 4 or more diagnostic procedures previously done
- 4 or more therapeutic modalities previously done
- 4 or more prescriptions given in the past

Contraindications to Triggerband Technique are mostly relative, and a partial list is offered below. Each physician should, of course, use his/her best judgment before employing this or any other treatment modality. Fortunately, I have never seen any complications of the treatment itself, but each physician should be aware that they can occur and could potentially be anything from stroke to phlebitis. Again, each physician should decide what he or she feels comfortable treating with each individual patient.

Partial List of Contraindications

Edema	Cancer
Cellulitis	Previous Strokes
Osteomyelitis	Open Wounds
Vascular Diseases	Aneurysms
Arteriosclerosis	Hematomas
Skin Wounds	Bone Fractures
Collagen Vascular Diseases	Bleeding Disorders
Poor Doctor-Patient Rapport	Litigious Patient Profile
Treatment of Abdomen or Pelvis During Pregnancy	Infectious Arthritis
Osteogenesis Imperfecta	Phlebitis

Side Effects

Pain: This occurs close to 100 percent of the time in nonathletes. Athletes rarely have this complaint. In chronic pain there may be localized tenderness after the treatment for three or four days. In acute dysfunction pain is generally only present during the treatment. Note that any pain after the treatment is much less than the pain during the treatment.

Erythema of the Skin: This occurs close to 100 percent of the time.

Brusing: This occurs in 5-10 percent of patients and is temporary.

Typical Steps in Treating a Chronic Patient

Once the physician has determined that Triggerband Technique is to be employed on a patient, he or she may need to go through specific steps to insure that proper attention is paid to certain details so that mistakes in diagnosis and treatment are avoided. For most chronic pain syndromes several days should be allowed in between treatments and four to six sessions may be needed. Progress should occur at each visit.

1) Rule out organic cause of pain.
2) Review all previous records.
3) Listen carefully to the patient's history.
4) Mentally or graphically map out patterns of pain.
5) Physical examination.
6) Make the proper diagnosis of distorted fascial syndrome.
7) Ask the patient about possible contraindications.
8) Discuss the treatment with the patient, and state in no uncertain terms that it will be painful.

continued on page 28

Software Review for The AAO Journal:
IBIS (Interactive Bodymind Information System)

reviewed by J. M. McPartland, DO, MS
Director, AMRITA (no relation)

IBIS is a huge database of alternative medicine. Its core lists 282 common "conditions" that bring patients to our offices. Most of the conditions are diseases, ranging from benign ones (acne and such) to medical emergencies (like myocardial infarction). Many nonpathological conditions are also covered (e.g., menopause, contraception).

Features:

Diagnostic criteria for each of the 282 conditions are provided, and linked with eight treatment modalities. Physical Medicine is the first treatment modality. It covers manipulation, exercises, hydrotherapy, and electrical/oscillating therapies. Nutrition includes dietary recommendations (foods to add or avoid), vitamin and micronutrient supplements, and a set of recipes. Botanical Medicine concerns Western herbs and formulae; Chinese Formulae does the same for Eastern herbs. Acupuncture suggests treatments for western diagnoses, coupled with Chinese diagnoses. IBIS uses the point numbering system in Dan Bensky's Acupuncture: a Comprehensive Text. Homeopathy details a mini-repertory with rubrics for each condition. Bach's flower remedies are listed in the next section, Vibrational Therapies, which also embraces color, sound, crystals, and electromagnetic techniques. Psychospiritual Approaches has two sections, "metaphors and correlations" and therapies. Therapies include behavior modification, process-oriented therapy, hypnotherapy, affirmations, visualizations, even medical astrology.

Other Operations:

IBIS provides a Materia Medica, full of additional treatment information. It supplies dosages and toxicology, details manual techniques, and provides references for further reading. Note Cards allows you to customize IBIS by adding data on therapeutics; the Research Module creates a patient database. Additional IBIS operations permit linking of cards, analyzing databases, finding key words, indexing key words, and printing information for patient education handouts.

Strengths:

IBIS is designed for primary care physicians. In the last month, I've encountered only one condition not listed among IBIS's 282 varieties-trigeminal neuralgia. Nevertheless, using "Find" and "Index" features, I found treatments for trigeminal neuralgia described in the Materia Medica.

IBIS contains a vast amount of information, which is well organized and easy to locate. Despite its ponderous size, IBIS searches are performed quickly. Shortcuts are available using command keys and floating palettes. Using the program is intuitive and easy to learn. Documentation accompanying the software is well-written and comprehensive. Technical help is available by telephone during working hours, Pacific time.

Of the eight modalities, Acupuncture, Botanical medicine, and Chinese formulae seem the strongest. The Psychospiritual section is daring - it comes with more cautions and contraindications than the other sections. The weakest section is Physical Medicine, especially manipulation. This weakness is a strength for osteopathic physicians. Indeed, IBIS seems tailor-made for DOs who don't need help with manipulative therapies. No indications for manipulative treatment are listed for nearly a third of the 282 conditions! Granted, OMT may not be indicated for hirsutism or scabies (at least HCFA thinks not), but other conditions miss the boat (e.g., metrorrhagia, intercostal neuralgia, post-surgical sequelae). IBIS only lists six conditions for craniosacral treatment, and rarely mentions visceral manipulation. IBIS champions Chapman's reflexes, but Muscle Energy, Counterstrain, Functional, and Myofascial techniques are unknown to the naturopaths who designed the program. Techniques they include under manipulation are sometimes mysterious ("nasal specifics" for Downs), and sometimes not manipulation (wiping with cotton instead of toilet paper for pruritus ani).

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Weaknesses:

To click into a condition requires the practitioner to adopt IBIS's terminology. This can be idiosyncratic. For instance, gallstones are not listed (see cholecystitis), but kidney stones are. Some of the listed conditions seem redundant, such as "intestinal parasites," "intestinal worms," "intestinal flora imbalance," and "intestinal dysbiosis." Since IBIS was written by several authors, treatments are not consistent. This is especially evident in the nutrition section. For instance, ten nutritional supplements are offered under "tachycardia," but none are listed for "arrhythmia."

IBIS does not rank remedies. Look up botanical remedies for prostatitis, and you'll find saw palmetto buried in a list of 18 different herbs. This lack of priority arises in all the modalities. The choices become overwhelming, and sometimes contradictory (e.g., foods listed in both "add" and "avoid" columns). The IBIS diagnosis section does not incorporate osteopathic information. The acupuncture section does not map point locations (except extra points), so you can't learn acupuncture from IBIS.

Bottom Line:

IBIS is immensely useful for physicians who wish to diversify their treatment modalities. It also serves as a reference tool to investigate things your patients may be taking or doing. IBIS version 1.2 is available for Macintosh or IBM-compatible computers. The Mac version requires over 15 MB of disc space and 4 MB of RAM on System 7. IBIS uses a HyperCard engine (version 2.1) with pull-down menus, pop-up text, and mouse controls. It costs $895. Student discounts and interest-free payment plans are available. Demo discs are free. IBIS is produced by AMR'TA (Alchemical Medicine Research and Teaching Association), P.O. Box 14641, Portland, OR 97214, phone (800) 627-6851 or (503) 228-6851; fax (503) 228-6904. ❑

In Memoriam

Donald C. Siehl, DO

Dr. Donald Siehl passed away on September 30. He graduated from the Kirksville College of Osteopathic Medicine in 1943 and served both an internship and orthopedic residency at Doctors Hospital in Columbus, Ohio. He practiced as an orthopedic surgeon in Dayton, Ohio until his retirement. Dr. Siehl was one of only four DOs to serve in the U.S. Public Health Service as a commissioned medical officer during World War II.

A dedicated and loyal Academy member since 1943, Dr. Siehl delivered the Academy's T.L. Northup Lecture in 1974 and the Scott Memorial Lecture in 1982 He earned his Fellowship in the American Academy of Osteopathy in 1978.

Dr. Siehl was certified in orthopedic surgery by the American Osteopathic Board of Surgery, later serving as its secretary-treasurer and orthopedic representative. He also served as president of the American College of Osteopathic Surgeons and American Osteopathic Academy of Orthopedics. He served as president of the American Osteopathic Association in 1978-1979.

He is survived by his wife, Susan, and seven children. Four of his brothers were also osteopathic physicians. Friends may write his family at 1500 Westbrook Road, Dayton, Ohio 45415 Memorial contributions may be made to Grandview Hospital/Donald Siehl Memorial Fund or to a charity of the donor's choice.❑

David M. Davidson, DO

Dr. Davidson of Kettering, Ohio, passed away April 16, 1994. He was a retired radiologist with 35 years of service and remained active in osteopathic medicine.

Dr. Davidson graduated from Kirksville College of Osteopathy and Surgery in 1938. As well as being certified in diagnostic roentgenology, he was a fellow of the American Osteopathic College of Radiology.

Memberships included The American Academy of Osteopathy and life membership in the American Osteopathic Association, among others.

Dr. Davidson was survived by his wife, Lillian L, Davidson of Kettering, two sons, David M. Davidson of Boston, MA and Edwin S. Davidson of Kettering.

The Academy sends its deepest condolences to his family.❑

From the Archives

"Who is to Blame?" Revisited

by Philip E. Greenman, DO, FAAO

Editor's Note: *the following excerpts are from Dr. Greenman's Thomas L. Northup, DO, Memorial Lecture presented in conjunction with the American Osteopathic Association's annual convention, November 15, 1989.*

"Having practiced osteopathy for more than 40 years – having watched the growth of the profession through the years in all of its unfolding and development, in its graduates, its conventions and its publications – I am free to confess that my disappointment in our osteopathic development as an osteopathic profession is indeed very great – we have traded our birthright for a mess of pottage.

We have progress, but not the osteopathic progress that reasonably could have been expected of us. Our schools have grown, and the number of graduates has multiplied, but the kind of graduate of today is very disappointing – he does not rely on his knowledge of osteopathy to cure his patients. He gives internal medicine to such an extent that many of his patients do not know that he is an osteopathic graduate; he has such an inferiority complex that he is ashamed to be known as an osteopathic physician and wants to be known as a physician and surgeon. Is this the kind of growth we have expected of our graduates? Are we satisfied with this kind of progress?" These insightful, challenging and disturbing comments were made by Orren E. Smith, DO, in the 1947 AAO Yearbook. Forty-two years ago he lamented what he saw as the decline of osteopathy within the profession.

How did the profession find itself in such difficulty? It is quite easy for one to point a finger at one or more of the elements of the profession and say that they are to blame.

...One is left with perception that there is clearly enough blame to be shared by all of us within the profession. In the words of the immortal bard, Pogo, "We have met the enemy, and they are us". Each of us share in the blame for the condition we find ourselves in today, and each of us must contribute to the solution if the profession is to survive into the twenty-first century.

It seems to me that each individual osteopathic physician, each osteopathic hospital and each osteopathic organization can share the blame for our problems of today. It also seems to me that each individual practitioner, each hospital and each organization can contribute to the solutions to our problems if we apply a basic osteopathic approach to the solutions. We all must function as "osteopathic" practitioners so that our organizational structures will truly reflect osteopathic health care and osteopathic public policy. I believe the osteopathic profession has the inherent capacity for self-regulation that has allowed it to meet the challenges of the past and to adapt to changes in the future, if we demonstrate the will to do so. As we approach the twenty-first century, the challenges to the health care delivery system in our society are tremendous. We must find ways to harness the burgeoning and costly highly sophisticated technological aspects of medicine control overall health costs provide access to quality health care to all of our citizens and, assure a personalized form of health care which our public seems to desire. The osteopathic profession has many things to contribute to the solution of these problems, particularly in the area of primary care, holistic care and the role of the musculoskeletal system in health and disease. We have an opportunity to influence the majority school of medicine and health policy makers at multiple levels about the valuable things of the osteopathic experience of the last century. Andrew Taylor Still formed the osteopathic profession as a reform school of medicine to meet the challenges of his day. I would submit to you that there still is need for reform in our health care delivery system, albeit different from Still's day, but it is certainly worth pursuing. If we, as members of the osteopathic profession, support the basic tenets of osteopathic philosophy and its concepts, then we should strive diligently to see that they are a part of all health care. If you will, we should strive to make all physicians "osteopathic" physicians. To do less denies the heritage of Andrew Taylor Still. Let us all become part of the solution, rather than part of the problem, so that 40 years hence another lecturer does not need to revisit "Who is to blame"?❑

From the AOBSPOMM Files

AAO Case History
Immune System

by Elaine M. Wallace, DO

Introductory Statement:

This case outlines the vital role that manipulative therapy has upon the immune system. This patient had HIV infection diagnosed in 1987 and has been consistently healthy through a combination of exercise and manipulative therapy.

Identification: N.R. is a 38 year old black male

Chief Complaint: "I need to deal with my positive HIV test"

History of Present Illness:

Patient found out a past lover was HIV positive so had himself tested at a free health clinic two months ago. He had the test redone and it was (+) a second time. He has heard I treat HIV patients and is seeking my care.

Medical History: Negative – for cardiac, pulmonary, HEENT, GI, lymphatic, pulmonary, neurological, musculoskeletal systems –Specific negative answers to hepatitis and mononucleosis.

Patient does report previous infections of nonspecific urethritis ("a few"), GC - urethogential and oropharyngeal and syphilis X 1.

Surgical History: negative

Family History:

Father:	Status unknown
Mother:	↑Hypertension, previous breast carcinoma
Grandmother (maternal):	↑ Heart attacks
Grandfather (maternal):	↓ Heart attacks
Grandmother (Paternal):	↓ Heart attack
Grandfather (Paternal):	↓ Stroke
2 Brothers:	one older-Hyperthyroidism one younger-healthy
1 Sister:	healthy

Social History: N.R. is a city government employee whose job is reading gas meters. He describes it as low stress. He sets his own hours and gets exercise (job related) daily. He has performed this job for six years.

N.R. does not smoke and drinks alcohol rarely. He has a past drug history (described as recreational) that includes; "poppers", cocaine (snorting), marijuana and quaaludes. He describes this activity to have been social and confirmed to his teens and early 20s. Incidence - weekly to 2 x week - variable. He presently does no drugs and has not for 4 years. This drug termination was self imposed.

N.R. describes himself as bisexual. He presently is involved in a stable homosexual relationship (4 years duration) with a married (although separated) male. They live in separate domicles.

N.R. also reports an active (x 4 years) in his mid-twenties when he was a male hooker. His partners were predominantly males - contacts made on the streets or in parks. None of the activities at that time were performed with condoms. His present partner has not been tested. They are practicing safe sex.

N.R. exercises daily (jogs 5 miles) and teaches aerobic exercise 8 hours/ week.

Allergies: No known drug or food allergies. Reports mild lactose intolerance.

Medications: 2 multiple vitamins/ day; 3000 mg. Vitamin C/day;2 B Complex/day

Review of Symptoms: Patient has no specific complaints or symptoms.

He specifically denies hot flashes, night sweats, diarrhea, easy bruiseability, rashes, weight loss or rectal bleeding.

Physical Examination:

Blood Pressure	118/70
Pulse	76
Height	6'1"
Weight	200 lbs

Body type: Mesomorph

Skin: Cool and dry with no appreciable dermatologic lesions.

Head/Neck:

Head: Normocedphalic

Neck: Supple with no apparent masses. No cervical or clavicular adenopathy appreciated.

Thyroid: Of normal size with no palpable masses.

EET Examination: Demonstrates normal ocular examination. Tympanic membranes clear to visualization with good light reflex. Dentation grossly intact with dental work appreciated in inferior posterior molars. There are no appreciable mucous membrane lesions in the mouth. There are no bruits auscultated in the caroltid region.

Heart: Rhythm and rate are regular without murmurs. Apical beat appreciated at intercostal space 5 on left.

Lungs: Clear to auscultation in all fields.

Breast Exam: Appropriate for male examination and negative for masses.

Axilla bilaterally demonstrates no anterior or posterior adenopathy.

Abdomen: Exam reveals firm musculature with no guarding or tenderness to palpation.

Liver: margin is normal.
Spleen: Small although palpable
Kidneys: normal - no organomegaly or masses appreciated

Extremities/BV: Both arms demonstrate slight valgus presentation at elbows. Normal arterial blood supply and venous blood supply appreciated in upper and lower extremities. Good tendon reflexes appreciated in upper and lower extremities (+2/+4).

Gait: Patient has a normal gait without antalgic steps. Weight bearing appears appropriate and mid-line. Patient absorbs weight in heel and transfer weight appropriately from lateral foot to hallux.

Biomechanical: (Structural)

Spinal: Patient shows extremely well developed musculature in all areas of body. There is a normal, although decreased cervical lordosis, a normal kyphosis and a decrease in lumbar lordosis. All ranges of motion in gross evaluation of the cervical, thoracic and lumbar areas are normal. There is no gross structural asymmetry noted.

Patient demonstrates mid-thoracic muscular tension at the areas of the rhomboids bilaterally.

There is an accompanying T7 & 8 somatic dysfunction rotated (R) sidebent (R), held in flexion.

Pelvis: There is a decreased respiratory motion noted in the sacrum with the sacral base held posterior (sacral extension-biomechanical/ sacral flexion-cranial). Bilateral anterior rotation of the ilium with slight inflaring bilaterally.

Cranial: Cranial rhythm is appreciated and evaluated to be slow (8 impulses per minute). The occiput is held in slight extension.

X-Rays: not applicable.

Laboratory:

	Normal	'87	'89	'90
Total T cells	65-95	77	85	76
Mature T cells	55-85	69	78	68
Helper T cells	32-55	31	29	32
Suppressor T cells	14-38	32	44	43
Helper/ Suppressor ratio	1.1-2.55	.97	.66	.74

Diagnosis:

1. Healthy black male positive HIV laboratory test.
2. Somatic dysfunction of sacrum-bilateral sacral extension.
3. Somatic dysfunction of ilium-bilateral iliac rotation in inflare.
4. Somatic dysfunction of thorax-T7 & 8, FR(R) SB(R)
5. Slow-Normal cranial rhythm.
6. Cranial base in extension

Treatment Planned: It was recommended to this patient that proper diet, exercise and manipulative threatment would be appropriate in his life planning-therapy for HIV infection.

Course of Therapy: This patient receives regular osteopathic manipulative threatments on a two week basis. His manipulative threatment includes treatment of specific somatic dysfunctions as evaluated at the time of visit. For the patient's initial evaluation, high velocity-low amplitude thrusting was performed at the thoracic region. Soft tissue was performed at all muscular tension sites. Muscular energy was performed on the sacrum and the ilium. Balancing of membranous tension was performed in the cranium.

In addition to treatment of specific somatic dysfunction this patient undergoes a complete treatment protocol of lymphatic techniques. The lumphatic treatment begins with thoracic pump techniques (repetitive pump) followed by pectoral traction, rib raising, doming of the diaphragm, abdominal pump, effleurage and petrissage of all extremities. This is followed by complete cervical and cranial lymphatic treatment including anterior and posterior cervical lymph node stroking, occipital node stroking, pre and post auricular node stroking, Galbraith's Technique (raking the face), submandibular node treatment, anterior cervical chain treatment, supra and infra hyoid node treatment, and repetition of the thoracic pump (both repetitive and vacuum type). This patient also receives pedal pump at the time of repeat thoracic pump. Special attention is also given to first rib dysfunctions and release of the superior thoracic inlet.

As part of this patient's exercise protocol, this patient continues to jog 5 miles per night, lifts weights in a balance directed manner and continues to teach aerobics on a weekly basis.

His dietary supplement continues to include the above mentioned vitamins as well as anti-oxidants. He takes no medication other than the above mentioned vitamins.

Prognosis: Stable

Discussion: This case demonstrates the efficacy of osteopathic manipulative treatment when used on a regular basis in the ancillary treatment of HIV infection. This patient's exercise regime, as well as structured manipulative threatment regime insures maximal functioning of the lymphatic system. This patient has been stable in his HIV infection since his original diagnosis in 1987.

Plan of Therapy:

This patient will continue to be seen by me on a regular basis for manipulative threatment for the remainder of his adult life. ❑

Goes Platinum in

Music City USA

American Academy of Osteopathy

1995 Annual Convocation

March 22-25, 1995

Opryland Hotel Nashville, Tennessee

Program

Wednesday, March 22, 1995

7:45 - 8:00	Welcome Ann Habenicht, DO, Program Chairperson Eileen DiGiovanna, DO, FAAO, AAO President
8:00 - 8:45	Neuroanatomy of Pain Frank Willard, PhD
8:45 - 9:30	Trauma Vectors Judith O'Connell, DO
9:30 -10:00	**Break/Exhibits**
10:00 -10:45	Physiatrist's Role in Chronic Pain James Lipton, DO, FAAO
10:45 -11:30	Reflex Sympathetic Dystrophy & Sympathetic Dystonia Robert Kappler, DO, FAAO
11:30 -12:00	Psychiatric Aspects of Chronic Pain Andrew Lovy, DO
12:00 - 1:30	**Lunch**
1:30 - 3:00	**Workshops** A-Back to Basics OMT: HVLA for Chronic Pain Robert Kappler, DO, FAAO B-Back to Basics OMT: Facilitated Positional Release Stanley Schiowitz, DO, FAAO C-Back to Basics OMT: Ligamentous Articular Release M. Denise Speed, DO and Conrad Speece, DO D-Torque Unwind (runs entire PM) Elaine Wallace, DO E-Fellows Forum FAAO and NUFA Harold Magoun, DO, FAAO
3:00 - 3:30	**Break/Exhibits**
3:30 - 5:00	**Workshops** F-Back to Basics OMT: HVLA for Chronic Pain Robert Kappler, DO, FAAO G-Back to Basics OMT: Facilitated Positional Release Stanley Schiowitz, DO, FAAO H-Back to Basics OMT: Ligamentous Articular Release M. Denise Speed, DO and Conrad Speece, DO I-Education Committee Forum Boyd Buser, DO, and AAO Education Committee

New Horizons in Pain Management

Thursday, March 23, 1995

7:45 - 8:00	Morning Convocation Update Ann Habenicht, DO
8:00 - 8:45	Pharmacology in ChronicPain William Elliott, MD, PhD
8:45 - 9:30	Oh, No, Fibromyalgia! Mark Cantieri, DO
9:30 -10:00	**Break/Exhibits**
10:00 -10:45	Chronic Foot and Ankle Pain Thomas Ravin, MD
10:45 -11:30	Discogenic vs. Non-Discogenic Pain Manuel Pinto, MD
11:30 -12:00	Acupuncture in Chronic Pain Kenneth Lubowich, OMD
12:00 - 1:30	**Lunch**
1:30 - 3:00	**Workshops** K-Back to Basics OMT: Muscle Energy Boyd Buser, DO L-Back to Basics OMT: Counterstrain John Glover, DO M-Torque Unwind (runs entire PM) Elaine Wallace, DO N-Treatment of Chronic Foot & Ankle Pain Thomas Ravin, MD O-Faculty Development: The Physician as a Researcher, Documenting Outcomes Research John Hohner, DO
3:00 - 3:30	**Break/Exhibits**
3:30 - 5:00	**Workshops** P-Back to Basics OMT: Muscle Energy Boyd Buser, DO Q-Back to Basics OMT: Counterstrain John Glover, DO R-Treatment of Chronic Foot & Ankle Pain Thomas Ravin, MD S-Faculty Development: How to Represent Osteopathy to Third-Party Payors Judith Lewis, DO & AAO Medical Economics Cmte

Friday, March 24, 1995

7:45 - 8:00	Morning Convocation Update Ann Habenicht, DO
8:00 - 8:45	Nutritional Needs in Chronic Pain Stephen Elsasser, DO
8:45 - 9:30	Reducing Gravitational Strain Pathophysiology Michael Kuchera, DO, FAAO
9:30 -10:00	**Break/Exhibits**
10:00-10:45	Chronic Pelvic Pain Melicien Tettambel, DO, FAAO
10:45-12:00	Exercises for Chronic Pain interactive lecture with audience participation Karen Gadja, DO
12:00-12:30	New Ideas Forum (two 15-minute presentation time slots available; prospective presenters must submit outlines to the EDCOM for selection)

Saturday, March 25, 1995

7:45 - 8:00	Morning Convocation Update Ann Habenicht, DO
8:00 - 8:45	Anesthesia's Role in Chronic Pain Management Larry Harker, DO
8:45 - 9:30	Migraine Cephalgia Hal Pineless, DO
9:30 -10:00	**Break/Exhibits**
10:00 -10:45	Facial Pain: Bell's Palsy & Trigeminal Neuralgia William Wyatt, DO
10:45 -11:30	Chronic Cervical Spine Pain Karen Steele, DO
1:00 - 5:00	Conclave of Fellows

Ann Habenicht, DO,
Program Chairperson

CME Hours:
25 Hours Category 1-A

continued from page 12

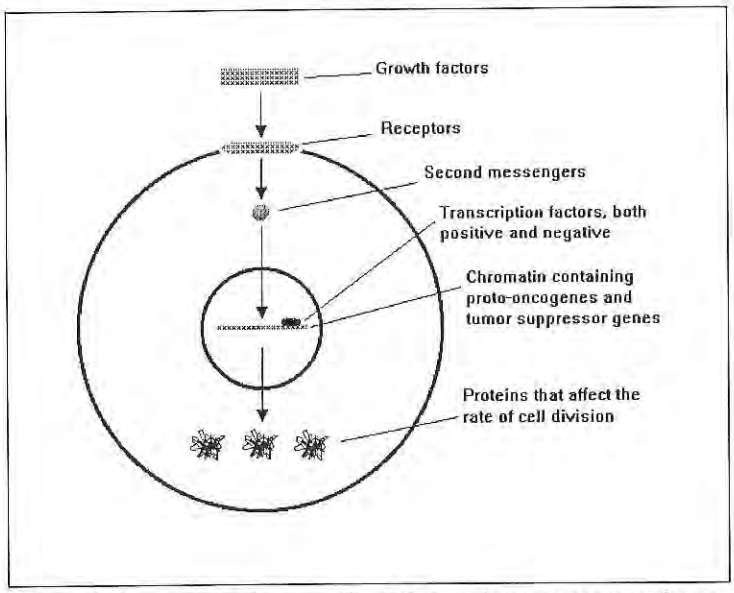

Figure 1. *Componets of a normal cell that are important in transformation, first, to a normal cell and then to a neoplasm.*

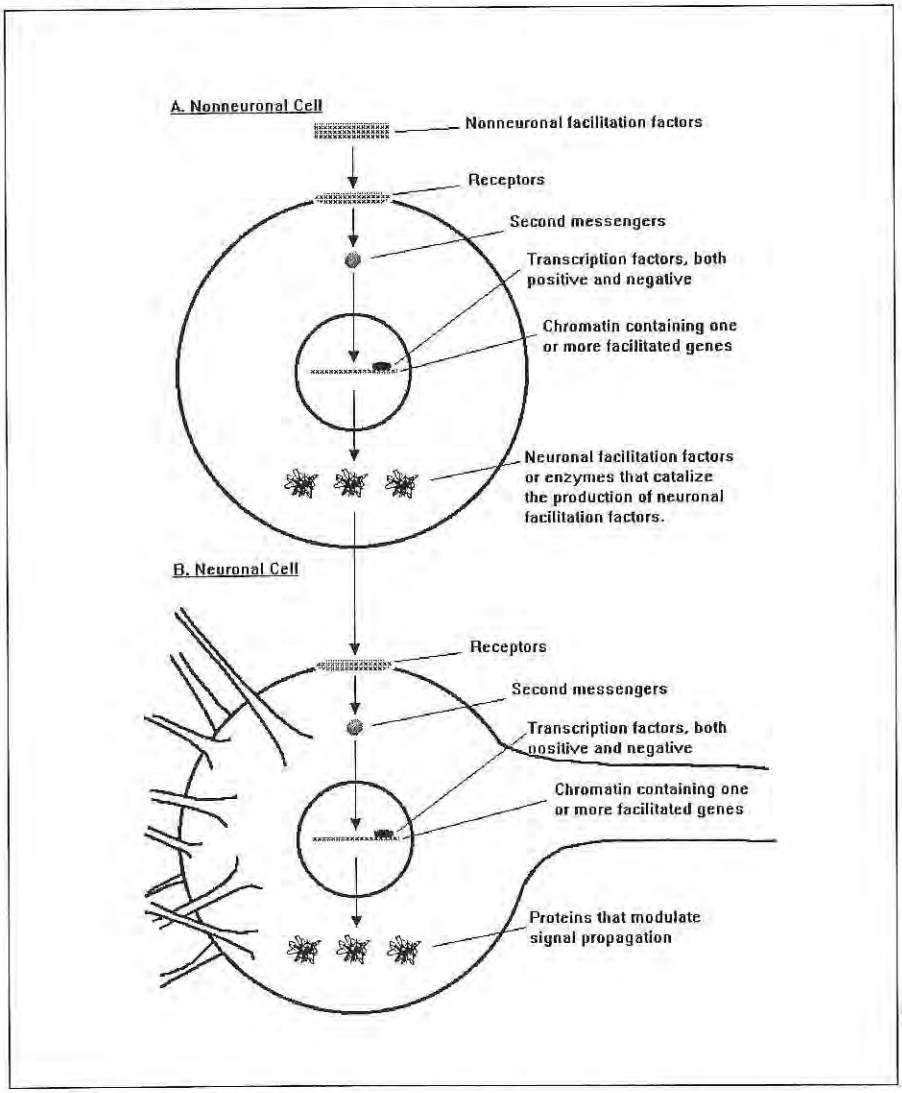

Figure 2. Example showing the case where both nonneuronal and neuronal cells have at least one facilitated gene.

Corollary #1:

People are predisposed to develop specific facilitated segments.

Some people inherit a predisposition to cancer but most cancers require several somatic mutations; the exact number of mutations is not known. However, colon cancer contains between 4 to 7 mutations and the initial tumorigenesis requires at least one oncogene and the loss of both alleles of a tumor suppressor gene.[40,41]

If facilitated genes contribute to producing a facilitated segment, some individuals would be genetically predisposed to develop facilitated segments. Consider two people exposed to the same environmental circumstances. One individual would display the phenotype of somatic dysfunction because of the existence of a certain set of facilitated genes. The other individual would not develop symptoms because of a lack of a set of facilitated genes.

Furthermore, since most somatic mutations would be expected to be limited to certain areas of the organism, only those areas of the organism containing facilitated genes would be predisposed to develop a facilitated segment. Thus, each individual would have a unique set of predisposed segments, and be predisposed to develop specific facilitated segments.

Corollary #2:

Somatic dysfunctions presenting in a similar manner may be due to different sets of physiological changes.

The hypothesis also predicts that individuals with a somatic dysfunction affecting the same region may have different sets of facilitated genes. Most specific types of cancers have been shown to contain different sets of oncogenes and tumor suppressor genes. What is diagnosed as colon cancer is actually a large set of colon cancers as judged by the different sets of oncogenes and tumor suppressor genes present in each individual. For example, the *ras* oncogene is found in 40 to 50 percent of all colon cancers.[42] This means that it is missing in 50 to 60 percent of all colon cancers. Several tumor suppressor genes are absent in many cases but not all cases of colono-rectal cancer. The MCC (mutated in colono-rectal cancer) gene on chromosome #5 is often absent. The DCC (deleted in colon cancer) gene is absent 70 percent of the time on chromosome #18 and the p53 tumor suppressor gene is absent 75 percent of the time on chromosome #17. Only 40 percent of the colon carcinomas have mutations in 3 of the 4 genes just mentioned.[8] By analogy, anatomically similar facilitated segments in two individuals may be due to different sets of facilitated genes with their own unique pathology.

Corollary #3:

Somatic dysfunction will not respond to a single intervention.

The cell biology of cancer and our hypothesis offers another corollary. Historically, researchers and clinicians have looked for a magic bullet to cure a single process gone awry. Now cancer is understood in terms of multiple genes and processes. The oncogenes and the sequence of their appearance are not identical from one type of cancer to the next. Also the cellular environments supporting different types of cancer vary greatly. It seems likely that what physicians call a facilitated segment will turn out to be many types of facilitated segments. Each of these facilitated segments may, in turn, be facilitated in a variety of ways and by a variety of cells. At present, the problem appears quite complex. However, as complex as cancer appeared to be, it is now better understood in terms of oncogenes, tumor suppressor genes, and multiple growth factors. It may be that the eventual identification of facilitated genes and cells will expand our understanding of the facilitated segment and direct us toward more specific therapeutic interventions.

Corollary #4:

The alterations causing a facilitated segment may be permanent and persist for the lifetime of the individual.

One of the most obvious implications of the hypothesis is that the underlying conditions, i.e. genetic changes, may be permanent. This does not bode well for the efficacy of therapy. In fact, should support for this hypothesis be forthcoming, it would alter the way we approach somatic dysfunction. The possibility that lesions may become permanent should lead osteopathic physicians and researchers to focus more on preventive measures than symptom related therapy.

Summary

A hypothesis has been presented that the physiological mechanisms involved in the formation of a facilitated segment may have features in common with the molecular mechanisms responsible for the development of cancer. This synthesis has been developed as the result of an increased understanding of the common mechanisms of cell function. We have derived a number of clinically relevant corollaries from this hypothesis. It is now incumbent upon our laboratory and others to develop evidence to either support or reject this hypothesis. In either case, the knowledge derived will be valuable for our further understanding of the facilitated segment.

References

1. Denslow JS: An analysis of the variability of spinal reflex thresholds. J. Neurophysiol, 1944;7:207-15.

2. Denslow JS: An analysis of irritability of spinal reflex arcs. JAOA, 1945;4:357-62.

3. Denslow JS, Korr IM, Krems AD: Quantitative studies of chronic facilitation in human motoneuron pools. Amer J Physiol, 1947;150:20-38.

4. Korr IM, Wright HM, Chace JA: Cutaneous patterns of sympathetic activity in clinical abnormalities of the musculoskeletal system. Acta Neurovegetativa, 1964;25(4):589-606.

5. Korr IM, Wright HM, Thomas PE: Effects of experimental myofascial insults on cutaneous patterns of sympathetic activity in man. Acta Neurovegetativa, 1962;23(3):329-355.

6. Van Buskirk, RL: Nociceptive reflexes and the somatic dysfunction: a model. JAOA, 1990;90;792-809.

7. Richardson GE, Johnson BE: The Biology of Lung Cancer, Seminars in Oncology 1993;20(2):105-127.

8. Story, MT: Polypeptide Modulators of Prostatic Growth and Development. Cancer Surveys 1991;11:123-146.

9. Fearon ER, Jones PA: Progressing toward a molecular description of colorectal cancer development. FASEB J, 1992;6:2783-2790.

10. McMahon SB, Monroe JG: Role of primary response genes in generating cellular responses to growth factors. FASEB J 1992;6:2707-2715.

11. Coffey RJ, McCutchen CM, Graves-

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Deal R, Polk WH Jr. J Cell Biochem, Suppl 16G, 1992;111-118.

12. Aaronson SA, Tronick SR: Growth factors, in Holland JF, Frei E III, Bast RC Jr. et al. (eds) Cancer Medicine, 3rd Ed, Philadelphia, Pa, 1993;1:33-48.

13. Akbasak A, Sunar-Akbasak B: Oncogenes:cause or consequence in the development of glial tumors. J Neurol Sci 1992;111:119-133.

14. Hunt SP, Pini A, Evan G, Induction of c-fos-like protein in spinal chord neurons following sensory stimulation. Nature 1987;328:632-634.

15. Menetrey D, Gannon A, Levine JD, Basbaum AI: The expression of c-fos protein in presumed nociceptive interneurons and projection neurons of the rat spinal chord: Anatomical mapping of the central effects of noxious somatic, articular and visceral stimulation. J Comp Neurol 1989;285:177-195.

16. Carbone DP, Minna JD: Antioncogenes and Human Cancer. Annu Rev Med 1993;44:451-64.

17. Cho KR, Vogelstein B: Genetic Alterations in the AdenomaCarcinoma Sequence. Cancer 1992;70:1727-31.

18. Westermark B, Heldin C-H: Platelet-derived growth factor in autocrine transformation. Cancer Research 1991;51:5087-92

19. Henderson BE, Bernstein L, Ross RK: Hormones and the Etiology of Cancer, in Holland JF, Frei E III, Bast RC Jr. et al (eds) Cancer Medicine 3rd Ed, Philadelphia, Pa, 1993;1:223-232.

20. Dickson RB and Lippman ME: Molecular Determinants of Growth, Angiogenesis, and Metastases in Breast Cancer. Seminars in Oncology, 1992;19(3):286-298.

21. Weinstein IB: The Origins of Human Cancer:Molecular Mechanisms of Carcinogenesis and Their Implications for Cancer Prevention and Treatment. Cancer Research 1988;48:4135-4143.

22. Moore RW, Mokler DJ, Winterson BJ: Intrathecally administered N-methyl-D-aspartate increases persistent hindlimb flexion in rat. Neuroscience Letters 1992;146:223-226.

23. Abe K, Xie F, Saito H: Epidermal growth factor enhances short-term potentiation and facilitates induction of long-term potentiation in rat hippocampal slices. Brain Res 1991;547:171-174.

24. Hennings H, Glick AB et al.: Critical aspects of initiation, promotion, and progression in multistage epidermal carcinogenesis. Proc Soc Exp Biol Med 1993;202(1):1-8.

25. Radeke HH and Resch K: The inflammatory function of renal glomerular mesangial cells and their interaction with the cellular immune system. Clin Investig 1992;70:825-842.

26. Wilkin GP, Marriott DR, Cholewinski AJ et al. Receptor activation and its biochemical consequences in astrocytes. Ann NY Acad Sci 1991;633:475-88.

27. Evans T, McCarthy KD, Harden TK: Regulation of cyclic AMP accumulation by peptide hormone receptors in immunocytochemically defined astroglial cells. J Neurochem 1984;43:131-138.

28. Kimura H, Okamoto K, Sakai Y: Modulatory effects of prostaglandin D2, E2 and F2a on the postsynaptic actions of inhibitory and excitatory amino acids in cerebellar purkinje cell dendrites in vitro. Brain Res 1985;330:235-244.

29. Murphy S, Pearce B et al.:Astrocytes as eicosanoid-producing cells. GLIA 1988;1:241-245.

30. Clemens MJ: Cytokines, in Read AP, Brown T, (eds): Medical Perspectives Series, BIOS Scientific Publishers Ltd., Oxford UK, 1991.

31. Malipiero W, Frei K, Fontana A: Production of hemopoietic colony-stimulating factors by astrocytes. J Immunol 1990; 144(10):3816-3821.

32. Lieberman AP, Pitha P et al.: Production of tumor necrosis factor and other cytokines by astrocytes stimulated with lipopoly-saccharide or a neurotropic virus. Proc Natl Acad Sci USA 1989;86:6348-52.

33. Farrar WL, Kilian PL, et al.: Visualization and characterization of interleukin 1 receptors in brain. J Immunol 1987;139(2):459-63.

34. Merril JE: Tumor necrosis factor alpha, interleukin 1 and related cytokines in brain development:Normal and Pathological. Dev Neurosci 1992;14:1-10.

35. Prados MD, Wilson CB: Neoplasms of the central nervous system, in Holland JF, Frei E III, Bast RC Jr. et al (eds) Cancer Medicine, 3rd Ed, Philadelphia, Pa, 1993;1:1080-1119.

36. Reynolds BA, Tetzlaff W, Weiss W: A multipotent EGFresponsive striatal embryonic progenitor cell produces neurons and astrocytes. J Neurosci 1992;12(11):4565-74.

37. Pons S, Torres A: Basic fibroblast growth factor modulates insulin-like growth factor-I, its receptor, and its binding proteins in hypothalamic cell cultures. Endocrinology 1992;131(5):2271-78.

38. Smits A, Kato M, Westermark B, et al.: Neurotrophic activity of platelet-derived growth factor (PDGF):Rat neuronal cells possess functional PDGF beta-type receptors and respond to PDGF. Proc Natl Acad Sci USA 1991;88(18):8159-63.

39. Ota A, Shen-Orr Z, et al.: TPA-induced neurite formation in a neuroblastoma cell line (SH-SY5Y) is associated with increased IGF-I receptor mRNA and binding. Molecular Brain Research 1989;6:69-76

40. Cho KR, Vogelstein B: Genetic alterations in the adenomacarcinoma sequence. Cancer Supplement, American Cancer Society, J.B. Lippincott, Philadelphia, 1992;70(6):1727-31.

41. Stein WDL Analysis of cancer incidence data on the basis of multistage and clonal growth models, in Holland JF, Frei E III, Bast RC Jr. etal. (eds) Cancer Medicine 3rd Ed, Philadelphia, Pa, 1993; 1:1080-1119.

42. Klein G: Oncogenes, in Holland JF, Frei E III, Bast RC Jr. et al. (eds) Cancer Medicine, 3rd Ed, Philadelphia, Pa, 1993;1:65-77.❑

continued from page 18

9) Use Triggerband Technique. Begin by going to the 'Crossband'* of the most painful triggerband.
10) Treat other affected bands.
11) On the second or third treatment consider OMT or other modalities.
12) Give home instructions of *ice,* **no heat**, and other appropriate activities. In chronic pain four days of rest are usually needed in between treatments. In acute pain the patient can usually be retreated in 24 to 48 hours.
13) Answer any patient questions.
14) Record and evaluate progress in the chart by using both subjective and objective criteria.

Clinical Examples of Commonly Seen Triggerbands

Two examples of commonly seen Triggerbands are shown in Figures 8 and 9. Fascial shoulder injuries and their treatments are then discussed in the final portion of this paper. Before treating any fascial distortion first check and record abduction, internal and external rotation, flexion and extension or other motions of the affected area, and then recheck each of these motions at the end of treatment. This demonstrates to the patient objective improvement, so he or she can appreciate your work. Failure to respond means either the diagnosis was wrong, or the treatments were not forceful enough. A complete reevaluation should be done on any atient that does not respond.

Triggerband Technique requires certain palpatory skills that take some time to develop. Be sure to allow the patient to guide your treatment. If you are unsure of where the triggerband is, ask, "Am I on it?" In a short time, with a little practice and experience,

'Star' Triggerband Pathway for Upper Thoracic Pain

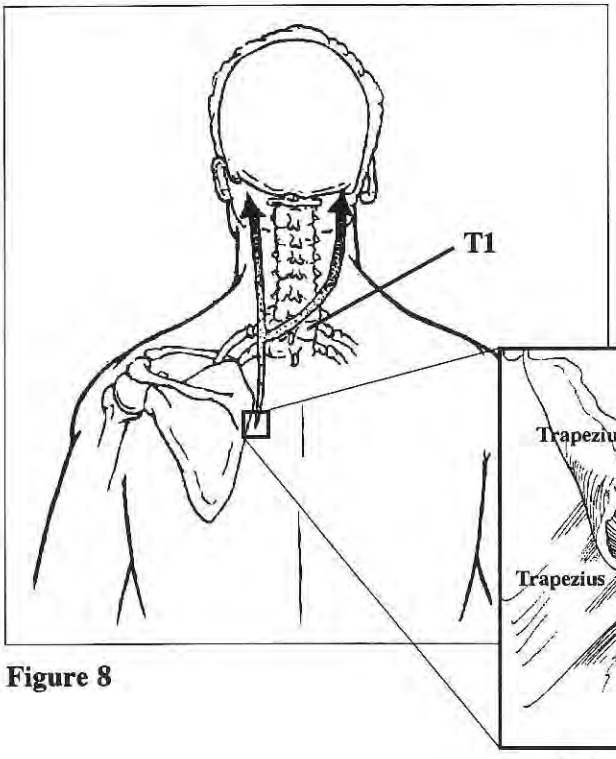


Figure 8

Pathway for Lower Back Pain with Posterior Thigh Tightness

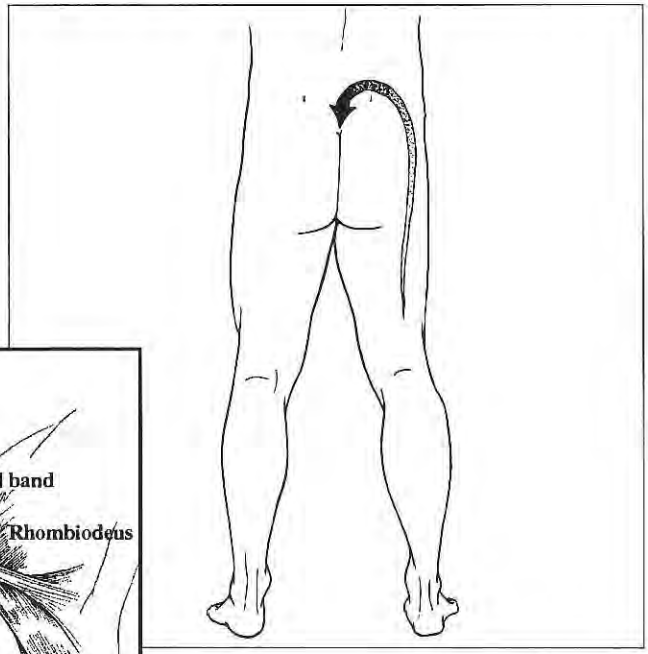
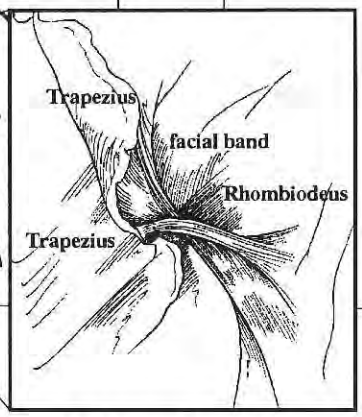


Figure 9



Cadaver Dissection

*Crossbands are the anatomical starting place of triggerbands. They are typically strong fasical fibers that are found in the same plane and at an angle to the triggerband. In figures 8, 9, 11 and 12, the crossbands are present where thearrows originate.

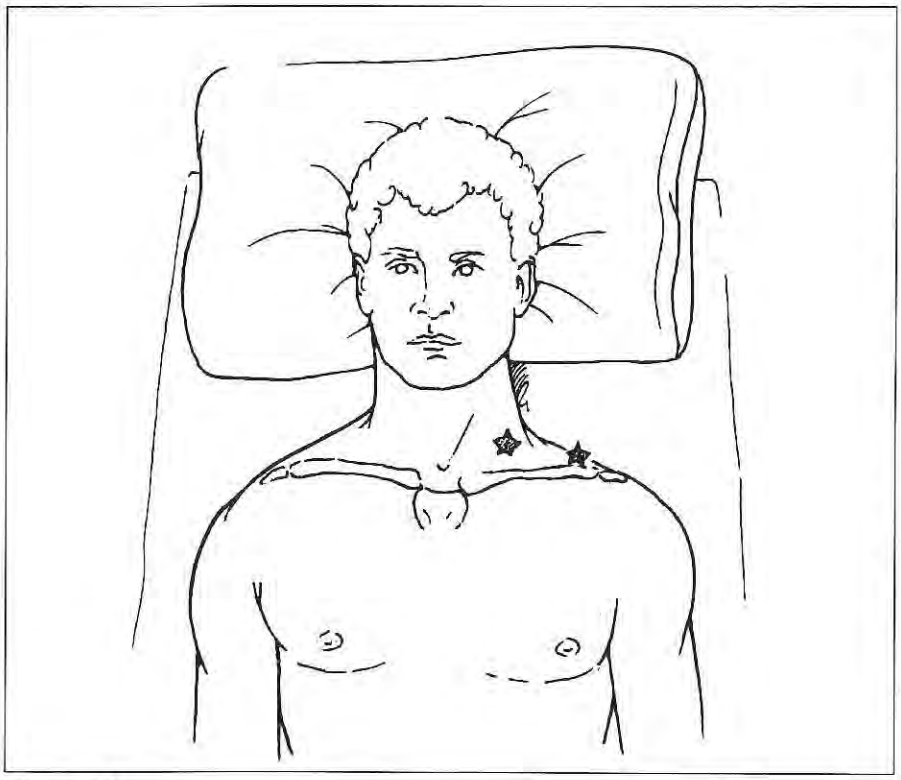


Figure 10

Triggerband Technique can be easily done with a minimal amount of time involved.

Treatment of the Injured Shoulder

Before and after each step check abduction, and internal and external rotation.

Step I: The supraclavicular triggerpoints are shown in **Figure 10** and should be treated in all shoulder pain patients. The medial is far more important than the lateral. Treat the medial first by palpating at the base of the neck between the clavicle and scapula. The amount of tension varies widely. Find the triggerpoint by feeling for an area that feels like a 'boggy marble'. Use firm pressure and push the triggerpoint in a downward and slightly medial direction. Gently 'milk' it as the pressure variant seems to change. Follow the maximum 'bogginess' and hold it firmly. After 15 seconds to 3 minutes (with an average of 1.5 minutes) the triggerpoint will begin to release. This release is dramatic but gradual. It may take as long as one-half minute for the complete release. Hold it with constant or increasing pressure, and 'milk' it until it has completed its entire release. The patient will have a strong sensation of this release as well. The lateral supraclavicular triggerpoint is more difficult to treat and is less important. For most beginners, treatment of the lateral supraclavicular triggerpoint should be skipped.

Step II: Triggerband Technique is the next portion of the treatment. If the pain is anterior in the shoulder along the biceps groove, then triggerband technique is done by using the anterior shoulder pathway. Refer to **Figure 11**. First, find a tender area in the anterior proximal lateral forearm. Then feel for an irregularity in the surrounding fascia. Once this is found, forcefully push it superiorly toward the shoulder. This tender area will move up the forearm, then up the arm and into the bicipital tendon area. Here it will be the most painful. Once it passes the biceps area it will continue to move into the supraclavicular fossa or along the clavicle. Then it will pass up the neck along the margins of the sternocleidomastoid muscle up to the mastoid where it terminates.

Step III: After the anterior shoulder pathway is completed, check internal rotation. Many times it is normalized. If not, ask the patient again where the pain is. If he/she specifies the pain is still in the biceps groove area, then repeat the above **more forcefully.** If instead the pain is more superiorly on the shoulder or more posteriorly, then the posterior shoulder pathway needs to be done. See **Figure 12**. This triggerband begins more laterally and is on the posterior surface of the proximal forearm. It is treated in the same manner as the anterior shoulder triggerband pathway except that it passes along the lateral arm and into the upper margins of the trapezius muscle to the base of the neck. Then it crosses over at T^1 to the opposite side and moves up the neck along the capitus muscle until it terminates at the mastoid. It often becomes buried under the occiput en route to the mastoid. Once this is corrected, check internal rotation again. If it is not normalized ask the patient where the pain is. If it is still in the posterior shoulder area, repeat this triggerband **with more force.**

→

Anterior Shoulder Pathway

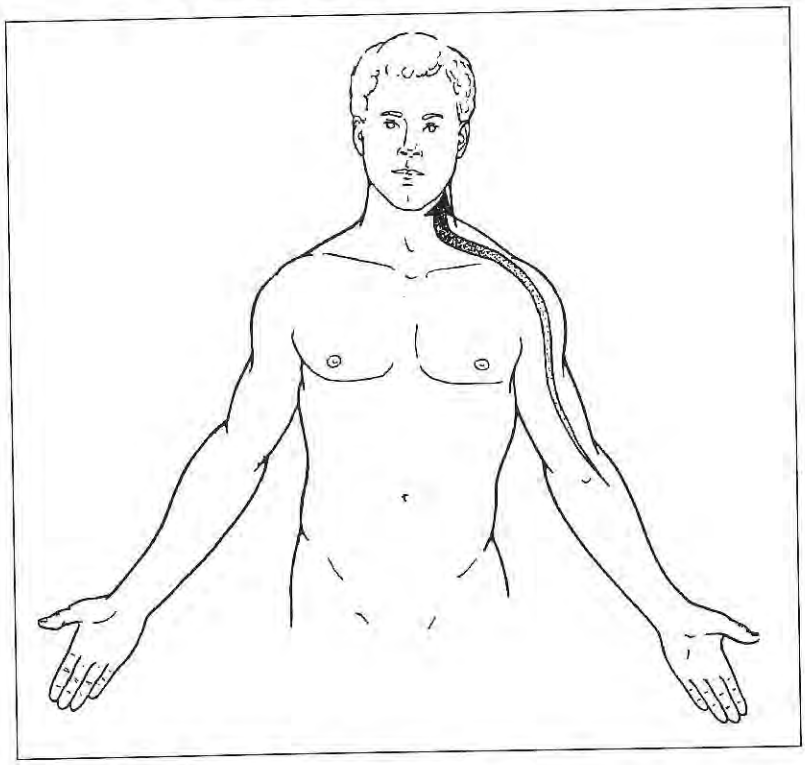


Figure 11

Posterior Shoulder Pathway

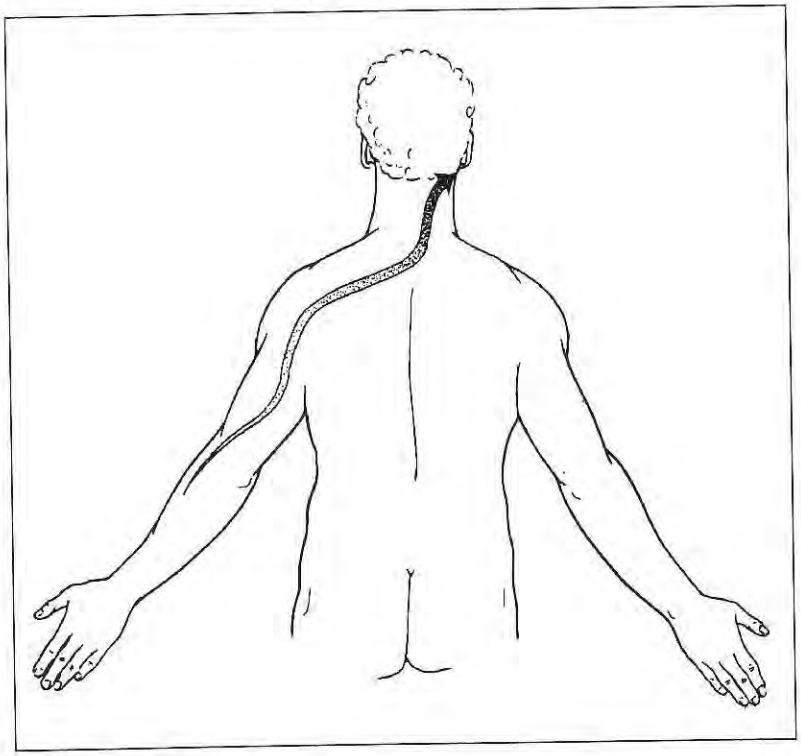


Figure 12

Step IV: Several short triggerbands or continuum distortions* may still be holding the shoulder and keeping it from complete motion. These are commonly found in the positions indicated in **Figures 13a, b, and c**. To treat, first guide the shoulder into the direction of its decreased range of motion and ask the patient to tell you where it hurts. Then while still holding the shoulder in the painful position, place your thumb on the point of maximum pain. Hold that spot until release. Then recheck the range of motion and repeat. Some of these fascial distortions are triggerbands and some of are continuum distortions . If it is a continuum distortion it will release. If it is a triggerband then follow it to the mastoid, where it terminates. The 'star' area often needs to be treated as well. After this step most frozen shoulders are much improved and have normal range of motion.

Step V: Most patients now have normal or close to normal range of motion and have had one, two or three treatments. However, many still feel a slight tightness or tug in the shoulder. These patients should then receive high velocity-low amplitude osteopathic manipulation. First, the thoracic spine should be manipulated in the standing position (hallaluya). Then, thoracic HVLA should be attempted in the chair to correct any lateral fixations. Following this, HVLA in the anterior-posterior direction and the posterioranterior direction to the thoracic spine may be needed.

After HVLA to the thoracic spine, the shoulder should also be

*See accompanying papers, *Continuum Technique* and *Introducing the Fascial Distortion Model.*

manipulated. Have the patient seated pin a chair and stand behind and reach around and grab his/her flexed elbow of the sore shoulder with both palms. As the patient drops the shoulder, manipulate with a quick thrust in the superior-posterior direction. Usually a loud 'crack' or 'pop' is felt or heard. Many times motion is dramatically improved.

Step VI: The patient is rechecked in several days. At times, the entire procedure may need to be repeated. Any shoulder that does not respond should be thoroughly reevaluated. Once corrected, exercises and physical therapy are considerations but are usually unnecessary. At home these patients should practice "dropping" the injured shoulder while watching in the mirror. Ice should be used to reduce the tendemess and any application of heat should be strongly discouraged. ❑

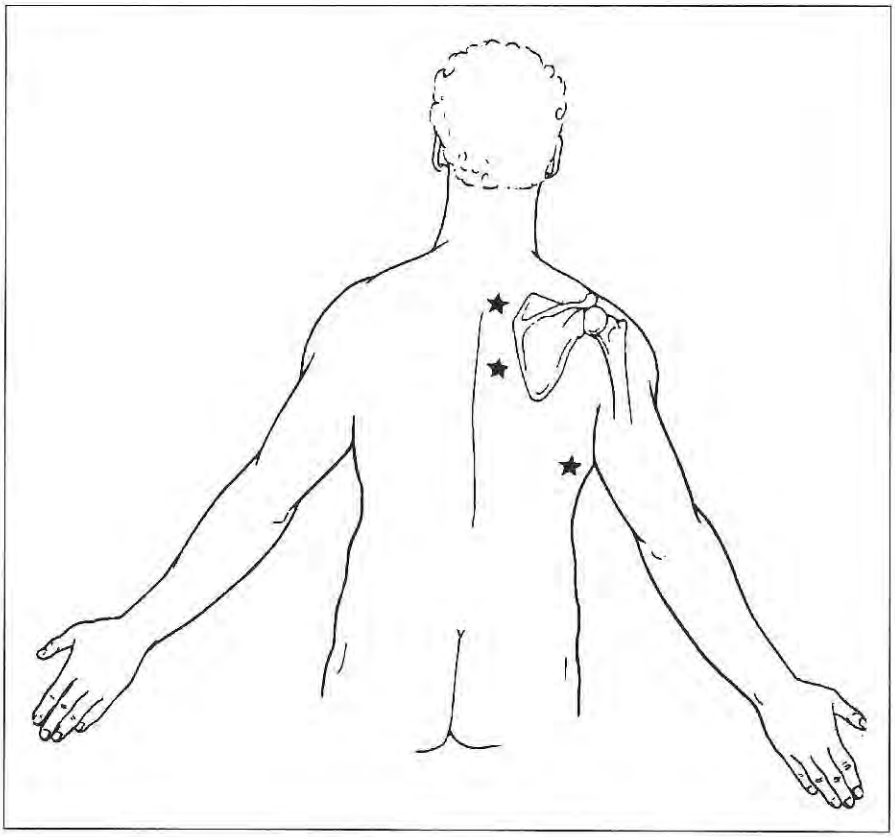


Figure 13a

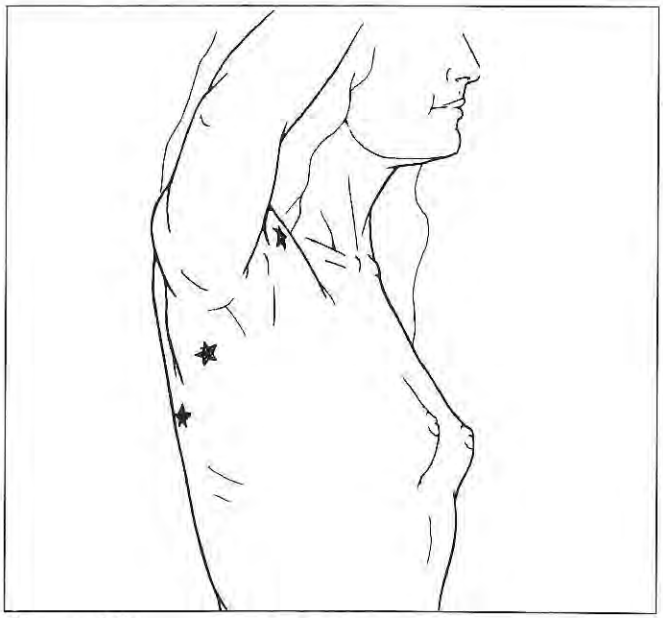


Figure 13b

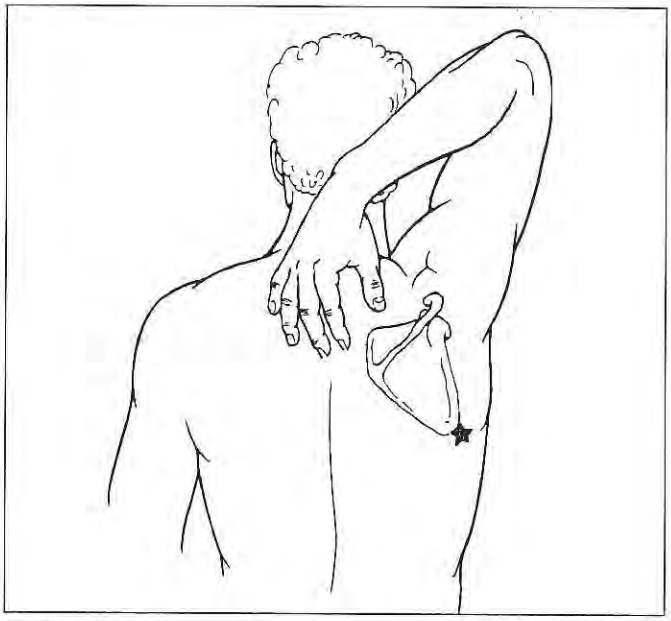


Figure 13c

References

1 Gerlach, U. J., Lierse, W.: Functional construction of the superficial and deep fascia system of the lower limb in man. Acta Anat (Basel) 1990;139(1):11-25.

Classifieds

OMM/ Physical Medicine Physician

to join busy, well established practice in Colorado Springs, Colorado. Partnership or solo option available. Call Vincent Conner at (719) 260-8179.

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Phyiatrist practicing orthopedic medicine (osteopathic techniques, prolotherapy) seeks DO to ultimately take over practice. Large physical therapy department; new medical office building 15 minutes from downtown New Orleans. Contact: Edna Doyle MD, 4224 Houma Boulevard, Suite 470, Metairie, Louisiana, 70006; (504) 456-5160.

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Classified Ad Deadline
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Calendar of Events

JANUARY

28-29
Use of the Advance Percussion Vibrator in Adults or Children (Advance Course)
Indiana Academy of Osteopathy
Location: The Nutritional Center
724 West Bristol Street, Suite A
Elkhart, IN
Hours: 16 Category 1-A
Contact: Max Hostetler, DO
(219) 262-9612

FEBRUARY

3-5
Visceral Manipulation (Basic Course)
Eastmoreland Hospital, Dept of OMM and Northwest Osteopathic Medical Foundation
Portland, Oregon
Hours: 20 Category 1-A
Fee: $350 (space limited)
Course Director: Daniel Benksy, DO
Contact: Al Turner, DO
Director, OMM Dept
Eastmoreland Hospital
(503) 230-2501

11-12
Winter OMT Update
"Application of Osteopathic Concepts in Clinical Medicine" and preparation for OMM Boards
Adams Mark Hotel
Indianapolis, Indiana
CME: 18 Hours, Category 1-A
Contact: Diana Finley, AAO
Associate Executive Director
(317) 879-1881

18-19
Use of the Advance Percussion Vibrator in Adults or Children (Advance Course)
Indiana Academy of Osteopathy
Location: The Nutritional Center
724 West Bristol Street, Suite A
Elkhart, IN
Hours: 16 Category 1-A
Contact: Max Hostetler, DO
(219) 262-9612

25-26
Ski & CME Midwinter Conference
Colorado Society of Osteopathic Medicine
Keystone Lodge & Resort
Keystone, Colorado
Contact: Patricia Ellis
(303) 322-1752

MARCH

1-5
34th Annual Convention & Scientific Exhibit Show of the Osteopathic
Physicians and Surgeons of California
Anaheim Hilton and Towers
Anaheim, California
Hours: 46 Category 1-A
Contact: Kimberley Bauer
OPSC
(916) 447-2004

9-12
92nd Annual Convention
Florida Osteopathic Medical Assn
Doral Ocean Beach Resort
Miami Beach, Florida
Hours: 30 Category 1-A
Contact: FOMA
(904) 878-7364

22-25
AAO Annual Convocation
Program: New Horizons in Pain Management
Opryland Hotel
Nashville, Tennessee
Contact: Diana Finley, AAO
Associate Executive Director
(317) 879-1881

APRIL

22-23
Sutherland's Methods for Treating the Rest of the Body
Hours: 16 Category 1-A
Contact: Conrad Speece, DO
10622 Garland Road
Dallas, TX 76218
(214) 321-2673

28-30
AAO Muscle Energy Course
Ramada Plaza Hotel
Indianapolis, Indiana
CME Hours: 20 Category 1-A
Contact: Diana Finley, AAO
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The Academy Invites its Component Societies to send in their course schedules to be published in the AAO Journals and the AAO Newsletters.

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"Functional Methods: A Manual for Palpatory Skill Development in Osteopathic Examination and Manipulation of Motor Function

by William J. Johnston, DO, FAAO and Harry D. Friedman, DO

This manual will be a valuable text for any osteopathic physician from which he/she could learn and use this functional method in the practice of osteopathic medicine.

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"The text includes and explains many of the concepts of the osteopathic profession and explains advantages of manipulation in an honest, matter-of-fact, non-threatening manner. It should make non-users wonder why they aren't cashing in on the advantages of providing manipulation for improved patient care."

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Co-author of *Osteopathic Principles in Practice*

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