NINCDS Monograph No. 15

The Research Status of Spinal Manipulative Therapy

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service National Institutes of Health

NINCDS Monograph Series

The NINCDS Monograph Series, edited and issued by the Office of Scientific and Health Reports, National Institute of Neurological and Communicative Disorders and Stroke, presents contributions to knowledge in the neurological and sensory disease fields, particularly material that is extensive, detailed, or specialized.

The following publications in this series are available from the NINCDS Office of Scientific and Health Reports:

NINDS Monograph No. 1: Epilepsy—A Review of Basic and Clinical Research. 69 pp., 1965.

NINDS Monograph No. 3: Minimal Brain Dysfunction in Children, Terminology and Identification, 18 pp., 1966.

NINDS Monograph No. 9: Central Processing Dysfunctions in Children: A Review of Research Needs, 148 pp., 1969.

NINDS Monograph No. 10: Human Communication and Its Disorders—An Overview, 176 pp., 1970.

NINDS Monograph No. 11: Reading Forum: A Collection of Reference Papers Concerned With Reading Disability. 256 pp., 1972.

NINDS Monograph No. 12: Studies on the Clinical Efficacy of Marketed Antiepileptic Drugs. 36 pp., 1971.

NINDS Monograph No. 14: The Epidemiology of Epilepsy: A Workshop. 167 pp., 1972.

NINCDS Monograph No. 15

THE RESEARCH STATUS OF SPINAL MANIPULATIVE THERAPY

A Workshop held at the National Institutes of Health, February 2–4, 1975.

Murray Goldstein, Editor

DHEW Publication No. (NIH) 76–998

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service National Institutes of Health
National Institute of Neurological and Communicative Disorders and Stroke
Bethesda, Maryland 20014

1975

WORKSHOP PLANNING COMMITTEE

Murray Goldstein, *Chairman*
Philip E. Greenman
Scott Haldeman
Horace W. Magoun
James H. McElhaney
Donald B. Tower
Henry G. West, Jr.
Andrew B. Wymore

NIH LIAISON

Philip S. Chen
Howard P. Jenerick

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, D.C. 20402 - Price $4.75
Stock Number 017-049-00060-7

Workshop Participants

Ballantine, H. Thomas, Jr., M.D.: *Senior Neurosurgeon, Massachusetts General Hospital; Associate Clinical Professor of Surgery, Harvard Medical School, Boston, Massachusetts*

Bromley, William, D.C.: *Audubon, New Jersey*

Burke, Robert E., M.D.: *Laboratory of Neural Control, National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, Maryland*

Chase, Thomas N., M.D.: *Director of Intramural Research, National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, Maryland*

Cleveland, Carl S., Jr., D.C.: *President, Cleveland College of Chiropractic, Kansas City, Missouri*

Cornfield, Jerome: *Professor of Statistics, George Washington University; Biostatistics Center, Bethesda, Maryland*

Cyriax, James, M.D., M.R.C.P.: *St. Thomas's Hospital London, England*

Day, William S., D.C.: *Spokane, Washington*

Denslow, J. S., D.O.: *Vice President, Kirksville College of Osteopathic Medicine, Kirksville, Missouri*

DiChiro, Giovanni, M.D.: *Head, Section on Neuroradiology, National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, Maryland*

Drum, David C., D.C.: *Department of Chiropractic Science, Canadian Memorial Chiropractic College, Toronto, Ontario, Canada*

Dubbs, Gustave, D.C.: *Hempstead, New York*

Frank, Karl, Ph. D.: *Chief, Laboratory of Neural Control, National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, Maryland*

French, Lyle A., M.D., Ph. D.: *Vice President for Health Sciences Affairs, University of Minnesota, Minneapolis, Minnesota. Member, National Advisory Neurological Diseases and Stroke Council, National Institutes of Health*

Gitelman, Ronald, D.C., F.C.C.S.: *Canadian Memorial Chiropractic College, Toronto, Ontario, Canada*

Goldstein, Murray, D.O., M.P.H.: *Director, Extramural Activities Program: Associate Director, National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, Maryland*

Greenman, Philip, D.O.: *Professor and Chairman, Department of Biomechanics, College of Osteopathic Medicine, Michigan State University, East Lansing, Michigan*

Gurdjian, E. S., M.D., Ph. D.: *Professor Emeritus, Neurological Surgery, Wayne State University, Detroit, Michigan*

Guth, Lloyd, M.D.: *Head, Section on Neuronal Development and Regeneration, Laboratory of Neurochemistry, National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, Maryland*

Haldeman, Scott, D.C., Ph. D.: *Vancouver, B.C., Canada*

Howe, Joseph W., D.C.: *Professor and Chairman, Department of Roentgenology, The National College of Chiropractic, Lombard, Illinois*

Janse, Joseph, D.C.: *President, The National College of Chiropractic, Lombard, Illinois*

Jenness, Martin, E., D.C., Ph. D.: *Director of Research, Palmer College of Chiropractic, Davenport, Iowa*

Johnston, William L., D.O.: *Professor, Department of Biomechanics, College of Osteopathic Medicine, Michigan State University, East Lansing, Michigan*

Kent, Christopher, B., D.C.: *Investigator, Research Department; Instructor in X-Ray, Anatomy and Chiropractic Science, X-Ray Department, Palmer College of Chiropractic, Davenport, Iowa.*

Klatzo, Igor, M.D.: *Chief, Laboratory of Neuropathology and Neuroanatomical Sciences, National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, Maryland*

Kleynhans, Andries M., D.C.: *Chairman, Committee on Research, The National College of Chiropractic, Lombard, Illinois*

Weis, Edmund B., Jr., M.D.: *Division of Orthopaedics, Ohio State University, Columbus, Ohio*
West, Henry G., Jr., D.C.: *Pocatello, Idaho*
White, Augustus A., III., M.D.: *Associate Professor, Department of Orthopaedic Surgery, Yale University School of Medicine, New Haven, Connecticut*
Wymore, Andrew B., D.C.: *Osawatomie, Kansas*

Contents

Chapter I.

Introduction, Summary, and Analysis

Murray Goldstein, *General Chairman*

Introduction, Summary, and Analysis

MURRAY GOLDSTEIN
Director, Extramural Activities Program
National Institute of Neurological and
Communicative Disorders and Stroke
National Institutes of Health

As part of the Senate Report on the Fiscal Year 1974 Appropriation for the National Institute of Neurological Diseases and Stroke (NINDS) of the National Institutes of Health (NIH), the Senate Appropriations Labor-HEW Subcommittee specified that . . . "this would be an opportune time for an 'independent, unbiased' study of the fundamentals of the chiropractic profession. Such studies should be high among the priorities of the NINDS. . . ."

In order to provide the substantive scientific base necessary for this evaluation, the NINDS convened a "Workshop on the Research Status of Spinal Manipulative Therapy." The Workshop was held at the NIH on February 2–4, 1975 and focused specifically on the documentation and evaluation of research results and clinical investigative experience. Spinal manipulative therapy was chosen as the theme of the Workshop since it is the primary therapeutic modality of chiropractic and would serve as a base for evaluating the scientific data about the fundamentals of chiropractic including the anatomy and pathophysiology of subluxation and methods of chiropractic diagnosis and therapy. The Workshop agenda included a detailed review of the history of manipulative therapy and discussion of the scientific issues of spinal geometry and kinematics, the intervertebral foramen, spinal root compression, spinal root and peripheral nerve pain, the pathophysiology of back pain, the concept of spinal vertebral subluxation, the clinical diagnosis of subluxation, and the evaluation of the efficacy of spinal manipulative therapy.

Workshop participants included 58 scientists and clinicians of national and international stature, including 16 Doctors of Chiropractic (D.C.), 24 Doctors of Medicine (M.D.), 7 Doctors of Osteopathic Medicine (D.O.), and 11 basic scientists (usually Ph. D.); seven of the aforementioned clinical participants were holders of both the Ph. D. and a clinical degree. Participants came from the United States and eight foreign countries. The Workshop was open to representatives of other Federal agencies and to the public, with approximately 200 persons monitoring the discussions.

The *history of manipulative therapy* is an ancient one with evidence of its use dating from Hippocrates. Through the ages, both orthodox physicians and folk practitioners in Europe and the Middle East wrote about and debated the usefulness of manipulation, particularly for the treatment of actue back pain and for "spinal curvatures" (i.e., kyphosis and scoliosis). By the late 19th century, most of the basic concepts and clinical principles of modern day usage had been formulated and documented. It seems probable that the genesis of the modern theory and practice of manipulative therapy as used by chiropractors, osteopathic physicians and medical physicians arose from concepts generally acceptable to many eminent 19th century medical practitioners and scientists since it was during this period that the role of the spinal cord in health and disease was being vigorously explored and discussed. Since that time, however, the three contemporary clinical professions utilizing manipulative therapy (M.D., D.O., D.C.) have developed in relative isolation from one another. Each group evolved primarily in a clinical setting with a self-generated terminology specific to the history of the clinical school. Individuals

from each clinical school of therapy have crossed professional lines as students and as instructors, but this has usually been done in secrecy; if done openly, it usually resulted in professional criticism. As a result of this isolation, a major problem of the present is the difficulty of sharing clinical experiences and scientific results because of self-developed terminology and continued inter-profesional isolation.

Review and discussion of *spinal biomechanical and anatomical research* demonstrate these to be ongoing areas of scientific interest and productivity. Many of the more recent biomechanical studies have focused on the consequences to the human spine of the biped erect stance and the consequences of trauma, particularly in association with rapid acceleration or deceleration. These problems have given emphasis to the need for mathematical and mechanical studies to provide methods for three-dimensional, quantitative descriptions of the complexity of normal spinal biomechanics. The anatomy of the intervertebral foramen is now quite well documented and the dynamic relationships of the para- and intervertebral structures are being elucidated. The roles of paravertebral supportive, vascular, and innervate tissues are becoming clearer, demonstrating the dangers of traction and manipulation, particularly to vascular and neural elements. However, despite attempts to describe "subluxation" biomechanically and anatomically, present data lack reliable quantitative precision and qualitative reproducibility.

A review of studies of the *pathophysiology and pathology of spinal root compression* is characterized by the lack of available data. The spinal root and its pathology have not been areas of widespread research interest. Available data are primarily focused on issues of gross injury secondary to major trauma or significant bony or cartilaginous impingement. The role of more subtle "pressure" or entrapment of specific types of nerve fibers in the spinal root is just being explored, as are the effects of metabolic changes in the neuron on peripheral and autonomic functions; this latter area of research, the cellular metabolism of the neuron, includes some of the most exciting explorations in modern neuroscience and is being pursued vigorously in laboratories in this country and abroad. The phenomena of referred pain and of reflex somatic and visceral dysfunction secondary to visceral disorders are generally accepted by scientists and clinicians. However, more controversial is the hypothesis that somatic disorders reflexly influence visceral function through somato-sympathetic reflexes. A relatively small but sound body of data is evolving that gives credence to this hypothesis. However, data were not presented which substantiate a direct relationship between manipulative therapy and clinical improvement by means of influencing segmental neuronal interactions. "Spinal man"—the paraplegic and tetraplegic—provides a useful model for examining the role of spinal viscero-somatic and somatico-visceral reflexes and pathways.

Back pain continues to be a clinical problem affecting large numbers of people. Acute back pain can result in complete immobilization; chronic back pain at the least is a continuing irritant and at the worst leads to physical incapacitation and to emotional degeneration. Most tissues have nociceptors, the stimulation of which often is experienced as pain. The mechanisms underlying the most generally recognized causes of back pain are reasonably well understood and include visceral disease (e.g., renal pathology), intervertebral disc herniation, dislocations and fractures; however, ligamentous and capsular pathology and intra-articular displacement are thought to be causes of the more common type of back pain, but these hypotheses suffer from lack of substantive evidence. Questions posed during the Workshop include: How do mechanical forces bring about the long-term discharge of nerve fibers? What is the origin of the neural discharge? Where are the receptors? This unclear situation is reinforced by the paucity of fundamental information about the mechanism of pain *per se*. Recent research opportunities generated by the interest in and clinical use of dorsal column stimulation and acupuncture may help to improve the conceptual framework necessary to approach the problem(s) of the treatment of pain, but pain continues to be a poorly understood phenomenon. The presently available evidence indicates that we do not yet understand the cause or pathophysiology of many cases of back pain. The several theories presented are conceptually viable, some are scientifically attractive, but all remain unsubstantiated.

A definition of the chiropractic *subluxation* was offered as: "an alteration of the normal dynamic, anatomical or physiological relationships of contiguous articular structures." Etiologic (e.g., traumatic, inflammatory, reflex, etc.) symptomatic

(e.g., fixed, hypermobile, painful, etc.) and pathophysiologic (e.g., primary, secondary) characteristics of the "nidus" were presented. To avoid semantic misunderstandings, the term "nidus," meaning focus of a morbid process, is used to incorporate the various clinical terms used by the several professions. The use of specific adjustive thrusts and the general techniques of spinal manipulation were described as methods for the reduction of positional abnormalities (subluxations), increasing movement at a "locked joint" and/or the "reduction" of disc lesions, with each therapeutic maneuver requiring the attainment of motion between vertebral segments to achieve its goal. The evidence for the pathophysiologic consequences of the proposed "nidus" remains unclear. The etiology of disc disease is still not completely understood and the chain of events resulting in pain and incapacity is surmised rather than demonstrated. Because of their rich sensory innervation, the posterior spinal articular facets and their associated ligaments are suggested by some as a "nidus" for back pain and the locus of the subluxation phenomenon. Flexion and/or rotation subluxations are described in anatomical and biomechanical terms, but their pathophysiologic effects on spinal roots, paravertebral tissues, interarticular capsules or segmental neural transmission are presented as hypotheses rather than the results of experimental studies. The lack of a relevant and reproducible animal model may be one important obstacle to clarification of these issues. The pathophysiologic role of subluxation as a cause or concomitant of organic disease (e.g., diabetes mellitus) was not presented or discussed. Like the biomedical evidence for the cause of back pain in general, the evidence for the chiropractic subluxation is inferential rather than experimental, the scientific references being selected from a broad and dispersed literature; however, the evidence at this time for disqualification of the hypothesis also is inferential. Thus, subluxation remains a hypothesis yet to be evaluated experimentally.

At this point, *a word of caution* is needed about interpretation of the preceding paragraphs. Many physical and emotional disorders of man are not understood etiologically or pathophysiologically; despite this, society demands relief. Historically, physicians have provided therapy based on empiricism and accumulated experience guided by that portion of the Hippocratic Oath that pledges:

> ". . . I will prescribe regimen for the good of my patients according to my ability and my judgment and never do harm to anyone. . . ."

The modern therapy for essential hypertension, stroke, cancer, the common cold—and back pain—fall within these guidelines for the treatment of disorders of unknown etiology. Several empiric therapies including spinal manipulative therapy are suggested for the relief of back pain within the concepts of the Hippocratic Oath, an ethic fundamental to all schools of the healing arts.

The Workshop did not directly address the issue: *Does spinal manipulative therapy do harm?* There are no experimental studies or controlled clinical trials of which the NINCDS is aware, documenting the evidence for injury. There are individual case reports in the literature documenting specific instances of misdiagnosis, poor clinical results and serious and occasional lethal aftereffects of spinal manipulative therapy—as there are for most modes of diagnosis and therapy offered by all clinical disciplines and all clinical professions. The clinical research issue which the subsequent sections of the Workshop did address is: *Does spinal manipulative therapy do good?*

The applicability of spinal manipulation hinges on the precision of the *diagnosis* of the underlying pathology described variously as a "subluxation" by chiropractors, as an "osteopathic lesion" by osteopathic physicians and as an "intra-articular displacement" by some medical physicians. The clinical skill of *palpation* is offered by each as a major diagnostic tool. Palpation is used clinically to: localize the anatomical site of the "nidus"; describe its anatomic properties; identify its static and dynamic biomechanical characteristics; evaluate paraspinal tissue "texture"; and elicit pain or discomfort. Like most techniques of physical diagnosis including the other classical diagnostic techniques of inspection, percussion and auscultation, palpation often is described as a skill passed from teacher to student, with the interpretation of findings based on clinical experience. Other than clinical and often anecdotal experience, no evidence was presented attesting to the specific reliability of palpatory findings in accurately describing the anatomical or biomechanical characteristics of the "nidus," or its paraspinal or painful properties. Studies were reported and data reviewed about both electrophysiologic and sudatory changes

associated with palpatory findings. However, discussion suggested that these findings are more significant at this time as experimental tools than as clinical techniques. The Workshop presentations and discussion indicated a lack of evidence about the interexaminer reliability of palpatory findings. Also, there was a paucity of evidence correlating palpatory with other diagnostic or pathological studies relevant to the "nidus." However, skilled clinicians claimed they could accurately differentiate by palpation between the presence or absence of a "nidus." The lack of data about the adequacy of palpatory findings needs consideration in the context of the status of similar types of experimental information about most classical physical diagnostic techniques.

The presentations on *X-ray examination* of the patient with a suspected "nidus" polarized discussion. Those utilizing X-ray examination for this purpose presented slides and quoted references attesting to the efficacy and safety of this diagnostic technique; others just as authoritatively presented slides and quoted references attesting to the inaccuracy and dangers of using the technique for this purpose. No evidence utilizing controlled retrospective or prospective trials was presented; no animal data were presented. The issues of efficacy and safety remain unresolved after the presentations and discussions. If the intensity of the discussion is an indication of the priority of this subject for focused investigative attention, it would receive a priority score of 1 on a decreasing scale of 1 to 5. *Other "objective" diagnostic modalities* were also presented but stimulated minimal discussion; included were thermography, electromyography, the spinogram and techniques for evaluating posture.

The evidence for the efficacy of spinal manipulative therapy in the *treatment of pain*, particularly low back pain and neck pain, was discussed in great detail. Three types of evidence were presented: case histories; clinical experience; inference. Established and prestigious medical and osteopathic physicians and chiropractors provided testimonial evidence in support of the efficacy and safety of manipulative therapy. This was held to be true for properly diagnosed cases of both acute and chronic pain. Orthopedic and neurological surgeons found the evidence unconvincing and by inference from other diseases of spinal structure, pathophysiologically unacceptable. Again, polarity of discussion occurred. No data derived from animal studies or from controlled clinical trials were presented for or against therapeutic efficacy for low back pain. The NINCDS is aware of a recent population study in the United States demonstrating equal results in overall outcome when chiropractic and conservative medical management of low back pain were compared; however, the study suffers from the problems of a research design based upon retrospective case history analyses. A prospective study of manipulative therapy is reported to be in progress in Great Britain but no data were available. Preliminary data were offered from a controlled clinical trial of manipulative therapy in the treatment of chronic obstructive pulmonary disease now in progress. The data suggest but do not establish improvement in pulmonary function; however, patients claim subjective improvement in work capacity.

During general discussion, there was a plea for precise definitions of terms (e.g., subluxation; clinical improvement) so that communication could be even more meaningful in the future. It was also suggested that precision of terminology would provide a base for focused investigations, comparisons of results and cooperative efforts among investigators.

The NINDS Workshop on the Research Aspects of Spinal Manipulative Therapy and staff review and analysis of available data clearly indicate that *specific conclusions cannot be derived from the scientific literature for or against either the efficacy of spinal manipulative therapy or the pathophysiologic foundations from which it is derived.* The efficacy of spinal manipulative therapy is based on a body of clinical experience in the "hands" of specialized clinicians. Chiropractors, osteopathic physicians, medical manipulative specialists and their patients all claim spinal manipulation provides relief from pain, particularly back pain, and sometimes cure; some medical physicians, particularly those not trained in manipulative techniques, claim it does not provide relief, does not cure, and may be dangerous, particularly if used by nonphysicians. The available data do not clarify either view. However, most participants in the Workshop felt that manipulative therapy was of clinical value in the treatment of back pain, a difference of opinion focusing on the issues of indications, contraindications and the precise scientific basis for the results obtained. No evidence was presented to substantiate the usefulness of manipulative therapy at this time in the

treatment of visceral disorders.

Biological and medical research focused on the issues of the biomechanics of the spine, kinesiology, spinal reflexes and pain are all areas just beginning to recapture the interests and attention of the scientific community. This is a reflection of the development of a cadre of investigators technically prepared to design meaningful experiments in these areas, the availability of new technologies and instrumentation providing methods for quantitative measurement, and the availability of research grant funds through the competitive research grant programs of the NIH. Moreover, a body of experience is now available for the development and conduct of controlled clinical trials of diagnosis and therapy; these are expensive undertakings requiring the investment of the coordinated efforts of scientists, biostatisticians, clinicians, patients and administrators over relatively long periods of time. Yet controlled studies provide the only scientific means presently available for the solution of the clinical research issues identified in this analysis.

Finally, the Workshop represents the beginnings of an interprofessional dialogue among chiropractors, physicians and biological scientists on the "neutral" and commonly-shared issues of science and research. Interprofessional rivalries characterized by the issues of accreditation, licensure, fee reimbursements and professional perogatives were turned aside at the Workshop as unacceptable bases for biomedical scientific exchange.

Thus the Department of Health, Education, and Welfare needs to consider both chiropractic and manipulative therapy from the viewpoints of strategy and priority. The fundamentals of chiropractic and of the other schools of manipulative therapy are founded on a century of clinical experience. There are little scientific data of significance from which to evaluate this clinical approach to health and to the treatment of disease. An obvious strategy would be the fostering of biological and clinical research so that answers to the questions of clinical indications and therapeutic efficacy of manipulative therapy can be approached more meaningfully. The issue of priority is a more difficult one. With limited national resources for research and research training, what relative priority should be placed upon this targeted area? Should funds and personnel presently assigned to other research areas be reassigned? Should additional research funds be made available specifically for this area? Should research on chiropractic and manipulative therapy compete on scientific merit with other areas of research for funds already available for biomedical research? The answers to these issues of health policy will determine the scope and intensity of the research effort.

Chapter II.

Evolution and Development of the Concepts of Manipulative Therapy

Donald B. Tower, *Chairman*

597-036 O - 76 - 2

Manipulative Therapy: A Historical Perspective from Ancient Times to the Modern Era

ELIZABETH LOMAX
Neuro-Psychiatric Institute
The Center for the Health Sciences
Los Angeles

This brief survey of manipulative therapy will serve as an introduction to the authoritative presentations to follow. For the sake of conservation of time, and in light of an abundant history, I shall skim the ancient and medieval periods, and concentrate on more recent events.[1] Yet the classical texts surely deserve further attention if only to decide whether the Greeks merely attempted to reposition vertebrae displaced through violence, or whether they actually performed manipulations of slightly luxated vertebrae as therapy for a wide variety of dysfunctions. The latter opinion, which transforms chiropractic as enunciated by D. D. Palmer into a literal rediscovery of ancient practice, has been advanced by Dr. Kleanthes A. Ligeros of Greece in a book published in 1937.[2] I hardly dare argue with a native, but Ligeros does seem to have read more into the Hippocratic writings than is obvious to the outsider using translations.

In conventional interpretation, Hippocrates distinguished between incurable humpback due to disease and posterior curvature caused by a fall, for which reduction though rarely successful might be attempted.[3] However, he disapproved of the popular method of succussion head down from a ladder, not because this treatment seemed unnatural but because it had been abused: "It is disgraceful in any art, and especially in medicine, to make parade of much trouble, display, and talk, and then do no good." Instead he recommended combined extension and pressure, exerted on the patient lying prone on a wooden bed. To quote:

> Such extension would do no great harm, if well arranged, unless one deliberately wanted to do harm. The physician, or an assistant who is strong and not untrained, should put the palm of his hand on the hump, and the palm of the other on that, to reduce it forcibly, taking into consideration whether the reduction should naturally be made straight downwards, or towards the head, or towards the hip. This reduction method is also very harmless; indeed, it will do no harm even if one sits on the hump while extension is applied, and makes succussion by raising himself; nay, there is nothing against putting one's foot on the hump, and making gentle succussion by bringing one's weight upon it.[4]

Even more effective was the use of a piece of wood as a lever with which to apply pressure on the hump beneath.

Later authors such as Galen, Celisus, and Oribasius, gave essentially the same advice, but, by the sixth century A.D., spinal curvature was also being treated by means of open wounds or eschars, induced on either side of the deformity.[5] The treatment of pain by invoking local suppuration was favored by Arab physicians and, as we shall see later, again used extensively in the late 18th century. Such treatment was compatible with the humoral theory of disease, but whether in this

instance its use was an Arab innovation, or one derived from Hellenistic sources, I have been unable to discover. Certainly the extension method was not lost, for it was again expounded at length by Albucasis in the 10th or 11th century, and by Guy de Chauliac in the 14th century.[6]

Luxations of the spine were discussed in some detail by the great French barber-surgeon, Ambroise Paré. He was not at all in favor of tying patients to ladders and dropping them from roof tops, but otherwise his methods of restoration differed little from those described by the ancients.[7] What he added was the steel corset to support the back after manipulation, and the apparently novel suggestion that much chronic deformity was caused by faulty posture during childhood rather than by luxation secondary to injury. The same theme was expounded two centuries later by Nicholas André who, as explained by Bick, synthesized the term "orthopaedia" "from the Greek roots *orthos* (straight) and *paidos* (child), to express his belief in the theory that many of the deformities of adolescence and adult life originate in childhood."[8] Curvature of the spine could be avoided by attention to posture and exercise during youth. Once deformity had occurred André, like many of his contemporaries, recommended rest, head suspension, and the wearing of corsets whenever ambulant.[9]

Following Percival Pott's pronouncement that caries of the spine was far commoner among young children than ever previously suspected, many physicians went to the extreme of diagnosing all cases of early scoliosis or lordosis as due to scrofula.[10] These they treated by rest and by creating local discharge through the use of issues, while condemning extension and manipulation as both useless and dangerous.

By the beginning of the 19th century, a plethora of literature existed on disorders of the spine, but absolutely no consensus as to differential diagnosis and treatment. Some, including Shaw, and Dods, thought that muscle weakness was the prime cause of deformity, and therefore recommended either complete rest or active exercise.[11] Others implicated the vertebrae, as being carious or simply too soft, and treated everyone with rest and induced local ulceration. Each group produced numerous case histories to support its contentions, and all indulged in polemics to silence any opposition.[12]

Even in this atmosphere some still prescribed manipulation, for example Dr. Edward Harrison, a graduate of Edinburgh University in 1784. His career was unusual, even when judged by the standards of his time.[13] In the late 18th century, he studied in Paris, a city which apparently then abounded with institutions for the cure of spinal distortion. Many methods were used, including traction and kneading of the hump, much to the horror of scrofula conscious British critics.[14] Later Harrison spent a couple of decades as medical practitioner in Lincolnshire, a county then famed for its numerous bone-setters and empirics.[15] All in all he had plenty of opportunity to learn the art of spinal manipulation.

In 1817 Harrison came to London. In 1821 he began writing a series of articles for the *London Medical and Physical Journal* entitled: "Observations respecting the Nature and Origin of the common Species of Disorders of the Spine: with Critical Remarks on the Opinions of former Writers on this Disease." Having delivered the necessary attack on the views of Pott, Harrison expressed his own as follows:

> According to this view of the subject, we must direct our attention to some other tissue to discover the true cause of spinal complaints; and I am of opinion that we shall find it in the connecting ligaments, "which seem to have lost part of their power of holding the bones together." These get relaxed, and suffer a single vertebra to become slightly displaced. The column now losing its natural firmness, other bones begin to press unduly upon the surrounding ligaments: they, in turn, get relaxed and elongated, by which the dislocation is increased and the distortion permanently established. The direction becomes lateral, anterior, or posterior, according to circumstances; but the malady has in every instance the same origin, and requires the same mode of cure.[16]

Thus, the mischief did not originate in the vertebrae, as taught by Pott, nor in the cartilage as suggested by others, but in the ligaments. For this reason people often recovered, an impossible outcome if the more serious pathology were indeed present. Harrison was full of optimism for the prospects in early cases, so long as his system was employed. Usually he was none too clear about the

actual treatment applied but, on one occasion, gave the following account:

> The patient, being placed upon a couch, as usual, had her back and chest well rubbed with an emollient liniment for more than an hour, while the spine was stretched in the machine formerly referred to. It is constructed of steel, upon the principle of the windlass of a ship, and fixed to the bottom of the crib. By means of soft leathers surrounding the arms, and connected with the top of the couch, and other leathers attached to the ankles, which are fixed to the machine, almost any degree of stretching may be safely resorted to, by turning the roller of the machine, provided the force be gradually increased. The prominent vertebrae and ribs were then pressed, and driven in the direction of their natural situations, with an instrument held in the right hand. . . . I formerly used my thumbs only for pressure; but, finding the other contrivance much more powerful and easier to be borne, I have for a long time given it the preference. A firm bandage was afterwards fastened round the chest, to prevent the bones from returning. This bandage being adjusted, she was laid flat upon the back, and directed to remain constantly in the same position.[17]

These manipulations were repeated daily for about 3 weeks, by which time, according to our author, "the contour of the spine was entirely restored."

In 1827 Harrison wrote a popular book on the subject.[18] On the whole this met with professional disapproval mainly, it would seem, because the doctrine of vertebral luxation and consequent need for manipulation was taboo. The *Medico-Chirurgical Review* began by expressing veiled criticism: "God forbid that we should impute to Dr. Harrison, or to any other doctor, the remotest idea of charlatannerie; but we do believe that it would be difficult to devise a system better adapted to the practice of that art, than the spinal pathology here delineated."[19] It ended its review article much more expressively:

> After this specimen of the "bolder flight" which Dr. Harrison has taken from the low ranks of his brethren, to the GENERAL READER we must lay down the pen. We are positively ashamed (if it will be believed that a REVIEWER can have any sense of shame) to record such a passage from the writings of a physician of the present period, on our pages. But the above extract will characterize the book under review, better than any thing which we would say. The pathology is erroneous—the practice is, for the most part, concealed—and the plates are more calculated to frighten the GENERAL READER than to convey any information to the MEDICAL PRACTITIONER.

Two added factors probably contributed to official disapproval of Harrison. First, he had developed a lucrative practice in London, having established a clinic favored by many English dignitaries and even by the Queen.[20] Second, since 1804, while in practice in Lincolnshire, he had become leader of a movement for medical reform, particularly reform of the antique statutes of the Royal College of Physicians which prevented anyone not a graduate of Oxford or Cambridge from practicing medicine within seven miles of the city of London. A low-grade battle rumbled on until 1827, when the College summoned Harrison to give an account of himself and obtain the required license. This he refused to do, so the College prosecuted him for illegal practice. The case came to court in 1828; Harrison defended himself on the ground that he had been practicing surgery not medicine and, although this was patently untrue, the jury found in his favor. From then on he worked unmolested in London until his death in 1838.[21]

For such reasons Harrison was probably the most notorious British physician to advocate and use manipulation in the treatment of spinal disease. But he was not the only one. In 1824 Andrew Dods had published his *Pathological Observations on the Rotated or Contorted Spine, etc.*, in which he argued that the immediate cause of deformity was "a peculiar affection of the muscles of the back" whose "perverted action" led to rotation or twisting of the spine.[22] Treatment was mainly prophylactic—plenty of exercise for children of both sexes—but once the condition was established, both friction and manipulation had a part in treatment.

In his popular work *On Spinal Weakness and Spinal Curvatures*, published in 1868, W. J. Little favored both Dods' theory of spinal rotation and

the proposition that manipulation had a place in therapy. Since Little was an important figure among mid-century London orthopedists, his opinion will be quoted at length:

> During recumbency, manipulations may with great advantage be resorted to. Very superficial observation of the beneficial effects of attempting to press the spinal column into a straighter direction will, unless the patient be much advanced in life, encourage the patient and her friends to persevere in a proper and sedulous use of this means.
>
> The person employed to effect these manipulations needs to be expressly taught in what manner they can be most advantageously carried out. The surgeon can impart the necessary instruction by showing where the pressure and counter-pressure upon the several curves require to be made. Dr. Harrison, a well-known practitioner thirty years ago, adopted a peculiar means of endeavouring to press the rotated spinous and transverse processes into a more favorable position. The instrument used by him resembled that which is sometimes employed to compress the subclavian artery above the clavicle. Although the attempt to effect the object he had in view appears at first sight futile, the reiterated pressure upon particularly projecting parts of vertebrae is fairly indicated, and is not unattended by benefit.[23]

Manipulation was one of a battery of therapies advocated by Little for lateral curvatures of the spine. He differentiated this condition from the more serious angular curvatures, caused by necrosis, caries, inflammation or rheumatism of the vertebrae, which were to be treated only by rest or fusion.

Dods had at least one other disciple, a Mrs. Godfrey of Liverpool, who carried on a business originated by her husband, a "medical man." The fruits of her spinal practice, and her faith in divine providence, were expressed in a book first published in 1851.[24] The treatment consisted not of "friction," nor of "shampooing," but of manipulation, although "not such as that which has been previously performed." Rather, careful manipulation was used to prepare the muscles for special exercises which she then imposed. She believed that spinal curvature was often caused by slight displacements of various parts of the body, "which were produced, in some instances, by convulsions in infancy, and in others through sudden jerks and falls."[25] *The British and Foreign Medico-Chirurgical Review* did not think much of her explanations, but at least reviewed her book, in company with three others on deformities written by two Fellows and one Member of the Royal College of Surgeons.[26]

Such recognition was not usually given to traditional bone-setters, but, by the last third of the 19th century, this attitude was beginning to change. Early in 1867, the *British Medical Journal* reported James Paget's lecture: "On the cases that bone-setters cure," a lecture in which he warned his professional listeners that they should pay attention to the activities of empirics, if only to avoid losing patients.[27] More complimentary to bone-setting was a series of articles on the subject written by Wharton Hood for the *Lancet* of 1871.[28] This orthopedist confessed himself most impressed by the skills of a famous bone-setter, Mr. Hutton, with whom he had actually worked as an assistant, "I was astonished," he wrote, "and often no less mortified, at the number and variety of instances in which the manipulations I have endeavoured to describe were followed by almost immediate cure."[29] These articles were followed by a popular book on the subject, and Hood's persistence seems to have paid off, at least to the extent that a discussion on bone-setting was held at the fiftieth annual meeting of the British Medical Association in 1882.[30]

However, I must hasten to add that bone-setting was not exclusively practiced in the British Isles. In the United States, by the mid-19th century, the male members of the Sweet family of Rhode Island and Connecticut were reputed to possess a hereditary skill in bone-setting. According to a skeptic, "the beginning of this strange delusion happened in South Kingstown, in the State of Rhode Island, more than one hundred years ago."[31] More about the family and its abilities may be learned from *An Essay on the Science of Bone Setting*, published in 1829.[32] Here the author, Waterman Sweet, set out to prove that surgery and anatomy were intuitive sciences only intelligible to those who had a talent for the profession, and were divinely endowed with sufficient ability and the capacity for hard work. His own mastery of the art was

illustrated by numerous case histories of successful manipulations of peripheral joints. Apparently he had a thriving practice in spite of "evil reports" circulated by doctors and others. As mentioned above, the family was still doing well in the mid-19th century, but I have not been able to follow its fortunes further.

Before concluding this survey, reference should be made to other early 19th century concepts pertinent to the later emergence of osteopathy and chiropractic. A novel development was the generation of intense interest in functions of the spinal cord. Since Galenic times, this structure had been considered merely as a conduit for nerves linking the brain with the periphery. But following the enunciation of the Bell-Magendie law, and of Marshall Hall's theory of reflex action, pathology of the spinal cord was suddenly heralded as the obvious cause of much disease of previously unknown origin. To the fore came a new clinical entity, "spinal irritation," which embraced a variety of nervous symptoms, so long as one diagnostic sign could be elicited—tenderness on pressure over the vertebral spines. Excellent accounts on the history of the growth of this idea have been given by the American neurologist William Hammond in his 1871 *Treatise on Diseases of the Nervous System*, and, more recently, by the historian and neurologist, Francis Schiller, in an article entitled: "Spinal Irritation and Osteopathy."[33]

In 1828 Dr. Thomas Brown of Glasgow coined the term "spinal irritation," although he was not the first to suppose that subacute disease of the cord could precipitate a plethora of nervous complaints. From then onwards this diagnosis became very fashionable, to be used by such prominent physicians as Cruveilhier of Paris and Corrigan of Dublin.[34] Indeed the latter considered the discovery to be "the greatest improvement in practical medicine" that had taken place within his recollection. "Other improvements may have been more brilliant, but there has been none so useful." Nor did it take long before "spinal irritation" found its way across the Atlantic. Beginning in 1832 with an article by Dr. Isaac Parrish of Philadelphia, *The American Journal of Medical Sciences* repeatedly carried reports on diagnostic progress both in this country and in Europe.[35]

Taken *in toto* it would appear that there were few morbid phenomena which could not result from irritation of the spinal marrow. The following is a list of possible manifestations: mania, vertigo, amblyopia, nervous fevers, cough, dyspnoea, pleuritis, colic, vomiting, disorders of menstruation, hysteria, asthma and diabetes.[36] In any case the diagnosis could be clinched by finding tenderness of the appropriate vertebrae, i.e. those from which emerged the spinal nerves, or beneath which lay the sympathetic ganglia, whose dysfunction might reasonably account for the prevailing symptoms.

The treatment was to apply irritants, such as blisters, leeches, and cauteries, to the tender dorsal point. Today we may be amazed that such heroic measures could still be advocated in the eighteen thirties and forties, until we remember that the humoral theory of disease still prevailed.[37] Given this viewpoint the aim was to confine the disease by local depletion and blistering, and so effect a cure. Even harsher measures were at hand should general inflammation and fever appear, but, according to contemporary reports, local irritants were usually very successful, leading to speedy and complete recoveries.

According to Francis Schiller, Andrew Taylor Still was probably influenced by the doctrine of spinal irritation which continued to have eminent supporters until the end of the century.[38] The founder of osteopathy was probably revolted by the style of therapy described above, which fortunately was rapidly becoming obsolete by mid-century. In addition, the pathology of spinal irritation was pertinent, especially as expounded by William Hammond in 1871. This neurologist attributed the symptoms to anemia of the posterior columns of the spinal cord, and proposed as treatment the application of direct galvanic current, with the negative pole at a point above the seat of pain, and the positive at another, an equal distance below.[39]

It can therefore be argued that at their genesis, both the theory and the practice of osteopathy and chiropractic depended upon concepts acceptable to many eminent 19th century medical practitioners. This fact has become blurred since such doctrines, i.e. spinal irritation, have been abandoned by medical scientists. If it is remembered that a century ago the cord was visualized as the center for the multiplicity of functions, and even by some psychologists as the locus of a "spinal soul," one can regard the beginnings of osteopathy and chiropractic as legitimate offsprings of contemporary thought.[40]

REFERENCES

1. A fuller treatment and bibliography of the history of manipulative therapy may be found in: Eiler H. Schiotz, "Manipulation treatment of the spinal column from the medical historical viewpoint," *Tidsskr Nor Laegerforn, 78*, 359–372, 1958. (NIH Library translation available.)
2. Ligeros, Kleanthes A., *How Ancient Healing Governs Modern Therapeutics* (New York, G. P. Putnam's Sons, 1937.)
3. Hippocrates, *Hippocrates, with an English Translation by Dr. E. T. Withington, Vol. III* (Cambridge, Harvard University Press, 1959), pp. 278–307.
4. *Hippocrates, Vol. III*, p. 299 (n. 3).
5. Galen, *Galeno: I Quattro Commentari al Trattato delle Articolazioni di Ippocrate*, trans. by M. Abballe (Rome, De Luca, 1972), pp. 97–158; *Celsus: De Medicina with an English Translation by W. G. Spencer, Vol. III* (Cambridge, Harvard University Press, 1961), pp. 566–567; Oribasius, *Oeuvres d'Oriobase*, trans. by M. M. Bussemaker et Daremberg (Paris, J. B. Baillière, 1851), pp. 449–451.
6. Albucasis, *Albucasis: On Surgery and Instruments* by M. S. Spink and G. L. Lewis (Berkeley, University of California Press, 1973), pp. 812–818. Albucasis also advocated the use of cauterization for early hunchback, *Ibid.* p. 128. Guy de Chauliac, *La Grande Chirurgie de Guy de Chauliac*, ed. by E. Nicaise (Paris, F. Alcan, 1890), p. 374, pp. 516–518.
7. Paré, Ambroise, The Collected Works of Ambroise Paré, translated by Thomas Johnson, from the first English ed. London, 1634 (New York, Milford House, 1968), pp. 602–607; William Brockbank and D. L. Griffiths, *J. Bone & Joint Surg., 30B*, 556–559, 1948.
8. Bick, Edgar M., *Source Book of Orthopaedics* (Baltimore, Williams & Wilkins, 1948), p. 67.
9. de Boisregard, Nicholas Andry, *Orthopaedia* (facsimile of first ed. in English, London 1743, Philadelphia, Lippincott, 1961).
10. Pott, Percivall, *Remarks on that Kind of Palsy of the Lower Limbs which is Frequently Found to Accompany a Curvature of the Spine, and is Supposed to Be Caused by It, Together with its Method of Cure* (London, J. Johnson, 1779), also, *Further Remarks on the Useless State of the Lower Limbs in Consequence of a Curvature of the Spine* (London, 1782), both essays reprinted in *Medical Classics, 1*, 281–322, 1936–37.
11. Shaw, John, *On the Nature and Treatment of the Distortions of the Spine, etc.*, (London, Longman, Hurst, Reese, Orme, Brown and Green, 1823); Andrew Dods, *Pathological Observations on the Rotated or Contorted Spine, commonly called Lateral Curvature, deduced from Practice* (London, T. Cadell, 1824).
12. Some idea of the number of publications may be gained by examining the entries under: Spine, deformities and curvatures of, in the *Index Catalogue of the Surgeon-General's Office, 13*, 450–462, 1892.
13. An account of Harrison's Life may be found in: George Clark, *A History of the Royal College of Physicians of London, Vol. II* (Oxford, Clarendon Press, 1966), pp. 627–632, 664–666.
14. "Editorial," *Lancet, 11*, 347–349, 1826–27.
15. Harrison, E., in: *An Address delivered to the Lincolnshire Benevolent Medical Society at their Anniversary Meeting in 1809* (London, R. Rickerstaff, 1810), p. 7, stated that after circulating printed queries it was found "that in Lincolnshire Empirical pretenders exceeded the regular Faculty, in the proportion of nine to one."
16. Harrison, E., "Observations Respecting the Nature and Origin of the Common Species of Disorder of the Spine: with Critical Remarks on the Opinions of former Writers on this Disease," *London Med. & Phys. J., 45*, 103–122, 1821 (106–107).
17. Harrison, E., "Observations on the Pathology and Treatment of Spinal Diseases," *London Med. & Phys. J., 51*, 350–364, 1824 (354).
18. Harrison, E., *Pathological and Practical Observations on Spinal Diseases; illustrated with Cases and Engravings. Also, an Inquiry into the Origin and Cure of Distorted Limbs* (London, T. & G. Underwood, 1827).
19. "Book review," *Med.-Chir. Rev.* Ser. 2, *9*, 87–90, 1828 (89).
20. Rogers, E. C., "Treatment of spinal curvature," *Boston M. & S.J., 49*, 118–121, 1853–54.
21. For accounts of these activities see: George Clark, *A History of the Royal College of Physicians of London* (n. 13); E. A. Underwood, *A History of the Worshipful Society of Apothecaries of London, Vol. I 1617–1815* (London, Oxford University Press, 1963), 196–201.
22. See Reference No. 11.
23. Little, W. J., *On Spinal Weakness and Spinal Curvatures* (London, Longman, Green & Co., 1868), p. 57.
24. Mrs. Godfrey, *On the Prevention and Cure of Spinal Curvatures and Deformities of the Chest and Limbs; being the result of many years' experience* (London, 1851).
25. Mrs. Godfrey, *On the Nature, Prevention, Treatment, and Cure of Spinal Curvatures, etc.* (London, John Churchill, 1860), p. 4.
26. "Book review," *Br. & For. Med.-Chir. Rev., 10*, 209–219, 1852.
27. Paget, James, "Cases that bone-setters cure," *Brit. Med. J. i.*, 1–4, 1867.
28. Hood, Wharton P., "On the so-called 'bone-setting,' its nature and results," *Lancet, i*, 336–338, 372–374, 441–443, 499–501, 1871.
29. *Lancet, i*, 501, 1871 (see Reference No. 28).
30. Hood, W., *On Bone-setting, so called, and its Relation to the Treatment of Joints Crippled by Injury* (London, Macmillan, 1871).
31. King, Dan, "Hereditary skill in bone-setting," *Boston M. & S.J., 50*, 12–15, 1854. For the favorable article on the Sweet family which inspired the above rebuttal, see: Joseph Comstock, "On the study of living anatomy," *Boston M. & S.J., 49*, 500–503, 1854.
32. Sweet, Waterman, *An Essay on the Science of Bone Setting* (Providence, 1829).
33. Hammond, William A., *A Treatise on Diseases of the Nervous System* (New York, D. Appleton & Co., 1871), pp. 397–407; Francis Schiller, "Spinal irritation and osteopathy," *Bull. Hist. Med., 45*, 250–266, 1971.
34. See the report on M. Cruveilhier's opinion in *Am. J. Med. Sci., 21*, 465–466, 1837–38; on the clinical lecture delivered by Dr. Corrigan in *Lancet, ii*, 163–169, 1830–31.

35. Parrish, Isaac, "Remarks on spinal irritation as connected with nervous diseases," *Am. J. Med. Sci., 10*, 293–314, 1832.
36. "Remarks upon many symptomatic affections which are encountered in cases of irritation of a greater or lesser portion of the spinal marrow," *Am. J. Med. Sci., 18*, 483–486, 1836.
37. An explanation of contemporary thinking may be found in: Lester S. King, "The blood-letting controversy: A study in the scientific method," *Bull. Hist. Med., 35*, 1–13, 1961.
38. Schiller, Francis, *Opus cit.* (see Reference No. 33) proposed that the genesis of osteopathy could best be understood by taking into account "the contemporary background of spinal irritation."
39. Hammond, W., *A Treatise on Diseases of the Nervous System*, p. 423 (see Reference No. 33).
40. Evidence for and against the existence of a spinal cord Soul is discussed by E. G. T. Liddell in *The Discovery of Reflexes* (Oxford, Clarendon Press, 1960), pp. 60–87.

History of the Development of Medical Manipulative Concepts; Medical Terminology

JOHN McM. MENNELL
Chief, Rehabilitation Medicine Service
Veterans Administration Hospital
Martinez, California

Some in this audience are leaders in the contemporary development of medical manipulative concepts. Fundamentally our contributions parallel each other. However, there are areas in which we find ourselves in some disagreement, particularly when we address ourselves to spinal manipulative therapy—the topic of this meeting.

I am at a loss to discuss a single modality of therapy. I feel that such a discussion is pretty meaningless unless we know what we are treating, why we are treating it, how to use modality and, above all, when we should not use it. This leads to what is perhaps the more important part of my presentation—medical terminology.

I find historically that there are significant problems in discussing manipulative therapy even among those who practice it. It is not surprising then that we find difficulty in expressing to others what we are talking about.

Watson-Jones has written that there is no place for manipulation in orthopedic practice. In contrast, Wiles has published that manipulation should always be tried before any operation for back pain is undertaken. Neither of these authorities describe what they mean by manipulation.

Recently I asked an orthopedic surgeon in charge of a large residency training program what he really thought about manipulation. I did not say "spinal manipulation" yet that is how he heard my question. Paraphrasing his reply he said: "I use it. I twist people around and I really don't know what I am doing, but it is surprising how often it works."

When we ask what is meant by spinal manipulation, we find the following nonspecific terms in use which do not mean very much scientifically: The bone-setters by their treatment put a little bone back in place; the chiropractors treat nerve pressures by correcting subluxations; the osteopaths treat the osteopathic lesion which impairs the circulation. James Cyriax and I had fathers who were leaders in the development of medical manipulation. Those who followed the Senior Mennell and the Senior Cyriax initially treated a loss of involuntary movements in joints. Later when disc lesions became fashionable, the Junior Cyriax manipulated discs while I maintained the belief that the only structure which could be moved manipulatively was a synovial joint. More recently, in Europe, it has been suggested by Wolf that manipulation frees blockage of spinal synovial joint movement by freeing an intra-articular meniscus (in 1974 he suggested that it is meniscoid material) within a joint. Maigne leads a European school of thought that manipulation releases an intervertebral mobile segment. For the most part manipulators in Scandanavia and Europe work according to the osteopathic concept. In the paramedical field, Paris, a physical therapist, talks of manipulators in Scandinavia and Europe work born, teaching physical therapists, infer that they are treating joints. But the teaching physical

therapists cloud the issue by talking about degrees of manipulation using such terms as articulation and mobilization leading up to manipulation.

Where does all this leave us? The implication is that no two people understand what the other is communicating. Manipulation is used to mean anything from the most gentle examining movement through the reduction of a dislocation, the setting of a fracture to a gross assault on an unconscious patient. If medical endeavors were as inexact as medical terminology, we would be in the dark ages of vapors and humors, bleeding and purging.

That is how manipulation stands today. I think it unnecessary to labor this topic of terminology further. Unless we can somehow arrive at a common language which does not infringe upon the basic medical sciences, I cannot see how we will communicate successfully.

I therefore offer the language which I have been attempting to establish for many years with, I believe, some success. I urge the acceptance of this language for three reasons:

(1) it does not fly in the face of the basic sciences of medicine,
(2) it adds to the basic sciences of medicine concepts from the basic science of physics, and
(3) it is applicable to manipulation in every part of the musculoskeletal system.

We are all acquainted with the tenets of anatomy and physiology and the description of the functional movements of synovial joints. I ask you to add a concept from the science of mechanics. In everything man makes, there is built in play between the moving parts which ensures efficient function. So I postulate the following basic concepts:

(1) There is a normal anatomical range of mechanical play movements in synovial joints. It is prerequisite to efficient pain-free movement. This is joint play.
(2) Loss of joint play results in a mechanical pathological condition manifested by impaired (or lost) function and pain. This is joint dysfunction.
(3) Mechanical restoration of joint play by a second party is the logical treatment of joint dysfunction. This is joint manipulation.

The etiological factors which give rise to joint dysfunction are:

(1) Intrinsic joint trauma,
(2) Immobilization which includes therapeutic immobilization, disuse and aging,
(3) The healing of some more serious pathological condition in the musculoskeletal system.

The development of medical manipulative concepts has been held up by three major problems. The first problem is language, which I have already discussed. The second problem is perpetuated by the fact that this conference focuses on spinal manipulation. The third problem is the belief held by many that the pathological causes of pain in the back are different from those which cause pain in the extremities.

As a result of these problems it is not surprising that the development of manipulation is clouded with prejudice, bias, and ignorance.

The only well-documented history of the development of medical manipulative concepts in the first half of the 20th century comes from my father, the late James Mennell.

Originally, James Mennell was convinced that musculoskeletal pain—in the absence of signs of gross extrinsic trauma, inflammation, metabolic or systemic disease, neoplasm, congenital anomalies, vascular or neurological disease—arises from some mechanical internal derangement of a joint, regardless of the presence of an intra-articular meniscus.

His interest derived from dissatisfaction about residual pain resulting from orthodox treatment of fractures. He could not understand why so many patients had post-fracture joint pain after bone healing; nor could he accept the idea that this pain arose from "a little arthritis," as patients of all ages were affected, even in the absence of radiological evidence of arthritis. Conversely he noted that some people demonstrating radiographic arthritis in their joints suffered no pain after the healing of a fracture.

His observations led to the publication in 1912 of his first book "Treatment of Fractures by Mobilization and Massage," based on his personal experience of handling a number of fractures in an unorthodox way. He disclaims originality and ascribes his thinking and practice to Dr. Lucas Championiere of Paris whom he had visited.

He developed the hypothesis that there is an involuntary range of movement within joints. When lost, this causes pain and loss of function

without the usual signs of arthritis or other pathological changes. He further recognized that the usual anatomical descriptions of movement in topographical joint areas were inaccurate and pertained to the dead and not the living.

From his study of bones and their articulating facets and his clinical observations, he described the different voluntary movements of individual joints and something new—their involuntary movements. For instance, he drew attention to the fact that for the most part different functional movements occur at different individual joints in the wrist. Further, he showed that the involuntary movements were different in every joint but common to each joint of the same nature. These findings were incorporated into his second book "Massage—Principles and Practice" in 1920. In subsequent editions—there were seven of them—the title was changed to "Physical Treatment by Mobilization and Massage."

Independent of the teaching of chiropractic or osteopathic schools in the United States, he then considered the spine and concluded that, if there are synovial joints in the back, then they act and react in the same way to trauma and disease as they do in the extremities. He worked out the involuntary movement of spinal joints. These findings were published in his book "Backache" in 1931. He continued his research and refined his techniques. He had by now also developed satisfactory and safe techniques and therapeutic manipulation of synovial joints. These were brought together in his final outstanding publication *The Science and Art of Joint Manipulation*, Volume I, *The Extremities* in 1939, and Volume II, *The Spine*, in 1948.

Unfortunately, in his teaching, James Mennell never clearly differentiated his examination from his therapeutic techniques and it was easy for those who were disinterested to take the position: "Joint manipulation (the therapy) is all very well but what is it done for?"

What contribution I have made is clearly to classify differential diagnosis of causes of synovial joint pain. I emphasize a method of using examining manipulative techniques in the assessment of musculoskeletal pain in general and joint pain in particular. To the basic sciences of anatomy, physiology and pathology, I have added the basics of mechanics to explain the syndrome of symptoms and signs characteristic of a mechanical cause of pain. I may have added some innovative therapeutic techniques but therapeutic techniques are quite unimportant for, in the end, joints can only move in one way and that is the way they are designed to move. Anyone's technique is as good as another's in therapy so long as they do not try to invent movements. Invented movements or forced overmovements are bound to create injury to joint structures and to other vital structures which are in close relation to joints.

I have little evidence other than logical inference which supports my unswerving belief that the Mennell concepts are right. But with reference to the back, I have been able to demonstrate by cineradiography that a facet joint—on the side responsible for a patient's complaint of neck and arm pain—failed to open. The joint was at the level at which pain was elicited on examination. After manipulating this patient, I demonstrated radiographically opening of the same joint and the patient was free of his symptoms. This joint opening was the only apparent change in the radiographic study. The patient had been unable to work as a plumber for 4 years. He returned happily to work within a month.

I am unable to endorse the hypothesis that manipulation does anything to the intervertebral disc. I have personally performed 32 lumbar discograms in living patients and noted no appreciable change in the appearance of living disc spaces before and after moving a spinal segment.

Further, I can find no basis in theory nor in the accepted tenets of neurology to change my acceptance of the principle that manipulation only moves synovial joints. We do not have to invent etiological factors to explain back pain. Were a manipulatable condition in the back to be shown to be associated with a raised protein in the spinal fluid, which results from dural irritation, or if an annulus defect were demonstrable by epidurography and/or discography, then I could accept this hypothesis.

But the mechanical triad which I present is common to every synovial joint in the body, whether in an extremity or the back. It is the only hypothesis I know to which there are no exceptions. The musculoskeletal system is one system. The back is just a part of that system. I believe that the intervertebral disc, when prolapsed, gives rise to neurological signs and, if the prolapse is central, to dural pain. But the symptom of dural pain is central or bilateral whereas almost in-

variably the pain which responds to manipulative therapy is unilateral.

Just as when an intraarticular meniscus is injured in a joint in an extremity with resulting mechanical blockage of motion, I do believe that an injured disc blocks facet joint movement at the level of injury. This would result in joint dysfunction and the back pain from this should be and is relieved by joint manipulation. The sacroiliac joints are synovial and may be the seat of pain from any pathological joint condition of which one is joint dysfunction. The costovertebral joints are synovial and are frequently a source of pain in the back with or without radiation through or around the chest. The atlanto-occipital and atlanto-axial junctions have no discs, yet identical syndromes of pain arise from these junctions when they are affected by trauma and disease as they do from junctions where discs are present. The same thought pertains to the sacrococcygeal junction.

In the third quarter of the 20th century the Rockefeller Foundation sponsored a meeting to promote manipulation (amongst other things) called "Toward Better Understanding." This was between 1960–62. Unfortunately no better understanding resulted from it. In 1968 Secretary Wilbur Cohen's report to the U.S. Congress on Independent Practitioners under Medicare (page 197) advocates research in manipulation. This recommendation, to my knowledge, was never acted on. There have been two other potentially noteworthy developments in medical manipulative medicine during this period.

The first was the founding of the International Federation of Manual Medicine some 14 years ago. Since then there have been four international conferences sponsored by the Federation. Sixteen countries sent delegates to the Fourth International Meeting in Prague in 1974.

Until 1974 the Federation required that membership in the participating national societies should be limited to doctors of medicine. When one realizes that most of those who practice manual (manipulative) medicine were trained by osteopaths—some by chiropractors—we see this constitutional requirement as prohibitive rather than permissive: the teachers were excluded from membership by it. In 1974 the constitution was changed so that the membership of national associations might include American-trained osteopaths.

At the four conventions sponsored by the International Federation, little significant research was reported and the programs have almost exclusively been geared to spinal manipulation. Little attempt has been made by speakers to establish terminology or to elucidate the why and what of manipulation.

This leads me to another thought about the development of manipulation. It has been apparent through the years that many in the medical and paramedical professions who embrace manual medicine come to rely on the use of manipulative therapy exclusively in their practice. Except when treating pure joint dysfunction, it is my belief that anyone who thinks he can cure anyone of anything in musculoskeletal medicine by the use of one modality of treatment is being unrealistic.

If a patient suffers from a joint problem for any length of time you can be sure there are changes in the muscles, in the vascular system, in the lymphatic system, in the nervous system and in the integument system. It is not unlikely that he may also develop organ system dysfunctions and psychological embarrassments as well. Manipulation then becomes part of a comprehensive medical treatment program.

The second development which may turn out to be noteworthy has been the founding of an International Association of Manipulating Therapists in the past year. I am not convinced that the organizers are proceeding in a practical way. There is a certain defiance in this development. This is not surprising, since physical therapists have been excluded by those practicing physical medicine. I ask: what is manipulation if not a modality of physical therapy. I would tend to welcome therapists in order to avoid more fragmentation in medical care.

Historically, there has been little significant research in manipulative medicine, though recently there have been some thought-provoking contributions on the subject. In the sixties Cailett demonstrated that radiographically it is impossible to detect opening of a facet joint in the denuded lumbar spine even when one is propped open. Though this is a negative observation, it verifies the relative uselessness of diagnostic radiology in assessing changes in the interlaminar facet joints in the spine.

I mentioned earlier the suggestion by Wolf that there are intraarticular menisci (or at least meniscoid

material) in the facet joints of the spine. This hypothesis seems to have been contrived to explain certain clinical facts. In a personal communication, LeCore reported that he has never seen such a meniscus in over 300 dissections. I myself never saw such a structure when I was training in orthopedic surgery and operating on backs. But Dr. Wolf has contributed convincing material on the reversible deformation of joint cartilage by pressure, which doubtless has an adverse mechanical effect on joint motion. An English industrial physician, Glover, has recently performed arthography of the facet joints in the lumbar spine in post mortem subjects. He showed that there is a large superior aspect of the capsule which can be blown up by the injected fluid. Could the meniscoid of Wolf be the crenated, undistended capsule which may be pinched between the articulating facets?

Finally, a number of preliminary reports on clinical observations regarding the interplay of dysfunctions in the spine and organ dysfunctions were presented at the recent Prague meeting by European workers. Belatedly more clinical research is being undertaken especially in Czechoslovakia.

The National Institutes of Health provided 107 pages of material to help develop my thesis for this presentation. The material was based on 250 references to the literature; this must be an incomplete reference list, as there is no reference to my own books!

From this material I judge that a large number of orthopedic surgeons have advocated spinal manipulation. However, from personal observation, they cannot or do not define the term manipulation; neither are they specific in their indications for its use.

I have not given credit to several people in this audience who merit a place in the history of the development of medical manipulation. Dr. Maigne of Paris is foremost in introducing the teaching of manual medicine into the curriculum of a university medical school. Of comparable stature in education is Dr. Lewit of Prague who is not present.

Some may consider it unbecoming to belabor Mennell in this paper. I could find nowhere else to turn for recorded history. I have omitted many names popularly associated with the development of medical manipulation because their names are associated mainly with therapeutic techniques. It is immaterial, as I mentioned earlier, how one manipulates so long as the movements used in therapy are normal and confined to the restoration of lost joint play movements.

If we are to get anywhere in our deliberations we must avoid the use of named techniques. Please let us talk about joint manipulation and not spinal manipulation. Spinal manipulation has the connotation of topographical manipulation. There can be no such thing. Further, I do not believe that one should be taught spinal manipulation until proficient in handling the joints of the extremities. If teaching in manipulation is limited to spinal joints, then error is perpetuated, since such teaching implies that etiological factors in back pain are special and different.

In the late 19th century Sir James Paget stated: "Few of you will enter into practice today without having the so-called 'bone-setter' as a competitor. There is little point in presenting a lecture on the injuries which these persons cause; it is more important to consider the fact that their treatment can do some good. . . . Learn then to imitate what is good and avoid what is bad in the practice of bone-setters. . . . It is advisable to learn from one's opponent."

In a 1925 editorial in the *Lancet* about the work of Sir Herbert Barker, England's most noted bone-setter, the editor states, "The medical history of the future will have to record that our profession has greatly neglected this important subject (manipulation) . . . the fact must be faced that bone-setters have been curing multitudes of cases by movement (manipulation) . . . and that by our faulty methods, we are largely responsible for their very existence."

In 1889, Dr. William Osler made himself unpopular by trumpeting that there are sent out yearly scores of men called doctors who have never attended a case of labor and who are ignorant of the ordinary everyday diseases which they may be called upon to treat. In those days virtually all doctors learned their trade by apprenticing themselves to well established practitioners. The pros and cons of manipulation today depend upon the dogma of well established practitioners.

I close with one final remark. In an editorial in the *Archives of Physical Medicine* in 1947 the editor writes, "While he (James Mennell) is with

us in this country (America), we should take advantage of his special skills and experience to develop further the science and art of joint manipulation." Here we are in 1975—28 years later and I say: "I hope before I die that someone somewhere will take advantage of anyone's special skills and experience to develop the science and art of joint manipulation." Maybe some of you will be the people to do so and maybe Bethesda will be the place where it is achieved.

History of the Development of Chiropractic Concepts; Chiropractic Terminology

JOSEPH JANSE

President, The National College of Chiropractic
Lombard, Illinois

Contemporary definitions of chiropractic are:

1. "Chiropractic is the study of problems of health and disease from a structural point of view with special consideration given to spinal mechanics and neurological relations, presenting the hypotheses that:
   (a) disease may be caused or aggravated by disturbances of the nervous system.
   (b) disturbances of the nervous system may be caused by derangements of the musculoskeletal structures." [1]
2. "Chiropractic is a discipline of the scientific healing arts concerned with the pathogenesis, diagnostics, therapeutics and prophylaxis of functional disturbances, pathomechanical states, pain syndromes and neurophysiological effects related to the statics and dynamics of the locomotor system, especially of the spine and pelvis."[2]
3. "Chiropractic is the science concerned with defects in the mechanics, statics and dynamics of the human body".[3]
4. A definition by exclusion relates to chiropractic as "the system or method of treating human ailments without the use of drugs or medicines and without operative surgery." [4]

Development of Chiropractic Concepts:

Early chiropractic concepts were formulated during the time that Golgi, Cajal, Sherrington and Pavlov were thoroughly investigating the nervous system and making outstanding contributions.[5] It must be assumed that some of the early concepts such as Stephenson's model resembling the reflex arc [5,6] were influenced by these workers.

Original hypothesis: The original hypothesis of chiropractic was formulated by D. D. Palmer (1845–1913), a self-educated manipulative practitioner who found that various complaints of his patients recovered following vertebral adjustments.[5–9] A concept of homeostasis was expressed as follows:

> "Life is the expression of tone. Tone is the normal degree of nerve tension. Tone is expressed in functions by normal elasticity, activity, strength and excitability of the various organs, as observed in a state of health. Consequently, the cause of disease is any variation of tone."

Having very likely been influenced by the neurological thinking of the mid-19th century,[5,7,8] he believed that the nerve impulse originated in the brain and traveled as a vibrational wave[5,7] and could be influenced by intervertebral derangements causing facilitation or inhibition:

> "Displaced vertebrae, by impinging or stretching, cause contraction of nerve tissue—Tension more or less than normal causes an increase or decrease of vibration . . ."

This crudely presented concept of facilitation and inhibition was postulated by Palmer to be responsible for lowered tissue resistance of the innervated tissues, making an individual more vulnerable to infection and disease.[6–9] Probably under the influence of the vitalists, and with the concept of the "vis medicatrix naturae" still prevalent at the time,[10] he postulated the existence of an "innate intelligence," seated in the brain, and

597-036 O - 76 - 3

also the origin of the "mental impulse," traveling via the nerves.

It may further be stated that he had essentially a holistic concept, and therefore a Coan approach to the healing arts.[7]

B. J. Palmer (1881–1961), the son of D. D. Palmer and a very prolific chiropractic author, wrote the first book on the subject (1906).[9] He was obviously influenced by his father's thinking and hindered by his own lack of contact with the scientific community.[5] He placed undue emphasis on the concept of "innate intelligence" and still talked about mental impulses,[9, 11–15] but with recognition of their electrical properties which he attempted to measure.[11] He made technic procedures more specific by adjusting segments at the neuromeric levels from which specific organs are innervated, called the meric system (1908–1920),[12–14] and eventually settled on the upper cervical region as the one area that would be the key to all disease (1934).[5, 15]

The Nerve Compression Hypothesis:

Since the earliest beginnings of chiropractic, the theory that a nerve could become compressed through impingement from intersegmental spinal derangements has been given a great deal of attention,[7, 9, 16–23] and even thought to be a primary cause of disease.[9, 22] The chiropractic literature makes extensive reference to the importance of intervertebral foramen nerve root compression,[23–47] and to changes resulting from such compression.[28, 30, 31, 36, 45, 46]

Other investigators supplied bases or supporting evidence for the chiropractic compression theory by proposing possible mechanisms of compression,[48–68] and elucidating upon the effects of such compression.[69–105] Some investigators became involved in studying the phenomenon with an awareness of chiropractic hypotheses (Haldeman, Sharpless, McGregor, Luttges).[81, 82, 94, 96–100]

While extensive work has been done on the effects of nerve compression,[69–105] the exact mechanism of compression at various spinal and extravertebral sites leaves a great many unanswered questions. The influence of disturbed dural biomechanics, root sleeve fibrosis and adhesions appear to be promising areas of investigation.[35, 63–68]

The Proprioceptive Insult Hypothesis:

Proprioceptive insult is a concept referred to extensively in chiropractic writings[3, 8, 23, 29–31, 34, 35, 37, 40, 46, 47] to describe the undue irritation and stimulation of sensory receptors (to include proprioceptors) in the articular structures and parasegmental ligaments under stress from derangement of the intervertebral motor unit due to subluxation. It is thought that this afferent barrage into the nervous system could disturb equilibrium, cause aberrant somatovisceral, somato-somatic and somato-psychic reflexes as expounded upon in subsequent sections.

The Somatosympathetic Reflex Hypothesis:

The somatosympathetic reflexes have received extensive and repeated attention by the profession, and were postulated to be in part responsible for the visceral changes often observed after spinal adjustment, manipulation, trigger point and spondylotherapy.[3, 7, 8, 16–23, 47, 106–129] Chiropractic treatment aimed at influencing specific neuromeric levels has for years been known as the meric system, not unlike an approach, later referred to by Kunert (130). Evidence on these reflexes has been presented by Drs. Sato, Korr, Denslow and others.[131–167]

The Viscerosomatic Reflex Hypothesis:

Viscerosomatic reflexes have important diagnostic connotations for clinicians in most fields. These have been inferred to be important causes of subluxations,[3, 8, 23, 29, 35–37, 40, 106, 112, 113, 117, 118, 121] triggerpoints,[117, 118, 168] and acupunture points.[117, 154, 168, 169] Not only are these reflexes related to referred pain,[158, 168, 170–174] but are also responsible for spasm of the deep paraspinal musculature, causing asymmetrical muscle spasm,[183] and lead to the establishment of vicious cycles and even dermal changes.[8, 10, 23, 26, 34, 35, 45, 47, 55, 110, 111, 117, 118, 123, 128, 131, 138, 158, 163, 168–183] It is for this reason that it is important and necessary that in the overall management of patients with visceral symptoms and if only to break up aberrant cycles, manipulative therapy be made available, if necessary, in collaboration with physicians of other disciplines.

The Somato-somatic Reflex Hypothesis:

This mechanism may account for clinical results obtained through the manipulative treatment of patients with certain types of intractable somatic pain, triggerzones, and neurogenic dermatoses.[4, 5, 8, 10, 23, 24, 38, 44, 61, 109, 115, 117, 118, 125, 135, 153, 154, 184, 185, 187, 188–190, 193, 195, 198] Empirical clinical results lead us

to believe that this would be a useful area for investigation.

The Somatopsychic Hypothesis:

Somatopsychic relationships of clinical importance have been suggested by various chiropractic investigators.[199-212] The interrelationship between disturbed body mechanics, posture and psychological problems has been commented upon[199-203, 206-209, 212-216] and the chiropractic treatment of emotionally maladjusted individuals studied.[209, 212, 214] The results were encouraging and offer interesting possibilities for future investigation. The hypothesis has been presented that spinal subluxations have inimical effects upon the ascending pathways of the reticular activating system, causing stress-producing symptoms such as insomnia and restlessness.[201, 209]

The Neurodystrophic Hypothesis:

In its simplest form it may be reduced to D. D. Palmer's concept that "lowered tissue resistance is the cause of disease" and due to "too much or too little nerve energy".[7] The concept of lowered tissue resistance is strongly brought out by Dubos,[217] and, extensive studies done by Speransky [218, 219] and others[220] illustrate the important role of the nervous system in the control of immunogenesis.

Studies on the neuroreflex mechanisms regulating phagocytosis; electrophysiological changes in nerves during antigen stimulation of the skin, and other mechanisms elucidating this role of the nervous system,[218-220] often referred to in the chiropractic literature,[221-231] definitely require investigation.

Effects of stress, due to subluxation and its concomitants,[232, 233] require much future attention in view of results obtained by Selye and others in studying closely related mechanisms.[234-237]

Development of Biomechanical Concepts:

Biomechanical concepts in chiropractic developed through three phases, each characterized by related neurological concepts. The mechanical phase, introduced by D. D. Palmer, found expression in the concept of vertebral subluxation as a focus of nerve irritation and impingement.[3, 11, 15] Subluxations of the spine could be diagnosed through palpation and corrected through adjustment.

Clinicians and investigators have developed methods for analysis of subluxations of the spine, pelvis and extremities, utilizing various systems of mensuration.[238-267] The upper cervical area probably has received the greatest attention, both in analysis and treatment.[238, 239, 241, 249-250, 252, 253, 255, 257, 259] Tomographic,[266, 267] two dimensional[238, 239, 241-267, 269] and three dimensional, computer-assisted systems of roentgenologic measurement have been developed.[96, 100, 240, 268]

Empirical results as well as extensive clinical use of the methods of subluxation complex analysis dictate further investigation in this area. The system of measurement used by Suh[259] and the stereometric (orthogonal) system used by Frigerio and others hold greatest promise.[240, 268]

Phase two, concerned with the statics, was a contribution of Carver[3, 17, 269] who recognized the interrelatedness of all parts of the skeletal frame and considered the subluxation in a larger context. He emphasized gravitational strain and presented the all important principle of basic and compensatory distortion. It involves postural evaluation of total spinal biomechanics in terms of the static weight-bearing position in man, the biped, both through clinical postural and spinographic analyses to determine how the individual copes with the forces of gravity.[270, 309]

A four-way scale, developed by Illi;[288] employs the principle that rotational distortion of the spine can be expressed in terms of maldistribution of weight through the soles and heels, each placed on four separate scales read simultaneously. Use of a single scale for determinations of the center of gravity in various postures has also been initiated by Jenness.[291]

Recording and mensuration of spinal curvatures through the use of mechanical devices has also been done with the rachiometer[288] (and correlated with radiographic findings), the conformateur,[291] the posturometer[275, 276, 292-294, 295, 305] and Distortion Analyzer;[273] the latter two being used to arrive at a postural stress index (PSI), the posturometer having been tested on large samples.[275, 305, 306] Additional investigations in this area are needed. Systems of spinographic analysis involving full body and full spine radiographs were developed and warrant further investigation.[310, 330]

The phylogenetic and pathomechanical implications of bipedalism have been investigated[332-334, 338] and special attention given to the associated syndromes of postural faults and aberrant muscle conduct.[331-338] Future studies in this area would be significant to the clinician.

Mechanical problems induced by developmental defects and asymmetry have been studied [339-348] and the hypothesis formulated are: possible facilitation of afferent input from the articular facet joints; conversion of amphiarthrodial sacroiliac joints (primate) into a synovial arthrodial mechanism (Homosapiens) vulnerable to subluxation; the production of undue tension in the erector spinae muscle groups because of eccentric function of the motor units; articular instability in case of developmental defects and their possible role in disc syndromes, spondylotic change, disturbance of postural, righting and gait reflexes (especially in advancing years), neuralgias, intervertebral nerve root entrapment, reactionary fibrosis and formation of adhesions in the articular elements and myofascial planes-producing triggerpoints.

The dynamic or functional approach designates that phase concerned with the skeletal framework, especially the spine and pelvis, as a complex unit where weight-bearing, maintenance of posture and locomotion are tied in with the behaviour of the nervous and vascular systems, and the physiology of the viscera and even the emotive processes.[349-382]

Illi [278-288] was mainly responsible for the development of the concepts and instrumentations of this phase. He developed the orthodyn, a specialized treadmill allowing locomotion on a selective incline towards the cephalad or caudad end with concomitant lateral tilt to either side, enabling the investigator to observe alterations in kinematics as the center of gravity is caused to shift.

From studies involving some or all of the four-way scale, the orthodyn, rachiometer, cineradiography, mechanical and cadaveric models, and the torsolocomotor treatment device (Illi), the following observations were evolved:

> Sacroiliac movement does take place [278,350,383,387] (confirmed by Frigerio et al., [240] and it occurs with a figure of eight configuration. The sacroiliac mechanism functions as a gyroscope-like unit essential to effective spinal balance [278,279] concepts commanding further investigation because of its clinical importance.
>
> Torsion and flexion and torsion and lateral bending take place concommitantly, shown on cadaveric models [279, 288] and confirmed by Farfan;[388,389] a concept that warrants further study.
>
> That spinal deviation into a distortion takes place easier than out of a distortion [288] was confirmed recently.[391] This requires further elucidation, especially as it relates to the classification into six types of postural scoliotic deviation according to disturbed muscular dynamics.[288,392]
>
> The production of undue traction upon the dural root sleeves and the cauda equina by movement of the cervical spine with the occipito-atlanto-axial complex blocked together,[279,390] is an area that merits extensive further investigation because of the significance of disturbed nervous system biomechanics due to articular dysfunction.
>
> Determination of the locations of vertebral pivot points from which the bilaterally extending radii of vertebral movements arise, and which, if altered in length, size and position as in facet tropism will lead to the dysarthria of subluxation, should be further investigated because of its great clinical significance to the chiropractor and biomechanical importance.[279,383]

Development of Manipulative Procedures and Concepts:

Although it has been stated that the chiropractic technics were quite different from those used by any other profession,[9,12-14,370] it must be assumed that extensive interchange of ideas and methods took place.

The chiropractic manipulative format was probably broadened because of the influences of medical manipulators such as Hood, Mennel and Fisher,[394-396] as well as the osteopathic physicians [397] and the early American bone-setters who allied themselves with early chiropractors (Richer of Ohio, Reese of Pennsylvania and the Tieszens of the Dakotas):[398]

Traditionally, the chiropractic adjustment has been characterized by a direct thrust; an osseous process being utilized as a lever.[7,9] A straight arm-body drop procedure [16,19,21,23,114,242,399,403] or a very specific, highly developed toggle recoil [12, 15,16,23,399,403] or torsional or leverage technics [16, 19,21,23,114,242,279-284] are used. Technics have been developed and adapted to specific mechanical pathologies [15, 241, 249, 250, 266, 299, 353, 365, 370, 399, 401, 402

and for the treatment of specific syndromes.[61, 106, 120, 175, 187, 270, 288, 300, 341, 353, 370, 401, 402, 406-426]

Soft tissue procedures have been developed, such as triggerpoint, pressure point and reflex technics for physiological input and perhaps utilizing a mechanism such as dispersal of triggerpoints (Nimmo), reciprocal inhibition of muscles (Goodheart) or a gate control mechanism.[427-437]

The concept of somatic physiological input, as an aid in pain control, relief of tensions, muscle spasms, the management of visceral neuroses, and the dissipation of the circulus vitiosus of disturbed neurological function is not new to chiropractic thinking, but requires extensive future investigation. Procedures used have included paraspinal vibratory manipulations, pressure contact triggerpoint goading, pressure and massage.[435] Locke foot techniques and the plantar reflex concept and similar procedures have been used by different disciplines.[438] The mechanism involved is suggested to be that of restoration of exaggerated reflexes to homeostatic balance by affecting minute individual sites in the skin, deep fascia and muscle.[115, 118, 163, 435]

Autonomic Balancing: It has been hypothesized that the sympathetic or parasympathetic systems may be selectively influenced by specific types of adjustments at specific levels of the spine, resulting in autonomic balancing.[439-442] This approach is in part based on somatotyping.

Upper Cervical Specific: This involves specific adjustment of the upper cervical segments with postulated effects upon the vagus and other neurological elements,[249, 252, 253, 365, 421] and has been used as an exclusive approach.[15, 208, 285, 239, 241]

The neuromeric or meric approach: This has been used extensively and involves adjustment, manipulation or percussion at the neuromeric level from which an organ is innervated for the purpose of influencing that organ.[16, 26, 36, 89, 180] The question is raised whether or not the nerve cells within the dorsal, anterior and lateral gray cell columns are stimulated in addition to the dorsal root ganglion and proprioceptors in the articular structures.

Manipulation of the extremities has been practiced since the earliest beginnings of chiropractic and is still extensively practiced as an adjunct to spinal manipulation for the restoration of biomechanical integrity reflecting on the spine. Manipulation of visceral structures through the parieties and various spine-related muscles (psoas, piriformis) is also commonly practiced.[443-454]

Development of Analytical and Therapeutic Instrumentation:

Technological advances:

1. Analytical equipment—As early as 1920 Evins[455, 456] endeavored to use sensors in determining possible sites of disturbance of the neurovascular elements attending subluxations. Progressively various devices became available, the most popular being differential thermocouples employed for measuring symmetrical joint temperature differences across the spine[455-458] galvanometers[459] and infrared devices.[460-467] Unfortunately, overclaim by users placed the usage of some of these under question by some chiropractic clinicians as well as government agencies.[466-468] Conversely, utilization of infrared photography has become commonplace in clinical practice.[462, 466, 467] Other instruments have been referred to above (Illi[288]).
2. Therapeutic equipment—Distinct contributions to the manipulative art were made through the development of various adjusting tables enabling the physician to make effective and exacting corrective movements.

Outstanding tables were produced (McManis, B. J. Palmer, Thompson and others). Tables have been designed for correction of subluxations in specific areas such as C1 and C2 (Palmer, Thompson, Puckett) cervicals (Gonstead) thoracic and thoraco-lumbar area (Gonstead). The rehabilitation equipment of Illi has been referred to.[288, 470-472]

Development of Procedures Adjunctive to Manipulation:

Various adjunctive procedures have been used either preparatory or supplementary and supportive to adjustments and manipulations, or for pain control and the treatment of various conditions. These adjunctive procedures have been collectively called physiological therapeutics[473] and include electrotherapy, actinotherapy, hydrotherapy, orthotics, mechanotherapy and traction, spondylotherapy, massage, clinical nutrition, dietetics, fasting and herbology.[473-487]

Conclusion:

Chiropractic has become the subject of investigation of people from outside the healing arts profession such as Dr. Wardwell and others [489, 490] and has spread over many parts of the world with reputable texts appearing in many languages.[491-496] Honest and intense investigation into the science and art has been made by eminent members of the medical community in different countries,[497-505] some presenting us with astounding clinical observations holding great promise for future study.

There is a growing body of information suggesting that the functional role of inhibition and facilitation is profound in all sorts of neuronal subsystems. In view of the widespread effects of spinal manipulation on the body, it seems probable that the central nervous system is the arena of these effects even though a direct relationship between many of these changes and the site of manipulation is often obscure. The effect(s) of biomechanical aberrations (subluxations) on the phenomenon of inhibition in the central nervous system, and on the modulation of neuronal activity deserve extensive investigation. Information about spinal, pelvic and costal biomechanics and the pain phenomenon should be greatly augmented to maintain a sound rationale for corrective, preventive and rehabilitative procedures, applied in the best interest of the sick – the purpose of our deliberations.

It might well be that as this workshop proceeds, a commonality of interest, concept and knowledge will be brought into profile and provoke the mindfulness that an interchange of observations may benefit everyone and generate an interprofessional dialog.

CHIROPRACTIC TERMINOLOGY

Adjustment: The chiropractic adjustment is a specific form of direct articular manipulation utilizing a short lever and characterized by a dynamic, forceful, high velocity thrust of controlled amplitude.

Basic Technique: A chiropractic method wherein the correction of the sacrum is considered to be of primary importance.

Chiropractic Analysis: Relates to making assessments of disharmonies especially along the course of the spinal column and its attachments, including physical and clinical diagnosis.

Discopathy: Any pathological changes in a disc.

Discogenic: Caused by derangement of an intervertebral disc.

Discopathogenic: Abnormal action or function of a disc resulting in a disorder or condition; originating because of disc degeneration.

Note: although not in common usage, this is etymologically correct.

Disease: Disease may be defined as merely a summation of chemical reactions that have gone wrong.

Boyd, W.: Textbook of Pathology. 6th ed., Lea & Febiger 1958, p. 12.

Dynamics: (1) That phase of mechanics which deals with the motion of material bodies taking place under different specific conditions (Dorland).

(2) Science which treats of matter in motion (Oxford).

(3) (Including statics, kinetics.) The science of actions of forces in producing motion or equilibrium (Oxford).

Dysarthric Lesions: (See dysarthrosis.) A restrictive term used interchangeably (Del Gliesch).

Dysarthrosis: The strict meaning of joint motion restriction without the neurological connotations. It refers to kinetics.

Dyskinesia: Impairment of the power of voluntary movement, resulting in fragmentary or incomplete movements, aberrant motion.

Functional: (1)a. Of or pertaining to the function of an organ – not structural, affecting functions only (Oxford).

b. Of, or pertaining to a function: affecting the functions but not the structures (Dorland's).

(2) Joint mechanics showing area disturbances of function without structural change . . . subtle joint dysfunctions affecting quality and range of joint motion, with no obvious attending tissue changes. They are diagnosed with the aid of motion palpation, stress and motion radiography investigation. They are the best indication of altered joint physiology and are usually completely reversible. Nothing is out of place, so there is no subluxation or misalignment in the medical sense. There will be fixations (usually partial) and erratic motions within the normal range of joint movement.

Funiculitis: Spinal nerve irritation within the I.V.F. leading to radicular syndromes, e.g. – a lateral disc protrusion within the I.V.F. produces a funiculitis: osteophytic spurs may cause a chronic funiculitis leading to a local neuropathy.

HIO: Hole-in-one technique, first introduced by B. J. Palmer, wherein the adjustment of the atlas in relationship to the occiput and the axis is considered to be of primary importance.

Innate Intelligence: The intrinsic biological ability of a HEALTHY organism to react physiologically to the changing conditions of the external and internal environment. Probably derived from the term "vis medicatrix naturae".

Intervertebral Motor Unit: The anatomical elements uniting two adjacent vertebrae, including disc, all ligaments and soft tissues in which neurological elements are found.

(Note): (1) Under pathomechanics these neurological elements generate neuropathogenic reflexes.

(2) Since visualization of osseous structure is our only means of determining the status of the motor unit, it would be appropriate to include such structures in any discussion of the motor unit.

Listing: Designation of the spatial orientation of vertebrae in relation to adjacent segments: e.g., rotational or flexion malposition.

Manipulation: A maneuver employing long levers for the active, passive, and resistive movement of the body without the use of a dynamic thrust, aimed at remobilizing parts of the vertebral column.

Meric System: The treatment of visceral conditions through adjustment of vertebrae at the levels of neuromeric innervation to the organs involved.

Myodysneuria: A term for triggerpoint designating its neurological implications.

Nerve Tracing: The method of tracing the tenderness along the peripheral sensory extensions of spinal nerves back to the spinal level of subluxation, employed since the beginning of chiropractic, and not unlike the tracing of tenderness of a radiating neuralgia.

Neurodystrophic Processes: The production of diseases in various organs by irritating the central nervous system.

Malnutrition of tissues caused by the nervous system – it may occur in all organs and form part of every disease – Speransky.

Neurogenic: This word is often used to mean originating in nerve tissue, e.g., "the cause of the disorder is neurogenic." To meet this need see neuropathogenic.

Neuropathogenic: A disease within a tissue resulting from abnormal nerve performance, e.g., Barre-Lieou Syndrome resulting from neuropathogenic reflexes caused by pathomechanics of the cervical spine.

Neurophysiological Effects: A general term denoting functional or aberrant disturbances of the peripheral or autonomic nervous systems.

The term is used to designate nonspecific effects related to:

(a) Motor and sensory functions of the peripheral nervous system.

(b) Vasomotor activity, secretomotor activity and motor activity of smooth muscle from the autonomic nervous system, e.g., neck, shoulder, arm syndrome – the extremity becomes cool with increased sweating.

(c) Trophic activity of both the peripheral and autonomic nervous system, e.g., muscle atrophy in neck, shoulder, arm syndrome.

Pathomechanical States: Joint pathomechanics with structural changes . . . those architectural changes are the scars of imbalanced motion and weight bearing, trauma and biochemical changes associated with aging and deficiency states. These tissues changes may be revealed by static x-rays, biopsy, and definitely diagnosed with surgical exposure, e.g.,

(1) arthrosis
(2) spondylolisthesis
(3) disc degenerations

Prophylaxis: That branch of applied biology which seeks to reduce or eradicate disease by removing or altering the responsible etiologic factors. (Boyd's Preventive Medicine, 7th Edition.)

1. To prevent occurrence of subluxations due to poor postural hygiene, physical fitness and faulty body mechanics.

2. The prevention of recurrence with followup care, e.g., exercise.

3. Many subluxations are not curable and become quiescent with treatments; therefore, followup care to prevent further pathology, or at least to retard the pathomechanical process, is necessary.

Reflex Therapy: Treatment that aims at stimulating afferent impulses and evoking a given response, i.e., neuromuscular.

Retracing: Sometimes, following adjustments, patients redevelop earlier symptoms which have disappeared under previous treatment, usually of a suppressive nature.

Soft Tissue Technique: Manipulation of the muscles and fascia.

Short Leg: An anatomical, pathological, or func-

tional leg deficiency leading to a syndrome.

Statics: That phase of mechanics which deals with the action of forces and system of force on bodies at rest (Dorland).

—At rest—in equilibrium pertaining to laws of status.

—Pertaining to forces in equilibrium, or to bodies at rest (Oxford).

—The science that deals with bodies at rest or at equilibrium relative to some given state of reference.

—Relating to the weight and its mechanical effects and to the conditions of equilibrium (Oxford).

Spinography: A system of detection of subluxation by geometric analysis of radiography usually taken in the weight bearing position.

Spondylotherapy: The therapeutic application of percussion or concussion over the vertebrae to elicit reflex responses at the levels of neuromeric innervation to the organs being influenced.

Subluxation: D. D. Palmer insisted that the word subluxation be applied to the intervertebral disrelationships amounting to less than a locked dislocation. He maintained at the same time that there is a functional response within the nervous system resulting from this structural disrelationship.

Subluxation: A subluxation is the alteration of the normal dynamics, anatomical or physiological relationships of contiguous articular structures. Inman, O. B.: Basic Chiropractic Procedural Manual, Des Moines, American Chiropractic Association, 1973.

REFERENCES

1. Dept. of HEW: Independent practitioner study, ad hoc consultant group, USPHS, session on chiropractic, Nov. 21, 1968. *Ace—Federal Reporters*, Washington, D.C., pp. 4, 17.
2. *Official Definition*, Canadian Memorial Chiropractic College, Toronto, Ontario.
3. Levine, Mortimer: *The Structural Approach to Chiropractic*, Comer Press, New York, 1964.
4. *Illinois Med. Practice Act.* Dept. of Education and Registration, Springfield, Ill., p. 11, 1968.
5. Haldeman, S., and Hammerich, K.: The evolution of neurology and the concept of chiropractic, *ACA J. of Chiro.*, VII, S-57, August 1973.
6. Stephenson, R. W.: *Chiropractic Textbook*, Davenport, Iowa, Palmer School of Chiropractic, 1948.
7. Palmer, D. D.: The science, art and philosophy of chiropractic—*The Chiropractors Adjuster*. Portland Printing House, 1910.
8. Homewood, A. E.: *The Neurodynamics of the Vertebral Subluxation*, Willoweate, Ontario, Chiropractic Publishers, 1968.
9. Palmer, B. J.: *The Science of Chiropractic—its principles and philosophies*, Vol. I, Davenport, Iowa, Palmer School of Chiropractic, 1906 and 1910.
10. Hoag, J., Editor: *Osteopathic Medicine*, New York, Blakiston, 1969.
11. Palmer, B. J.: *Our Masterpiece*, Davenport, Iowa, Palmer College Press, 1966.
12. Ibid: *The Philosophy and Principles of Chiropractic Adjustments*, Davenport, Iowa, Palmer School of Chiropractic, Vol. III, 1908–1911.
13. Ibid: *The Science of Chiropractic—Causes Localized*, Vol. IV, Davenport, Iowa, Palmer School of Chiropractic, 1908.
14. Ibid: *The Science of Chiropractic*, Vol. II, Davenport, Iowa, Palmer School of Chiropractic, 1913–1917–1920.
15. Ibid: *The Subluxation Specific—The Adjustment Specific*, Vol. XVIII, Davenport, Iowa, Palmer School of Chiropractic, 1934.
16. Beatty, H. G.: *Anatomical Adjustive Technique*, 2nd Ed., Denver, Colo., 1937.
17. Carver, W.: *Carver's Chiropractic Analysis*, Oklahoma City, 1921.
18. Firth, J. N.: *A Textbook on Chiropractic Symptomatology*, Rock Island, Ill., 1914.
19. Forster, A. L.: *Principles and Practice of Spinal Adjustment for the Use of Students and Practitioners*, Chicago, 1915.
20. Gregory, A. A.: *Spinal Adjustment*, Oklahoma City, 1910.
21. Loban, J. M.: *Technic and Practice of Chiropractic*, Davenport, Iowa, 1912.
22. Logan, H. B.: *Textbook of Logan* Basic Methods Edited by V. F. Logan and F. M. Murray, St. Louis, 1950.
23. Janse, J., Houser, R. H., and Wells, B. F.: *Chiropractic Principles and Technic*, Chicago, National College, 1947.
24. Baker, F. A.: Speaking of subluxations, nerve pressures may exist away from the spine. *Chiro. J.* 8: Vol. IV, August 1935.
25. Coggins, W. N.: *Basic Technique*, Willowdale Ontario, Elco Pub., 1968.
26. Eisenberg, A.: An analytical study of the effects of nerve interference, *J. National Chiro. Assoc.*, Vol. XXX, p. 20, August 1960.
27. Green, L.: The intervertebral foramen in vertebral subluxation, *J. National Chiro. Assoc.*, XXVII, p. 13–14, January 1957.
28. Haldeman, S., and Drum, D.: The compression subluxation, *J. of Clin. Chiro., Archive* Ed. 1:10–21, Spring 1971.
29. Hayes, S.: The chiropractic subluxation—a new hypothesis for consideration (a series of seven publications), *J. National Chiro. Assoc.*, Vol. XXVII, pp. 9–12, July 1957.
30. Higley, H. G.: Physiology of a subluxation, Chirogram, Los Angeles College of Chiro., December 1966.
31 Ibid: How serious is a subluxation?, *ACA J. of Chiro.*, VII, S-41, June 1968.
32. Hviid, H.: A consideration of contemporary chiropractic theory, *J. National Chiro. Assoc.*, Vol. XXV, pp. 17–18, January 1955.
33. Janse, J.: Segmental disturbances and pathologies which cause cord and spinal nerve pressures, National Coll., *J. of Chiro.*, p. 5, December 1939.
34. Idem: A report on 500 cases of low back and sciatic involvement, Ibid, 27 No. 2:3, September 1955.
35. Idem: An evaluation—parasegmental effects of vertebral subluxation, *J. of Clin. Chiro.*, 2:68, Spring 1968.
36. Keller, R. M.: The integrity of the cell when impaired by nerve interference, *J. National Chiro. Assoc.*, Vol. XXIV, pp. 19–21, December 1954.

37. Kimmel, E. H.: A treatise on neurological implications of distortion. *J. National Chiro. Assoc.*, Vol. XX. 13–15, August 1950.
38. Nilsson, A. V.: The physical relationship between the heads of the ribs and the nervous and vascular content of the intervertebral foramina of the typical thoracic vertebrae, *Monograph*, Dept. of Anat., Los Angeles Coll. of Chiro., April 1937.
39. Idem: A few remarks on the intervertebral foramen, *Chirogram*, Los Angeles Coll. of Chiro., January 1955.
40. Raskin, L.: A scientific basis for chiropractic, *J. National Chiro. Assoc.*, Vol. XIX, p. 31, May 1949.
41. Rich, E.: Another instance of medical corroboration of chiropractic principles, *J. National Chiro. Assoc.*, Vol. XXII, p. 13, October 1952.
42. Schlenoff, M. B.: Electrotactic theory of chiropractic, *J. National Chiro. Assoc.*, Vol. XXIX, pp. 9–11, January 1959.
43. Scofield, A. G.: *Chiropractic*, London, Thorsons, 1968.
44. Sjoman, E. W.: The role of the nervous system in health and disease, *J. National Chiro. Assoc.*, Vol. XXVIII, pp. 24–26, July 1958.
45. Sorkin, H. M.: Intervertebral foramen diminution, *J. National Chiro. Assoc.*, Vol. XX, pp. 13–14, December 1950.
46. Squire, H., et al.: A detailed study of the physiology of a subluxation, *J. National Chiro. Assoc.*, January 1954.
47. Weiant, C. W., and Goldschmidt, S.: *Medicine and Chiropractic*, 4th edition, New York, 1966.
48. Brain, L.: Some aspects of the neurology of the cervical spine, *J. Fac. Radiol.*, 1956.
49. Brain, L., and Wilkinson, M.: *Cervical Spondylosis*, London, William Heinemann, 1967.
50. Epstein, J. A., et al.: Sciatica caused by nerve root entrapment in the lateral recess – the superior facet syndrome, *J. Neurosurg.*, May 1972.
51. Frykholm, R.: Cervical nerve root compression resulting from disc degeneration and root sleeve fibrosis, *Acta. Chir. Scand. Suppl.*, 160, 1951.
52. Hadley, L. A.: Constriction of the intervertebral foramen, *J.A.M.A.*, 140:5, 1949.
53. Idem: Intervertebral joint subluxation, bony impingment and foramen encroachment with nerve root change, *Am. J. of Roent. and Rad. Ther.*, 65:377, March 1951.
54. Idem: Anatomico-roentgenographic studies of the spine, Springfield, Charles C Thomas, 1964.
55. Hirsch, C., and Zotterman, Y.: *Cervical Pain*, New York, Pergamon Press, 81–88, 1971.
56. Holt, S., and Yates, P. O.: Cervical spondylosis and nerve root lesions, *J. Bone. Joint Surg.*, 48B:407–423, 1966.
57. Jackson, R.: *The Cervical Syndrome*, Springfield, Charles C Thomas, 1966.
58. Kovacs, A.: Subluxation and deformation of the cervical apophyseal joints, *Acta Radiologica* 43, January 1955.
59. Overton, L.M.: Cervical spine nerve root pain, *Am. Surg.*, 17:343, April 1951.
60. Richter, H. R.: The significance of entrapment neuropathy for the differential diagnosis of vertebrogenic pain syndromes, Manuelle Medizin, Heft 5, September–October 1971.
61. Stoddard, A.: *Manual of Osteopathic Practice*, New York, Harper and Row, pp. 168, 203, 1969.
62. Wright, J.: Mechanics in relation to derangement of the facet joints of the spine, *Arch. Phys. Ther.*, 201–206, April 1944.
63. Brieg, A.: *Biomechanics of the Central Nervous System*, Chicago, Yearbook 1960.
64. Brieg, A.: Overstretching of and the circumscribed pathological tension in the spinal cord – a basic cause of symptoms in cord disorders, *Biomechanics* Vol. 3, 7–9, 1970.
65. Reid, J. D.: Effects of flexion-extension movements of the head and spine upon the spinal cord and nerve roots, *J. Neurosurg. Psychiat.*, 23:214–221, 1960.
66. Sunderland, S., and Brodkey, K. C.: Stress and strain phenomena in human spinal nerve roots, *Brain*, 84, 1961.
67. Sunderland, S., and Brodkey, K. C.: Stress and strain phenomena in the human spinal nerve roots, *Brain*, 84, 120, 1961.
68. Sunderland, S., and Brodkey, K. C.: Stress and strain phenomena in the human spinal nerve roots, *Brain*, 84, 125, 1961.
69. Allen, F. M.: Effects of ligations on nerves of the extremities, *Ann. Surg.*, 1938.
70. Bentley, F. H., and Schlapp, W.: The effects of pressure on conduction in peripheral nerve, London, *J. Physiol.*, 102:62, 1943.
71. Idem: Experiments on the blood supply of nerves, Ibid, p. 72, 1943.
72. Burns, L.: *Pathogenesis of Visceral Disease Following Vertebral Lesions*, Chicago, Amer. Ost. Assoc., 1948.
73. Causey, G.: The effect of pressure on nerve fibers, *J. Anat.*, 82, 1948.
74. Causey, C., and Palmer, E.: The effect of pressure on nerve conduction and nerve fiber size, *J. Physiol.*, 109, 1949.
75. Daniell, H. W.: Fusiform swellings on terminal portions of peripheral nerves, *J. Neuropath. and Exper. Neurol.*, July 1954.
76. Droz, B., and Leblond, C. P.: Migration of proteins along the axon of the sciatic nerve, *Science*, 137:1047–1048, 1962.
77. Friede, T. L.: Transport of oxidative enzymes in nerve fibers; a histochemical investigation of the regenerative cycle in neurons, *Exper. Neurol.* 1:441–466, 1959.
78. Gelfan, S., and Tarlov, I. M.: Differential vulnerability of spinal cord structures to anoxia, *J. Neurophysiol.*, 170, 1955.
79. Idem: Physiology of spinal cord, nerve root and peripheral nerve compression, *Amer. J. Physiol.*, 185:217, 1956.
80. Gerard, R. W.: The response of nerve to oxygen lack, *Amer. J. Physiol.*, 92:489–541, 1930.
81. Haldeman, S.: *Changes in the Structure and Function of the Sciatic Nerve* caused by constriction, (M.Sc.-thesis), University of Pretoria, 1969.
82. Haldeman, S., and Meyer, B. J.: The effect of constriction on the conduction of the action potential in the sciatic nerve, *S.A. Med. J.*, 903, 1970.
83. Hollingsworth, D., and Ochs, S.: Dependence of fast axoplasmic transport in nerve on oxidative metabolism, *J. Neurochem.* 18:107–14, January 1971.
84. Korr, I.: Axonal delivery of neuroplasmic components to muscle cells, *J.A.O.A.*, 68, 1968.
85. Lewis, T., and Pochin, E. E.: Effects of asphyxia and pressure on the sensory nerve of man, *Clin. Sci.*, April 1938.
86. Ochs, S.: The dependence of fast transport in mammalian

nerve fibers on metabolism, *Acta Neuropathol.* (Berl) 5:Suppl, 5:86–96, 1971.
87. Ochs, S.: Rate of fast axoplasmic transport in mammalian nerve fibers, *J. Physiol.* (Lond), 227:627–45, December 1972.
88. Ochs, S.: Fast axoplasmic transport of materials in mammalian nerve and its integrative role, *Ann. N.Y. Acad. Sci.*, 193:43–58, 25 Aug. 72 (80 ref.)
89. Olsson, Y.: Studies on vascular permeability in peripheral nerve. Distribution of intravenously injected protein tracers in the peripheral nervous system of various species, *Acta Neuropath.* (Berl.) 17, 114–126, 1971.
90. Porter, E. L., and Wharton, P. S.: Irritability of mammalian nerve following ischemia, *J. Neurophysiol.*, March 1949.
91. Ranish, N., and Ochs, S.: Fast axoplasmic transport of acetylcholinesterase in mammalian nerve fibres, *J. Neurochem.*, 19:2641–9, November 1972.
92. Sabri, M. I., and Ochs, S.: Relation of ATP and creatine phosphate to fast axoplasmic transport in mammalian nerve, *J. Neurochem.*, 19:2821–8, December 1972.
93. Sabri, M. I., and Ochs, S.: Characterization of fast and slow transported proteins in dorsal root and sciatic nerve of cat, *J. Neurobiol.*, 4:145–65, 1973.
94. Sharpless, Seth K., and Cobb, S.K.: *A Neurophysiological Study of the Effects of Compression on Spinal Roots*, Univ. of Colorado, Boulder, Colo., 1958.
95. Smith, C. B., and Ochs, S.: Fast axoplasmic transport in mammalian nerve in vitro after block of glycolysis with iodoacetic acid, *J. Neurochem.*, 18:833–43, June 1971.
96. Suh, C.H.: Editor, *Proceedings of the Fourth Annual Biomechanics Conf. on the Spine*, University of Colorado, Boulder, Colo., 1974.
97. Idem: Editor, *Proceedings of the Fifth Annual Biomechanics Conf. on the Spine*, University of Colorado, Boulder, Colo., 1975.
98. Suh, C. H., and West, H. G.: Biomechanics of the spine, Part I, ACA *J. of Chiro.*, VIII, 581–596, June 1974.
99. Idem: *Biomechanics of the Spine*, Part II, Ibid: VIII, 597–1120, July 1974.
100. Idem: *Biomechanics of the Spine*, Part III, Ibid: VIII, 5121–5133, July 1974.
101. Symposium Proceedings, Summary: Various opinions about the mechanism of axoplasmic transport, *Neurosci. Res. Prog.* Bull. 5, 1968.
102. Thompson, I. M., and Kimball, H. S.: Effect of local ischemia upon human nerve fibers in vivo., *Proc. Soc. Exper. Biol. and Med.*, June 1936.
103. Weiss, P.: Endoneurial edema in constricted nerve, *Anat. Rec.*, 86:491–522, 1943.
104. Weiss, P., and Davis, H.: Pressure block in nerves provided with arterial sleeves, *J. Neurophysiol.*, 6, 1943.
105. Weiss, P., and Pillai, A.: Conduction and rate of mitochondria flow in nerve fibers; axonal flow as vehicle, *Proc. Nat. Acad. Sci.* Wash. 54:48–56, 1965.
106. Amodei, J. E.: Causes contributing to headaches and how they can be corrected, *J. National Chiro. Assoc.*, Vol. XXX, pp. 21–23, December 1960.
107. Budden, W. A.: Spinal subluxations—usually result in visceral pathology, *National Chiro. J.*, Vol. XII, pp. 9–10, March 1943.
108. Eisenberg, A.: The suprarenal response to a chiropractic adjustment, *J. National Chiro. Assoc.*, Vol. XXVI, p. 22, November 1956.
109. Gregory, A. A.: *Spondylotherapy Simplified*, Oklahoma City, Palmer-Gregory College, 1915.
110. Haldeman, S.: Interactions between the somatic and visceral nervous system, *ACA J. of Chiro.*, August 1971.
111. Haldeman, S.: Spinal and paraspinal receptors, *J. Canada. Chir. Assoc.*, June–July 1972.
112. Harper, W. D.: *Anything Can Cause Anything*, Pasadena, Tex., Texas College of Chir., 1966.
113. Harper, W. D., et al.: Chiropractic of today, *ACA J. of Chiro.*, p. S–83, November 1973.
114. Howard, J. F. A.: *Encyclopedia of Chiropractic*, Vol. 3, Chicago, Ill., National School of Chiropractic, 1915.
115. Janse, J.: Study of reflex and pressure technics, *National Chiro. J.*, Vol. XVI, pp. 9–10, December 1946.
116. Janse, J.: A scientific treatise on clinical chiropractic measures—section one, *J. National Chiro. Assoc.*, Vol. XXII, pp. 11–12, December 1952.
117. Kleynhans, A. M., and Aarons, M. W.: Myodysneuria and acupuncture, *Digest of Chiro. Econ.*, September–October 1974.
118. Nimmo, R. L., and Vannerson, J. F.: Specificity and the law of facilitation in the nervous system, *ACA J. of Chiro.*, March 1973.
119. Overton, R. M.: Chiropractic treatment for decongestion of the liver, *J. National Chiro. Assoc.*, Vol. XXV, pp. 19–20, August 1955.
120. Schmidt, E. G.: A presentation of a mode of therapy for treating rib lesions, *J. National Chiro. Assoc.*, Vol. XXX, pp. 11–12, December 1960.
121. Smith, A. R.: An anatomic and physiologic basis of chiropractic, *ACA J. of Chiro.*, S–73, October 1970.
122. Stein, K.: The first specific adjustment—confirmed by latest scientific researches, *National Chiro. J.*, Vol. XVIII, pp. 16–17, October 1948.
123. Stonebrink, R. D.: Neurological features of the chiropractic philosophy, *ACA J. of Chiro.*, May 1968.
124. Verner, J. R.: *Science and Logic of Chiropractic*, 4th ed., Englewood, New Jersey, 1950.
125. Watkins, R. J.: Anthropology in reflex technics, *National Chiro. J.*, Vol. XVIII, pp. 14–15, August 1948.
126. Watkins, R. J.: The upper thoracic complex, *J. National Chiro. Assoc.*, Vol. XX, p. 33, November 1950.
127. Weiant, C. W.: Some observations on blood pressure, *National Chiro. J.*, Vol. XIV, pp. 13–14, November 1944.
128. Wayte, K. M.: Disturbances of the nervous system as related to chiropractic, *ACA J. of Chiro.*, II, S–73–78, October 1968.
129. Weiant, C. W.: Effect of chiropractic on metabolism—a preliminary report, *National Chiro. J.*, Vol. XVI, p. 17, March 1946.
130. Kunert, W.: Functional disorders of internal organs due to vertebral lesions, *Ciba Symp.* 13:3, 1965
131. Beilke, M. C.; the clinical relationship of structural inadequacy to the somatico-visceral syndrome, *Acac. Appl. Ost. Yearbook*, Carmel, Calif., 1962.
132. Burns, Chandler, and Rice, et al.: The pathogenesis of visceral disease following vertebral lesions, Chicago, Ill., *The Am. Ostero. Assoc.*, 1948
133. Burton, J. E., Bloedel, J. R., and Gregory, R. S.: Electrophysiological evidence for an input to lateral reticular nucleus from collateral of dorsal spinocerebellar and

cuneocerebellar fibers, *J. Neurophysiol.*, 34:885–897, 1971.
134. Cole, W. V.: The osteopathic lesion complex, *J.A.O.A.*, 3:60–66, 49, November 1949.
135. Coote, J. H., Dowman, C. B. B., and Weber, W. V.: Reflex discharges into thoracic white rami elicited by somatic and visceral afferent excitation, *J. Physiolo.*, 202:147–159, 1969.
136. Denslow, J. S., and Hassett, C. C.: Central excitatory state associated with postural abnormalities, *J. Neurophysiol.*, September 1942.
137. Denslow, S.: Analysis of variability of spinal reflex thresholds, *J. Neurophysiol.*, July 1944.
138. Dittmar, E.: Cutaneo-visceral neural pathways, *Brit. J. Phys., Med.* 15:208, 1952.
139. Gardner, E.: Pathways to the cerebral cortex for nerve impulses from joints, *Acta Anat.* Supp., (56–1, 73) 203–216, 1969.
140. Gillum, G. N.: The effect of spinal manipulation on the heart rate and the temperature of the skin surface, *J.A.O.A.*, July 1940.
141. Hargrave, Wilson W.: The somatovisceral reflexes, *J.A.O.A.*, December 1935.
142. Hirsch, C., and Zotterman, Y.: *Cervical Pain*, Pergamon Press, 179–193, 1971.
143. Judovich, B.: *Pain Syndromes Treatment by Paravertebral Nerve Block*, 3rd edition, Philadelphia, Davis, 1949.
144. Kamieth, H.: Pathogenetic importance of the thoracic portion of the vertebral, *Post. Grad. Med.*, J.A.M.A., November 1958.
145. Korr, I. M.: Symposium on the functional implications of segmental facilitation, *J.A.O.A.*, January 1955.
146. Idem: The somatic approach to the disease process, *Acad. Appl. Ost. Yearbook*, Carmel, Calif., 201–205, 1963.
147. Idem: The neural basis of the osteopathic lesion, Ibid: 49–58, 1963.
148. Idem: Clinical significance of the facilitated state, Ibid: 133–138, 1963.
149. Kugelmass, I., (Ed.) Newton. The physiologic basis of osteopathic medicine, Symposium October 7, 1967, *Post. Grad. Inst. of Osteopathic Med. and Surg.*, New York, 1970.
150. Kuntz, A.: Anatomic and physiologic properties of the cutaneo-visceral vasomotor reflex arc, *J. Neurophysiol.*, 1945.
151. Kuntz, A., and Haselwood, L.A.: Circulatory reactions in the gastro-intestinal tract elicited by local cutaneous stimulation, *Am. Heart J.*, 1940
152. Lyons, R. H., and Calvia, F. S.: The clinical manifestations of autonomic dysfunction, *N.Y. State J.M.*, August 1950.
153. Mann, P. L., and Chen, C. H.: Mechanism of acupuncture anaesthesia, *Dis. Nerv. Syst.*, 33:730, 1972.
154. Matsumoto, T.: Acupuncture for physicians, *Charles C Thomas*, 1974.
155. Nelson, J.: Reflex relationship of paravertebral muscles, *Am. J. of Physiol.*, May 1961.
156. Northup, T. L.: Manipulative management of hypertension, *Acad. Appl. Osteop. Yearbook*, 1965.
157. Northup, G. W.: Osteopathic lesions, *J.A.O.A.*, Vol. 71, 854–45, 864–55, June 1972.
158. Pottenger, F. M.: Symptoms of visceral disease, *Mosby*, 1953.
159. Richins, C. A., and Brizzee, K.: Effect of localized cutaneous stimulation on circulation in duodenal arterioles and capillary beds, *J. of Neurophysiol.*, 1949.
160. Sato, A.: Spinal and medullary reflex components of the somatosympathetic reflex discharges evoked by stimulation of the group IV somatic afferents, *Brain Res.* 51:307–318, Mar. 15, 1973.
161. Idem: Spinal and supraspinal inhibition of somatosympathetic reflexes by conditioning afferent volleys, *Pflueg. Arch.*, 336, 1972.
162. Sato, A., Kaufman, A., Koizumi, K., and Brooks, C. M.: Afferent nerve groups and sympathetic reflex pathways, *Brain Res.*, 14:575.
163. Skillern, Penn-Gaskell: Clinical observations on cutaneo-visceral (somato-sympathetic) reflex arcs; the rate of hypermyotonia in bodily aches and pains, *J. of Nervous and Mental Dis.*, May 1947.
164. Travell, J., and Rinzler, S. H.: Relief of cardiac pain by local block of somatic trigger areas, *Proc. Soc. Exp. Bio. and Med.*, 1946.
165. Ussher, N. T.: Spinal curvatures – visceral disturbances in relation thereto, *California and Western Med.*, Vol. XXXVIII, 423–428, June 1933.
166. Ussher, N. T.: The viscerospinal syndrome – a new concept of visceromotor and sensory changes in relation to deranged spinal structures, *Annals of Int. Med.*, May 1940, 427–432.
167. Weiss, S., and Davis, D.: The significance of the afferent impulses from the skin in the mechanism of visceral pain. Skin infiltration as a useful therapeutic measure, *Am. J. Med. Sci.*, Vol. 176:517, October 1928.
168. Gutstein, R. R.: A review of myodysneuria (Fibrositis). *Amer. Practitioner and Dig. of Treatment*, 6:570–77, April 1955.
169. Mann, F.: *Acupuncture*, revised edition, Vintage Books., 1973.
170. Leikind, E. R., and Harlin, H. C.: *Treatment of Silent Prostato-vesiculism in General Practice*, New York, Julian Press, Inc.
171. Lewis and Kellegran, J. H.: Observation relating to referred pain, visceromotor reflexes and other associated phenomenon, *Clinic Sc.*, June 1939.
172. Pottenger, F. M., and Ussher, N. T.: The viscerospinal reflex, *Calif. West Med.*, 38 June 1933.
173. Gibson, J. W.: Significance of spinal reflexes due to visceral disturbances, *Western Osteopath*, August 1925.
174. Reiss, E. H.: Visceral disorders and their relationship to backache, *J. National Chiro. Assoc.*, Vol. XXX, pp. 17–19, October 1960.
175. Watkins, R. J.: A chiropractic explanation of migraine and how it can be corrected, *J. National Chiro. Assoc.*, Vol. XXI, p. 22, May 1951.
176. Denslow, J. S.: An analysis of the variability of spinal reflex thresholds, *J. Neurophysiol.*, July 1944.
177. Head, H.: On disturbance of sensation with special reference of visceral disease, *Brain*, 1893–1894.
178. Keller, R. M.: Inhibitory reflexes – confirmatory evidence on chiropractic principles, *Chiro. J.*, Vol. X, pp. 13–14, June 1941.
179. Gimmler, J. and W., and Kimmel, E. H.: The substantiation of chiropractic by scientific research, *J. National*

Chiro. Assoc., Vol. XXII, pp. 20–22, July 1952.
180. Korr, I. M., and Goldstein, M. J.: Dermatomal autonomic activity in relation to segmental motor reflex threshold, *Fed. Proc.*
181. Idem: Skin resistance patterns associated with visceral disease, *Fed. Proc.*, March 1949.
182. Miller, F. R., and Waud, R. A.: Visceromotor reflexes, IV, *Amer. J. Physiol.*, 73:329.
183. Albe-Fessard, D.: Central pathways for noxious stimuli, Wenner-Gren *Int. Sympos. Series*, 1971.
184. Bessou, P., and Perl, E. R.: Response of cutaneous sensory units with unmyelinated fibers to noxious stimuli, *J. Neurophysiol.*, 32, 1969.
185. Burgess, P. R., and Perl, E. R.: Myelinated afferent fibers responding specifically to noxious stimulation of the skin, *J. Physiol.*, 190, 1967.
186. Bessou, P., and Perl, E. R., Burgess, P. R., and Taylor, C. B.: Dynamic properties of mechanoreceptors with unmyelinnated C fibers, *J. Neurophysiol.*, 34, 1971.
187. Foutz, C. A.: Your role in the relief of headaches of spinal origin, *J. National Chiro. Assoc.*, Vol. XXX, pp. 21–22, October 1960.
188. Hagbarth, K. E., Hongel, et al.: General characteristics of sympathetic activity in human skin nerves, *Acta Physiol. Scand.*, Vol. 84, 1971, 1972.
189. Heinbecker, P., Bishop, G. H., and O'Leary, J.: Pain and touch fibers in peripheral nerves, *Arch. Neurol. and Psychiat.*, 29, 1933.
190. Hirsch, C., and Zotterman, Y.: *Cervical Pain*, Peragamon Press, 157–164, 1971.
191. Josey, Izard: Headache associated with pathologic changes in the cervical spine, *J.A.M.A.*, July 1949.
192. Kellgren, J. H.: On distribution of pain arising from deep somatic structures with charts of segmental pain areas, *Clin. Sci.*, June 1939.
193. Perl, E. R.: Myelinated afferent fibers innervating the primate skin and their response to noxious stimuli, *J. Physiol.*, 197, 1968.
194. Sargent, J. W.: Chiropractic—present and future, *J. National Chiro. Assoc.*, Vol. XV, p. 14, June 1945.
195. Vallbo, A. B., and Hagbarth, K. E.: Activity from skin mechanoreceptors recorded percutaneously in awake human subjects, *Exp. Neurol.* 21, 1968.
196. Zotterman, Y.: Touch pain, and tickling; an electrophysiological investigation on cutaneous sensory nerves, *J. Physiol.* (London), 95, 1939.
197. Sullivan, J. E.: Backache due to visceral lesions of the chest and abdomen, *Clin. Orth.*, 26, 1963.
198. Keller, G.: The significance of changes in the small vertebral joints as cause of local back pain, *Zeitschrift für Orthopädie*, 83 : 67, 1953.
199. Amber, R. B.: Structural and neurological bases for chiropsychotherapy-II, *J. National Chiro. Assoc.*, Vol. XXVI, pp. 30–31, December 1956.
200. Boyce, W.: A psychological study, *Chiro. J.*, Vol. VI, pp. 42–43, June 1940.
201. Janse, J.: Interrelation between psychic disturbances and body balance, National Coll. *J. of Chiro.*, Vol. 14, 4:3–6, December 1941.
202. Janse, J.: Nerve reactions—causes of psycho-neurosis are outlined, *Chiro. J.*, Vol. XI, pp. 25–27, February 1942.
203. Janse, J.: Your emotions and your health are associated, *Chiro. J.*, Vol. XI, pp. 17–18, August 1942.
204. Hollod, M. L.: Theory and practice of chiropsychiatry, *National Chiro. J.*, Vol. XIV, p. 23, November 1944.
205. Killeen, J. T.: A discussion on emotions and their relation to the autonomic nervous system, *National Chiro. J.*, Vol. XV, p. 18, October 1945.
206. Klein, L.: Psychoneurotic manifestations in physical symptoms, *J. National Chiro. Assoc.*, Vol. XXVIII, pp. 32–33, July 1958.
207. LaLonde, R. J. H.: Hypersensitivity and its relationship to pathology—a new concept, *National Chiro. J.*, Vol. XVIII, pp. 26–27, July 1948.
208. Mears, Donald B.: Mental disease and cervical spine distortions, ACA *J. of Chiro.*, pp. 13–15, September 1965.
209. Schwartz, Herman S., Editor: *Mental Health and Chiropractic*, Sessions Publishers, New York, 1973.
210. Weiant, C. W.: The neuroses and the psychoses, *National Chiro. J.*, Vol. XII, pp. 14–15, September 1943.
211. Weiant, C. W.: The neuroses and the psychoses, *National Chiro. J.*, Vol. XII, pp. 11–12, October 1943.
212. Weiant, C. W.: A new chiropractic and postural approach to neuropsychiatry, *J. National Chiro. Assoc.*, Vol. XX, pp. 24–25, September 1950.
213. Feldenkrais, M.: *Body and Mature Behaviour*, International Universities Press, Inc., 1949.
214. Gutmann, G.: *Die Chiropraktik als Rationelle Therapie*, Hippokrates, Sept. 15, 1957.
215. Popielianski, Ia. Iu.: Some psychopathological manifestation of the vertebral artery syndrome in patients with cervical osteochondrosis, The Osteochondrosis of the Vertebral Column, *Novoksuetsk*, pp. 111–118, 1962.
216. Travell, J., and Bigelow, N. H.: Role of somatic trigger areas in patterns of hysteria, *Psychosom. Med.* 9:353–363, November–December 1947.
217. Dubos, Rene: *Biochemical Determinants of Microbial Diseases*, Cambridge, Mass., Harvard Univ. Press, 1954.
218. Speransky, A. D.: *A Basis for the Theory of Medicine*, New York, International Pub., 1943.
219 Freidrich, R.: *Frontiers of Medicine*, "The revolutionary pathology of A. D. Speransky," New York, Liveright, 1961.
220. Gordienko, A. N., Editor: *Control of Immunogenesis by the Nervous System*, (A compilation of 21 papers), Rostov-On-Don, Translation from Washington, Off. of Tech. Serv., U.S. Dept. of Commerce, 1958.
221. Edgars, N. K.: Speransky in light of developments, *National Chiro. J.*, Vol. XIV, pp. 21–22, March 1944.
222. Keller, R. M.: Considerations of the electronic factor in irritation, *J. National Chiro. Assoc.*, Vol. XXIII, pp. 13–14, February 1953.
223. Lee, H. A.: A concise summary of the work of A. D. Speransky, *J. National Chiro. Assoc.*, Vol. XXV, pp. 10–11, February 1955.
224. Nelson, G. E.: A study of chiropractic and the neurodystrophic process, *National Chiro. J.*, Vol. XVIII, pp. 30–33, December 1948.
225. Oleshey, I. H., and Parker, A. M.: Our basis for chiropractic survival, *National Chiro. J.*, Vol. XII, pp. 21–23, December 1943.

226. Schriber, T.: Neurophysiology triumphant, *J. National Chiro. Assoc.*, Vol. XIX, pp. 21–22, October 1949.
227. Verner, Weiant, and Watkins: *Rational Bacteriology*, Privately Published, Weiant, New York, Peekskill, 1953.
228. Watkins, R. J.: A study of typical neurodystrophies under chiropractic treatment, *J. National Chiro. Assoc.*, Vol. XXI, p. 16, June 1951.
229. Weiant, C. W.: Lessons from Speransky – some specific applications to chiropractic, *Chiro. J.*, Vol. XI, pp. 11–12, December 1942.
230. Weiant, C. W.: Lessons from Speransky – some specific applications to chiropractic, *Chiro. J.*, Vol. XII, pp. 13–14, January 1943.
231. Watkins, C. O.: Interference with nerve function, *National Chiro. J.*, Vol. XV, pp. 19–20, April 1945.
232. Dittrich, R. J.: Somatic pain and autonomic concomitants, reflex sympathetic dystrophy, *Am. J. of Surg.*, January 1954.
233. Evans, J. A.: Reflex sympathetic dystrophy, *Am. J. of Int. Med.*, pp. 417–426, March 1957.
234. Appley, M. H., and Trumbull, R.: *Psychological Stress, Issues in Research*, New York, Appleton-Century-Crofts, 1967.
235. Proceedings – Conference of the Soc. for Psychosomatic Res: *The Nature of Stress Disorder*, London, Hutchinson, 1958.
236. Levi, Lennart: *Stress, Sources, Management and Prevention*, New York, Liveright, 1967.
237. Selye, Hans: *The Stress of Life*, Longmans, 1957.
238. DeVore, W. W., and DeVore, F. N.: An illustrated anatomic analysis of displacement of the atlas vertebra, *J. National Chiro. Assoc.*, Vol. XXIV, pp. 15–16, May 1954.
239. Dickholtz, M.: *Handbook for X-ray Alignment*, NUCCA, Privately published, Chicago, 1971.
240. Frigerio, Stowe, and Howe: Movement of the sacroiliac joint, *Clinical Orthop. and Related Research*, May 1974.
241. Gregory, R. R.: *The Upper Cervical Monograph*, Monroe, Mich., Vol. 1, No. 1–5, 1972–1974.
242. Gonstead, C. S.: *Seminar of Chiropractic*, Monographs, Mt. Horeb, Wisconsin, 1974.
243. Hildebrandt, R. W.: Synopsis of chiropractic postural roentgenology, Lombard, Ill., 2nd edition, National-Lincoln Sch. of *Postgrad. Ed.*, 1974.
244. Howe, J. W.: Some consideration in spinal X-ray interpretation, *J. Clin. Chiro.*, 75–98, Spring 1971.
245. Howe, J. W.: Facts and fallacies, myths and misconceptions in spinography, *J. Clin. Chiro.*, 34–45, Winter 1972.
246. Howe, J. W.: The chiropractic concept of subluxation and its roentgenological manifestations, *J. Clin. Chiro.*, 64–70, September–October 1973.
247. Janse, J.: The vertebral subluxation, *National Chiro. J.*, Vol. XVIII, pp. 9–11, October 1948.
248. Kuhn, J.: *Protractorscope and Stereo-protractorscope Spinograph analysis*, Nashville, Parthenon, 1956.
249. Mears: Analysis and adjustment of the occiput and cervical spine, *The Digest of Chiro. Econ.*, January 1970.
250. Messer, A. R., et al.: The cervical subluxations: Its correction and effects upon the body, *J. Clin. Chiro.*, 122–134, Winter 1972.
251. Oetteking, B.: Spondylometry: Means and ways (1) General vertebral morphology, *National Chiro. J.*, Vol. XVI, pp. 15–16, February 1946.
252. Orrick, R. L.: Some considerations in upper cervical laterality, *J. National Chiro. Assoc.*, Vol. XXII, 20–22, December 1952.
253. Pettibon, B. R.: *Pettibon Method of Cervical X-ray Analysis and Instrument Adjusting*, Tacoma, Wash., 1968.
254. Poehner, W. G.: Reading of spinographs, *National Chiro. J.*, Vol. XVI, pp. 24–25, December 1946.
255. Rehberger, L. P.: A spinographic study of the atlas and axis, *J. National Chiro. Assoc.*, Vol. XXII, pp. 18–20, March 1952.
256. Remier, P. A.: *Modern X-ray Practice and Chiropractic Spinography*, 3rd edition, Davenport, Iowa, Palmer School of Chiropractic, 1957.
257. Sausser, W. L.: Spinograph and X-ray, occiput-atlas-axis listings, *Chiro. J.*, 32: Vol. IV, May 1935.
258. Sausser, W. L.: A treatise on the use of X-ray in spinal analysis, *National Chiro. J.*, Vol. XVII, pp. 25–27, September 1947.
259. Suh, C. H.: The fundamentals of computer aided X-ray analysis of the spine, *J. Biomech.*, 7:2:161–169, March 1974.
260. Viadeff, T.: The theory of the fixation points, *National Chiro. J.*, 20–23, June 1945.
261. Weiant, C. W.: Constancy of vertebral subluxation, *National Chiro. J.*, Vol. XV, pp. 12–14, February 1945.
262. Weiant, C. W.: Comparative study of spinographic methods, *National Chiro. J.*, Vol. XVI, pp. 11–12, June 1946.
263. Fox, E. A.: Roentgenological studies of lumbosacral distortion, *J. National Chiro. Assoc.*, Vol. XXIV, pp. 24–27, August 1954.
264. Wunsch, L. E.: Radiological diagnosis, its importance to the doctor of chiropractic, *Chir. J.*, 9: Vol. IV, September 1937.
265. Winterstein: *Chiropractic Spinographology*, Lombard, Ill., National College Chiro., February 1970.
266. Illi, F. W.: *Highlights of 45 Years of Experience and 35 Years of Research*, Geneva, Institute for the study of the statics and dynamics of the human body, 1970.
267. Sandoz, R.: A tentative classification of the functional disorders of the intervertebral motor unit, *Ann. Swiss Chiro. Assoc.*, V:93–180, 1971.
268. Herron, R. E.: Biostereometric measurement of body form, *Yearbook of Physical Anthropology*, 16:80–120, 1972.
269. Carver, F. J.: An important discovery that will greatly benefit all chiropractors, *Chiro. J.*, 17: Vol. IV, October 1937.
270. Beech, R. A.: The fundamentals of the short leg syndrome, *Ann. Swiss Chiro. Assoc.*, III: 7–36, 1965.
271. Bourdillon, J.: *Spinal Manipulation*, Appleton Century-Crofts, New York, 1970.
272. Bradbury, P.: *The Mechanics of Healing*, London, Peter Owen, 1967.
273. Deming: *Posture Analysis*, Dubuque, Iowa, 1964.
274. Dishman, R. W.: A study of differences in leg lengths – the anatomical short leg, *J. National Chiro. Assoc.*, Vol. XXI, pp. 21–22, August 1951.

275. Drum, C.: An introduction to the study of postural and spinal mechanics, Toronto, Ontario, *Canadian Coll. of Chiro.*, September 1970.
276. Drum, C.: The nature of the problem on subluxation, *New England J. of Chiro.*, 29–31, May 1973.
277. Grice, A. S.: Posture and postural mechanics, *J.C.C.A.*, July 1970.
278. Illi, F. W.: Sacroiliac mechanism, keystone of spinal balance and body locomotion, *National Coll. J. of Chiro.*, 13, 1940.
279. Illi, F. W.: *The Vertebral Column Life Line of the Body*, Chicago, National College of Chiro., 1951.
280. Illi, F. W.: The statics and dynamics of the spine and pelvis in health and disease, *J. National Chiro. Assoc.*, Vol. XXI, pp. 13–15, October 1951.
281. Idem: *Chiropractic Locomotion and Spinal Statics*, Geneva, Institute for the study of human body statics and dynamics, 1952.
282. Illi, F. W.: On the functional unit spine-pelvis, *Hippokrates, 27, 1956.*
283. Illi, F. W.: The functional unity of the spinal column and the pelvis, *J. National Chiro. Assoc.*, Vol. XXVII, pp. 21–22, March 1957.
284. Idem: Lumbar statics and the trochanter phenomenon, *Ann. of the Swiss Chiro. Assoc.*, 1:60, 1960.
285. Idem: Paper submitted to the *Danish Chiro. Defense Committee*, Geneva, Institute for the study of the statics and dynamics of the human body, 1970.
286. Idem: Morbid predisposition mechanical origin inherent to the phylogenesis man, *ACA J. of Chiro.*, 1965.
287. Idem: Vertebral and sacroiliac arthrosis, *ACA J. of Chiro.*, January 1968.
288. Janse, J.: The concepts and research of Dr. Fred W. Illi, National Coll. *J. of Chiro.*, 28:1 pp. 10–34, June 1956.
289. Janse, J.: Postural studies – body types and their diatheses, *Chiro. J.*, Vol. XII, pp. 11–12, January 1943.
290. Janse, J.: A common pelvic mechanical pathology, *J. of National Coll. of Chiro.*, 22:14–19, May 1950.
291. Jenness, M. E., et al.: Use of the conformateur and the line of gravity apparatus in a new technique for assessing posture, *The Digest of Chiro. Econ.*, November–December 1974.
292. Johnson, A. C.: An efficient method of chiropractic examination and diagnosis, *Chir. J.*, 8: Vol. IV, August 1936.
293. Johnston: The paradox of the functional spine, *ACA J. of Chiro.*, April 1967.
294. Johnston: Structural advantages of the spiral spine, *Digest of Chiro. Econ.*, November 1967.
295. Gleeson, G. M.: A report of findings and observations regarding the use of the posturometer and posturizer, *J. Canad. Chiro. Assoc.*, 1957.
296. Kenel, F.: The body posture of children and adolescents, *Ann. Swiss Chiro. Assoc.*, pp. 78–104, 1961.
297. Kenel, F.: Flat feet – a cause of static insufficiency of the body, *Ann. Swiss Chiro. Assoc.*, V:197–206, 1971.
298. Lind, G. A.: *Auto Traction*, Linköpings University Medical Dissertations, No. 17, Sweden, 1974.
299. Mennell, J.: *Back Pain; Diagnosis and Treatment Using Manipulative Techniques*, Boston, Little Brown, 1960.
300. Idem: *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques*, Boston, Little Brown, 1964.
301. Reinert, O. C.: *Chiropractic Procedures and Practice*, Rev. 3rd edition, Florissant, Mo., 1972.
302. Smith, R. K.: Therapeutic possibilities of manual treatment, *N.Y. Med. J.*, Mar. 22, 1913.
303. Steinbach, L. J.: Spinal balance, what we know about correction of lateral balance, *Chir. J.*, 9: Vol. IV, April 1937.
304. Steinbach, L. J.: The sacroiliac mechanism – some important new scientific facts are revealed, *Chiro. J.*, Vol. X, p. 11, January 1941.
305. Vear, H. J.: A survey of postural indices in adolescents, *J. Canad. Chiro. Assoc.*, April 1970.
306. Vilholm, F.: Statistical evaluation of posture in 1000 school children, *Ann. Swiss Chiro. Assoc.*, V:55–80, 1971.
307. Vladeff, T.: A report on leg inequalities, *National Chiro. J.*, Vol. XVIII, pp. 17–18, January 1948.
308. Widmann, B. J.: Electromyography for chiropractic, *Annals of the Swiss Chiro. Assoc.*, 3:132–160, 1965.
309. Widmann, B. J.: Neurofunctional aspects of postural disorders, *Ann. Swiss Chiro. Assoc.*, pp. 105–120, 1961.
310. Beilke, M. C.: Roentgenological spinal analysis and the technic for taking standing X-ray plates, *J.A.O.A.*, May 1936.
311. Bosshard, R.: Spinal hygiene, *Ann. Swiss Chiro. Assoc.*, IV, 163–184, 1969.
312. Buehler, M. T.: Sway and round back complexes, *ACA J. of Chiro.*, November 1971.
313. Denslow, J. S., Chase, J. A., Gutensohn, et al.: Methods on taking and interpreting weight-bearing X-ray films, *J.A.O.A.*, July 1955.
314. Glogowski, G.: A correct placement for the so-called chiropractic roentgendiagnosis, *Münchener medizinische Wochenschrift*, 98, 1956.
315. Hildebrandt, R. W.: Spinographic analysis of the sacro-innominate triangle: a statistical survey, *J. Clin. Chiro.*, 62–74, Spring 1971.
316. Hildebrandt, R. W.: The clinical basis of chiropractic postural roentgenology, *J. Clin. Chiro.*, 22–37, Spring 1973.
317. Howe: Research on several parameters relating to full spine radiography, *ACA J. of Chiro.*, September 1970.
318. Janse, J.: Evaluating 500 cases of low-back involvement, *J. National Chiro. Assoc.*, Vol. XXVI, pp. 28–29, August 1956.
319. Jochumsen, Ole.: The curve of the cervical spine, *ACA J. of Chiro.*, August 1970.
320. Kissinger, R. N.: Findings in low-back and sciatic neuralgia syndromes, *J. National Chiro. Assoc.*, Vol. XXX, p. 32, July 1960.
321. Kraus, E. A.: A clinicoradiological consideration of low back pain, *J.A.O.A.*, February 1948.
322. Kraus, E. R.: Postural roentgen study, *J.A.O.A.*, January 1949.
323. Lindner, H.: Precoxarthrosis and coxarthrosis incipiens, *Ann. Swiss Chiro. Assoc.*, III:185–200, 1965.
324. Poehner, W. G.: Roentgenology, first erect spinograph, *Chiro. J.*, Vol. XI, pp. 18–19, February 1942.
325. Sausser, W. L.: Achievement, entire body X-ray technique is perfected, *Chiro. J.*, 17–18: Vol. IV, February 1935.
326. Sausser, W. L.: Spinograph and X-ray, Chiro. J., Vol. VI, pp. 18–20, November 1938.

327. Sausser, W. L.: The spine viewed and recorded roentgenologically in 4584 cases, *National Chiro. J.*, Vol. XVII, pp. 18–23, August 1947.
328. Sausser, W. L.: The pelvis viewed and recorded roentgenologically in 4584 cases, *N.C.J.*, August 1941, 9–7–54.
329. Seitz, K.: Chiropractic and osteopathy in the light of functional statics, *Erfahrungsheilkunde*, 7, 1958.
330. Weiant, C. W.: Investigation of low back syndrome, *J. National Chiro. Assoc.*, Vol. XIX, pp. 15–23, February 1949.
331. Amodei, J. E.: Outline of a complete examination of the low back, *J. National Chiro. Assoc.*, Vol. XXX, pp. 21–24, April 1960.
332. Jaquet, P.: The upright position of man: its phylogenetic and psychological aspects and some therapeutic considerations of interest, *Ann. Swiss Chiro. Assoc.*, IV:185–196, 1969.
333. Janse, J.: A study of man, the biped, *National Chiro. J.*, Vol. XVII, pp. 11–12, June 1947.
334. Jenkins, F. A.: Chimpanzee bipedalism cineradiographic analysis and implications for the evaluation of gait, *Science*, November 1972.
335. Lupica, B.: Treatise on inherent spinal distortion, *National Chiro. J.*, Vol. XVI, p. 21, September 1946.
336. Oetteking, B.: The spine—the natural and acquired curvatures of the human vertebral column, *National Chiro. J.*, Vol. XV, pp. 11–12, August 1945.
337. Oetteking, B.: The spine—two crucial loci in spinal evolution, *National Chiro. J.*, Vol. XV, pp. 13–14, October 1945.
338. Wright, H. M.: Health and the biped stance, *J.A.O.A.*, 51:531–532, July 1952.
339. Bailey, H. W. and Bechwith, C. G.: Short leg and spinal anomalies, *J.A.O.A.*, March 1937.
340. Bollier, W.: The relative irrelevance of morphologic changes in the production of symptoms, *Ann. Swiss Chiro. Assoc.*, 102–111, 1960.
341. Janse, J.: The mechanical pathologies involved in the low back and sciatic neuralgia syndrome, *J. National Coll. Chiro.*, 25:3–21, January 1953.
342. Kaufman, J.: The relationship of spinal anomalies to low back pain, *ACA J. of Chiro.*, July 1974.
343. Phillips, R. B.: Clinical and roentgenographic considerations of congenital spinal anomalies important to the chiropractor, *ACA J. of Chiro.*, October 1974.
344. Sandoz, R. W.: About three cases of a typical spondylolysis, *Ann. Swiss Chiro. Assoc.*, pp. 198–208, 1961.
345. Schmorl and Junghanns: *The Human Spine in Health and Disease*, Trans. and Edit. by S. P. Wilk and L. S. Goin, New York, Grune and Stratton, 1959.
346. Ulrich, H. O.: Congenital abnormalities and variations, *J. National Chiro. Assoc.*, Vol. XXVI, pp. 26–27, July 1956.
347. Valentini, E.: The occipito-cervical region, *Ann. Swiss Chiro. Assoc.*, IV:225–232, 1969.
348. Weiant, C. W.: Research on vertebral asymmetry, *National Chiro. J.*, Vol. XIV, p. 11, January 1944.
349. Cailliet, R.: *Low Back Pain Syndrome*, Philadelphia, Davis, 1974. Idem: Neck and arm pain, Ibid, 1974.
350. Colachis, Warden, Bechlol, and Strohm: Movement of the sacroiliac joint in the adult male: a preliminary report, *Arch. of Phys. Med. and Rehab.*, September 1963.
351. Conley, R. N.: Stress evaluation of cervical spinal mechanics, J. Clin. Chiro., 1–3:46–62, 1974.
352. Cramer, A.: On the function of the iliolumbo-sacral union, *Erfahrungscheilkunde*, V, 1956.
353. Dankmeyer, J., and Rothmeir, B. J.: The lateral movement in the atlanto-axial joints and its clinical significnace, *Anat. Lab. and Roent. Dept.*, Keiden, Holland, 1943.
354. Denslow, J. D.: Palpation of the musculoskeletal system, *J.A.O.A.*, August 1964.
355. Frymann, V. M.: Palpation, *AAO Yearbook*, 1963.
356. Gillet, H.: Theoretical considerations on spinal balance, *National Chiro. J.*, Vol. XVII, pp. 22–23, October 1947.
357. Gillet, H.: Vertebral fixations, an introduction to movement palpation, *Ann. Swiss Chiro. Assoc.*, pp. 30–33, 1960.
358. Grice, A. S.: Mechanics of walking, development and clinical significance, *J. CCA*, October 1972.
359. Grice, A.: A preliminary study of muscle tone, changes pre and post manipulation, *J. Canad. Chiro. Assoc.*, 4th quarter, Vol. XVIII, No. 4.
360. Gutmann, G.: Vertebral fixation and its roentgenological demonstration, Hippokrates-verlag, 1960.
361. Hildebrandt, R. W.: The functional mechanics of the pelvis and lumbar spine, *Dig. Chiro. Econ.*, January 1970.
362. Howe, J. W.: Preliminary observations from cineroentgenological studies of the spinal column, *ACA J. of Chiro.*, October 1970.
363. Howe, J. W.: Cineradiographic evaluation of normal and abnormal cervical spine function, *J. Clin. Chiro.*, 76–88, Winter 1973.
364. Howe, J. W.: Cineradiographic evaluation of normal and abnormal cervical function, *J. Clin. Chiro.*, 1–4, 42–54, 1974.
365. Hviid, H.: The influence of chiropractic treatment on the rotary mobility of the cervical spine—a kinesiometric and statistical study, *Ann. Swiss Chiro. Assoc.*, V:31–44, 1971.
366. Hviid, H.: Functional radiography of the cervical spine, *Ann. Swiss Chiro. Assoc.*, Vol. III, 37–65, 1965.
367. Lane, G. F.: Cervical spine: its movement and symptomatology, *J. Clin. Chiro.*, 128–145, Spring 1971.
368. Liekens, M.: Movement palpation, *Ann. Swiss Chiro. Assoc.*, 34–42, 1960.
369. Macconaill, M. A.: Some aspects of vertebral function, *Bull. of Eup. Chiro. Union*, 1974.
370. Maigne, R.: Orthopedic medicine, Springfield, Ill., *Charles C Thomas*, 1972.
371. McAndrews, J. E.: Spinal motion examination, *ACA J. of Chiro.*, May 1969.
372. Nachemson, A.: The effect of forward leaning on lumbar intradiscal pressure, *Acta Orthop. Scand.*, 1965.
373. Nachemson, A.: In vivo discometry in lumbar discs with irregular nucleograms, *Acta. Orthop. Scand.*, 1965.
374. Roaf, R.: Rotation movements of the spine with special reference to scoliosis, *J. of Bone and Joint Surgery*, May 1958.
375. Sandoz, R. W.: Technic and interpretation of functional radiography of the lumbar spine, *Ann. Swiss Chiro. Assoc.*, III:66–106, 1965.

376. Steffen, E. J.: Lateral curvature of the spine, *National Chiro. J.*, Vol. XIV, pp. 17–18, July 1944.
377. Steindler, A.: *Mechanics of Normal and Pathological Locomotion in Man*, Charles C Thomas, Springfield, Ill. 1935.
378. Taylor, D. B.: Biomechanics of the spine under stress, *J. Clin. Chiro.*, 1:2, 61–67, November–December 1973.
379. Van Dusen: *Chiropractic Relationship to Gravitational Force*, Sodus, N.Y., 1968.
380. Watkins, R. J.: Pelvic mechanics, *J. National Chiro. Assoc.*, Vol. XXX, p. 26, March 1960.
381. Weiant, C. W.: First X-ray movies of spine and pelvis in action shown, *J. National Chiro. Assoc.*, Vol. XXVII, p. 17, May 1957.
382. Widmann, B. J.: Electromyography for chiropractic, *Ann. Swiss Chiro. Assoc.*, III:132–160, 1965.
383. Fryette, H.: Some reasons why sacroiliac lesions recur, *J.A.O.A.*, November 1936.
384. Kamieth, H.: Distortions of the sacroiliac joints in chiropractic, *Fortschrift Röntgenstr.*, 89, 1958.
385. Solonen, K. A.: The sacroiliac joint in the light of anatomical, roentgenological and clinical studies, *Acta Ortho. Scand.*, 1957.
386. Weisl, H.: The articular surfaces of the sacroiliac joint and their relation to the movement of the sacrum, *Acta. Anat.*, 1954.
387. Weisl, H.: The movements of the sacroiliac joint, *Acta. Anat.*, 1955.
388. Farfan, et al.: The effects of torsion on the lumbar intervertebral joints. The role of torsion in the production of disc degeneration, *J. of Bone and Joint Surg.*, April 1970.
389. Farfan, H. F.: *Mechanical Disorders of the Low Back*, Lea and Febiger, Phil., 1973.
390. Reid: Effects of flexion and extension movements of the head and spine on the spinal cord and nerve roots, *J. Neurosurg. Psychiat.*, 1960.
391. Yochum, R. T.: Stress studies of thoracolumbar scoliosis, *J. of Clin. Chiro.*, September–October 1973.
392. Pyzik, L. C.: Stress correction of scoliosis, *J. Clin. Chiro.*, 46–63, September–October 1973.
393. Farfan, et al.: Instantaneous center of rotation of the third lumbar intervertebral joint, *J. Biomech.*, 1971.
394. Fisher, A. G.: Timbrell: *Treatment by Manipulation*, Lewis & Co., 1948.
395. Mennel, J. B.: *Manual Therapy*, Springfield, Ill., Thomas, 1881.
396. Mennel, J. B.: *The Science and Art of Joint Manipulation*, Vol. 1: *The Extremities;* Vol. 2: *The Spinal Column*, London, Churchill, 1939 and 1949.
397. Schitz, Eiler H.: Manipulation treatment of the spinal column from the medical-historical viewpoint, *Tidsskr Nor Laegeforn*, 78: in several numbers, 1958.
398. Tieszen, D. Waldo: *The Tieszen Technique*, Tieszen Clinic, Marion, S.D., 1937.
399. Beatty, H. G.: Chiropractic technique—an analysis of various types of treatment, *Chiro. J.*, Vol. XI, pp. 11–12, January 1942.
400. Biedermann, F.: Fundamentals of chiropractic from the standpoint of a medical doctor, Translated by L. C. J. Iekeler, Davenport, Iowa, *Internation. Chiro. Research Comm.*, 1959.
401. Droz, J. M.: Indications and contra-indications of vertebral manipulations, *Ann. Swiss Chiro. Assoc.*, V:81–92, 1971.
402. Jaquet, P.: *The Importance of Laboratory Methods in Chiropractic Diagnosis*, V:215–264, 1971.
403. Grecco, M. A.: *Chiropractic Technic Illustrated*, New York, Jarl Publ. Co., 1953.
404. Lorez, E.: Some thoughts on manipulation, *Ann. Swiss Chiro. Assoc.*, V:7–14, 1971.
405. Sandoz, R.: The significance of the manipulative crack and of other articular noises, *Ann. Swiss Chiro. Assoc.*, 4:47–67, 1969.
406. Sandoz, R. W.: Problems pertaining to the choice of indications for chiropractic therapy, *Ann. Swiss Chiro. Assoc.*, III:201–213, 1965.
407. Spears, L. L.: *Spears Painless System of Chiropractic and Other Developments*, 5th edition rev., Denver, 1950.
408. States, A. Z.: *Spinal and Pelvic Technics*, National Coll. of Chiro., Lombard, Ill., 1968.
409. Janse, J.: Cervical syndromes, *National Coll. of Chiro. J.*, 31:3–13, April 1959.
410. Janse, J.: Chiropractic methods of correcting masor pelvic distortions, *National Coll. of Chiro. J.*, 23:4–10, January 1951.
411. Janse, J.: Basic clinical phenomena as it relates to the art of specific reduction of spinal segmental dysarthrias, *National Coll. of Chiro.* Monograph, 1973.
412. Beech, R. A.: A history of the intervertebral disc syndromes as it applies to manipulative practice, *Ann. Swiss Chiro. Assoc.*, pp. 7–19, 1961.
413. Beyeler W.: Scheuerman's disease and its chiropractic management, *Ann. Swiss Chiro. Assoc.*, 170–177, 1960.
414. Beyeler, W.: Experiences in the management of asthma, *Ann. Swiss Chiro. Assoc.*, III:111–117, 1965.
415. Bosshard, R.: The treatment of acute lumbago and sciatica, *Ann. Swiss Chiro. Assoc.*, pp. 50–61, 1961.
416. Cox, J. M.: Lumbar disc herniation: statistics on an innovative diagnostic and therapeutic approach, *J. Clin. Chiro.*, 20–45, September–October 1973.
417. Drum, D. C.: Conservative management of lumbar disc degenerations, *J. Clin. Chiro.*, 96–113, Winter 1972.
418. Egli, A. B.: Long-time prognosis of spondylarthrosis and chondrosis under classic and chiropractic therapy, *Ann. Swiss Chiro. Assoc.*, V:181, 1971.
419. Fernandez, P. G.: Spondylolisthesis, *J. Clin. Chiro.*, 28–32, Winter 1972.
420. Grillo, F.: The differential diagnosis and therapy of headache, *Ann. Swiss Chiro. Assoc.*, pp. 121–166, 1961.
421. Janse, J.: Cervical and occipital involvements and their management, *National Coll. of Chiro. J.*, July 1958.
422. Valentini, E.: The rate of success of chiropractic manipulations in lumbar herniated discs, *Ann. Swiss Chiro. Assoc.*, IV:79–86, 1969.
423. Weiant, C. W.: Some observations on blood pressure, *National Chiro. J.*, Vol. XIV, pp. 13–14, November 1944.
424. Weiant, C. W.: Effect of chiropractic on metabolism, a preliminary report, *National Chiro. J.*, Vol. XVI, p. 17, March 1946.
425. DeJarnette, M. B.: *Sacro-occipital Technic of Chiropractic*, Nebraska City, 1952.

426. Ibid. *Sacro-occipital Technic of Spinal Therapy*, Nebraska City, 1940.
427. Goodheart, G. J.: Cross pattern crawling and muscle spasm, *Dig. Chiro. Econ.*, March–April 1969.
428. Idem: *The Intrinsic Muscles and the Persistent Subluxation*, Ibid.
429. Idem: *The Principle of Muscle Balancing*, Ibid. March–April 1970.
430. Idem: *Sacroiliac and Ilio Sacral Problems*, Ibid, January–February 1972.
431. Idem: *The Cervical Challenge*, Ibid. September–October 1972.
432. Janse, J.: Study of reflex and pressure technics, *National Chiro. J.*, Vol. XVI, pp. 9–10, December 1946.
433. Janse, J.: A scientific treatise on clinical chiropractic measures—section one, *J. National Chiro. Assoc.*, Vol. XXII, pp. 11–12, December 1952.
434. Kenel, F.: Winkler's reflex therapy, *Ann. Swiss Chiro. Assoc.*, V:262–266, 1971.
435. Nimmo, R. L.: Receptors, effectors and tonus—a new approach, *J. National Chiro. Assoc.*, Vol. XXVII, pp. 21–23, November 1957.
436. Watkins, R. J.: Anthropology in reflex technics, *National Chiro. J.*, Vol. XVIII, pp. 14–15, August 1948.
437. Watkins, R. J.: The upper thoracic complex, *J. National Chiro. Assoc.*, Vol. XX, p. 33, November 1950.
438. Macdonald, J.: Dr. Locke—healer of men, Williamsburg, Ontario, 1933.
439. Chillcott, R. L.: The autonomic nervous system in chiropractic practice, *J. National Chiro. Assoc.*, Vol. XXVI, pp. 15–16, July 1956.
440. Davids, S.: The subluxation as an adaptive autonomic regulator, *National Chiro. J.*, Vol. XVIII, p. 14, April 1948.
441. Müller, R. O.: *Autonomics in Chiropractic*, Toronto, Canada, Chiro. Pub. Co., 1954.
442. Pitzer, B. E.: An interesting study of the autonomic nervous system, *J. National Chiro. Assoc.*, Vol. XXVII, p. 11, April 1957.
443. Baker, F. A.: Speaking of subluxations, nerve pressures may exist away from the spine, *Chiro. J.*, 8: Vol. IV, August 1935.
444. Curchod, G. A.: Sciatic pain and the foot, *Ann. Swiss Chiro. Assoc.*, V:207–215, 1971.
445. Eisenberg, A. M.: Diversified chiropractic technique, *J. Clin. Chiro.*, 24–61, Spring 1971.
446. Janse, J., and Kissinger, R. N.: The relationship of the mechanical integrity of the feet, lower extremities, pelvis and spine to the proper function of the various structural and visceral systems of the body, *J. of National Coll. Chiro.*, 26:6–19, May 1954.
447. Schmidt, E. G.: A presentation of a mode of therapy for treating rib lesions, *J. National Chiro. Assoc.*, Vol. XXX, pp. 11–12, December 1960.
448. Fox, E. A.: Psoas major muscle, *ACA J. of Chiro.*, 541–548, July 1967.
449. Fox, E. A.: Let us consider the importance of psoas muscle contraction, *J. National Chiro. Assoc.*, Vol. XXVI, pp. 11–14, July 1956.
450. Fielder, S. L., and Pyott, W. H.: *The Science of Manipulative Surgery*, Bountiful, Utah, Carr Printing, 1953.
451. Finnell, F. L.: *Constructive Chiropractic and Endonasal-aural and Allied Office Techniques for Eye, Ear, Nose and Throat*, 3rd edition, Portland, Oregon, Ryder Printing, 1955.
452. Forster, A. L.: *Forster's System of Non-medicinal Therapy*, Vol. I and Vol. II, Chicago, National Publishing Assoc., 1919.
453. Lake, T. T.: *Treatment by Neuropathy and the Encyclopedia of Physical and Manipulative Therapeutics*, Philadelphia, 1946.
454. Maitland, G. D.: *Vertebral Manipulation*, London, Butterworth, 1964.
455. Dye, A. A.: *The Evolution of Chiropractic*, Republished by Richmond Hall, Inc., pp. 203–218, 1969.
456. Palmer, B. J.: *Chiropractic Clinical Controlled Research*, Vol. XXV, W. B. Conkey, publishers, 1951.
457. Haldeman, S.: First impressions of the synchro-therme as a skin temperature reading instrument, *JCCA*, April 1970.
458. Haldeman, S.: Observations made under test conditions with the synchro-therme, *Canad. Chiro. Assoc.*, October 1970.
459. Whitby, Morton, M.R.C.S.: *Theory of Life, Disease and Death*, Cutler Publishing Co., Chicago, 1945.
460. Adelman, G. N.: Visual nerve tracing, its importance to the chiropractic profession, *Chiro. J.*, 9–10: Vol. IV, August 1935.
461. Adelman, G. N.: Photography through the skin proves chiropractic a science, *J. National Chiro. Assoc.*, Vol. XXII, pp. 20–23, April 1952.
462. Dudley, W. N.: Facial thermography and adjustment, *ACA J. of Chiro.*, 54–56, August 1974.
463. Kimmel, E. H.: Analytical instruments in chiropractic practice, *JNCA*, p. 28, February 1961.
464. Kimmel, E. H.: The derma thermagraph, *JCC*, Vol. 1, No. 4, pp. 78–86, 1969.
465. Novick, N. D.: The VNT: Photo-electric instrument, *JCC*, Vol. 2, No. 3, pp. 78–83, 1969.
466. Aarons, M. W.: Electro-analytical instrumentation, *ACA J. of Chir.*, January 1974.
467. Ibid: Advanced diagnostic instruments, *ACA J. of Chiro.*, p. S–49, April 1974.
468. Maitland and Gerrard: The neurocalometer—a survey to asses its value as a diagnostic instrument, *Med. J. of Australia*, 1:464–468, March 1972.
469. Weiant, C. W.: Some preliminary observations on photo-electric visual nerve tracing, *J. National Chiro. Assoc.*, Vol. XXI, pp. 11–12, January 1951.
470. Cox, J. M.: *Chiro-manis Treatment Manual*, Privately published, Fort Wayne, Indiana, 1974.
471. Laufenberg, P. C.: *Thompson Technic Notes*, Privately published, Sacramento, California 1973.
472. McManis, J. V.: *McManis Table Technic*, Kirksville, Mo. 1938.
473. Janse, J.: The rationale and history of physical therapy in chiropractic, Monograph Dept. Clinical Practice, Lombard, Ill., *National Coll. of Chiro.*, 1974.
474. Bolier, W.: Electrotherapy in coxalgiae accompanied by vertebral pain, *Ann. Swiss Chiro Assoc.*, III:118–122, 1965.
475. Bolier, W.: The spine and internal diseases, *Ann. Swiss Chiro. Assoc.*, pp. 167–182, 1961.
476. Dreyfus, C. E.: The management of disuse atrophy in

597-036 O - 76 - 4

scoliosis, *Ann. Swiss Chiro. Assoc.*, III:123–131, 1965.
477. Droz, J. M.: Possibilities, limits and results of an elective vertebral mechanotherapy, *Ann. Swiss Chiro. Assoc.*, 202–216, 1960.
478. Droz, J. M.: The trophostatic syndrome of post-menopause, *Ann. Swiss Chiro. Assoc.*, III:171–184, 1965.
479. Jaquet, P.: The psychosomatic approach to the treatment of backache, *Ann. Swiss Chiro. Assoc.*, pp. 183–197, 1960.
480. Johnson, A. C.: Corrective therapeutics enhances the value of chiropractic treatments, *National Chiro. J.*, Vol. IX, p. 23, May 1940.
481. Keller, F.: Simple auxiliary measures in the treatment of lumbalgias, *Ann. Swiss Chiro. Assoc.*, 191–196, 1960.
482. Kenel, F.: Purpose of sole and heel lifts and their effects on spine and pelvis, *Ann. Swiss Chiro. Assoc.*, III:161–170, 1965.
483. Overton, R. M.: Chiropractic treatment for decongestion of the liver, *J. National Chiro. Assoc.*, Vol. XXV, pp. 19–20, August 1955.
484. Pasqua, V.: Intensive chiropractic day care, *J. Clin. Chiro.*, 74–101, Spring, 1973.
485. Sandoz, R., and Lorez, E.: Presentation of an original lumbar technic, *Ann. Swiss Chiro. Assoc.*, 43–55, 1960.
486. Sandoz, R. W.: Suspension plaster cast, *Ann. Swiss Chiro. Assoc.*, pp. 70–77, 1961.
487. Widmann, B.: Experiences with intermittent traction of the lumbar spine, *Ann. Swiss Chiro. Assoc.*, 178–186, 1960.
488. Lindlahr, H.: *Philosophy of Natural Therapeutics*, Chicago, Lindlahr, 1918.
489. Wardwell, W. I.: *A Marginal Professional Role: The Chiropractor*, Doctoral Dissertation, Harvard, 1951.
490. White, M., and Skipper, J. K.: The chiropractic physician: a study of the career contingencies of a marginal occupation, *J. Clin. Chiro.*, 148–161, Winter 1972.
491. Betge, Giorgia: *La terabia Chiropractica*, 2nd edition, Lugano, 1974.
492. Janse, J.: *Chiropractic Principles and Technic; For Use by Students and Practitioners*, Trans. into Japanese by Shoji Fujii, Tokyo, Japan, Kazakushimoun-Sha., 1969.
493. Lofgren, H.: *Orsak och verkan*, Stockholm, Seelig, 1959.
494. Moller, E.: *Kiropratik; Haender og Helbred*, Kobenhaven, Danampress 1968.
495. Paper, W.: *Technik der Chiropraktik*, 6 Verb. Aufl Ulm, Donau, Haug, 1958.
496. Pecunia, A. L.: *Reboutement Chirosomatotherapie; Cinesiologie Passive Occasionnelle*, 2nd edition, Paris, Malone 1971.
497. Cramer, A.: Possibilities of manual and instrumental treatment of vertebral disturbances in the segment of motion, *Therap. woche*, 7:1956–1957.
498. Gutzeit, K.: *The Spine and Internal Medicine*, Münchener medizinische Wochenschrift, 1953.
499. Ibid: The spine as a factor in disease from a therapeutic point of view, *Therapie der Gegenwart*, 92:41, 1953.
500. Ibid: The vertebrae in research and practice, *Therap. woche* 7, 1956–1957.
501. Hochrein and Schleicher: On the question of the vertebral origin of angina pectoris, *Medizinische Klinink*, 15, 1953.
502. Kamieth, H.: The chiropractic view of disease in internal organs, *Medizin.*, 1957.
503. Rabe, R.: *Manual Therapy, Technics of the Chiropractors, Osteopaths, Bone-setters* and *Others*, Berlin, Corvus-Verlag, 1958.
504. Seuss, W.: *Therapy for High Blood Pressure Through Chiropractic*, Heilkunst, 73, 1960.
505. Zukschwerdt, L.: *Gynecology and Chiropractic*, Geburtshilfe und Frauenheilkunde, July 1955.

History of the Development of Osteopathic Concepts; Osteopathic Terminology

GEORGE W. NORTHUP
Director
Center for Rehabilitation Medicine
Mesa General Hospital
Mesa, Arizona

From the time of the ancient Egyptians the use of manipulations[1] as a therapeutic modality appears, disappears, and reappears like a thread woven through the tapestry of medical history. In its broadest connotation, manipulation ranges all the way from the manual reduction of fractures, dislocations, subluxations, and more simple joint dysfunctions to the application of manually applied external cardiac massage.

The role of manipulation in the history and development of osteopathic medicine has been both important and controversial. Andrew Taylor Still, the founder of osteopathic medicine, was essentially a medical philosopher. He lived at that time of history when the medical world was just emerging from 18th-century confusion. "The tubercle bacillus would not be introduced for 8 years. Lister, then in his prime, was trying to introduce antiseptic surgery against much conservative opposition. The use of diphtheria antitoxin and the X-ray would not be introduced for several years. Pasteur, then 54 years of age, had established the germ theory of disease only 10 years before. Osler, a young man of 25, was beginning his work as a professor at McGill University. Another 25 years would pass before Harvey Cushing would bring the first blood pressure measuring instrument to the United States."[2]

It was also the time when Dr. Still opened his first college of osteopathic medicine, although, as modern-day medical commentators have remarked, medical schools were numerous and "a disgrace to the profession." Willis, although critical of Still's postulates, wrote that "Still's claim that medicines were poison and surgery a means of murder were less ridiculous at that time than medical historians like to admit."[3]

Still was one of the first to recognize the role of the musculoskeletal system in health and disease. He believed that health and disease were not dependent on sharply compartmentalized anatomical or self-limiting physiological systems of the body. He believed that the body functioned as a unit, and that to understand health and to search for the causation and treatment of disease one had to consider the body as a total unit. He further believed that the total unity of the body could not be understood by excluding 60 percent of the body mass, namely, the musculoskeletal system.[4]

In its 100 years of existence, osteopathic medicine has not attempted to build a system of medical practice around a single therapeutic modality. It is significant that Still did not write a book specifically on manipulative therapy. But in the early days of osteopathic medicine, manipulative therapy was to play a crucial role.

To understand the place of manipulation in osteopathic medicine either then or today, it must be viewed in the context of the profession's basic philosophy. The Still thesis, reduced to its essence, consists of five major points.[5]

1. The unity of the body

Still believed in the interrelationship of all body

systems *including* the musculoskeletal system. He believed that each system, both in function and dysfunction, depends upon every other.

2. The healing power of nature

Still believed that there were substances *within* the body, which when in proper balance preserved health and protected against disease. He believed that these same substances, when properly mobilized, were useful in the treatment of disease.

3. The somatic component of disease

The musculoskeletal system is truly the "machinery of life," and its reciprocal communication with the other systems of the body is an important anatomical and physiological principle in medical care.

4. Structure-function concept

Structure and function cannot be separated in human physiology. There is an interdependence. Structure governs function, and function influences structure. In fact, structure and function are so closely related they can be considered a single component.

5. Manipulative therapy

The application of manipulative therapy to restore and maintain normal structure-function relationships in the musculoskeletal system is important not only to the function of the musculoskeletal system itself but through its neural-hormonal communication with other body systems. It is a potentially useful therapeutic medium for both the maintenance of normal function and the correction of dysfunction.[6]

It should be reiterated that the role of manipulative therapy in osteopathic medicine must be viewed in the totality of its philosophy and not as a thing set apart.

From a clinical standpoint, joint dysfunction in the musculoskeletal system is related to three broad clinical areas.[7]

First, the local musculoskeletal effects of these joint strains and sprains resulting in pathological limitations of normal joint motion manifest their more obvious effects in the local area of the lesion. Musculoskeletal problems such as mechanical low-back pain, functional torticollis, and similar disorders are more readily understood than those joint dysfunctions affecting other areas and systems of the body.

Secondly, joint dysfunction and the accompanying system may mimic the symptomatology of disease in more remote body systems. This mimicking effect becomes important to clinicians, regardless of their specialty. The mimicking of diseases of other organ systems is a consideration of great importance.

Finally, the possibility that local disorders in the musculoskeletal system affect the function of other body systems is a clinical area for further investigation. For example, one might cite our growing understanding of the symptomatology produced in cervical spinal strains in a whiplash type of injury. These are often misunderstood, but recent clinical research has shown that these cervical spinal joint dysfunctions cause such widely diverse symptoms as palpitation, headache, vertigo, vague gastrointestinal disturbances, and other visceral phenomena.[8,9,10] These investigations have shown that the stress-strain injury to the somatic component of the neck reflects itself in other body systems through an effect on the autonomic nervous system. If such a phenomenon occurs in the area of the cervical spine, it is not hard to understand that mechanical stresses and strains in other parts of the body can also affect visceral function.

The somatic-visceral reflex is admittedly a gray area in clinical evaluation. And, it is in this particular area that there has been a need for increased research efforts to further evaluate and understand the usefulness and specificity of manipulative therapeutics.

From its early beginnings, osteopathic medicine considered itself to be the complete practice of medicine. For example, the first college of osteopathic medicine in Kirksville, Mo., installed one of the earliest diagnostic X-ray machines (1898) to be found west of the Mississippi.[11] It is a fact that the first arteriography ever performed was done at the Kirksville College of Osteopathic Medicine in 1898.[11] Total medical care, including surgery, was part of the practice of osteopathic medicine from its beginning.[12]

However, it is also a fact that manipulative therapy was widely used and recommended by Dr. Still and his early colleagues. Still divorced himself from the pharmacopeia of his day because he alleged that those medicinal agents were either placeboes or were outright toxic to an already disturbed psysiology.

Some of Still's early antidrug statements are embarrassing to contemporary physicians. Here is the *kind* of thing he said: "One of the first duties of the physician is to educate the masses not to take medicine. Man has an inborn craving for medicine. Heroic dosing for several generations has given his tissues a thirst for drugs. The desire to take medicine is one feature which distinguishes man, the animal, from his fellow creatures." However, these particular statements were made not by Still but by one of his contemporaries—Sir William Osler.[13]

To the osteopathic clinic in Kirksville at the turn of the century, patients came by the hundreds to be relieved. Some responded through the simple expedient of removing the toxic drug component of their treatment. In a recent article Still was credited as being one of those who contributed to the abolition of calomel as a therapeutic agent.[14] Patients also began to show improvement because, for the first time in their medical care, an important component of their body function was now being treated.

Early osteopathic terminology was as faulty as early medical terminology. In those days as now, disease names and clinical processes bore only an occasional resemblance to the problem to which they referred.

The development of an osteopathic terminology to describe disorders of the musculoskeletal system was, from its beginning, at best confusing and less than descriptive. In speaking of joint dysfunctions, particularly in relationship to synovial joints, osteopathic physicians have traditionally referred to them as "osteopathic lesions." Recent efforts on the part of a study committee made up of representatives of the American Osteopathic Association and the American Academy of Osteopathy have been accepted by the Hospital Adaptation of International Classification of Disease, Adapted (H-ICDA), a commonly accepted publication of medical terminology (Appendix I).

This terminology more accurately, albeit sometimes rather broadly, refers to these joint dysfunctions of synovial joints as *somatic dysfunctions*. Therefore, what used to be referred to as an "osteopathic lesion" involving joint dysfunction of the zygapophyseal joints between the spinal segments T-3 and T-4 would now be referred to as "somatic dysfunction" involving certain anatomical locations. Essentially, a somatic dysfunction (osteopathic lesion) of the synovial joint is a biomechanical process occurring in the musculoskeletal system with structural-functional changes occurring in the articular and periarticular tissues resulting in subjective and/or objective signs and symptoms.[7]

The manifestations of these biomechanical lesions are mediated through the neurocirculatory system and may appear local to the lesion, in segmentally related but remote portions of the musculoskeletal system, or as functional disturbances in other body systems.

Joint dysfunction can occur either from other somatic lesions or from visceral dysfunction or both.

Much of the manipulative treatment of early osteopathic physicians centered around joint manipulation and mobilization. Still also paid particular attention to disturbances in the soft tissue, muscles, and fasciae. Many of his treatments for upper respiratory diseases were similar to physio therapeutic methods recommended today in the management of respiratory problems.

It is correct to say that in the time of Still and his early followers both too much and at times too little was claimed for the therapeutic efficacy of manipulative care. It is quite probable that the same weakness exists today.

Further medical research into the mechanisms of cause and effect of disorders of the musculoskeletal system and total body function are sorely needed. Animal experiments were conducted early in the profession's history; in 1898 research experimentation was done on dogs at the American School of Osteopathy, parent school of the present Kirksville College of Osteopathic Medicine. Clinical observations began to be collected, correlated and recorded at an early period as well. In 1903, Dr. Louisa Burns, a graduate of the Pacific College of Osteopathy in California, began the first of her many research investigations.

At the annual convention of the American Osteopathic Association in 1911 it was proposed that a research institute be established and endowed to conduct research into "osteopathic phenomenon." (This was actually formalizing an institution already in existence.) With the founding of the A. T. Still Research Institute in Chicago, the first organized attempt by the profession was inaugurated to study the physiological processes responsible for the effectiveness of manipulative treatment.

The institute existed until 1936 when it was merged with the American Osteopathic Associa-

tion. Since that time research programs have been developed and expanded in all osteopathic colleges. A large part of the research has been directly involved in the study of the mechanisms and responses to joint dysfunction and the response to manipulative treatment.[15]

Research, both medical and osteopathic, in those early days of the profession, was a far cry from the more skilled and sophisticated methodologies we know today. Many research projects conducted in both medical and osteopathic institutions would scarcely be called convincing in the light of modern knowledge. But from the beginning, research into the function and dysfunction of the musculoskeletal system, and its mechanisms of effect and response, attracted the attention and efforts of the osteopathic profession.[16]

The modern era of osteopathic research was inaugurated by Denslow (1938) and further implemented and expanded by Korr, his associates,[17,18,19] and others.

There is a crucial need for expanded research programs both to study the functions and dysfunctions of the musculoskeletal system and relate them to clinical practice. But as Tinbergen,[20] the 1973 Nobel prize winner for physiology or medicine, has said, "Medical practice often goes by the sound empirical principle of 'the proof of the pudding is in the eating.'" In his speech of December 12, 1973, when Dr. Tinbergen, professor of animal behavior of Oxford University, received the Nobel prize and presented his lecture, he reported on the amazing influence of the musculoskeletal system in a wide variety of clinical problems. He reported from personal experience that "corrective manipulation of the entire muscular system beginning with the head and neck and then very soon the shoulders and chest—and finally the pelvis, legs, and feet was considered until the whole body is under scrutiny and treatment."

Dr. Tinbergen, his wife, and daughter reported that "striking improvements in such diverse things as high blood pressure, breathing, depth of sleep, overall cheerfulness, mental alertness, resilience against outside pressures, and also in such a refined skill as playing a stringed instrument" were results of a special kind of treatment directed to the body musculature. He determined that there must be some relationship to the proper use of body mechanics, posture and body movement, and the function of other body systems.

One can do no better than heed Tinbergen's words and emphasis on "the importance for medical science of openminded observation—of 'watching and wondering.' This basic scientific method is still too often looked down on by those blinded by the glamor of apparatus, by the prestige of tests, and by the temptation to turn to drugs. But it is by using this old method of observation that . . . general misuse of the body can be seen in a new light; to a much larger extent than is now realized. . . ."

He concludes, "Medical science and practice meet with a growing sense of unease and of lack of confidence from the side of the general public. The causes of this are complex, but at least in one respect the situation could be improved: A little more openmindedness, a little more collaboration with other biological sciences, and a little more attention to the body as a whole and to the unity of the body and mind could substantially enrich the field of medical research."[20]

In summary: The osteopathic profession has traditionally and presently thought of manipulative therapy as something inclusive of, not exclusive to, the total practice of medicine.

The importance of the musculoskeletal system, its structural diagnosis and the usefulness of manipulative therapy in the total practice of osteopathic medicine can only be totally understood when placed in context to the basic philosophical principles of osteopathic medicine.

The importance of clinical observation and clinical reporting cannot be minimized. However, further basic research to achieve a better understanding of the function and malfunction of the musculoskeletal system and the effect of manipulative treatment both on that system and other related systems of the body should be pursued vigorously.

There is a continuing need, as there is in all of medicine, to progress further toward common agreement on terms and their meanings. One of the barriers to the more general acceptance of the efficacy of manipulative treatment in the practice of medicine can be summed up in the two words: research and communication.

To paraphrase Tinbergen's[20] concluding statement, it can be said with truth of manipulative therapy that, "a little more openmindedness, and a little more collaboration with other biological sciences, and a little more attention to the body as a whole and to the unity of the body and mind could substantially enrich the field . . ."

REFERENCES

1. Breasted, J. H., *The Edwin Smith Surgical Papyrus*, Vol. I, Chicago, Ill., University of Chicago Press, 1930, pp. 425–426.
2. Page, L., *Principles of Osteopathy*, Journal Printing Co., Kirksville, Mo., 1952.
3. Willis, P. A., *Man's Back*, Springfield, Ill., Charles C Thomas, 1953, p. 142.
4. Northup, G. W., *Osteopathic Medicine: An American Reformation*. American Osteopathic Association, 1966.
5. Northup, G. W., Osteopathic Medicine: A Medical Reformation, *J.A.O.A.*, 64:787–794, April 1965.
6. Northup, G. W., The Role of Manipulative Therapy in the Practice of Medicine, *J.A.O.A.*, 71:537–542, February 1972.
7. Northup, G. W., Osteopathic Lesions, *J.A.O.A.* 71:854–865, June 1972.
8. Stoddard, A., *Manual of Osteopathic Practice*, New York, N.Y., Harper & Row, 1969.
9. Cailliet, R., *Neck and Arm Pain*, Philadelphia, Pa., A. Davis Co., 1964.
10. Mennell, J., *Back Pain*. Boston, Mass., Little, Brown & Co., 1960.
11. Peterson, B., 1898: Radiology in Kirksville, *J.A.O.A.*, 74:167–172.
12. Still, A. T., *Philosophy of Osteopathy*, Kirksville, Mo., A. T. Still, Publisher, 1899.
13. Bean, W. B., Ed.: *Sir William Osler Aphorisms: From His Bedside Teachings and Writings*. Charles C Thomas, Springfield, Ill., 1961.
14. Risse, G. B., Calomel and the American Medical Sects During the Nineteenth Century, *Mayo Clinic, Proc.* 48:57–64, January 1973.
15. Wright, H. M., *Perspectives in Osteopathic Medicine, Kirksville College of Osteopathic Medicine*, 1974, pp. 40–46. (Monograph prepared for publication.)
16. Hoag, J. M., Cole, W. V. and Bradford, S. G.: *Osteopathic Medicine*, McGraw-Hill Book Co., New York, 1969.
17. Denslow, J. S., and Clough, H. G.: Reflex Activity in Spinal Extensors. *J. Neuro Physiol.* 4:430–437, September 1941.
18. Korr, I. M.: Neural Basis of Osteopathic Lesion. *J.A.O.A.* 47:191–198, December, 1947.
19. Korr, I. M.: Axonal Delivery of Neuroplasmic Components to Muscle Cells, *Science* 155:324–344, January 20, 1967: reprinted *J.A.O.A.* 66:1057–1061, May 1967.
20. Tinbergen, N., Ethology and Stress Diseases, *Science 185:20–27, July 1974.*

APPENDIX

Osteopathic Terminology

Records for office and hospital use—Revised June 1974

The terminology most commonly required for requesting payment of third-party carriers for osteopathic procedures is outlined on this sheet. Three types are commonly used: **H-ICDA** (Hospital Adaptation of International Classification of Disease, Adapted); **ICDA** (International Classification of Disease, Adapted); and **CPT** (Current Procedural Terminology). Each of these complete classifications is available in book form at minimal cost.* The material presented here is excerpted or adapted from those publications.

The osteopathic terminology in **H-ICDA** was worked out through the cooperation of the American Academy of Osteopathy and the American Osteopathic Association and was first published in September 1970. The committee agreed on the following general points:

A relatively uniform method for recording distinctive diagnostic and therapeutic procedures used by osteopathic physicians requires the use of standard anatomic, physiologic, kinesiologic, and pathologic terms.

The term "somatic dysfunction" is used to designate "impaired or altered function of related components of the somatic (body framework) system; skeletal, arthrodial, and myofascial structures, and related vascular, lymphatic, and neural elements." The term can then be amplified to indicate specific areas of the body that are involved. The term is further amplified to denote the specific dysfunction by using such terms as "myositis," "neuralgia," or "limited arthrodial motion." The term can be further amplified to indicate associated visceral pathology, such as "colitis" or "pneumonitis." Thus, one could indicate on a specific patient, for instance, the diagnosis of "lumbar and sacral somatic dysfunction with lumbar myositis and right sciatic neuralgia."

The term "osteopathic manipulative therapy" is defined as "a form of manual treatment" applied by a physician to eliminate or alleviate somatic dysfunction and related disorders. The term can be amplified to include the area treated and a specific type of treatment under this classification of therapy. Thus, one could indicate "osteopathic manipulative treatment, soft tissue, and corrective to entire cervical area."

The above can be put into lay terms in the following example: "Somatic dysfunction is a malfunction of a segment or segments of the spinal column which may produce limited motion in an area, muscle spasm, pain, tenderness and even remote symptoms." The osteopathic manipulative treatment could be described in lay terms as "a manipulative or manual type of treatment administered to alleviate or eliminate the result of somatic dysfunction and related disorders."

---

*H-ICDA: **Hospital Adaptation of ICDA.** Second Edition, 1973. Commission on Professional and Hospital Activities, 1968 Green Road, Ann Arbor, Michigan 48105, $8.50 (two volumes), $7.50 if prepaid.

ICDA: Eighth Revision, International Classification of Diseases, Adapted for use in the United States. U.S. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics, 1968. For sale by Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402. Publication no. 1693, $7.75 (two volumes).

CPT: Physicians' Current Procedural Terminology, Third Edition, 1973. American Medical Association, Circulation and Records Department, 535 North Dearborn Street, Chicago, Ill. 60610, $5.00.

Osteopathic terminology – H-ICDA

SOMATIC DYSFUNCTION (OSTEOPATHIC) (719)

719 Somatic dysfunction
Requires: additional code for specific condition
Excludes: vertebrogenic pain syndrome (728.0–728.9)
Definition†: impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial, and myofascial structures, and related vascular, lymphatic, and neural elements
Additional terms†: osteopathic lesion, bony lesion, joint orthodysarthric lesion
Etiology†: strains, viscerosomatic reflexes, postural faults, occupational habits and stress
Symptoms†: pain, burning, neuralgia, functional visceral disturbance
Signs†: hyperalgesia, autonomic nervous system disturbances manifested in the skin (hot, cold, dry, wet, smooth, rough), edema, contracture or contraction of muscles, hypermobility or hypomobility, maybe positional changes
Complications†: neuralgia, neuritis, disability, visceral dysfunction
Laboratory data†: electrical skin resistance, thermography
X-ray†: not definitive
Pathology†: inflammation, congestion, edema, fibrous reaction, fibrosis

719.0 Head region
occipitocervical; temporomandibular

719.1 Cervical region cervicothoracic

719.2 Thoracic region
thoracolumbar

719.3 Lumbar region
lumbosacral

719.4 Sacral region
sacrococcygeal; sacroiliac

719.5 Pelvic region
hip; pubic

719.6 Lower extremities

719.7 Upper extremities
acromioclavicular region; sternoclavicular region

719.8 Rib cage
region: costochondral; costovertebral sternochondrial

719.9 Abdomen

95 MISCELLANEOUS PROCEDURES RELATED TO . . . MUSCULOSKELETAL SYSTEM

95.8 Osteopathic manipulative therapy
Excludes: manipulation for:
reduction of dislocation (79.0–79.8)
reduction of fracture (79.0–79.8)
release of adhesions (83.9, 85.0)

ICDA – possibilities for use by osteopathic physicians

DIAGNOSIS

717.9 Other muscular rheumatism, fibrositis, and myalgia

726 Affection of sacroiliac joint

728 Vertebrogenic pain syndrome

728.0 Cervicalgia
Pain in neck

728.1 Cervicocranial syndrome
Posterior cervical sympathicus syndrome

728.2 Diffuse syndrome cervicobrachial
Cervicobrachial syndrome (diffuse)
Pain, cervicobrachial

728.3 Radicular syndrome of upper limbs
Brachial radiculitis

728.4 Cervical myelopathy
Spondylogenic compression of cervical spinal cord NOS
Vertebral artery compression syndrome

728.5 Pain in thoracic spine
Thoracic radiculitis (with visceral pain)

728.6 Thoracic myelopathy
Spondylogenic compression of thoracic spinal cord NOS

728.7 Lumbalgia
Low back pain

728.8 Radicular syndrome of lower limbs
Lumbar vertebral syndrome
Lumbosacral radiculitis

728.9 Other and unspecified
Backache NOS
Back pain NOS
Coccygodynia
Radicular syndrome NOS

846 Sprains and strains of sacroiliac region
Lumbosacral (joint)

847 Sprains and strains of other and unspecified parts of back

---

Note†: This represents a change from the 1970 system, which had numerous subclassifications.

847.0 Neck
Cervical vertebrae

847.8 Other
Coccyx
Spine (lumbar) (sacral) except cervical
Other specified parts of back

847.9 Unspecified
Back NOS

848 Other and ill-defined sprains and strains

PROCEDURES

88.8 Stretching of fascia, muscle and tendon
Includes: manipulation for stretching

R9.9 Other nonsurgical procedures

CPT – possibilities for use by osteopathic physicians

PROCEDURES

22500 Manipulation of spine, any region, during office visit

27270 Manipulation of sacroiliac joint (without anesthesia), including office visit

OFFICE MEDICAL SERVICES, ESTABLISHED PATIENT

90040 Brief service
Brief period of time, minimal effort or judgment by physician.

90050 Limited service
Limited effort or judgment, such as abbreviated or interval history, limited examination or discussion of findings and/or treatment.

90060 Intermediate service
Level of service such as complete history and physical examination of one or more organ systems, or an in-depth counseling or discussion of the findings, but not requiring comprehensive examination of the patient as a whole.

90070 Extended service
Requiring an unusual time, effort or judgment but not a complete examination of the patient as a whole.

90080. Comprehensive (adult) service
In-depth evaluation of the patient.

Note†: Similar listings are given for extremity manipulation as needed, or for manipulation under anesthesia as needed.

Note‡: Similar listings are available for new patients and for various age groups.

† Additional information supplied by the AOA–AAO committee to help with use of this system of terminology.

Discussion: The Impact of Spinal Manipulative Therapy on the Health Care System

WALTER I. WARDWELL
Professor of Sociology
College of Liberal Arts and Sciences
University of Connecticut

Since this session is focused on concepts and terminology, I will suggest a shorthand designation for the phrase "spinal manipulative therapy." Following the precedent set by osteopaths, who refer to osteopathic manipulative treatment (or therapy) as "OMT," I shall use the abbreviation "SMT" as a shorthand for "spinal manipulative therapy." I understand John Mennell's point that the concepts and techniques of manipulative therapy should not be limited to the spine, and I note that the word "spinal" is omitted from the titles of the other papers on this panel; however, since the topic for the entire conference is *spinal* manipulative therapy, and, as John Mennell observes, the term "manipulation" is often understood to mean *spinal* manipulation, I shall leave in the "S" for "spinal" and use SMT, recognizing at the same time that the concepts and principles that I am discussing may apply as well to the manipulation of other parts of the body. So much for concepts and terminology.

It was a stroke of genius to focus this conference on the research status of SMT rather than on the scientific basis of chiropractic. This allows a more precise and a more suitable topic for discussion. More important, it directs attention away from the secondary questions of relative professional competence, legal privilege, patient responsibility, and interprofessional rivalry, which have caused so much controversy and organizational jockeying throughout the past century. (It was in 1874 that Andrew Taylor Still first attempted, unsuccessfully, to present his osteopathic theories to the medical authorities at Baker University in Texas.[1]) The focus on SMT separates the question of its scientific merit as a therapeutic technique from the question of who should practice it and under what conditions. For too long a time disputes over the second question have prevented serious attempts to address the first question. I should like to add this fact as a fourth point to John Mennell's three reasons why the development of manipulative concepts has been long delayed.

An unquestioned virtue of science is the universalism of its concepts and empirical conclusions once definitional problems and semantic confusions are resolved. The previous papers have brought us a long way toward that goal. The speakers in the remaining sessions now have a group of concepts and terms with which to debate the research status of SMT. However, since my topic is the impact of SMT on the health care system, I must consider the practitioners themselves, their relationships with each other and with organized medicine, their reception by the public, and the kind of judgments which legislators and government officials have made about them.[2] Therefore, I note first that several types of practitioners of SMT are not represented here. There were several earlier varieties of practitioners of SMT, some of which no longer exist—such as neuropaths, naprapaths, spondylotherapists, mechanotherapists and naturopaths. And John Mennell has referred to the physical therapists, a few of whom are beginning to practice SMT. Since the fate of some of these groups logically and politically depends on the outcome of this conference, they should perhaps have been offered an opportunity to con-

tribute to it, and they should be considered when the policy question is faced of who should practice SMT and under what legal, professional, and reimbursement conditions.

Now to the topic of the impact of SMT on our health care system. I conceive the health care system as categories of health practitioners and their assistants, plus practice facilities and the patterns of recruiting, training, paying, supervising, and motivating them; also their respective locations within the system, their numbers, their legal or conventional rights, privileges, and responsibilities; and their professional relationships with each other —conflict, competition, cooperation, subordination, or whatever. The attitudes and behavior of the public, the consumers of health services, are also relevant. For example, most chiropractic patients, while aware of the disapproving attitudes of organized medicine, appear to be satisfied with chiropractic treatment. Many patients regard their chiropractor as equal to other doctors, and some even consider chiropractors to be specialists like orthopedists or obstetricians.

The impact of SMT on the health care system has been mediated through legislative action. Despite the fact that its scientific and clinical value has been challenged by the American Medical Association, the legislatures of all 50 States and the District of Columbia have licensed chiropractors. All the States have authorized reimbursement in Workmen's Compensation cases. Thirty-one States have legislated equal treatment of chiropractors by insurance companies, while most insurance companies have decided independently to pay chiropractors for services rendered to patients. And finally Congress has authorized payments to chiropractors under both the Medicare and Medicaid programs. Symbolically important was the decision last year by the U.S. Office of Education to designate an official accrediting agency for chiropractic colleges—a group called the Chiropractic Commission on Education. There are about 6,000 students currently studying in 12 chiropractic colleges; 850 chiropractors were graduated from 10 of these colleges last year. These figures compare with 3,156 students at 9 osteopathic colleges, which graduated 587 osteopaths last year.

It is estimated that about 17,500 practicing chiropractors treated 2½ percent (i.e., over 5 million) of the population last year, which, in addition to the unknown number of patients receiving SMT from osteopaths, medical doctors, and physical therapists, is an impressive measure of the impact of SMT on our health care system. I assume that nearly every chiropractic patient receives SMT.

Another type of impact can be seen in the increased acceptance of osteopaths by the American Medical Association as qualified professional associates. While such recognition has been attained in part because most osteopaths no longer use OMT as their principal therapeutic technique, such recognition by the AMA as professional colleagues carries considerable symbolic impact.

Equally important is the North American Academy of Manipulative Medicine, which John Mennell helped to found about 10 years ago, for now there exists a dedicated group of manipulators within the medical profession itself. The importance of this group is also partly symbolic because it still has relatively few members, it lacks acceptance by more orthodox colleagues in physical medicine and orthopedics, and it has had to suffer not a few indignities for its venturesomeness.

Another recent development is the establishment by a group of physical therapists devoted to manipulation of a Section on Orthopedic Physical Therapy within the American Physical Therapy Association.

Despite these developments there is still a great deal of ignorance and bias in medical circles concerning osteopaths and chiropractors. The official position of the American Medical Association until 1961 was that osteopaths practice sectarian medicine; and the decision by the AMA House of Delegates in that year did not really reverse that judgment but merely left it to each State medical society to decide for itself whether the osteopaths in their State practice sectarian medicine or not.[3] The AMA's judgment on chiropractic has been even more severe. For example, as recently as May of last year the Chairman of the Committee on Quackery, Dr. Thomas Ballantine, in testimony before the Advisory Committee on Accreditation and Institutional Eligibility of the U.S. Office of Education quoted from an earlier report which "describes the cult of chiropractic as involving the purported 'healing' through methods or according to theories which do not have a scientifically based foundation," and goes on to state: "Medical authorities unanimously agree that chiropractic has no validity. The cult's theories have never been supported by objective evidence, and they have been thoroughly refuted by medical science." Further-

more, "Chiropractic treatment often produces actual physical damage to patients." It is my strong impression that such statements cause the AMA to lose credibility and are probably self-defeating as regards the attainment of AMA objectives. I realize, of course, that "SMT" is not identical to "chiropractic theories," which, particularly in the past, have sometimes emphasized spinal subluxations and their correction to the exclusion of other causes of disease and therapies. However, the AMA statements distort reality when they identify chiropractic with the oldest and the most cultist views that some chiropractors have held.

The AMA's statements on chiropractic education are also misleading. While chiropractic colleges have lagged behind medical and osteopathic colleges in lengthening their course of instruction and improving the quality of their curriculum and graduates, all chiropractic colleges today require 2 years of preprofessional college credits for admission and a 4-year course for a D.C. degree. Indeed, the laws of 38 States now require a minimum of 6 years of postsecondary education for a chiropractic license while 15 States require that chiropractors pass the same examinations in basic science subjects that medical and osteopathic candidates must pass. I am speaking of minimum requirements. The percentage of students entering chiropractic colleges who already have the baccalaureate has continued to rise; in Joseph Janse's college this past fall, 43 percent had a bachelor's degree.

The stereotypes are still with us, however. A physician, who is one of the most well informed I know on the subjects of SMT and chiropractic, referred to a chiropractor who is participating in this conference in these words: "He is not really a chiropractor." When I inquired what he meant, he said: "He (meaning the chiropractor) does not believe in using chiropractic to treat every disease." What he could have said is: "Some chiropractors are not chiropractors," or perhaps even: "Some of my best friends are chiropractors!"

In my capacity as panel discussant, I would like to make a few comments on the preceding papers. I consider sociology to be one of the basic medical sciences, but since I am not a historian or trained in the medical sciences, my remarks must be limited with regard to technical questions.

I have a feeling of genuine gratitude to Elizabeth Lomax for the excellent job of historical scholarship which she has performed. I wish we also had the benefit of a medical anthropologist's survey of the variety of primitive and folk practices which resemble SMT, such as the mili-mili and the lomi-lomi of the native Hawaiians.

George Northup's paper presents a clear statement of the position which I would expect from a "true, tried, and qualified osteopath," to use Andrew Taylor Still's phrase. The sharpest differences in conceptualization lie between the Northup and Janse papers, on the one hand, and John Mennell's on the other. For John Mennell advances a mechanical explanation of "joint dysfunction," and he discounts the role of impaired circulation and of nerve irritation or impingement. I should like to learn his reaction to Northup's and Janse's arguments that osteopathic lesions, subluxations, and joint dysfunctions, if they are the same thing, also involve blood circulation and neural inhibition or stimulation; and his opinion of the "reports on clinical observations regarding the interplay of dysfunctions in the spine and organ dysfunctions" to which he refers near the end of his paper.

Based on the papers presented this evening and on my own investigations I have reached the following conclusions:

(1) Osteopathy and chiropractic were not new discoveries by Andrew Taylor Still and Daniel David Palmer respectively. On the contrary, there was a long tradition of irregular practitioners, mainly "bonesetters," who treated patients by putting little bones that were "out" back in place by manipulation. Elizabeth Lomax's conclusion that one can only regard osteopathy and chiropractic as legitimate offspring of contemporary thought explains such metaphysical expressions as innate intelligence and also where osteopathy and chiropractic derive some of their therapeutic theories and techniques. As dentists, podiatrists, optometrists, and mental healers arose as providers of specialized services that orthodox practitioners were unable or unwilling to perform, so osteopaths and chiropractors found gaps and niches in the health care system which they could fill.

(2) Theories advanced to explain the clinical benefits of SMT have been numerous and various. The four previous papers all document this.

(3) Opposition to SMT by regular medical practitioners has been strong for at least 150 years, despite its advocacy at fairly frequent intervals by physicians like Harrison, Little, Paget, Hood, the Mennells, and the Cyriaxes. A wide gap in com-

munication and professional interaction has therefore persisted between manipulators and regular practitioners. Even the rapprochement between medicine and osteopathy that has been growing in the past decade appears to have resulted because osteopaths have become more like allopathic physicians rather than because allopathic physicians have begun to accept SMT. Actually this relationship seems more to be an organizational maneuver than the product of intellectual conviction, as attested to by the resentment of traditional osteopaths toward their California brethren who converted to medicine.

(4) The major impact of SMT on our health care system appears to have been the development of several separate and distinct professions outside orthodox medicine—not only osteopathy and chiropractic but also naturopathy. Although other healing traditions such as herbalism in America and natural healing methods from Europe exerted an independent effect on all three of these professions, such influences appear to have been especially strong in the case of naturopathy. Due to the strength of the opposition by the AMA, even the modest amount of accommodation between medicine and these other health professions that has been taking place in Canada, Germany, and several other parts of the world has not occurred in the United States.

(5) SMT has remained almost completely neglected by orthodox medicine, probably to the latter's disadvantage. A few physicians abandoned medicine over the years in order to practice as osteopaths or chiropractors. Those who remained in medicine and practiced SMT were often stigmatized and discriminated against. The laboratory and clinical research that should have been done to evaluate the efficacy of SMT was not done. The recent publication in *Lancet* of a Utah study comparing favorably the relative success of chiropractic and of medical treatment in 232 Workmen's Compensation cases with back or spinal problems stands nearly alone in the medical literature.[4] Consequently the benefits of SMT (whatever they may be) have not been generally available to the patients of medical practitioners.

(6) Because of the hostility of organized medicine, osteopathy and chiropractic have remained more sectarian. Chiropractic became defined not only as "separate and distinct" from medicine but as the diametric opposite. A process of polarization occurred in which differences were exaggerated and common elements minimized. A new terminology suggested that chiropractors not *diagnose* disease but *analyze* the spine in order to discover the *cause* of disease. Drugs and inoculations became defined as poisons antipathetical to true chiropractic. While these policies may have provided some minor legal advantages (e.g., it could be argued that since chiropractic is not the practice of medicine, it therefore should be separately regulated), they appear mainly to have aided B. J. Palmer's strategy for condemning his opponents and retaining his dominant position in the profession. The present division among chiropractors still hinges on the question of how different from and opposed to medicine chiropractic is held to be.

Since osteopathy was not so strongly opposed to the use of drugs, it could more easily evolve in a medical direction. And the fact that the osteopaths who strongly emphasized OMT risked being identified with chiropractors may have pushed osteopathy further in a medical direction. Nevertheless, while osteopathy theoretically could have monopolized the common ground between medicine and chiropractic and thus perhaps have bridged the gap, it has tended to bifurcate through being torn between medicine and SMT. That is the dilemma still faced by osteopathy today.[5]

(7) Since SMT is a highly developed and specialized skill requiring hundreds of hours of theoretical and practical training for its mastery, it would not be practical to make every medical school graduate a competent manipulator. A possible alternative would be for specialists in physical medicine or orthopedics or neurology to become proficient manipulators. But apart from the reluctance of many such specialists to master SMT, there simply are not sufficient numbers of specialists to meet the needs of all the patients requiring SMT (assuming that the consensus of this conference will be that SMT is valuable therapy).

Another possibility would be for the physician's traditional assistants in physical medicine—i.e., physical therapists—to become manipulators, which a few of them are now beginning to do. The difficulty with this solution, however, is that it would be as inefficient for a physician to prescribe manipulation to be carried out by a technician as it would be for a surgeon to stand by while an assistant performs his surgery. The manipulator should be able to evaluate the patient's suitability

for SMT, and make a differential diagnosis; and the manipulator should know the contraindications of SMT, when to refer a patient to another practitioner or to a specialist, and when to modify treatment. This requires diagnostic and clinical skill. To incorporate these skills into the training of physical therapists would result in duplication of the length and type of training now offered in chiropractic colleges. As an alternative, chiropractors could practice under or in cooperation with general medical practitioners or specialists in physical medicine, orthopedics, etc., but such a solution would have to overcome very strong resistance. There is, therefore, no easy solution to the question of how specialized practitioners of SMT, like chiropractors, can best be fitted into our health care system.

(8) Compared to drug therapy and surgery, the risks of SMT are minimal, if one can judge from the paucity of reported cases of harm done to patients. The cost of malpractice insurance for chiropractors has remained low while that for medical practitioners, particularly surgeons, has gone up sharply. When SMT is carried out by a skilled practitioner, there is little risk to the patient, provided of course that a proper differential diagnosis has revealed that SMT is indicated and that treatment by some other means or for some other condition (e.g., a malignancy) is not prevented or delayed.

(9) Consideration should be given to the possible role SMT can play in health maintenance and illness prevention. Philosophically most of us would probably agree with Rene Dubos' thesis that Hygeia, the goddess of right living, should take precedence over Asclepius, the god of healing.[6] Certainly the contemporary ethos has swung far in that direction. I am referring to the concern especially among young people over environmental pollution, clean air, clean water, pesticides, organic gardening, natural foods, aerobic exercises, and a simple life. These themes have long been emphasized by osteopaths, chiropractors, and naturopaths, as well as by many medical doctors. SMT on a regular prophylactic basis has been advocated as a means by which a healthy person can become even healthier. Although the practitioners of prophylactic SMT have sometimes been charged with giving excessive or unnecessary treatment, a real possibility exists that such use, along with other natural healing methods, will result in better overall health. This possibility would seem to justify designing a large-scale prospective epidemiological study in which the incidence of cancer, cardiovascular disease, mental breakdown, and other degenerative and contagious diseases would be assessed in two carefully controlled groups of subjects from the same population—one taking regular SMT treatments, the other not. It would be necessary to control for the effects of other relevant variables, especially diet, exercise, smoking, etc., in order to isolate the effects of SMT. The hypothesis would be that since disease is a process in which the resistance and level of functioning of the host is a critical factor, efforts at health maintenance should be directed at the natural resources of the human organism as an integral system comprising musculo-skeletal, circulatory, neurological, and other subsystems. If "the body is its own laboratory," as the osteopaths have said, and is vitally dependent on its structural and neurological integrity, as both osteopaths and chiropractors have claimed, then a prospective epidemiological study could test the value of SMT as a prophylactic strategy.

To summarize, the principal impact of SMT on our health care system so far has been social, economic, and political in that it has resulted in several jealously competing professions with different legal standings, therapeutic philosophies, and modalities of practice. Although a few medical physicians like the Mennells, the Cyriaxes, Robert Maigne, and others have used SMT, most practitioners of it are still outside the medical mainstream. As a result, the vast majority of Americans have not been exposed to SMT and thus have only vague, possibly erroneous, and generally skeptical attitudes toward it. Basic research has been limited and the planning of clinical trials is just beginning (in Toronto, for example). More research is needed. It is clear that most of the impact of SMT on our system of health care has yet to occur.

REFERENCES

1. Northup, George W., *Osteopathic Medicine: An American Reformation.* Chicago, Ill. American Osteopathic Association, 1966, p. 14.
2. My publications in this area include: *Social Strain and Social Adjustment in the Marginal Role of the Chiropractor.* (Ph. D. thesis, Department of Sociology, Harvard University) 1951; "A Marginal Professional Role: the Chiropractor." *Social Forces,* 30:339-348, 1952; "Public

597-036 O - 76 - 5

Regulation of Chiropractic." *J. Natl Med Assoc.* 53:166–172, 1961; "Limited, Marginal, and Quasi-Practitioners." in Howard E. Freeman, Sol Levine, and Leo G. Reeder (Eds.) *Handbook of Medical Sociology.* New York, N.Y. Prentice-Hall, 1972.

3. "Osteopathy: Special Report of the Judicial Council to the AMA House of Delegates." *JAMA,* 177:774–776, 1961.
4. Kane, Robert L., et al., "Manipulating the Patient." *Lancet,* 7869:1333–1336, June 29, 1974.
5. New, Peter Kong-ming, "The Osteopathic Students: A Study in Dilemma." in E. Gartley Jaco (Ed.), *Patients, Physicians, and Illness.* Glencoe, Ill. Free Press, 1958.
6. Dubos, Rene, *The Mirage of Health.* Garden City, N.Y. Anchor, 1961.

Chairman's Summary: Evolution and Development of the Concepts of Manipulative Therapy

DONALD B. TOWER
Director, National Institute of Neurological and Communicative Disorders and Stroke
National Institutes of Health
Bethesda, Maryland

In discussion it was pointed out that an important figure in the history of manipulation – one who provided inspiration for both chiropractic and osteopathy – had not been mentioned. He was J. Evans Riadore, a London physician who wrote a treatise on "Irritation of the Spinal Nerves" in 1843. He attributed many diseases to this condition, stating: ". . . if any organ is deficiently supplied with nervous energy or of blood, its functions immediately, and sooner or later its structure, become deranged . . . ."

This was a viewpoint subsequently echoed by osteopaths and chiropractors. Riadore went on to describe root pain arising from compression in the intervertebral foramen; disc degeneration and manipulation to treat these conditions. Thus, it was suggested that he ought to be considered a source for much of what is under discussion at this workshop.

The discussion concluded with a plea to establish observable criteria and definitions of terminology. These were perceived to involve questions about the kinds of observable conditions treated with spinal manipulative therapy; about ways in which such conditions could be observed by groups of independent, unbiased observers; and about the influences of the therapy and the means for recognizing and evaluating these influences. Such considerations seemed essential to any scientific discourse on the problem.

Chapter III.

What Do the Basic Sciences Tell Us About Manipulative Therapy? (Anatomical and Biomechanical Studies)

Philip Greenman, *Chairman*

The Vertebral Motor Unit and Intervertebral Foramen

DAVID C. DRUM
Canadian Memorial Chiropractic College
Toronto, Ontario, Canada

INTRODUCTION

The main goal of all scientific research is to develop adequate conceptual models about the relevant problem area.[1] The vertebral motor unit (bewegungssegment – motion segment) is Junghanns' term for his conceptual model of the segmentally arranged functional unit of the spine.[2,3] There are usually 23 typical motor units found from the junction of C2–C3 to that of L5–S1, each composed of an intervertebral disc, neurovascular elements concentrated within the neural foramen and intervertebral foramina, posterior spinal joints and all the connective and muscular tissues supporting and limiting intersegmental motion. The posterior articulations and disc form the mobile elements; the ligaments and muscles form a system that ties them together. Certain relatively constant modifications associated with different spinal regions are encompassed within the model of the typical vertebral motor unit. (e.g. Vertebral artery and joints of Luschka[4-6] in the cervical spine, costovertebral articulations[7] and sympathetic chain in the dorsal spine and the transforaminal ligaments and cauda equina in the lumbar spine.) The concept of the functional unit has also been effectively applied to the pelvic girdle.[8,9]

In an attempt to shift prevailing attitudes toward spinal disorders from the skeletal system to the soft tissues, Junghanns created a concept in 1950 that has great relevance to manipulating spinal therapists and bioengineers. It gave them a functional concept of vertebral subluxations by stressing the close relationship between the morphological features of the motor unit and its dynamics.[10-18] Was the vertebral motor unit loosened (unstable, hypermobile), fixated (blocked, hypomobile) or displaying more complex erratic motion? These questions have encouraged clinical investigations into the relative efficacy and priority of immobilization and manipulatory techniques and inquiry into therapeutic measures directed at avoidance of damage to neurovascular elements.[19-29]

THE TYPICAL VERTEBRAL MOTOR UNIT

Anatomical Components

Junghanns' unit is made up exclusively of soft tissues schematically represented in figure 1.

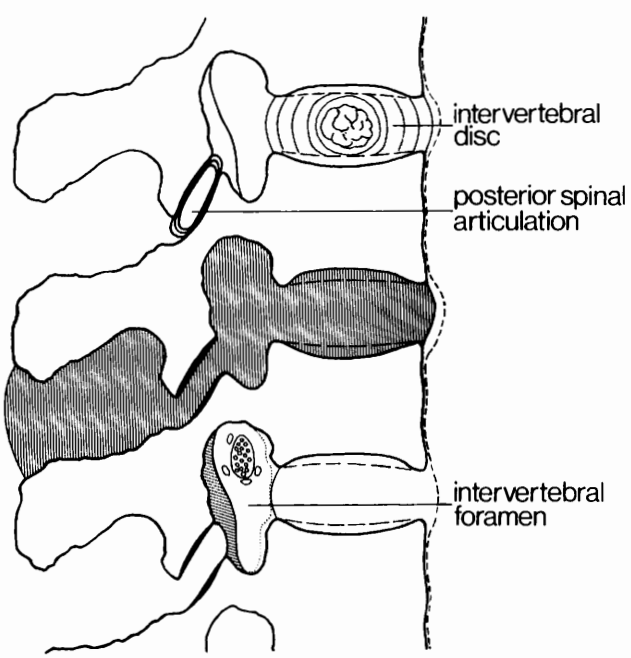


Fig. 1. The Vertebral Motor Unit (Junghanns).

Theoreticians of spinal manipulation, at one time or another, have implicated virtually every anatomical component of the vertebral motor unit in their attempts to explain the mode of action of their therapy. Studies identifying the pain sensitive tissues of the back have, on occasion, been used to formulate rigid theories that must be considered inadequate if for no other reason than their overemphasis on single anatomical features. Conceptual dogmatism has done little to facilitate interdisciplinary dialogue. In the light of new data some cherished models will have to be abandoned altogether; others expanded to incorporate these findings.[30, 31]

The Usefulness of the Vertebral Motor Concept in Spinal Manipulation

Employing the concept of the spinal organ system containing functional units, clinical procedures have been evolved to discover altered mechanics (loosening, blocking, erratic motion) displayed by the motor unit before there are visible radiographic changes. Discovering such early functional impairment is of particular importance in the biomechanical therapies because most are reversible, many permanently, with conservative care.

Except through surgical exposure and special radiographic techniques using contrast media, soft anatomical components of the motor unit cannot be adequately visualized, their status generally being inferred from the positional and dynamic relationships of adjacent vertebrae. This explains Parke and Schiff's [32] incorporation of the opposing superior and inferior halves of each vertebra (which also represents an embryologic somite) and Depalma and Rothman's [33] inclusion of the adjacent osseous segments in their vertebral motor unit models. The early manipulators' "bone out of place" concept clearly overlooked the significance of evaluating the integrity of the radiolucent soft elements and failed to recognize their pathogenetic precedence—a regrettable limitation, for as Maigne reminds us, a manipulation acts only upon these mobile structures.[34] (Jackson [35] in an analysis of more than 5,000 patients with symptoms referable to the cervical spine found their injuries in 75 percent of the cases confined to the soft tissue structures.) But this oversight was in the days before the assimilation and application of Junghanns' concept on an international, multidisciplinary basis.

Lewit,[16] Drum,[14] and Howe [13] have reminded therapists using manipulation that many congenital, traumatic, degenerative, or adaptive positional disrelationships of vertebral motor units may well be irreversible; that there are permanent intervertebral subluxations that cannot be manipulated back into "proper alignment." Markedly abnormal motor units are distorted as a result of advanced breakdown of their stabilizing elements. A manipulation would have to instantaneously restore these degenerated tissues to "hold." This is not to suggest that manipulation is contraindicated in all architecturally modified motor units. But the therapeutic goals are more sophisticated and an advanced level of sensory (palpatory) and motor (manipulative) skills is necessary to insure the delicacy of the maneuver, which even in the hands of an experienced clinician is not without risk.[36] Novices in vertebral manipulation, regardless of their intellectual appreciation of the problems, are encouraged to develop their neuromuscular and proprioceptive abilities by mobilizing vertebral fixations and avoiding the unstable segments altogether. To an inexperienced observer all spinal-pelvic manipulations may look the same but they are not, and while the subtleties of discrimination in technique application may not be discussed at this conference, they do exist.

Fisher,[37] an orthopedic specialist writing about the results of manipulation in lumbar intervertebral disc lesions warns: "The results depend upon careful selection of cases and upon the skill and experience of the manipulator. I strongly suspect that many of the sad examples regularly brought forward by opponents of manipulation are due to the lack of the necessary qualities in the operator and to the use of brute force." If such advice [28-30] is disregarded in the present atmosphere of enthusiasm toward spinal manipulation, I predict an escalation in the already proliferating reports by neuro and orthopedic surgeons describing serious complications of ill-advised, ill-performed manipulations. Darbert et al.[38] warn of the danger of combining two therapeutic modalities, back manipulation and anticoagulant medication, without appropriate interprofessional communication, reporting a case of spinal meningeal hematoma.

Specialists in manipulation have been interested in the observations of Sandoz,[10] namely, that reversible, episodic intervertebral motor unit functional impairment affecting motion in one dimension may be superimposed on grossly malaligned segments and that manipulation may improve their function without alleviating their previous instability. Such patients are, of course, prone to

recurrence. Froning and Frohman [39] note that an abnormal spinal joint with qualitative abnormal motion may still move through a full arc of motion, while Jirout [17] describes rotatory blocks (fixation) in lateral cervical inclination coincident with pathological increase in flexion-extension in the same vertebral motor unit. Gillet and Liekens [11] also write of hypo and hypermobility manifesting during different planes of movement of the same motor unit and refer to the complexities of incomplete fixations that may reveal themselves in erratic, jumpy motions at some point during the arc of movement. German manual medicine specialists hold the opinion that reducing fixations of anatomically intact and degenerative motor units still capable of function and compensation is the main objective of manipulative therapy.[40] If these findings are substantiated, vertebral motor unit dynamics are much more complex than hitherto suspected, and it may be naive to rely on an "all or nothing" polarity functional classification (i.e. hypo-hypermobile) of the motor unit, which is, of course, multiaxial.

The vertebral motor unit concept redirected attention from the bony confines of the intervertebral foramen to its soft tissues and stressed the developmental and topographic interdependence between the fibrous structures surrounding the intervertebral foramen and the function of the structures passing through it. Inquiry has been stimulated into the sequential influences of congenital and acquired disorders of a single major component of any one of the spinal motor segments on both the functions of the other components of the same unit and other levels of the spine.

THE INTERVERTEBRAL FORAMEN

The intervertebral foramen is the elipsoid aperture, more of a short canal than orifice, that gives exit to the segmental spinal nerves and entrance to the vessels and nerve branches that supply the bone and soft tissues of the vertebral canal. Its neurovascular elements may be influenced by the intervertebral disc and other motor unit ligaments, the posterior spinal articulations and intrinsic alterations in their own tissues.

Neurovascular Components

Part of the originality of the chiropractic concept, and most criticized, has been the hypothesis that spinal disturbances are capable of generating malfunctions in areas remote from the spine. Interest in the intervertebral foramina as a prime area for potential neuromechanical conflicts has increased since Gutzeit,[41] Wendt,[42] Duus and Kahlau,[43] Reischauer[44] and Pillokat[45] in the 1950's claimed that secondary irritation of the cervical ganglia may lead to organ lesions and functional disorders. Chiropractors have from their beginning drawn attention to the intervertebral foramina[46] in spite of cadaver studies claiming they were anatomically unchanging.[47]

Breig[48] considers the hind brain, medulla oblongata, and cord as forming a functional unit together with their nerves and ligaments. A remarkable mobility of the neural structures (nerve roots, posterior root ganglia, spinal nerves), their meningeal sleeves and fibrous sheaths in the normal intervertebral foramen permits this "radicular complex" to adjust to the great range of spinal movements. The space between the definitive sheaths of the neural structures and the foramen is occupied by fine connective tissue with no thickenings attaching the radicular complex to bone. Sunderland[49] has recently discovered that only the fourth, fifth, and sixth cervical spinal nerves have strong attachments (to the transverse processes). He suggests this is because of the additional strain of shoulder girdle movements. He further describes a dynamic "plugging" action of the dural funnel at the intervertebral foramen limiting the movement of a laterally tractioned spinal nerve. Traction on the dura is transmitted via the dentate ligaments to the spinal cord. The great lengths of the lumbosacral nerves and nerve roots dissipate stresses[50] efficiently except perhaps the first sacral nerve which Hollinshead[51] reports to be relatively taut and unyielding with its dural sheath attached to the margins of the intervertebral foramen. Frykholm,[52] in illustrating malformation of root pouches and spinal nerve angulation, reminds us that a perfect functional anatomy is seldom found, except in a small privileged group of individuals.

Neuromechanical conflicts in the intervertebral foramina rarely involve pure statics; advanced radiographic degenerative changes are frequently found in asymptomatic spines. Rosomoff and Rossman[53] estimated 75 percent of persons over 50 have some narrowing of the cervical intervertebral foramina and encroachment from joint of Luschka hypertrophy is well documented.[54] We may use the analogy of a "one-two knockout punch," the

static reduction of a cushioning, shock-absorption mechanism upon which is superimposed dynamic demands of motion of the spine and extremities. The significance of considering dynamics along with tissue alteration is at least honored by the universally observed practice of removing the mechanical component by suggesting rest and immobilization devices. The theory of cord, root, and dural tension opens a new field for investigations. (Breig[48] has shown the spinal cord does not move up and down axially in the canal, but adapts itself to varying canal lengths by plastic deformation that affects its cross-sectional area. Its blood vessels are kept open by stiff fibers first described by Key and Retzius.[55]) Chronic compression of a radicular nerve by cervical intraforaminal disc protrusion may initiate root-sleeve fibrosis with thickening of the adjoining dural tissue. This places the nerve root under tension even during physiological head and neck movements.[56-60]

Other vertebral motor unit changes may contribute to neurovascular irritation and perhaps compromise hemodynamics. The role played by the venous drainage system of the vertebral column is, according to Batson,[61] as important as that of the other main venous systems in the body—caval, portal, and pulmonary. They have soft, almost valveless walls and are tortuous. It may be appreciated that venous blood flow in the spinal complex depends to a large degree upon motion in the vertebral motor unit as these veins are deprived of the benefits of active muscular contraction and relaxation, which elsewhere assist in the propulsion of venous flow. Fixation subluxations may therefore assume an, as yet, unexplored role in pathohemodynamics.

Impediments to venous flow, caused by varices, obliteration, congestion, are of both morphological and functional importance, inasmuch as the vertebral venous system in an adult of average build holds about 200 ml. of blood; in the presence of congestion, this volume may be two to three times greater.[61] The extensive anastomoses of the vertebral venous system with other body venous systems and the potential for reversal of their blood flow (as for example in raised intra-abdominal pressure) involve them in circulation disorders originating in other venous regions. Brain[62] claims venous compression in cervical spondylosis results in blood stasis and vascular dilatation causing additional compression on neural elements and Jones implicates venous congestion in influencing the sympathetic plexus surrounding the vertebral artery.[63] Macnab[64] questions whether the pain of osteoporosis may not be due to venous stasis in the vertebral bodies. Olsson[65] associates mechanical root pressure with edema formation in the sheaths and endoneurium. He theorizes the sudden elaboration and subsequent reabsorption of this edematous fluid may be mirrored by the acute exacerbation-remission clinical pattern. The small blood vessels of the roots and ganglia, unlike those in the central nervous system, are surrounded by large extracellular spaces and do not exhibit the "blood-brain barrier phenomenon."[66] Olsson suggests that their easy permeability in combination with the extracellular spaces facilitates diffusion of edema. Others[67] relate the familiar gradual reduction of vibratory sense in aging people to the unusual arterial supply to the spinal ganglia where the ganglionic vessels branch at right angles or by way of recurving arcs against the direction of blood flow in the parent spinal artery. Japanese investigators[68] have suggested that inflammatory processes play an important role in the production of symptoms of cervical and lumbar disc lesions and that radicular pain is closely related to the pathophysiological state of the epidural space.

The Intervertebral Disc and Other Vertebral Motor Unit Ligaments

Interest in the ligamentous elements of the vertebral motor unit has centered on their ability to compress neurovascular structures, their contribution to shock absorption ("joint-play"[69]) and stabilization by limiting intersegmental movements, and their role in the pathogenesis of motor unit hypermobility.

Farfan[70] considers a degenerated disc as a "joint" suffering from a chronic sprain and its annulus as the main ligament joining two adjacent vertebrae. Investigations[71-77] centered on the intervertebral disc over the last four decades have been proliferating to the point where "mesmerization"[78] has been suggested. Shealy,[79] in speaking of 700 "flunk-outs" of disc surgery, comments on the frequency with which patients report their pain to be exactly the same as it was prior to the first of their average of four lumbar surgeries. German physical medicine specialists[80] state that only in the lower lumbar spine do true disc herniae deserve their present focus of attention. They reason that since manipulation is also effective in segments without discs (e.g. the atlanto-occipital region), the action principle of manipulation must involve more

than reduction of disc fragment displacement. Junghanns[2] and Armstrong[81] describe internal disc derangements with displacement and incarceration of a disc sequestrum within the disc space causing motor unit locking and de Sèze[82] believes all fixations are secondary to a disc lesion. While admitting that disc derangement is undoubtedly responsible for much lumbar pain, Cover and Curwen[83] feel that the pendulum has swung too far and diagnosis of a "disc lesion" is apt to be made too readily. McRae [84] shows that although disc protrusions are common, they seldom produce symptoms – that overemphasis on the importance of cervical disc protrusions has led to operations in patients with minor abnormalities in the myelogram. Many lumbar discs suspected of compressing nerve roots show no annular rupture but simply bulge posterolaterally.[85]

Bick[86] has referred to disc changes inducing motor complex loosening and Macnab[87] has illustrated hyperextension subluxations from degeneration of the anterior annular fibers. He also indicates a mechanism of discopathogenetic subluxation caused by loss of intervertebral disc height where the posterior joints subluxate and the vertebral body moves downward and backward. Similar disc narrowing is implicated by Kunert [88] and Nilsson[7] in loosening the rib's costovertebral and costotransverse attachments through a "settling" of the opposed vertebrae with irritation of the sympathetic ganglia located immediately anterior to the costovertebral joints as a potential sequela.

The elastic ligamenta flava, between the laminae, may bulge forward to encroach on the intervertebral foramen and cervical cord[89-91] but Breig[48] considers the volume of the ligamenta flava too small to cause cervical myelographic defects, interpreting these as transverse dural folds.

Various pathologies affecting the ligaments may compress neural elements[92] while others may lead to ligamentous softening.[93] The pelvic and lower spinal motor units may be rendered excessively mobile by the hormonal environment of pregnancy and minor ligamentary strains may only cause discomfort premenstrually.[94] It should be recalled that vertebral osteophytosis is an osteogenic process representing a local response to ligamentous strain.[95]

Lumbar ligamentous variation, although commented upon by Macnab,[87] is not well documented in standard anatomy texts. Golub and Silverman,[96] in an anatomical study of the intervertebral foramina of 10 spines, found[47] anomalous transforaminal ligaments, most cases grossly diminishing the space available to the emerging nerve root. These are strong, unyielding structures of varied width and thickness ranging from 2 to 5 millimeters.

It has been the author's repeated observation that the most common error in the application of the modality of spinal manipulation is the failure to recognize the hypermobile vertebral motor unit. Newman [97] defines instability as the loss of integrity of the soft tissue intersegmental control. The motor units are bound together by ligamentous systems under tension; degeneration of any one will alter the dynamic equilibrium of the entire complex. Jirout [98] has recently investigated the role of the nuchal ligament in cervical spine dynamics and White and Hirsch [99] that of the ligamentum flavum in restraining axial rotation in the thoracic spine.

Vertebral Motor Unit Innervation

The posterior rami of the spinal nerves supply sensory fibers to the fascia, ligaments, periosteum and apophyseal capsules and motor fibers to the muscles.[100-101] The sinuvertebral nerve, a recurrent branch of each spinal nerve, has a dual spinal and autonomic composition. (Occasionally these two components are distinct when they enter the foramen, but usually they are reflected as a common bundle.) It contains sensory fibers and supplies the posterior and anterior longitudinal ligaments, dura mater, periosteum, articular connective tissues including the capsules of the "joints" of Luschka,[102] vascular structures [103] and, according to some,[104-109] the posterior part of the annulus. Even if the debate goes against those who have demonstrated nerve endings within the outermost laminae of the disc, their distortion could certainly produce tensions in the overlying highly innervated connective tissues. The ascending and descending branches of the sinuvertebral nerve (the usual distribution being one segment up and two down) contributes to difficulties in localizing vertebral motor unit lesions in the absence of signs of nerve root compression.[110] Macnab [64] emphasizes that pressure on a nerve root by itself is not painful, but secondary inflammatory, congestive or chemical changes will produce pain (i.e. chemical plus mechanical factors in combination). He further cites the work of Hirsch and Bobechko [111] suggesting that an autoimmune response may take place in the disc. The occipito-atlanto and atlanto-axial joints are innervated by the

anterior branches of the first two cervical nerves. [112] The spinal ganglia are usually in the intervertebral foramina immediately lateral to the sites where the nerve roots perforate the dura mater. Each anterior branch of the spinal nerve is joined by a grey ramus communicans from the corresponding ganglion of the sympathetic trunk while the anterior branches of the spinal nerves from T1–L2 each contribute a white ramus communicans containing preganglionic sympathetic fibers arising from lateral horn cells to the corresponding sympathetic ganglion. The clinical significance of these sympathetic relations is a fertile area for future investigation.[88, 16, 40]

The Posterior Spinal Articulations

The posterior spinal joints are similar to peripheral joints in their construction. They have articular cartilage, a loose capsule with synovial membrane, small menisci and related muscles and ligaments. They are subject to the same insults as other joints [113] and show a higher frequency of degenerative changes than any other synovial joint in the body.[114] Zuckschwerdt [115] in 1960 rediscovered small, semilunar "menisci" filled with adipose cushions penetrating the intra-articular space of the posterior spinal joints.[116] He proposes "blocking" from meniscal jamming. Schmorl [2] suggests incarceration of an articular villus or meniscus and Kraft [117] and Hadley [118] incriminate synovial impingement in the etiology of sudden, severe low back pain. Badgley [119] finds no essential difference in the pathomechanics associated with free body formation in apophyseal joints from those of knee joint derangement. Hirsch [120] and Lazorthes [121] have confirmed the extensive nerve supply while Shealy [122] and Rees [123] have reawakened interest in apophyseal changes' etiological significance to back and leg pain [124] by surgically destroying the posterior branches of the dorsal roots supplying the facets.

The posterior articular processes assist in providing motor unit stability [125] and normally bear no appreciable spinal load [126] but may become weight-bearing in certain postural distortions.[127] Jung, Brunschwig [128] and Vele [129] conclude that much of the proprioceptive stimuli for spinal balance arises from the holding elements of these articulations.

Those concerned with spinal manipulation are reminded of the following: (1) If primary disc changes have led to distortion and abnormal movement within the associated facet joints the reduction of secondary apophyseal joint lesions does not guard the patient from recurrence;[130] (2) the common occurrence of articular tropism [131] should make it obvious that the concept of normality or abnormality related to median or off-centered vertebral positions is obsolete;[132] (3) the aim of "realigning" offending vertebrae with posterior joint arthroses by thrusting toward the side of arthrotic block is utopic [132] and in violation of Maigne's rules;[19] and (4) the tendency of too quickly condemning the apophyseal joint when the pain may be coming from the mechanically or chemically irritated tissue of attachment of muscle, tendon, intermuscular septum, and ligament to sensitive spinal periosteum must be guarded against.[133-134]

THE ATYPICAL MOTOR UNIT

The occipito-atlanto and atlanto-axial motor units are atypical and may suffer additional complication from the presence of congenital and developmental variations. Anomalies may render any typical motor unit atypical as commonly occurs in the spinal-pelvic transitional segments. Acquired architectural remoulding of the motor unit may progress throughout life to the point where it has been fundamentally altered and could no longer be considered typical. Consideration of its morbid anatomy and pathomechanics is outside the scope of this paper.

The Occipito-atlanto-axial Atypical Vertebral Motor Units

Failure to appreciate the structural and dynamic intricacies of the upper cervical motor units, recently reviewed by von Torklus and Gehle in their text, *The Upper Cervical Spine*,[135] has contributed to complications from their manipulation.[136-143] The incidence of serious iatrogenic injury has been small and appears associated with vertebral artery abnormalities [144-147] and ligamentous-capsular laxity. Jirout [148] compares the unusual mobility of the atlas in the occipito-axial dynamic system to that of a meniscus; Paul and Moir [149] refer to "physiological" lateral displacements of the atlas during lateral inclination of the head while Cattel and Filtzer,[150] commenting on the hypermobility of the immature cervical spine, describe "pseudo-subluxation" of the axis ventrally on forward head-neck flexion in children. Houle [151] judiciously suggests the performance of a modified Adson's maneuver to assist in the assessment of

vertebro-basilar hemodynamics prior to the performance of any upper cervical manipulation.

The Vertebral Motor Unit With Congenital-developmental Variations

While the clinical and pathogenetic significance of anatomical variations of the osseous boundaries and soft tissue elements of the vertebral motor unit are still a matter of debate,[152-157] their presence often confuses diagnosis, leading to excessive therapeutic intervention. Practitioners of manipulative therapy must evaluate the unusual dynamics associated with vertebral motor unit variation and modify their standard mechanical interventions accordingly. For example, if one attempts to attain "normal" range of movement of a motor unit influenced by imperfect segmentation and rudimentary disc, the manipulation will predictably result in sprain. Any facet tropism requires alteration in customary patient positioning so the manipulative thrust will parallel the unusual mechanical axes, otherwise apophyseal joint trauma results. The subtleties of manipulative technique application to anomalous motor units have not been described in chiropractic or osteopathic literature and may never be exhaustively analyzed due to the myriad variations of spinal morphology.[2, 118, 158, 159]

Interest in the configuration of the intervertebral disc,[157, 160, 161] spinal canal [162-165] and neural arch [166-168] is generally shared by all clinicians concerned with spinal syndromes. Less attention has been focused on neurovascular [169-176] and ligamentous variations.[96] This is unfortunate because it is injury of these soft tissue variations, particularly those of the vertebro-basilar vessels, that has led to the most serious complications of spinal manipulation.[177-181]

The Pelvic Motor Unit

As Junghanns [2] claims, chiropractors [9, 182, 183] in particular emphasize pain caused in the position and stability of the sacroiliac joints and have applied the concept of the functional unit to the pelvic girdle, the sacroiliac joints and pubic symphysis forming the functional unit. Many investigations of sacroiliac movements [184-189] have been performed since von Luschka [190] first considered the sacroiliac articulations as true diarthrodial joints in 1854. These have discussed their shock absorption capacity in dampening the effect of lower limb locomotor activity,[8, 9] the physiologic ligamentous laxity induced through hormonal influences,[191-193] pelvic motor unit instability (hypermobility) after removing iliac bone for grafting,[194] sacroiliac conditions simulating low lumbar disc syndromes,[195] and sacroiliac ligament trauma caused by misguided, vigorous, repeated manipulations of a joint that is already fully mobile [196] or even hypermobile (as may be found in children, patients with generalized ligamentous laxity, and those engaged in activity placing very high functional demands on the sacroiliacs – ballet, acrobatic and modern dancers, gymnasts, golfers).

Illi's biomechanical concepts [8, 9] explaining the genesis and influence of blocked (fixated) and asymmetric sacroiliac articulation(s) on the spine merit further investigation. More research is required into the functional significance of Mitchell's observation [197] that spinal stability is favored by a more vertical sacral position; on the mechanical relevance of accessory sacroiliac articulations described by Trotter [198, 199] and Hadley,[118] and into the validity and hopefully preventive value of hypotheses based on male-female anatomical differences in hip joint placement.[200]

Limitations of the Motor Unit Concept – Areas for Additional Research

The muscles limiting and coordinating ranges of motion of individual and multiple motor units have been neglected, but their complexities make even a cursory description of their attachments too lengthy for consideration here. Additional information is required on the dynamic mechanical response of the whole body system to steady state and impulsive mechanical forces to determine the effect of this energy within the spine and on various neural receptors.[201] Breig's monograph, *Biomechanics of the Central Nervous System*,[48] reporting physiologic deformation of the cord and hind brain is an excellent beginning. The role of thoracic-abdominal pressures [202, 203] and trunk musculature in transmitting forces generated in loading the spine and the deforming effects of forces generated by muscle and gravity on the viscoelastic elements of the vertebral motor unit also require more investigation,[204-209] as does Johnston's work [210] with three-dimensional spinal configurations. A limitation of the vertebral motor unit concept is its emphasis on segmental analysis, and yet, Junghanns certainly never intended his model to be autonomous. In the introduction to his text he refers to the "organ system spine" as appearing "no longer, as in the past, as an isolated,

segmental, skeletal organ. Today the spine appears in the light of many mutual relationships with the total body; with its equilibrium it exerts influences and also receives forces all of which are interwoven with the far-reaching chain of motion. In addition, the spine is able to exercise considerable influence upon neighbouring structures as well as upon remote organs by its action upon nerves and blood vessels."

To fully exploit Junghanns' and other anatomico-mechanical concepts in clinical practice will require the continuation of interdisciplinary dialogue enhanced greatly by this conference.

SUMMARY

The usefulness of Junghanns' concept of the functional unit of the spine (vertebral motor unit) to those concerned with spinal manipulatory theory and practice is presented along with a review of its applied anatomy with special reference to the clinical significance of the relations and contents of the intervertebral foramen.

REFERENCES

1. Sub, C. H., West, H. G.: Biomechanics of the spine. *J. of the Canad. Chir. Ass'n, 12–22 (reprinted from the ACA J. of Chir.)*, November 1974.
2. Schmorl, G., Junghanns H.: *The Human Spine in Health and Disease*. 2nd Amer. Ed. (translated by E. F. Besemann) Grune & Stratton, New York, 1971.
3. Junghanns, H.: Der lumbosakralwinkel. *Dtsch. Zschr. Chir.*, 213–322, 1929.
4. Orofino, C., Sherman, M. S., Schechter, D.: Luschka's joint, a degenerative phenomenon. *J. Bone Joint Surg.* 42-A:853, 1960.
5. Compère, E. L., Tachdjian, M. O., Kernahan, W. T.: The luschka joints, their anatomy physiology and pathology. *Orthopaedics* 1:159–68, 1959.
6. Tondury, G.: La colonne cervicale, son developpement et ses modifications durant la vie. *Acta Orthop. Belg.* 25:602–26, 1959.
7. Nilsson, A. V.: The physical relationship between the heads of the ribs and the nervous and vascular content of the intervertebral foramina of the typical thoracic vertebrae. *Monograph*, Dept. of Anatomy, Los Angeles College of Chiropractic. April 1937.
8. Illi, F. W.: *The Vertebral Column, Life-line of the Body*. National College of Chiropractic. (Chicago) 12–19, 1951.
9. Illi, F. W.: The phylogenesis and clinical import of the sacroiliac mechanism. *J. of the Canad. Chir. Ass'n*, 11–14, August–September 1965.
10. Sandoz, R.: Newer trends in the pathogenesis of spinal disorders, a tentative classification of the functional disorders of the intervetebral motor unit. *Annals of the Swiss Chiropractors' Ass'n*, 5:93–180, 1971.
11. Gillet, H., Liekens M.: *Belgian Chiropractic Research Notes*. (Published by the authors) 8th Ed. Belgium, 1970.
12. Inman, O. B.: Basic chiropractic procedural manual. (Printed by the *Amer. Chir. Ass'n)* 1973.
13. Howe, J. W.: The chiropractic concept of subluxations and its roentgenological manifestations. *J. Clin. Chir.* 64–70, September 1973.
14. Drum, D. C.: The nature of the problem, a functional concept of vertebral subluxations. *New Eng. J. of Chir.* 29–31, May 1973.
15. Rich, E. A.: Atlas of *Clinical Roentgenology*. Rae Publishing Co. (Indianapolis) 126, 1965.
16. Lewit, K.: The problem of manual and reflex neurotherapy. *Wien. Med. Wschr.* 23:473–77 (translated by M. Gauchat, Canad. Chir. College, Toronto), 1971.
17. Jirout, J.: Correlations of the functional disturbances in the cervical spine as seen roentgenographically in frontal and sagittal projections. *Svy. Univ. Carocinae Suppl.* 21:128–29, 1965.
18. Farfan, H. F.: *Mechanical Disorders of the Low Back*. Lea and Febiger (Philadelphia) 1973.
19. Maigne, R.: *Orthopedic Medicine, A New Approach to Vertebral Manipulations*. (Translated and edited by W. T. Liberson. Charles C Thomas (Springfield), 1972.
20. Droz, J. M.: Frequency and causes of recurrence of sciatica. *Annals of the Swiss Chiropractors' Ass'n.* 4:69–77, 1969.
21. Haldeman, S., Drum, D. C.: The compression subluxation. *J. of Clin. Chir. Arch.* Ed. 1, 1971.
22. Veleanu, C., Grun, U., Diaconescu, M., Cocota, E.: Structural peculiarities of the thoracic spine, their functional significance. *Acta Anat.* 82 : 97–107, 1972.
23. Hadley, L. A.: Subluxation of the apophyseal articulations with bony impingement as a cause of back pain. *Am. J. Roentgenol. & Rad. Therapy.* 33:209–13, 1935.
24. Stoddard, A.: *Manual of Osteopathic Practice*. Harper & Row, (New York) 114–26, 1969.
25. Harris, R. H.: *Subluxation and Distortion of Joints Without Fracture*. San Lucas Press, (Los Angeles) 35–44, 1961.
26. Cyriax, J.: *Text-book of Orthopaedic Medicine*, Vol. 2, "Treatment by Manipulation and Massage." 7th Ed. Cassell (London) 53–69, 1965.
27. Gillet, H., Liekens, M.: A further study of spinal fixations. *Annals of the Swiss Chiropractors' Ass'n.* 4:41–6, 1969.
28. Mennell, J.: *Back pain, Diagnosis and Treatment Using Manipulative Techniques*. Little, Brown and Co. (Boston-Toronto) 110, 1960.
29. Paris, S. V.: *New Zealand Med. J.*, 62:320, 1963.
30. Lacroix, G.: *Royal Commission on Chiropractic and Osteopathy*. Queen's Printer, (Quebec City) 73, 1965.
31. Weiant, C. W., Goldschmidt, S.: *Medicine and Chiropractic*. Published by the authors (New York) 4th Ed. 1966.
32. Parke, W. W., Schiff, D. C.: The applied anatomy of the intervertebral disc. *Orth. Clinics of North America*. 2: No 2, 320, July 1971.
33. DePalma, A. F., Rothman, R. H.: *The Intervertebral Disc*. W. B. Saunders Co. (Philadelphia) 59, 1970.
34. Maigne, R.: Ibid, p. 5.

35. Jackson, R.: The cervical syndrome. *Charles C Thomas* (Springfield) 3rd Ed. 1966.
36. Sutherland, D. C.: Medicine approves chiropractic manipulation, injudicious use by untrained physicians presents "grave danger". *J. of the Canad. Chir. Ass'n.* 3–5, October–November 1966.
37. Fisher, A. G. T.: Manipulation in lumbar intervertebral disc lesions. *The Practitioner* 187:327, September 1961.
38. Darbert, O., Freeman, D. G., Weis, A. J.: Spinal meningeal hematoma, warfarin therapy and chiropractic adjustment. *JAMA* 214: No. 11,2058, December 1970.
39. Froning, E., Frohman, B.: Motion of the lumbosacral spine after laminectomy and spine fusion. *J. Bone Joint Surg.* 50–A: No. 5, July 1968.
40. Wolff, H. D.: Remarks on the present situation and further development of manual medicine with special regard to chirotherapy. (Reduction of presentation to the German Society for Manual Medicine.) *Manuelle Medizin*, February 1972.
41. Gutzeit, K.: Wirbelsaule und innere krankheiten. *Munchener Med. Wschr.* 95:47, 1953.
42. Wendt: *Dtsch. Gesundheitswes*, 5, 14, 1951.
43. Duus, P., Kahlau, G.: Welche pathogenetische bedeutung hat der bandscheibenvorfall in bereich der lendenwirbelsaule? *Bruns' Beitrage zur Klin. Chir.* 180:1, 1950.
44. Reischauer, F.: Untersuchungen uber d. bandscheibenvorfall. Thieme, Stuttgart, 1949.
45. Pillokat: Die halswirbelsaule als krankheitsfaktor. *Dtsch. Gesundheitswes.* 6:909, 1952.
46. Eisenberg, A. M.: A basis for chiropractic. *J. of Clin. Chir. Arch.* Ed. 3:60–5, 1973.
47. Crelin, E. S.: A scientific test of the chiropractic theory. *Amer. Scient.* 61:574–80, 1973.
48. Breig, A.: *Biomechanics of the Central Nervous System, Some Basic Normal and Pathologic Phenomena Concerning Spine, Discs and Cord.* Almqvist & Wiksell, Stockholm, 1960.
49. Sunderland, S.: Meningeal-neural relations in the intervertebral foramen. *J. Neurosurg.* 40:756–63, June 1974.
50. Inman, V. T., Saunders, J. B.: The clinicoanatomical aspects of the lumbosacral region. *Radiol.* 38:669–78, 1942.
51. Hollinshead, W. H.: *Anatomy for Surgeons:* Vol. 3, the back and limbs. 2nd Ed. Harper & Row (New York) 176, 1969.
52. Frykholm, R.: *The Clinical Picture in Cervical Pain.* Pergamon Press (Editors Hirsh & Zotterman) 5–16, 1972.
53. Rosomoff, H. L., Rossman, F.: Treatment of cervical spondylosis by anterior cervical diskectomy and fusion. *Arch. Neurol.* 14:392, 1966.
54. Friedenberg, Z. B., Edeiken, J., Spencer, N., Tolentino, S. C.: Degenerative changes in the cervical spine. *J. Bone Joint Surg.* 41–A:61, 1959.
55. Key A., Retzius G.: *Studien in der Anatomie des Nervensystems.* Folio 166:1, P. A. Norstedt & Soner, Stockholm, 1875.
56. Frykholm, R.: Cervical nerve root compression resulting from disc degeneration and root-sleeve fibrosis. *Acta. Chir. Scand.*, Suppl. 160, 1951.
57. Oppenheimer, A.: Narrowing of the intervertebral foramina as a cause of pseudo-rheumatic pain. *Ann. of Surg.* 106:No. 3, September 1937.
58. Wilkinson, M.: Cervical spondylosis. *The Practitioner.* 204:537–45, April 1970.
59. Holt, S., Yates, P. O.: Cervical spondylosis and nerve root lesions. *J. Bone Joint Surg.* 48–B:407–23, 1966.
60. Kovacs, A.: Subluxation and deformation of the cervical apophyseal joints. *Acta Radiolog.* 43, 1955.
61. Batson, O. V.: The function of the vertebral veins and their role in the spread of metastases. *Ann. Surg.* 112: p. 138. See also Batson, O. V.: *Am. J. Roentg.* 78, 1957, and Warwick, R., Williams, P. L. *Gray's Anatomy.* 35th Ed. Longman (Edinburgh) Angiology, 1973.
62. Brain, L.: Some aspects of the neurology of the cervical spine. *J. Fac. Radiol.* 8:74–91, 1956.
63. Jones, R. T.: cited by Kleynhans, A. M.: Vascular changes occurring in the cervical musculo-skeletal system. *Bull. of the Eur. Chir. Union.* October 1970.
64. Macnab, I.: The mechanism of spondylogenic pain – cervical pain – proceedings of the international symposium held in Wenner-Gren Center, Stockholm, Pergamon Press, 89–95, 1971.
65. Olsson, Y.: Studies on vascular permeability in peripheral nerves, distribution of circulating fluorescent serum albumin in normal, crushed, and sectioned rat sciatic nerve. *Acta Neuropath.* (Berl.) 7:1–15, 1966. See also Neuropathological aspects on root affections in the cervical region (Olsson, Y., Sourander, P., Kristensson, K.).
66. Olsson, Y.: Studies on vascular permeability in peripheral nerves, distribution of intravenously injected protein tracers in the peripheral nervous system of various species. *Acta Neuropath.* (Berl.) 17:114–26, 1971.
67. Bergmann, L., Alexander, L.: Vascular supply of the spinal ganglia. *Archives of Neurol. and Psychiatry*, 46:761–82, November 1941.
68. Hasue, M., Ito, R., Suzuk, N.: Circulatory dynamics of the epidural space in cervical and lumbar disc lesions. *Clin. Orth. and Related Research.* 89:129–138, November–December 1972; also Fujiwara T.: An evaluation of the "vascular theory" of cervico-brachial syndrome in view of the vertebral arteriography and phlebography. *Central Jap. J. Orthop. Trauma. Surg.* 6:1, 1963, in Japanese – cited.
69. Mennell, J. B.: *The Science and Art of Joint Manipulation.* Vol. 2, Blakiston Co., Philadelphia, 1952.
70. Farfan, H. F.: *Mechanical Disorders of the Low Back.* Lea and Febiger (Philadelphia), 1973.
71. Nashold, B. S., Hrubec, Z.: *Lumbar Disc Disease, A Twenty-year Clinical Follow-up Study.* The C. V. Mosby Co., St. Louis, 1971.
72. Jayson, M. I. V., Carol, M. P., Barks, J. S.: Intervertebral discs, nuclear morphology and bursting pressures. *Ann. Rheum. Dis.* 32:308, 1973.
73. Hakelius, A., Hindmarsh, J.: The comparative reliability of preoperative diagnostic methods in lumbar disc surgery. *Acta Orthop. Scandinav.* 43:234–38, 1972.
74. Markolf, K. L., Morris, J. M.: The structural components of the intervertebral disc. *J. Bone Joint Surg.* 56A–A: No.4, 675–87, June 1974.
75. Stauffer, R.: The lumbar disc syndrome and its operative treatment. *Postgrad. Med.* 87–93, February 1971.
76. Drum, D. C.: Conservative management of lumbar disc degenerations. *J. of Clin. Chir. Archive Ed. 2*, 96–113, 1972.

77. DePalma, A. F., Rothman, R. H.: *The Intervertebral Disc.* W. B. Saunders Co., Philadelphia, 1970.
78. Scott-Charlton, W., Roebuck, D. J.: The significance of posterior primary divisions of spinal nerves in pain syndromes. *Med. J. Aust.* 2:945–48, 1972.
79. Shealy, C. N.: The role of the spinal facets in back and sciatic pain. *Headache.* 101–4, July 1974.
80. Wolff, H. D.: Remarks on the present situation and further development of manual medicine with special regard to chirotherapy. *Manuelle Medizin.* February 1972.
81. Armstrong, J. R.: Lumbar disc lesions. E. & S. Livingstone Ltd Edinburgh, 1965. See also Cyriax J.: Manipulation. *Clin. Med.* 73:37, August 1966.
82. De Seze: Maladies des os et des articulations. Ed. Editions medicales Flammarion. *Mise a jour,* 1970.
83. Cover A. B., Curwen I. H. M.: Low back pain treated by manipulation, a controlled series. *Brit. Med. J.* 705–7, March 1955.
84. McRae, D. L.: Asymptomatic intervertebral disc protrusions. *Acta Radiol.* 46, 1956.
85. Haley, J. C., Perry, J. H.: Protrusions of intervertebral discs, study of their distribution, characteristics and effects on the nervous system. *Am. J. Surg.* 80:394, 1950.
86. Bick, E. M.: Common degenerative diseases of the aging spine. *Geriatrics.* 19: No. 1, January 1964.
87. Macnab, I.: *A.A.O.S. Symposium on the Spine.* C. V. Mosby Co. (St. Louis), pathogenesis of symptoms in discogenic low back pain, 101, 1969.
88. Kunert, W.: *The Vertebral Column, Autonomic Nervous System and Internal Organs.* Enke, Stuttgart, 1963.
89. Naffziger, H. C., Inman, V., Saunders, J. B.: Lesions of the intervertebral disc and ligamenta flava. *Surg. Gynec. & Obst.* 66:288, 1938.
90. Dockerty, M. B., Love, J. G.: Thickening and fibrosis (so-called hypertrophy of the ligamentum flavum) a pathologic study of fifty cases. *Proc. Staff Meet., Mayo Clinic.* 15:161, 1940.
91. Stoltmann, H. F., Blackwood, W.: The role of the ligamenta flava in the pathogenesis of myelopathy in cervical spondylosis. *Brain* 87:45, 1964.
92. Nagashima, C.: Cervical myelopathy due to ossification of the posterior longitudinal ligament. *J. Neurosurg.* 37:653–60, December 1972.
93. Ornilla, E., Ansell, B. M., Swannell, A. J.: Cervical spine involvement in patients with chronic arthritis undergoing orthopaedic surgery. *Ann. Rheum. Dis.* 31: 364–68, 1972.
94. Jeffcoate, T. N. A.: Pelvic pain – part one. *Brit. Med. J.* 3431–3435, 1969.
95. Bick, E. M.: Vertebral osteophytosis, pathologic basis for its roentgenology. *Amer. J. Roentgenol.* 73:979, 1955.
96. Golub, B. S., Silverman, B.: Transforaminal ligaments of the lumbar spine. *J. Bone Joint Surg.* 51-A: No. 5, July 1969.
97. Newman, P. H.: Surgical treatment for derangement of the lumbar spine. *J. Bone Joint Surg.* 55-B: No. 1, 7–19, 1973.
98. Jirout, J.: The dynamic dependence of the lower cervical vertebrae on the atlanto-occipital joints. *Neuroradiol.* 7:249–52, 1974.
99. White, A. A., Hirsch, C.: The significance of the vertebral posterior elements in the mechanics of the thoracic spine. *Clin. Orthop.* 81:No.81, 2–14, November–December 1971.
100. Pedersen, H. E., Blunck, G. F. G., Gardner, E.: Anatomy of lumbosacral posterior rami and meningeal branches of spinal nerves (sinu-vertebral nerves) *J. Bone Joint Surg.* 38-A:377–91, 1956.
101. Wyke, B.: The neurological basis for thoracic spinal pain. *Rheum. & Phys. Med.* 10:No.7, 356–66, 1967.
102. Jung, A., Brungschwig, A.: Recherches histologiques sur l'innervation des articulations des corps vertebraux. *Presse Med.* 40:316–17, February 1932.
103. Parke, W. W., Schiff, D. C. M.: The applied anatomy of the intervertebral disc. *Orthop. Clinics of North Amer.* 2:No.2, W. B. Saunders Co. (Philadelphia), July 1971.
104. Roofe, P. G.: Innervation of annulus fibrosis. *Arch. Path.* 27:201–11, 1939.
105. Roofe, P. G.: Innervation of annulus fibrosis and posterior longitudinal ligament. *Arch. Neuro. Psych.* 44:100–3, 1940.
106. Wiberg, G.: Back pain in relation to nerve supply of intervertebral disc. *Acta Orthop. Scand.* 19:211–21, 1949.
107. Smyth, M. J., Wright, V.: Sciatica and the intervertebral disc, an experimental study. *J. Bone Joint Surg.* 40-A: 1401, 1958. See also Shutkin, N. M.: Syndrome of the degenerated intervertebral disc. *Am. J. Surg.* 84:162, 1952; Kaplan, E. B.: Anatomy of recurrent meningeal branch of spinal nerves. *Bull. Hosp. Joint Dis.* 8:108, April 1947; Lazorthes, G., Poulhes.: Etudes sur les nerfs sinu vertebraux lombaires. *C. R. Assoc. Anatomistes,* 317, 1947, and Haldeman, S.: Spinal and paraspinal receptors. *ACA J. of Chir.* 16: May 1972.
108. Jackson, H. C., Winkelmann, R. K., Bickel, W. H.: Nerve endings in the human lumbar spinal column and related structures. *J. Bone Joint Surg.* 48-A:1272–81, 1966.
109. Ferlic, D. C.: The nerve supply of the cervical intervertebral disc in man. *Bull. Johns Hopkins Hosp.* 113:347–51, 1963.
110. Edgar, M. A., Nundy, S.: Innervation of the spinal dura mater. *J. Neurol. Neurosurg. Psych.* 29:530–34, 1966.
111. Bobechko, W. P., Hirsch, C.: Auto-immunity reaction of nucleus pulposus. *J. Bone Joint Surg.* 47-B:574–80, August 1965.
112. Lazorthes. G.: Le rachis cervical, donnees anatomiques et physiologiques recentes. Journees d'Etude et de Perfectionnement des manipulations vertebrales. In *Ann. Med. Phys.,* 9, 3:193, 1966, also Lazorthes, G., Despeyrou, L., Juskiewenski, S.: Les branches posterieures des nerfs rachidiens et la medecine physique. *Ann. Med. Phys.,* 8, 1:67, 1965.
113. Warwick, R., Williams, P. L.: *Gray's anatomy.* 35th Ed. Longman (Edinburgh), 1973.
113a. Wright, J.: Mechanics in relation to derangement of the facet joints of the spine. *Arch. Phys. Ther.* 201–6, April 1944.
114. Kellgren, J. H., Lawrence, J. S.: Osteoarthrosis and disc degeneration in an urban population. *Ann. Rheumat. Dis.* (Lond.), 17:388–97, 1958.
115. Zuckschwerdt, Emminger, Biedermann, Zettel: Wirbelgelenk und Bandscheibe. Ed. *Hippokrates Verlag. Stuttgart,* 1960.

116. Schmincke, A., Santo, E.: Zur normalen pathologischen anatomie der jalswirbelsaule. *Zbl. Allg. Path. Anat.* 55:369, 1932.
117. Kraft, G. L., Levinthal, D. H.: Facet synovial impingement, a new concept in the etiology of lumbar vertebral derangement. *Surg., Gynec. & Obst.* 93:439, 1951.
118. Hadley, L. A.: *Anatomico-roentgenographic Studies of the Spine.* (Springfield), Charles C. Thomas. The posterior spinal articulations, 181, 1973.
119. Badgley, C. E.: The articular facets in relation to low back pain and sciatic radiation. *J. Bone Joint Surg.* 23:No.2, 481–95, April 1941.
120. Hirsch, C., Inglemark, B. E., Miller, M.: The anatomical basis for low back pain. *Acta Orthop. Scand.* 33: 1963.
121. Lazorthes, G.: Le rachis cervical, donnees anatomiques et physiologiques recentes. *Annales de Med. Phys.* 9:196, 1966.
122. Shealy, N. C.: The role of the spinal facets in back and sciatic pain. *Headache* 101–4, July 1974.
123. Rees, W. E. S.: Multiple bilateral subcutaneous rhizolysis of segmental nerves in the treatment of the intervertebral disc syndrome. *Ann. Gen. Prac.* 26:126–7, 1971.
124. Patti, V.: New conceptions in the pathogenesis of sciatic pain. *Lancet.* 2:53–60, 1927.
125. Sullivan, I., Farfan, H., Kahn, D.: Intervertebral joint instability, pathologic changes with intervertebral joint rotational instability in the rabbit, *Canad. J. Surg.* 14, January 1971. See also Farfan, H. F.: Effects of torsion on intervertebral joints. *Canad. J. Surg.* 12:336, 1969.
126. Nachemson, A.: The influence of spinal movements on the lumbar intradiscal pressure and on the tensile stresses in the annulus fibrosis. *Acta Orthop. Scand.* 33:183, 1963.
127. Drum, D. C.: The posterior gravity line syndrome, recurrent low back pain of postural origin. *J. of the CCA-4th quarter,* 1968.
128. Jung, A., Brunschwig, A.: L'innervation des articulations des corps vertebraux. *Press Med.* 40:316–17, 1932.
129. Vele, F.: The origin of proprioceptive information in the zygopophyseal joints and the processing of these afferences. Manuelle Medizin und ihre wissenschaftlechen Crundlagen. Wolff-Trier, Editor. Heidelberg, 1970.
130. Fisk, J. W.: Manipulation in general practice. *N.Z. Med. J.* 74:172–75, September 1971.
131. Farfan, H. F., Sullivan, J. D.: The relation of facet orientation to intervertebral disc failure. *Canad. J. Surg.* 10:179–85, 1967.
132. Sandoz, R.: Radiographic semiology of posterior vertebral arthroses. *Annals of the Swiss Chiropractors' Ass'n.* 4:105–54, 1969.
133. Scott-Charlton, W., Roebuck, D. J.: The significance of posterior primary divisions of spinal nerves in pain syndromes. *Med. J. Aust.* 2:945–48, October 1972.
134. Vvid, N.: Sylnasolbehandlung af ligamentosis interspinalis et supraspinalis Ugeskr. *Laeg.* 121:169, 1959.
135. von Torklus, D., Gehle, W.: *The Upper Cervical Spine, Regional Anatomy, Pathology and Traumatology, a Systematic Radiological Atlas and Textbook.* Butterworths (London) 21–84, 1972.
136. Kanshepolsky, J., Danielson, H., Flynn, R. E.: Vertebral artery insufficiency and cerebellar infarct due to manipulation of the neck. *Bull. of L.A. Neurol. Soc. 37,* April 1972.
137. Mehalic, T., Farhat, S. M.: Vertebral artery injury from chiropractic manipulation of the neck. *Surg. Neurol.* 2, March 1974.
138. Fields, W. S., Ratinov, G., Weibel, J., Campos, R. J.: Survival following basilar artery occlusion. *Arch. Neurol.* 15, 1966.
139. Pribek, R. A.: Brain stem vascular accident following neck manipulation. *Wisconsin Med. J.* 62:141–43, 1963.
140. Livingston, M.: Spinal manipulation causing injury, a three year study. *Clin. Orth. & Related Research.* 81, 1971.
141. Ford, F. R., Clark, D.: Thrombosis of basilar artery with softenings in cerebellum and brain stem due to manipulation of neck. *Bull. Johns Hopkins Hosp.* 98:37–42, January 1956.
142. Schwarz, G. A., Geiger, J. K., Spana, A. V.: Posterior inferior cerebellar artery syndrome of Wallenberg after chiropractic manipulation. *AMA Arch. Int. Med.* 97:352–54, 1956.
143. Schneider, R. C., Schemm, G. W.: Vertebral artery insufficiency in acute and chronic spinal trauma. *J. Neurosurg.* 18:348–60, 1961.
144. West, H. G.: Vertebral artery considerations in cervical trauma. *The ACA J. of Chir.,* December 1968.
145. Sheehan, S., Bauer, R. B., Meyer, J. S.: Vertebral artery compression in cervical spondylosis, arteriographic demonstration during life of vertebral artery insufficiency due to rotation and extension of the neck. *Neurology* 10:968–86, 1960.
146. Lewis, R. C., Coburn, D. F.: The vertebral artery, its role in upper cervical and head pain. *Missouri Med.* 53:1059–63, December 1956.
147. Nagashima, C., Iwama, K., Sakata, E., Miki, Y.: Effects of temporary occlusion of a vertebral artery on the human vestibular system. *J. Neurosurg.* 3388–94, October 1970.
148. Jirout, J.: Changes in the atlas-axis relations on lateral flexion of the head and neck. *Neurorad.* 6:215–18, 1973.
149. Paul, L. W., Moir, W. W.: Non-pathologic variations in relationship of the upper cervical vertebrae. *Amer. J. Roentgenol.* 62:519, 1949.
150. Cattel, H. S., Filtzer, D. L.: Pseudosubluxation and other normal variations in the cervical spine in children. *J. Bone Joint Surg.* 47-A:1295, 1965.
151. Houle, J. O. E.: Assessing hemodynamics of the vertebrobasilar complex through angiothlipsis. *J. of the Canad. Chir. Ass'n.* June–July 1972.
152. Janse, J.: The mechanical pathologies involved in the low back and sciatic neuralgia syndrome. *J. National College of Chiropractic.* 25: 1953.
153. Stinchfield, F. E., Sinton, W. A.: Clinical significance of the transitional lumbosacral vertebra. *JAMA* 157:1107–9, 1955.
154. La Rocca, H., Macnab, I.: Value of pre-employment radiographic assessment of the lumbar spine. *Canad. Med. Ass'n. J.* 101, October 1969.
155. Farfan, H. F., Sullivan, J. D.: The relation of facet orien-

597-036 O - 76 - 6

tation to intervertebral disc failure. *Canad. J. Surg.* 10:179–185, 1967.

156. Kovacs, A.: Subluxation and deformation of the cervical apophyseal joints. *Acta Radiol.* 43, 1955.
157. Farfan, H. F., Huberdeau, R. M., Dubow, H. I.: Lumbar intervertebral disc degeneration, the influence of geometrical features on the pattern of disc degeneration, a post mortem study. *J. Bone Joint Surg.* 54–A:No. 3, April 1972.
158. Epstein, B. S.: *The spine: radiographic text and atlas.* Lea & Febiger (Phila.) 3rd Ed., 1969.
159. Bornstein, P. E., Peterson, R. R.: Numerical variation of the presacral vertebral column in three population groups in North America. *Am. J. Phys. Anthrop.* 25:139–46, 1966.
160. Ehrenhaft, J. L.: Development of the vertebral column as related to certain congenital and pathological changes. *Surg., Gynec. & Obst.* 76:282, 1943.
161. Brav, E. A., Molter, H. A., Newcomb, W. J.: The lumbosacral articulation, a roentgenologic and clinical study with special reference to narrow disc and lower lumbar displacement. *Surg., Gynec. & Obst.* 87:549, 1948.
162. Murone, I.: The importance of the sagittal diameters of the cervical spinal canal in relation to spondylosis and myelopathy. *J. Bone Joint Surg.* 56–B:No. 1, February 1971.
163. Mayfield, F. H.: Cervical spondylosis, observations based on surgical treatment of 400 patients. *Postgrad. Med.* 345–57, October 1965.
164. Jones, R. A. C., Thompson, J. L. G.: The narrow lumbar canal, a clinical and radiological review. *J. Bone Joint Surg.* 50–B:595–605, 1968.
165. Ehni, G., Clark, K., Wilson, C. B.: Significance of the small lumbar spinal canal, cauda equina compression syndromes due to spondylosis (parts 1–4). *J. Neurosurg.* 31:490–512, 1969.
166. Beeler, W.: Further evidence of the acquired nature of spondylolysis and spondylolisthesis. *Amer. J. Roentgenol.* 108:796, 1970.
167. Hollinshead, H. W.: *Anatomy for surgeons, spondylolysis* and *spondylolisthesis,* Harper & Row (New York) 2nd Ed. 3:137–42, 1969.
168. Wiltse, L. L.: The etiology of spondylolisthesis. *J. Bone Joint Surg.* 44–A, 1962.
169. Horwitz, M. T.: The anatomy of (A) the lumbosacral nerve plexus—its relation to variation of vertebral segmentation and (B) the posterior sacral nerve plexus. *Anat. Rec.* 74:91, 1939.
170. Batson, O. V.: The function of the vertebral veins and their role in the spread of metastases. *Ann. Surg.* 112, 1940.
171. Norgore, M.: Clinical anatomy of the vertebral veins. *Surgery* 17, 1945.
172. Hassler, O.: Blood supply to the human spinal cord, a microangiographic study. *Arch. Neurol.* 15:302, 1966.
173. Turnbull, I. M., Breig, A., Hassler, O.: Blood supply of the cervical spinal cord in man, a microangiographic cadaver study. *J. Neurosurg.* 24:951, 1966.
174. Hutchinson, E. C., Yates, P. O.: Cervical portion of the vertebral artery, clinico-pathological study. *Brain* 79:319–331, June 1956.
175. Stopford, J. S. B.: Arteries of pons and medulla oblongata. *J. Anat.* 50:131–164, 1960.
176. Lindenberg, R.: Compression of brain arteries as pathogenetic factor for tissue necroses and their areas for predilection. *J. Neuropath. & Exper. Neurol.* 14:223, 1955.
177. Smith, R. A., Estridge, M. N.: Neurologic complications of head and neck manipulations. *JAMA* 182:528–31, November 1962.
178. Green, D., Joynt, R. J.: Vascular accidents to brain stem associated with neck manipulation. *JAMA* 170:522–524, May 1959.
179. Ford, F. R.: Syncope, vertigo, and disturbances of vision resulting from intermittent obstruction of vertebral arteries due to defect in odontoid process and excessive mobility of second cervical vertebra. *Bull. Johns Hopkins Hosp.* 91:168–173, September 1952.
180. Grossiord, A.: Les accidents neurologiques des manipulations cervicales. *Annales de Medecine Physique.* 9:283–298, 1966.
181. Lyness, S. S., Wagman, A. D.: Neurological deficit following cervical manipulation. *Surg. Neurol.* 2:121–24, March 1974.
182. Weiant, C. W., Janse, J.: The work of Dr. F. W. H. Illi, an analysis and preliminary evaluation. *The J. of the National Chir. Ass'n.,* December 1955.
183. Parker, B. L.: Biomechanics of sacroiliac union in walking. *The J. of Clin. Chir. Arch.* Ed. 3:40–8, Spring 1973.
184. Frigerio, N. A., Stowe, R. S., Howe, J. W.: Movement of the sacroiliac joint. *Clin. Orthol.* 100:370–77, 1974.
185. Solonen, K. A.: The sacroiliac joint in the light of anatomical, roentgenological, and clinical studies. *Acta orthop. Scadinav.* Supp. 27:9–126, May 1957.
186. Weisl, H.: The articular surfaces of the sacroiliac joint and their relation to the movements of the sacrum. *Acta Anat.* 22:1–14, 1954.
187. Weisl, H.: The movements of the sacroiliac joint. *Acta Anat.* 23:80–91, 1955.
188. Duckworth, J. W.: The anatomy and movements of the sacroiliac joints. *Manuelle Medizin und ihre wissenschaftlichen Grundlagen,* 1970.
189. Colachis, S., Worden, R., Bechtol, C., Strohm, B.: Movement of the sacroiliac joint in the adult male, a preliminary report. *Arch. of Phys. Med. & Rehab.,* September 1963.
190. von Luschka, H.: Die Kreuzdarmbeinfuge und die schambienfuge des menschen. *Virchows Arch. Path. Anat.* 7:299–316, 1854.
191. Abramson, D., Roberts, S. M., Wilson, P. D.: Relaxation of the pelvic joints in pregnancy. *Surg. Gynec. & Obst.* 58:596–613, March 1934.
192. Thorp, D. J., Fray, W. E.: The pelvic joints during pregnancy and labor. *J.A.M.A.* 111:1162–66, September 1938.
193. Borell, U., Fernstrom, I.: The movements at the sacroiliac joints and their importance to changes in the pelvic dimensions during parturition. *Acta Obst. & Gynec. Scadinav.* 36:42–57, January 1957.
194. Coventry, M., Tapper, E.: Pelvic instability, a consequence of removing iliac bone for grafting. *J. Bone Joint Surg.* 54–A:83–101, 1972.
195. Norman, G. F., May, A.: Sacroiliac conditions simulating

intervertebral disc syndrome. *W. J. of Surg. Gynec. & Obst.* 461–62, August 1956.
196. Stoddard, A.: *Manual of Osteopathic Practice.* Harper & Row (New York) 184–190, 1969.
197. Mitchell, G. A. G.: The lumbosacral junction. *J. Bone Joint Surg.* 16:233, 1934.
198. Trotter, M.: Accessory sacroliac articulations in East African skeletons. *Am. J. Phys. Anthrop.* 22:137–42, 1964.
199. Trotter, M.: Accessory sacroiliac articulations. *Am. J. Phys. Anthrop.* 22:247–55, 1937.
200. Tichauer, E. R., Miller, M., Nathan, I. M.: Lordosimetry, a new technique for the measurement of postural response to materials handling. *Amer. Indust. Hyg. J.* 1–12, January 1973.
201. Coermann, R. R., Ziengenruecker, G. H., Wittwer, A. L., von Gierke, H. E.: The passive dynamic mechanical properties of the human thorax-abdomen system and the whole body system. *Aerospace Med.* 31:No.6, 443–47, June 1960.
202. Bartelink, D. L.: The role of abdominal pressure in relieving the pressure on the lumbar intervertebral discs. *J. Bone Joint Surg.* (Brit.) 39–B:718, 1957.
203. Nachemson, A., Morris, J. M.: In vivo measurements of interdiscal pressure: discometry, a method for the determination of pressure in the lower lumbar joints. *J. Bone Joint Surg.* 46:1077–92, 1964.
204. Morris, J. M., Lucas, D. B., Bresler, B.: Role of the trunk in stability of the spine. *J. Bone Joint Surg.* 43:327–51, 1961.
205. Olsen, G. A., Allan, J. H.: The lateral stability of the spine. *Clin. Orthop.* 65:143–56, 1969.
206. Morris, J. M.: Biomechanics of the spine. *Arch. Surg.* 107, September 1973.
207. Hildebrandt, R. W.: The clinical basis of chiropractic postural roentgenology. *J. Clin. Chir. Arch.* Ed.3, 1973.
208. Michele, A. A.: *Iliopsoas, Development of Anomalies in Man.* Charles C Thomas, Springfield, 1962.
209. Northup, G. W.: Osteopathic lesions. *J.A.O.A.*, 71:854, June 1972.
210. Johnston, L. C.: The paradox of the functional spine. *J. Canad. Chir. Ass'n.* June–July 1966.

Spinal Geometry: Normal and Abnormal

EDMUND B. WEIS, JR.
Division of Orthopaedics
Ohio State University

INTRODUCTION

This report is an attempt to bring together diverse research studies and clinical experiences into a coherent consideration of the role of basic physical and physiologic factors in spinal geometry. It attempts to set up categories based on qualitative estimations of basic factors and then fits clinical entities into the categories. The fit is procrustean at best with little assurance that a vital part has not been cut off.

The analysis of spinal geometry must include consideration of pertinent anatomic, physiologic, and mechanical variables. Among the mechanical variables one might include at least a qualitative separation as to the size of deviations as well as to the time history. These separations are important before proceeding to quantification of factors. As a minimum consideration of anatomic factors one might include the presence or absence of abnormalities of the bone structure of the spine. Physiologic variables such as age and size are clearly important in treatment but may be deferred for consideration until mechanical and anatomical factors can be at least organized.

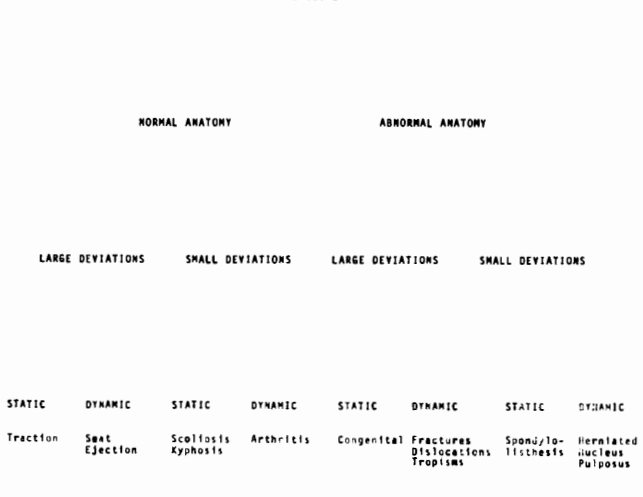


Fig. 1. Categorization of spinal geometry.

The considerations lead to the designation of the categories in figure 1. The remainder of this report will be devoted to presenting the practice and research in each of these categories.

1. TRACTION

Traction on the spine is a familiar clinical experience. It is probably the only static large deviation of spinal geometry with normal anatomy. The most spectacular clinical situation is the Halo-hoop apparatus [1] shown in figure 2. My own experience with this device is presented in figures 3, 4, and 5. This is a case of idiopathic scoliosis, long neglected in which an attempt to straighten the curve was made. The attempt failed inasmuch as an abducens nerve palsy supervened. Note the decrease in the curve shown in figures 4 and 5 and the dramatic change in intervertebral disc height shown in figure 3. There are many less dramatic demonstrations of traction in the spine but the widening of the cervical discs in this case and the abducens palsy imply many things about the future of such treatment.

2. SPINAL MODELS

High speed transportation has stimulated much study of the spine. The era of escape and restraint system research produced models by Weis,[2,3] Kaleps,[4] Orne [5] and King.[6] These models represent the large loads and deviations incurred in crash situations. Only Orne's model accounts directly for the spinal curvature as shown in figure 6 but gives

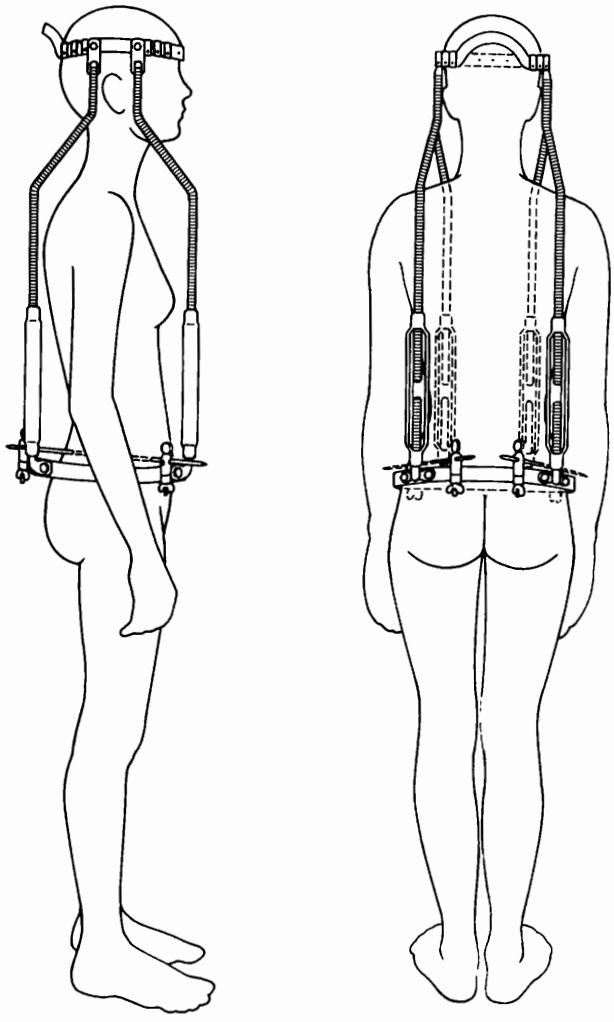


Fig. 2. Halo-hoop traction apparatus (redrawn from DeWald).

no consideration to the posterior elements. These models predict locations of high risks of injury in the spine as well as critical modes of exposure.

There are other studies of the physiologic aspects of crash situations such as by Weis.[7,8,9] These studies give tolerance limits for crash exposure and the tolerance limiting factors are closely related to the spine.

3. SCOLIOSIS

Studies of the spinal geometry in scoliosis are plentiful in the literature. Two types of these studies are pertinent. One is the quantification of curvature and rotation as for example by Weis.[10] This study derives mathematical representation of the curvature and rotation of the vertebra from X-rays of the spine. The data is obtained by direct measurement (figure 7), corrected for X-ray distortion, converted to unit vector representations for each vertebra and subjected to computer analysis for curvature and rotation of the unit vector (figure 8). This study points to the location of the apex of the curve for most patients with scoliosis (usually T8 or 9) as the location in the spine with the greatest curvature and rotation inherent in the normal and scoliotic spine.

The other type of study is that by Schultz.[11] This is a study of the structural and kinematic aspects of the intervertebral joints. It calculates the configuration assumed by the spine associated with changes in the components of the intervertebral connection (figure 9). The results of the calculations reveal, among other things, that when the vertebra are normal, scoliotic curves can be reproduced within the normal range of motion of the vertebra. It also confirms the coupling of deformation modes inherent in the spine as observed by White.[12]

4. DEGENERATIVE JOINT DISEASE

The class of normal anatomy, small deviation, dynamic movements of the spine is almost certainly the largest group of all. It includes most of the "back pain" patients seen in the practitioner's office. There is no quantitative way and few qualitative guides to include the majority of these patients in the discussion. Therefore, by going to what is very likely the end stage of all these processes or afflictions, it may be possible to give some basis for future studies.

Degenerative joint disease afflicts the spine in a remarkable way. It focuses on two areas, the lumbosacral spine and the cervicothoracic spine. These two areas correspond roughly to the location of complaints of the "back pain" patient. These locations are, mechanically, very significant. One, the lumbosacral area, is most surely a high stress, low deformation area in a mechanics sense. Some of the details of this reasoning are given by Weis.[13] The other, the cervicothoracic area is again a transition region, mechanically, and no doubt incurs stress because of that. The other transition region, the thoracolumbar is not a region of high incidence of complaints and of degenerative joint disease. It is, however, the most likely location of a fracture-dislocation.

These clinical observations can be interpreted in terms of the dynamics of a curved tapered beam which is the subject of ongoing research. The curved tapered beam model was applied to problems of

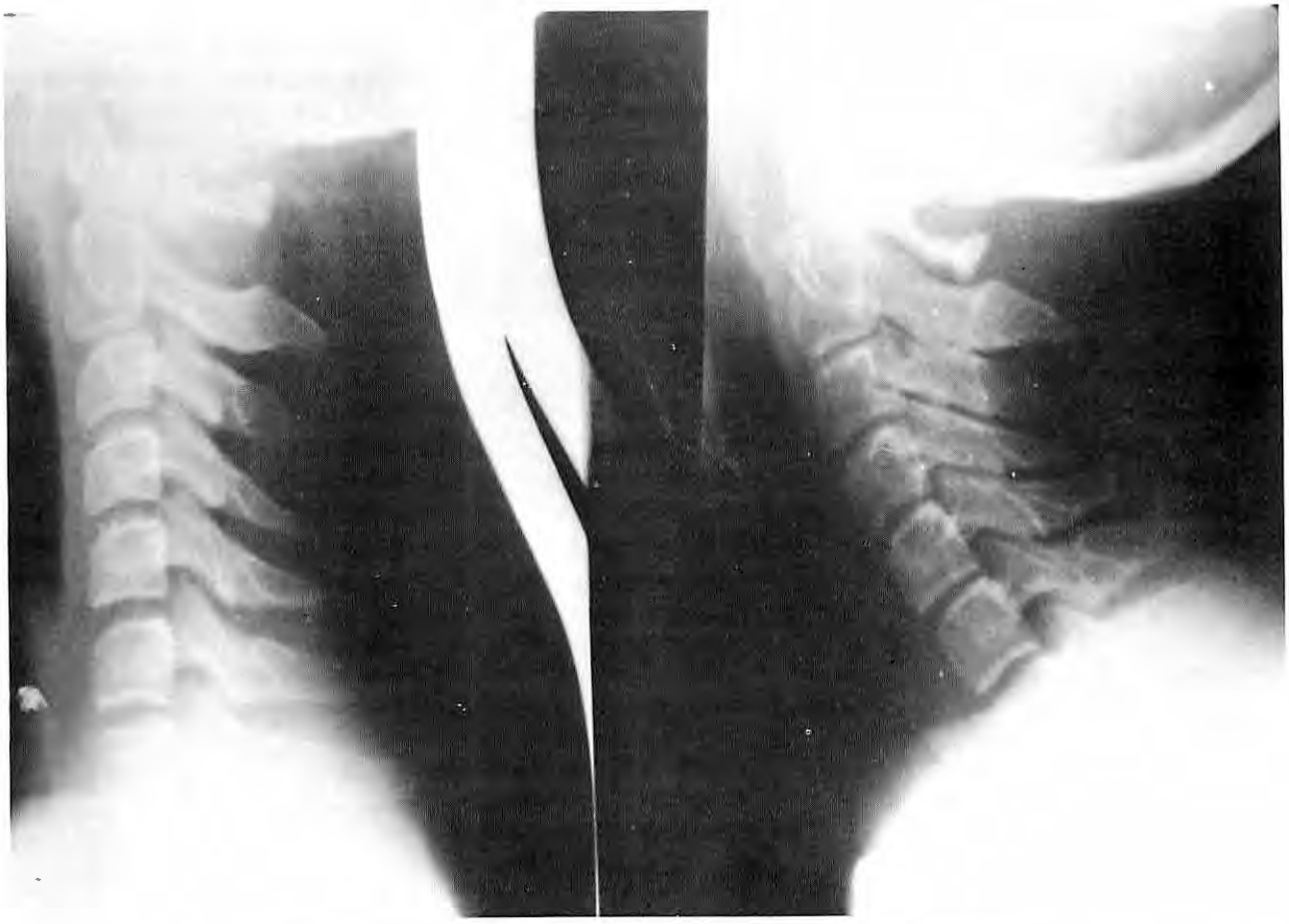


Fig. 3. Lateral cervical spine film in (left) and out (right) of halo-hoop traction.

spine injury by Benedict.[14] The pertinent derivations of this author are given in figure 10. Superficial inspection shows that bending cannot occur without rotation and lateral deviation and in fact, none of the motions is independent of the others. As mentioned earlier, Schultz and White have found this also. The curved tapered beam (figure 11) is intuitively satisfying as a model of the spine; study of the beam model shows that it has the same kind of critical locations as the spine. These are high stress, low deformation at the upper and lower ends and a low stress, high deformation location in the middle.

Studies, using this model, are devoted to measurement of the transmission characteristics of the cadaver torso from head to pelvis so that the coupling characteristics may be calculated. Ultimately the parameters for the curved tapered beam can be developed from this.

At this point in time, extrapolation of the curved tapered beam model to clinical problems of "back pain" may be interpreted in terms of inducement of a chronic healing reaction at the high stress, low deformation regions. The solution would seem to be the induction of significant long-term changes in such things as leg length, gait, weight, and posture.

5. CONGENITAL VERTEBRA ABNORMALITIES

Passing on to the situations of abnormal vertebra anatomy and using the same categorization scheme, consider the situation which arises when the vertebra are abnormal from the beginning and get worse as growth occurs. There are three basic types seen clinically; hemivertebra, block vertebra, and defects of the posterior elements. Hemivertebra leads to severe progressive scoliosis. Block vertebra leads to severe progressive kyphosis. Posterior element defects leads to a combination of scoliosis and kyphosis, both severe but not always progressive.

The problem of hemivertebra is illustrated in figure 12. This young child has been advised to be in a Milwaukee Brace immediately with careful ob-

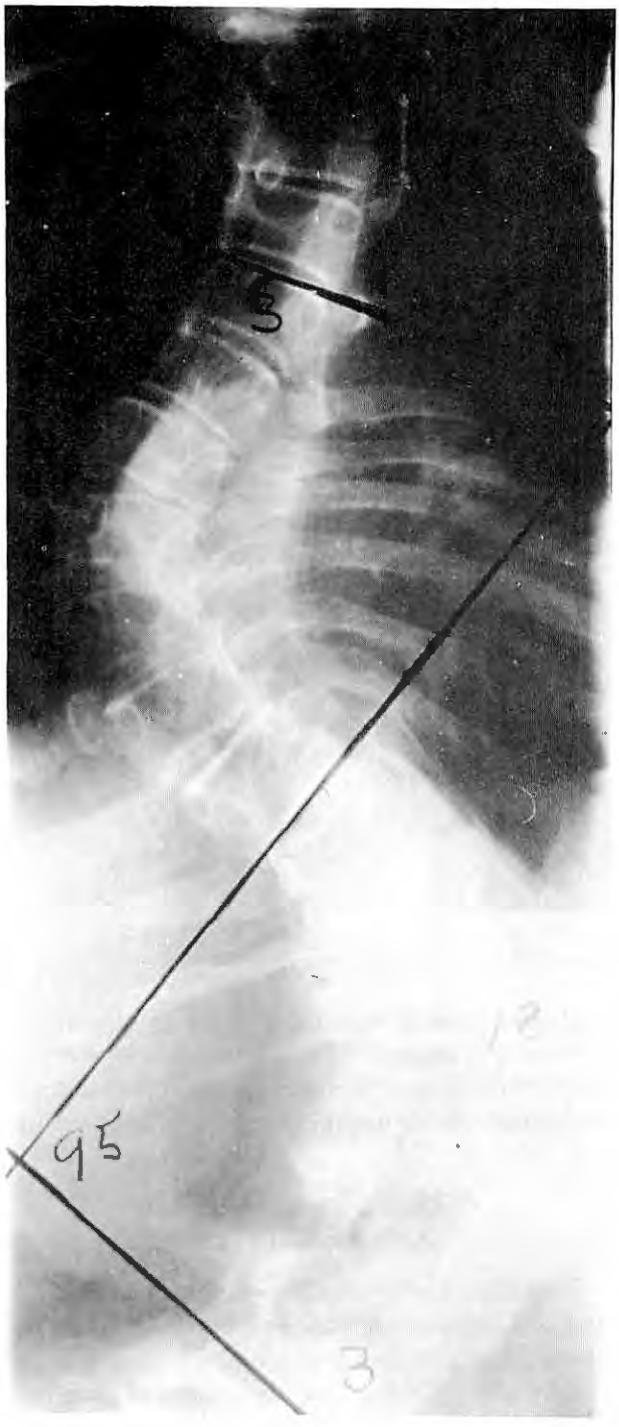


Fig. 4. Anterior posterior roentenogram of scoliosis before halo-hoop traction.

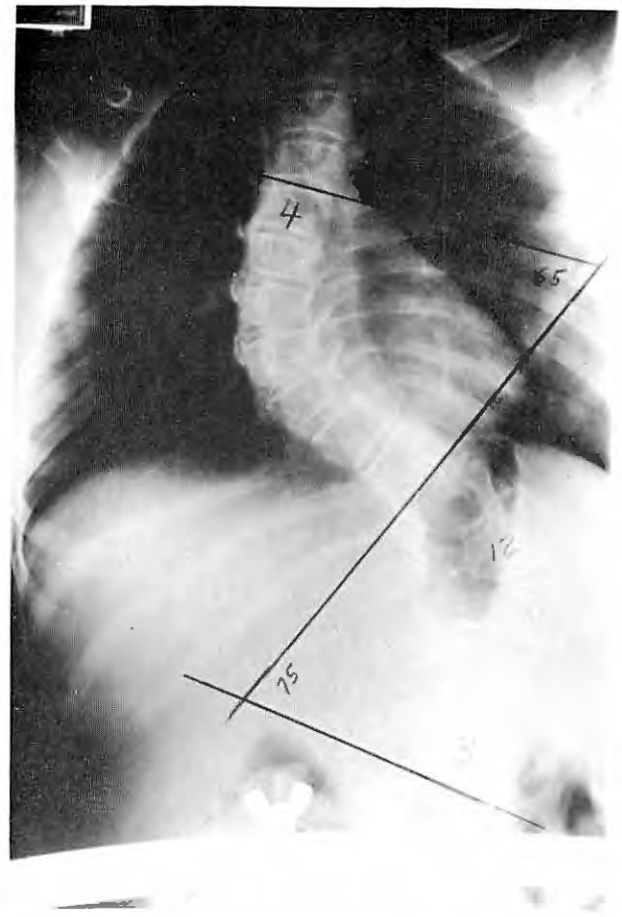


Fig. 5. Anterior-posterior roentenogram of scoliosis after halo-hoop traction.

servation and the almost sure expectation of extensive spine fusion.

The problem of block vertebra is illustrated in figure 13. This 20-year-old female should have had an anterior osteotomy and an anterior and posterior fusion years ago to prevent the extreme deformity. She has now undergone the same surgery with much higher risks and much poorer cosmetic effect.

The myelomeningocele has many problems above and beyond the spine deformity but it is instructive to consider the spine deformity because it is unstable. The defect is the absence of the posterior elements along with much of the neurological function. This leads to scoliosis and kyphosis which are only correctable surgically. This is desirable because wheelchair sitting is otherwise difficult and decubiti are common.

6. FRACTURES AND DISLOCATIONS

The basic question which must be answered regarding the spine in such cases is; is it stable? The high incidence is at T12–L1. Statistics about how many have neurological problems are difficult to interpret. It is small if the 10 to 30 percent compression fractures are included and high if they are not.

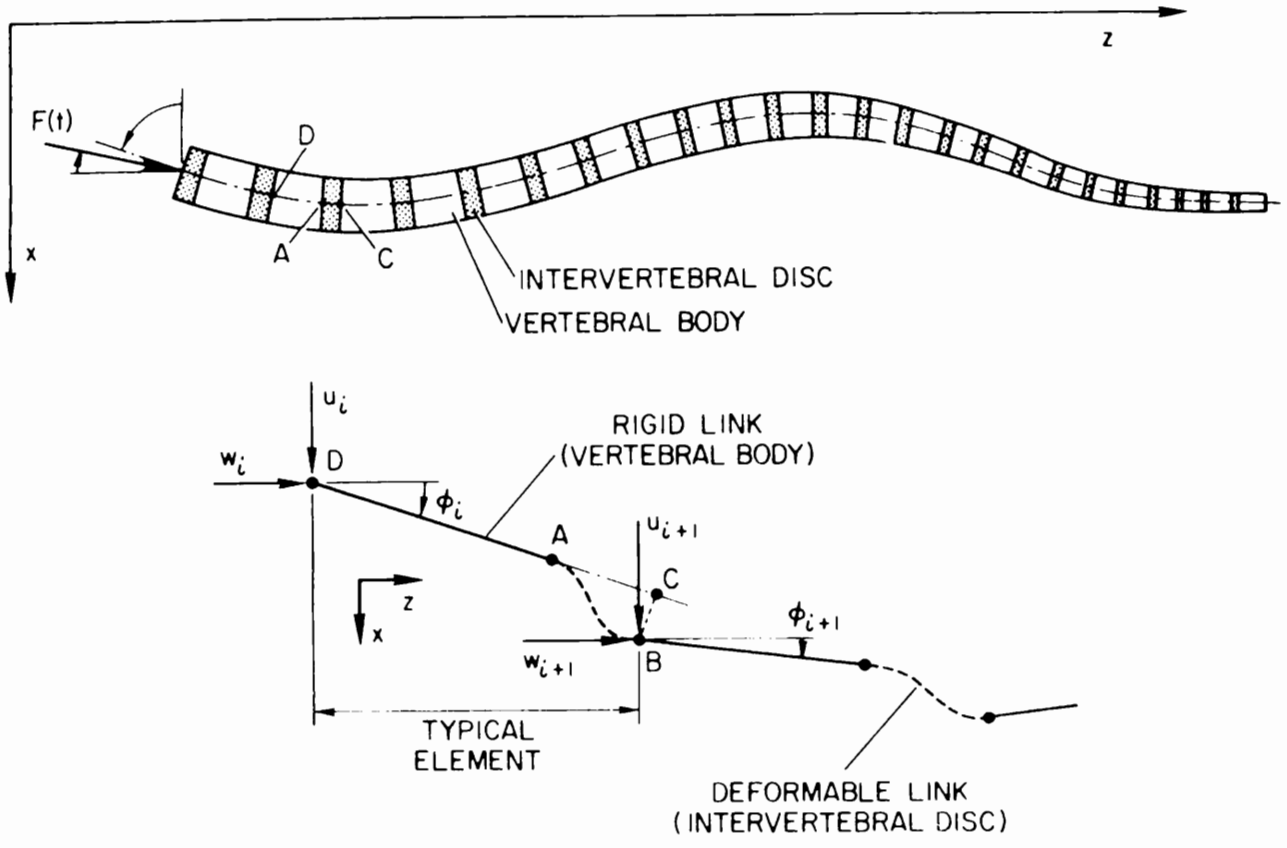


Fig. 6. Schematic illustration of spinal model by Orne.

The history of treatment of fractures and dislocations of the spine is pertinent.[15] In the past these injuries were treated aggressively by manipulation. The complication of neurological injury secondary to manipulation was recognized and manipulation was reduced to traction with hyperextension. Still more recently the deformities have been neglected if the assessment could be made that the injury is structurally stable from the onset or if it is expected to become stable by spontaneous fusion. Operative reduction at the time of fusion of dislocations is not often undertaken, particularly in the absence of a neurological lesion.

There is much uncertainty about the relationship between fracture or dislocation, including reduction, and neurological treatment. A case of fracture with extrusion of a portion of the vertebral body into the spinal canal is shown in figure 14. There was no neurological disease, no reduction or operative treatment was applied, and the patient is fine. A case of fracture-dislocation is shown in figure 15. The patient was immediately paraplegic but the disease receded to become a Brown-Sequard syndrome with little functional impairment. No reduction or operative treatment was applied. A case of fracture-dislocation is shown in figure 16. The patient was immediately paraplegic. Laminectomy was performed without benefit. Anterior fusion was carried out without reduction. The patient is still paraplegic.

7. SPONDYLOLISTHESIS AND SACRALIZATION

Whether acquired or congenital, abnormalities of the lumbosacral joint are a dilemma. A case of spondylolisthesis is illustrated in figure 17. This patient was symptomatic and had a posterior elementectomy and posterior interbody fusion and is now asymptomatic. There is no difficulty in finding a similar case in which the patient was not relieved. Nonsurgeons treating the same problems are often of the opinion that surgery is contraindicated and that physical therapy is the correct approach. There is no difficulty finding patients unrelieved by physical therapy.

Sacralization is only one of the variations of in-

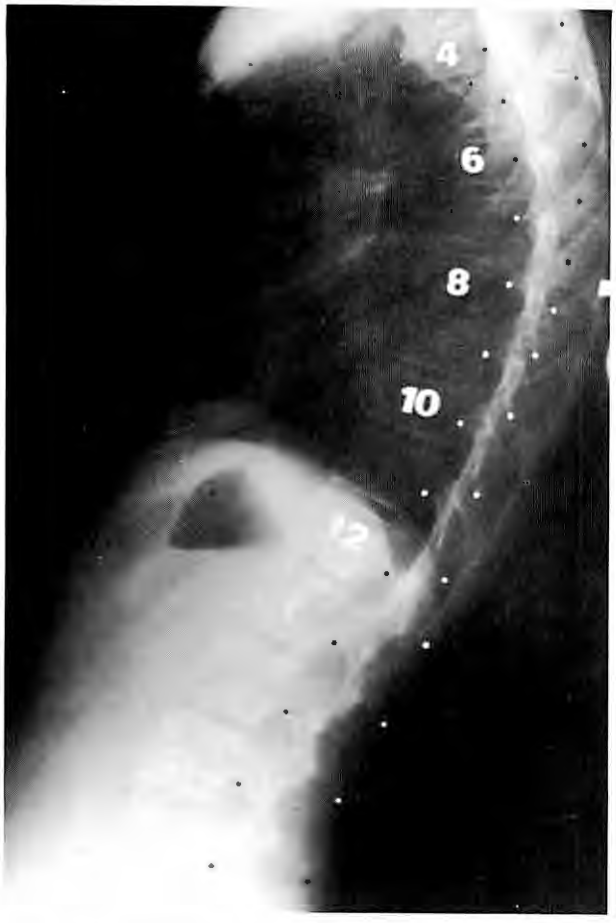


Fig. 7. Measurement X-ray for evaluation of curvature and rotation (Weis).

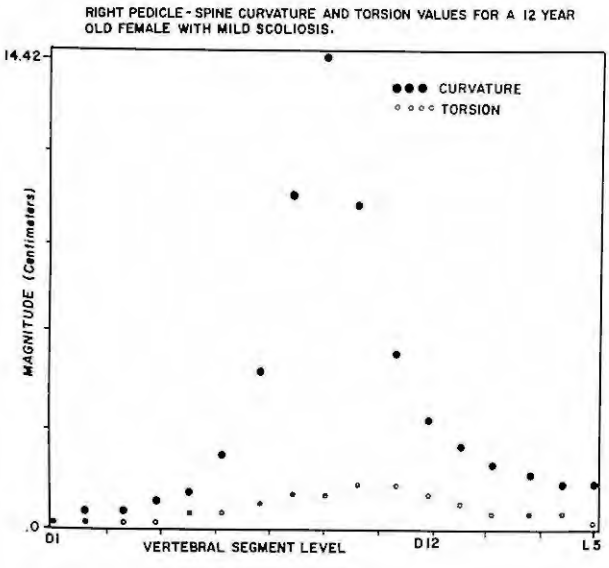


Fig. 8. Calculated curvature and torsion from case shown in Figure 7.

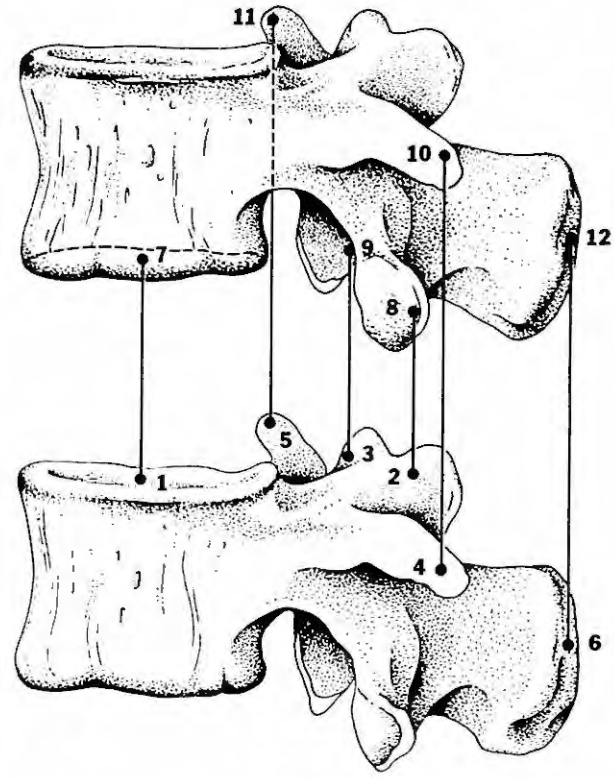


Fig. 9. Rigid links used in calculation of geometrical variations (redrawn from Schultz).

$$\text{I}\quad \frac{\partial P}{\partial z} = \rho A \frac{\partial^2 \bar{w}}{\partial t^2} + \rho A r \frac{\partial^2 \phi}{\partial t^2}$$

$$\text{II}\quad \frac{\partial}{\partial z}\left(EI_1 \frac{\partial \phi}{\partial z}\right) + kAG\left(\frac{\partial u}{\partial z} - \frac{\partial u_0}{\partial z} - \phi\right) = \rho I_2 \frac{\partial^2 \phi}{\partial t^2} + \rho A r \frac{\partial^2 w}{\partial t^2}$$

$$\text{III}\quad \frac{\partial}{\partial z}\left[kAG\left(\frac{\partial u}{\partial z} - \frac{\partial u_0}{\partial z} - \phi\right) + P\frac{\partial u}{\partial z}\right] = \rho A \frac{\partial^2 u}{\partial t^2}$$

$$\text{IV}\quad P = EA\left[\frac{\partial \bar{w}}{\partial z} + \frac{1}{2}\left(\frac{\partial u}{\partial z}\right)^2 - \frac{1}{2}\left(\frac{\partial u_0}{\partial z}\right)^2\right]$$

Fig. 10. Partial differential equation representing a curved tapered beam.

clusion of the last lumbar vertebra in the sacrum, exclusion of the first sacral vertebra from the sacrum and abnormal shape, size, and placement of the lumbosacral facet joints. Sacralization is seen in figure 18. Studies [16] of the relationship between X-ray abnormalities of the spine and symptomatology show a very poor correlation with abnormalities of L5 and S1 being the second abnormality when scoliosis is included and first when it is not. Given this uncertain situation it is nearly impossi-

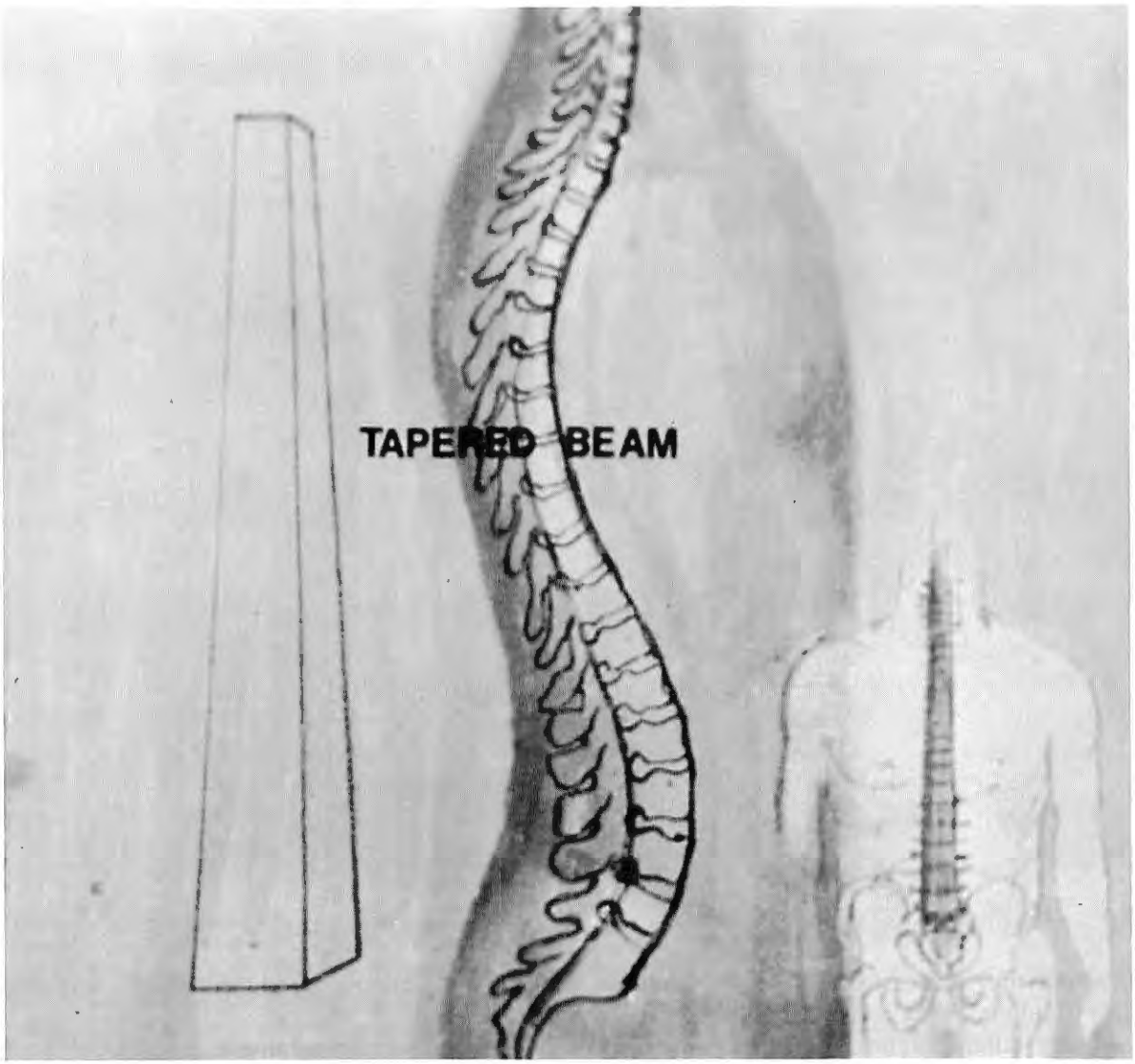


Fig. 11. Curved tapered beam representation of the spine.

ble to convince anyone that there is any benefit in treating symptoms thought to be related to radiological abnormality.

Studies of the mechanics of the lumbosacral junction have been undertaken by several authors (Weis [13] and Krause [17]). These tend to show that the margin of safety for this joint is quite low and that it is probably subjected to near failure loads in normal activity. It is not surprising then that interior linemen on football teams and Eskimos in their igloos have low back problems.[18,19]

8. HERNIATED NUCLEUS PULPOSUS

A case of radiculopathy of the first sacral nerve root is shown in figure 19. This patient was operated on and found to have a disc protrusion but it was composed mostly of spur formation secondary to the degenerative joint disease. He was relieved by the operative procedure. The case illustrates the coincidence of the two diseases or rather the transition from one to the other which may occur.

Studies of the intervertebral disc by Markolf[20,21] and Belytschko [22] are providing insight into the pathologic process of herniated disc. Markolf has presented his studies of the stiffness of the intervertebral disc (as shown in figure 20) and the change in the disc subjected to operative treatment. He defines the torsional strength variation along the spine and finds that operative treatment of disc disease does not change its mechanical character much. Belytschko is studying the stress distribu-

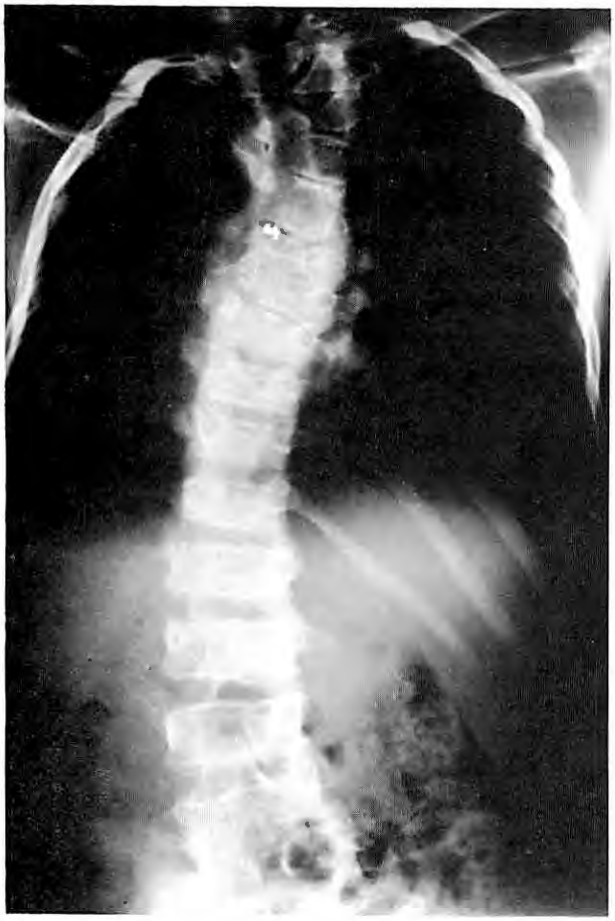


Fig. 12. Scoliosis due to congenital anomalies of the vertebra.

Fig. 13. Congenital kyphosis due to block vertebrae.

tion within the intervertebral disc by computer modeling and may be expected to contribute greatly to the understanding of the significance of the structure of the disc.

SUMMARY AND CONCLUSIONS

The parade of clinical entities given in the foregoing pages is distinguished by arrangement into categories based on attempts at quantification. It seems clear that because of the highest incidence of problems, the most interest should lie in the normal anatomy, small deviation, dynamic loading regimen. In particular, the overall response of the spine needs to be characterized better to allow one to assess therapy effects as well as to understand pathogenesis. The second critical area or regimen is for small deviations, dynamic loading, abnormal anatomy, usually produced by a degenerative process.

The role of manipulative therapy seems no more or less well established than surgical therapy. The efficacy of both are difficult to assess in patients in whom the relationship between anatomy, function, and symptoms is indistinct. The most hopeful approach seems to be to model the mechanics of an abnormality or condition, when it can be defined, by laboratory derivation of properties. The models of such measurements can then be manipulated by people and computers to study the effect of manipulative treatment in a gross way. Parallel prospective studies of the efficacy of manipulative therapy by statistically sound methods must be conducted to answer the question of whether there is any effect in addition to the "placebo effect."

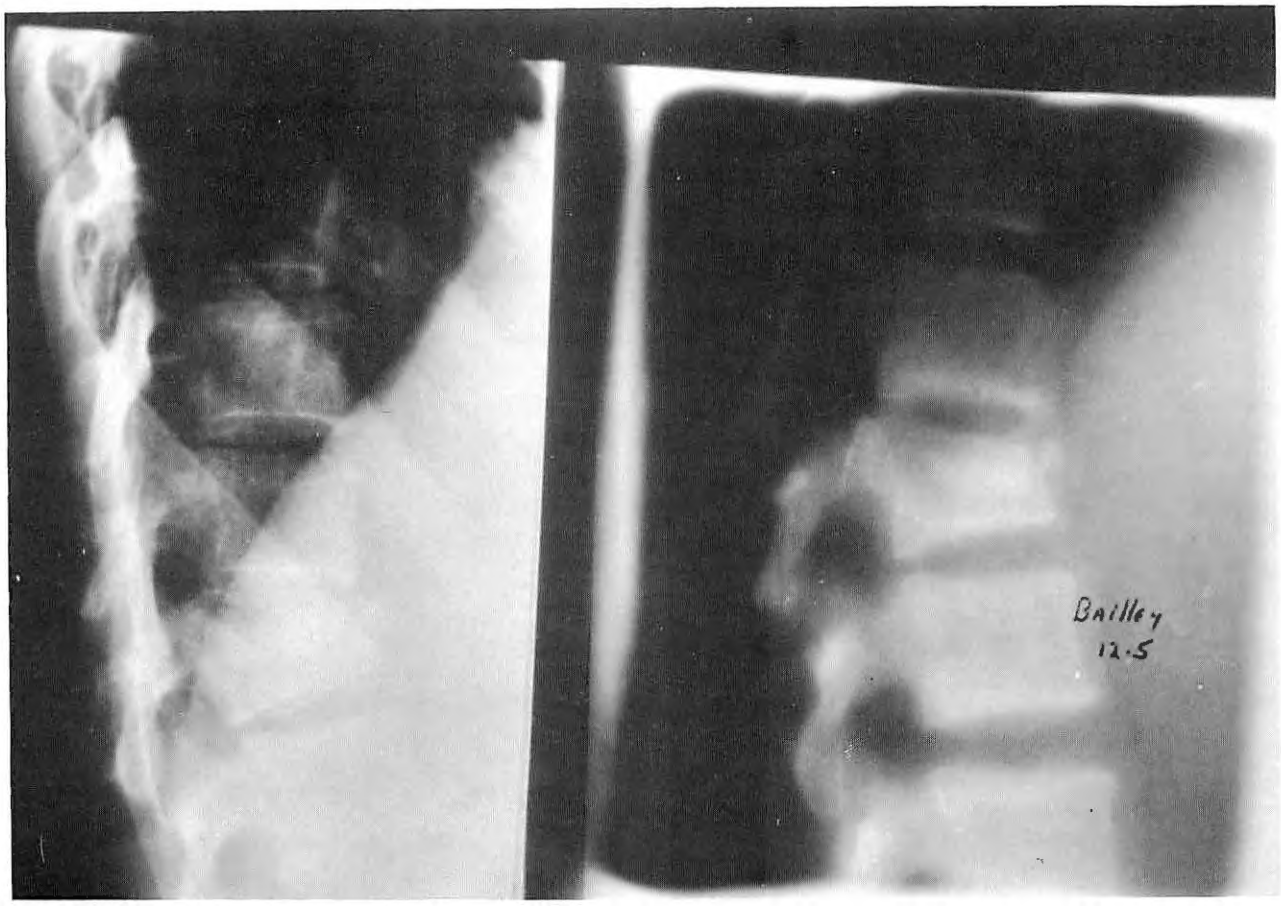


Fig. 14. Fracture of the L1 vertebral body with extrusion of a fragment into the spinal canal (laminogram right).

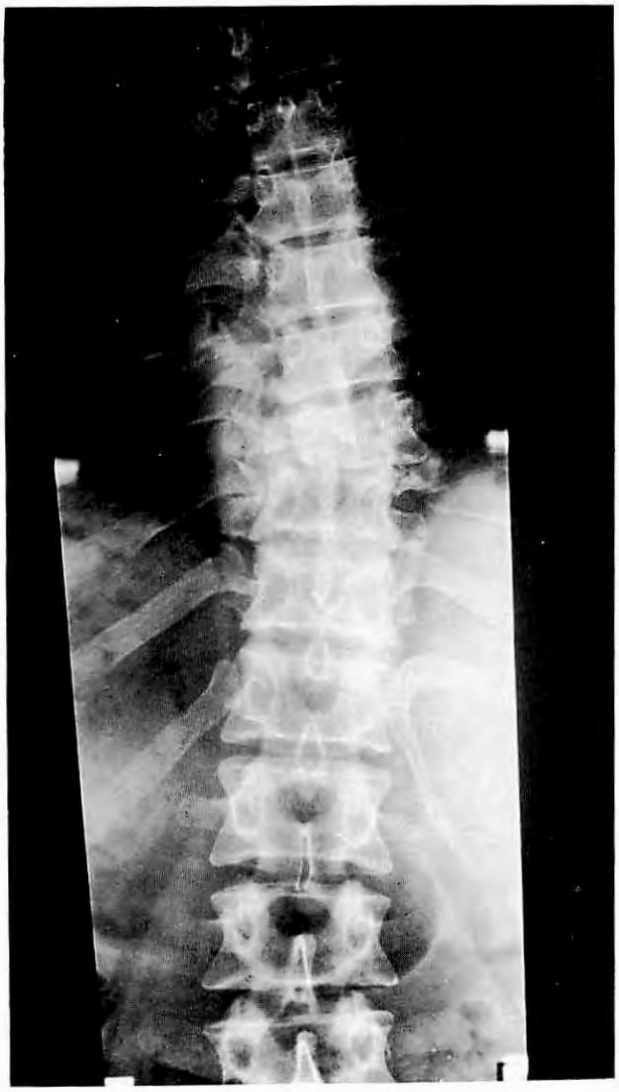


Fig. 15. T9–10 fracture-dislocation with Brown-Sequard syndrome.

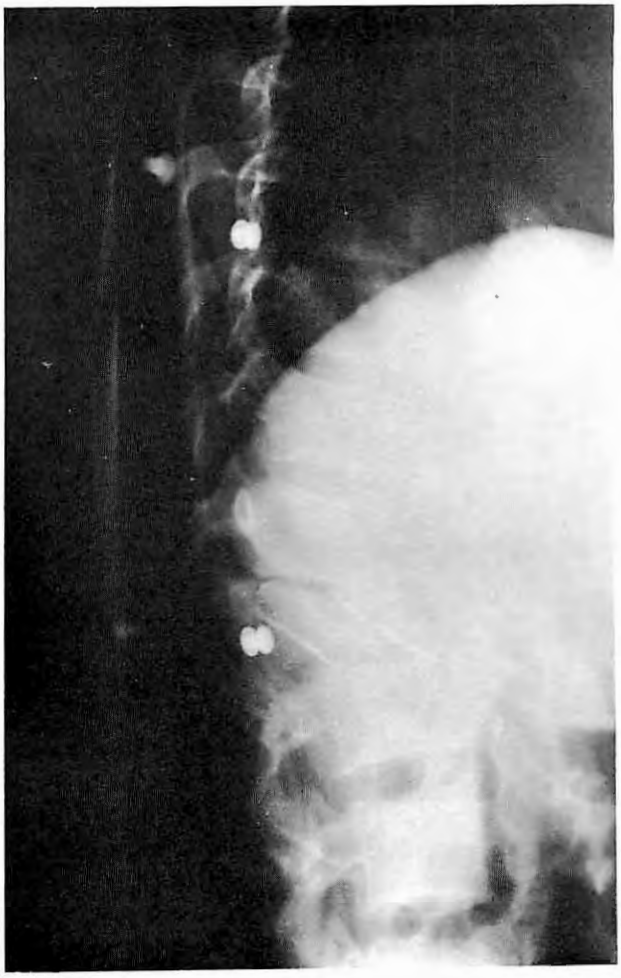


Fig. 16. Post-operative roentenogram of L1 fracture-dislocation (anterior decompression and fusion).

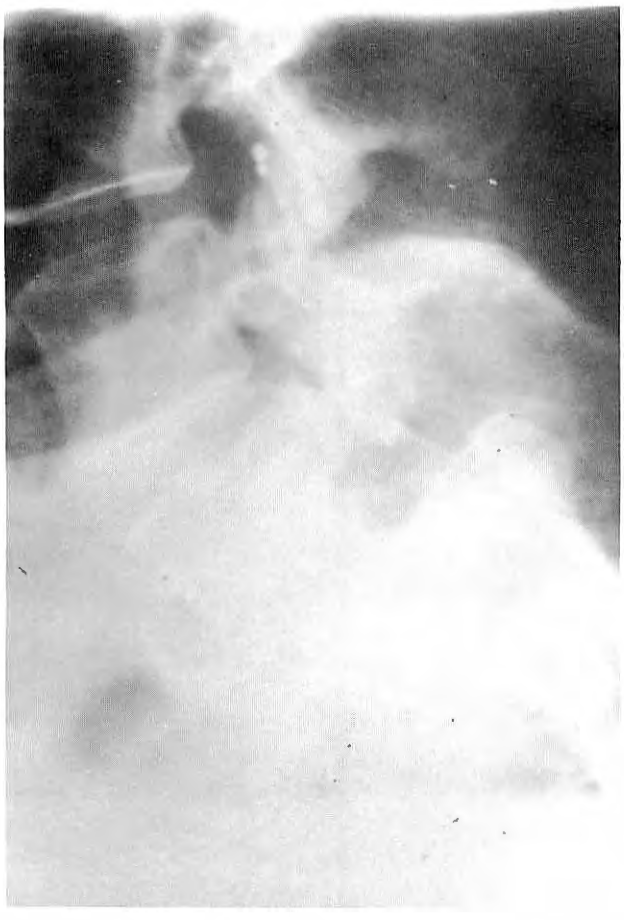


Fig. 17. First degree spondylolisthesis.

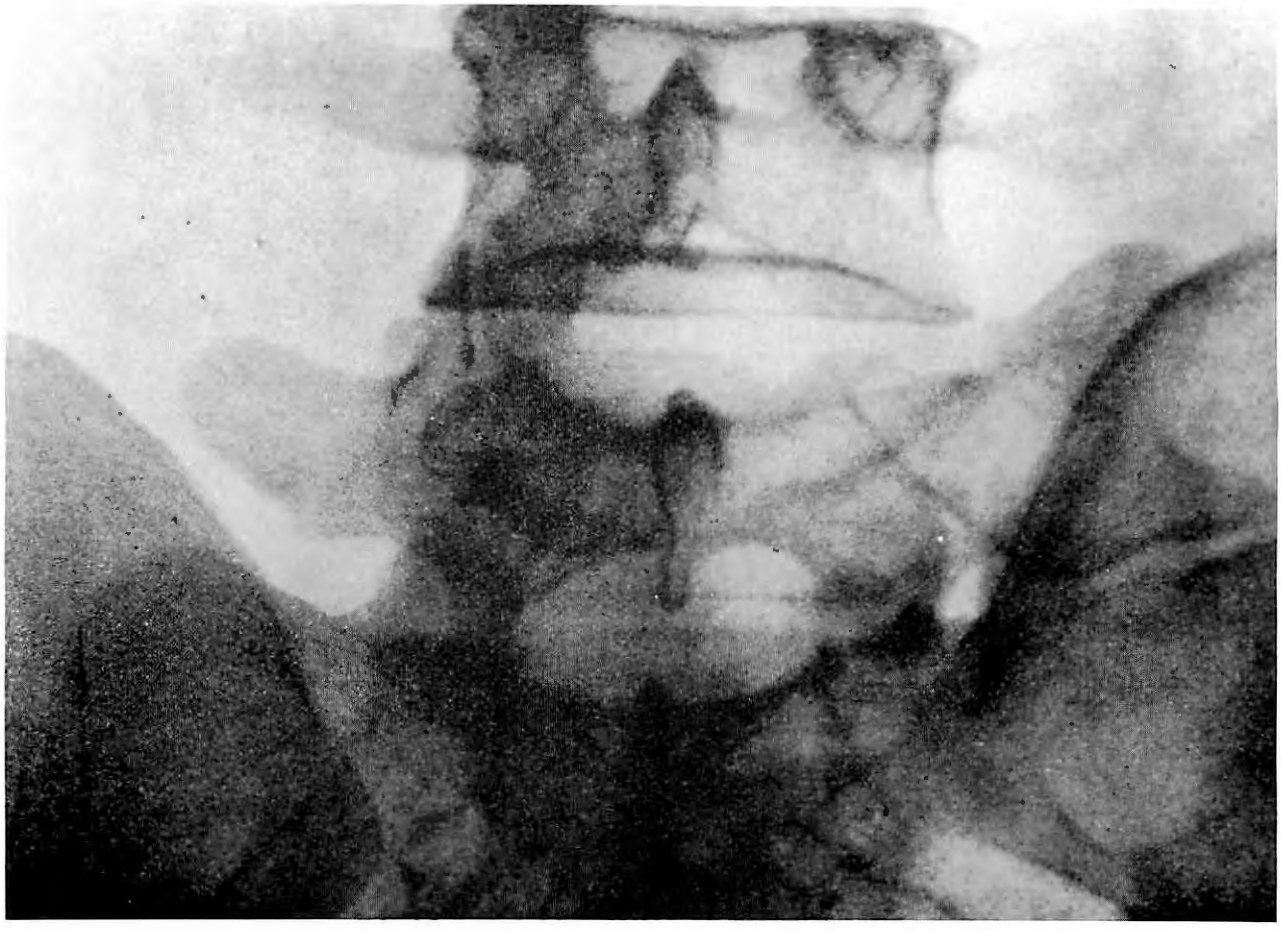


Fig. 18. Sacralization of the fifth lumbar vertebra.

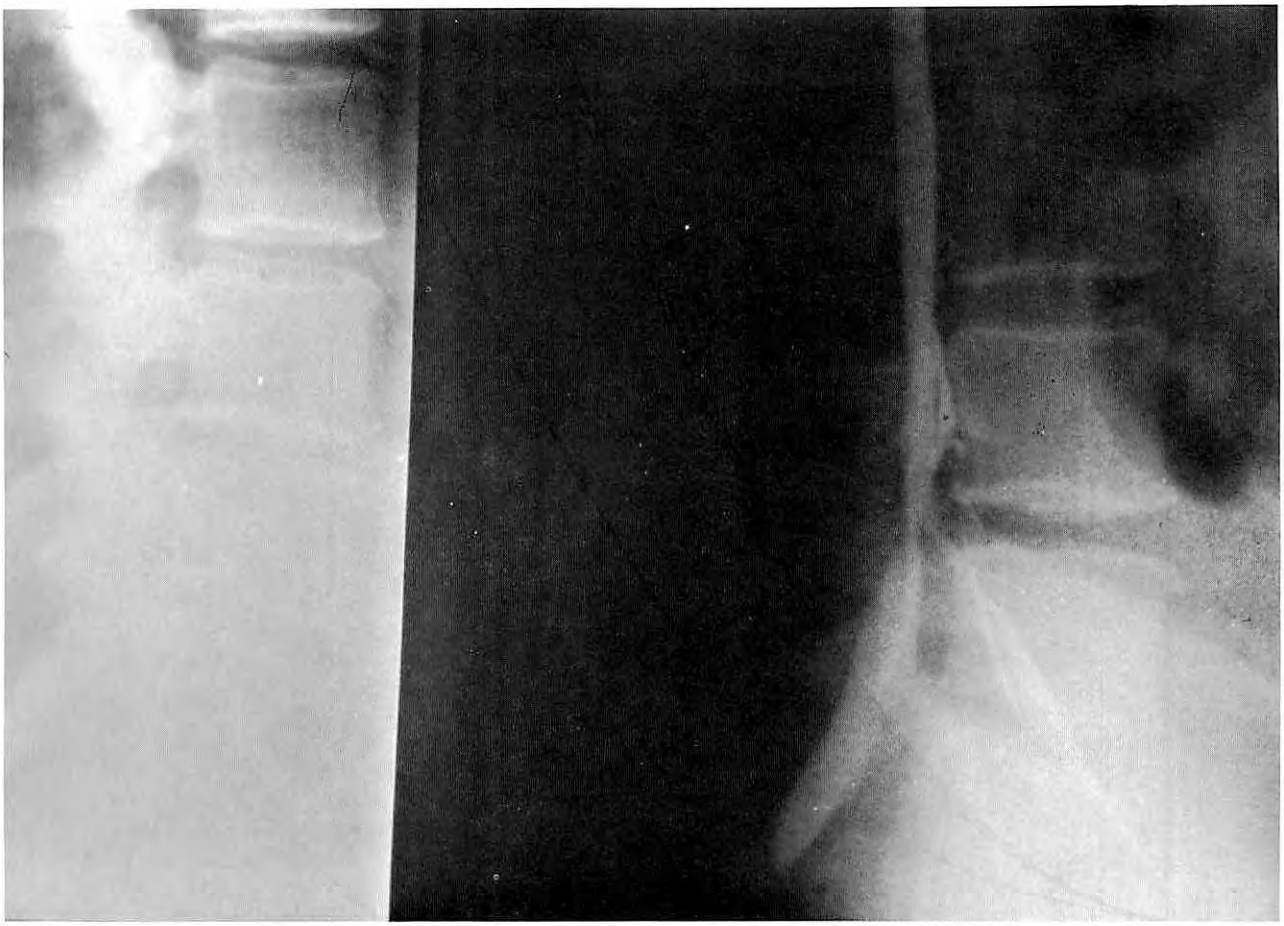
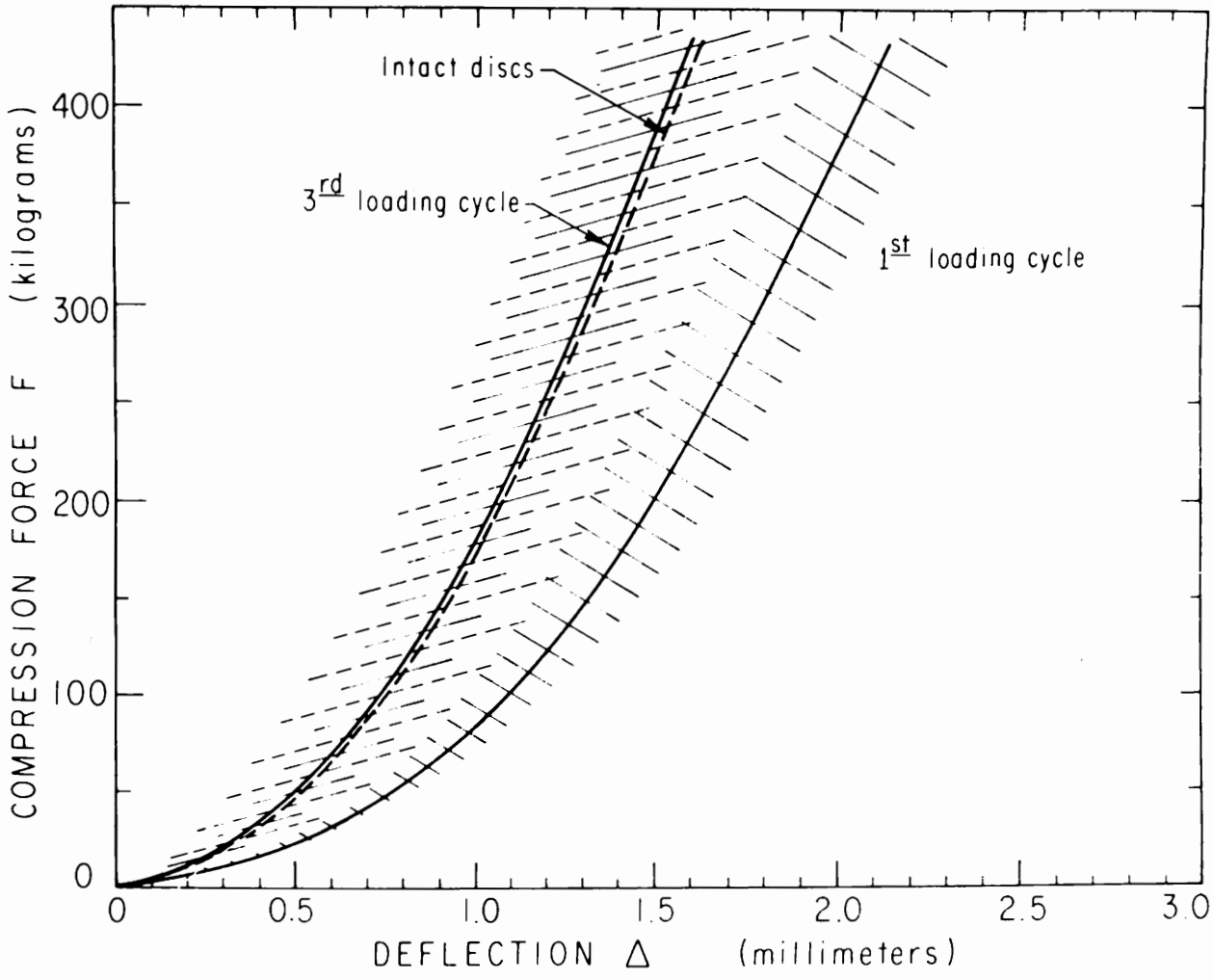


Fig. 19. Degenerative joint disease at L4–5–S1 with disc protrusion at L4–5.

597-036 O - 76 - 7



Fog. 20. Stiffness of the intervertebral disc (redrawn from Markolf).

REFERENCES

1. DeWald, R. L. and Ray, R. D., "Skeletal Traction for the Treatment of Severe Scoliosis," *J.B.J.S.* 52-A, March 1970, p. 233.
2. Weis, E. B., Clarke, N. P., Brinkley, J. W., and Martin, P. J., "Mechanical Impedance as a Tool in Human Response to Acceleration," *Aerosp. Med.* 35:10, October 1964, p. 945.
3. Weis, E. B. and Primiano, R. P., "Motion of the Human Center of Mass and Its Relationship to Mechanical Impedance," *Human Factors*, October 1966, p. 399.
4. Kaleps, I., Von Gierke, H. E., and Weis, E. B., "A Five-Degree-of-Freedom Mathematical Model of the Body," Paper No. 8, *Proceedings of the First Biodynamics Symposium*, Aerospace Medical Research Laboratory, Wright Patterson Air Force Base, October 1969.
5. Orne, D. and Liu, Y. K., "A Mathematical Model of Spinal Response to Impact," *Journal of Biomechanics*, Vol. 4, pp. 49–71, 1971.
6. King, A. I. and Vulcan, P., "Elastic Deformation Characteristics of the Spine," *Proceedings of Workshop on Bio-engineering Approaches to Problems of the Spine*, September 1970, p. 15.
7. Weis, E. B. and Mohr, G. C., "Cineradiographic Analysis of Human Visceral Responses to Short Duration Impact," *Aerosp. Med.* 38(10), October 1967, p. 1041.
8. Weis, E. B., Pearce, W. L., and Shoenberger, R. W., "Measurement of Human Responses to Short Duration Impact," *Journal of Biomechanics*, Vol. 6, 1973, pp. 719–728.
9. Weis, E. B., Clarke, N. P., and Brinkley, J. W., "Human Response to Several Impact Acceleration Orientations and Patterns," *Aerosp. Med.* 34(12), December 1963, p. 1122.
10. Weis, E. B., "Quantitation of Curvature and Rotation in X-rays of the Spine," *Proceedings of Workshop on Bio-engineering Approaches to Problems of the Spine*, September 1970, p. 177.
11. Shultz, A. B., Larocca, H., Galante, J. O., and Andriachhi, T. P., "A Study of Geometrical Relationships in Scoliotic Spines," *Journal of Biomechanics*, Vol. 5, No. 4, July 1972, p. 409.

12. White, A. A., "Analysis of the Mechanics of the Thoracic Spine in Man," *Acta Orthop. Scand.*, Supp. 127, 1969.
13. Weis, E. B., "Stresses at the Lumbosacral Junction," *Orth. Clin. N.A.*, January 1975.
14. Benedict, J. V., "The Investigation of Vertebral Injury Sustained During Aircrew Ejection," *Final Technical Report, Contract NAS2-5062*, Technology, Inc., San Antonio, Tex.
15. Bick, E. M., *Source Book of Orthopaedics*, Hafner Publishing Co., N.Y., 1968, p. 306.
16. Splitoff, C. A., "Roentgenographic Comparison of Patients With and Without Backache," *J.A.M.A.*, 152: 1610, 1953.
17. Krause, H., Robertson, G., and Farfan, H. F., "On the Mechanics of Weight Lifting," *Proceedings of the New England Bioengineering Conference*, April 1973.
18. Ferguson, R. F., McMaster, J. H., and Stanitski, C. L., "Low Back Pain in College Football Lineman," *J. Spts. Med.*, Vol. 2, No. 2, 1974, p. 63.
19. Kettlekamp, D. B. and Wright, D. G., "Spondylolysis in the Alaskan Eskimo," *J.B.J.S.* 53-A, No. 3, April 1971, p. 563.
20. Markolf, K. L., "Deformation of the Thoracolumbar Intervertebral Joints in Response to External Loads," *J.B.J.S.* 54-A, No. 3, April 1972, p. 511.
21. Markolf, K. L. and Morris, J. M., "The Structural Components of the Intervertebral Disc," *J.B.J.S.* 56-A, No. 4, June 1974, p. 675.
22. Belytschko, J., Kulak, R. F., and Schultz, A. B., "Finite Element Stress Analysis of an Intervertebral Disc," *Journal of Biomechanics*, Vol. 7, 1974, pp. 277-285.

Spinal Kinematics

AUGUSTUS A. WHITE, III
MANOHAR M. PANJABI
Engineering Laboratory for Musculoskeletal Diseases
Section on Orthopaedic Surgery
Yale University School of Medicine

INTRODUCTION

Kinematics is that phase of mechanics concerned with the study of motion of particles and rigid bodies with no consideration of the forces involved.

To describe the kinematics of the spine, the analogy of the train used by Lovett [1] is employed and adjusted to the imagination of the present authors. If each vertebra is thought of as a train, one could begin to describe its kinematics with answers to the following questions. What points does the train run between? What course does it take? How far apart are these points? What alterations of the train will change the course it takes, the distance it travels, or the smoothness of the ride? How far outside its usual course can it travel without being in danger of malfunctioning? What happens to the route taken and the distance traveled as the train progresses from a new to an old train? What are the methods for precisely defining the course and distances traveled by a given train? As this presentation develops it will become apparent how answers to these hypothetical questions describe the normal kinematics of the spine and relate it to some of the important abnormal situations of clinical interest.

There is a considerable amount of information available on this complex topic. In order to stay within the limitations of this presentation, only the major aspects of the subject will be covered and not in detail.

TERMS AND DEFINITIONS

This section contains some guidelines and ground rules for clear and accurate communications. Its contents, in part or in toto, will no doubt be received with gratitude, disdain, confusion, or indifference, depending on the individual reader.

Coordinate System

The right-handed orthogonal (Cartesian) coordinate system is recommended.[2] Its orientation in space is shown in figures 1 and 2. Motion is described in terms relative to the subadjacent vertebra as shown in figure 2.

Motion Segment

The motion segment which is the traditional unit of study in spinal kinematics is constituted by two adjacent vertebrae and their intervening soft tissues.

Translation

A body is said to be in translation when movement is such that all particles in the body at a given time have the same velocity relevant to some reference.

Rotation

A body is said to be in rotation when movement is such that all particles along some straight line in the body or a hypothetical extension of it have a zero velocity relative to some reference.

Degrees of Freedom

One degree of freedom is motion in which a rigid body has the possibility of translating back and forth in either direction along a straight line. Or if a given body can rotate back and forth clockwise and counterclockwise in either direction, that too is a degree of freedom. Figure 3 gives examples of one, two, and three degrees of freedom. Vertebras have the possibility of six degrees of freedom.

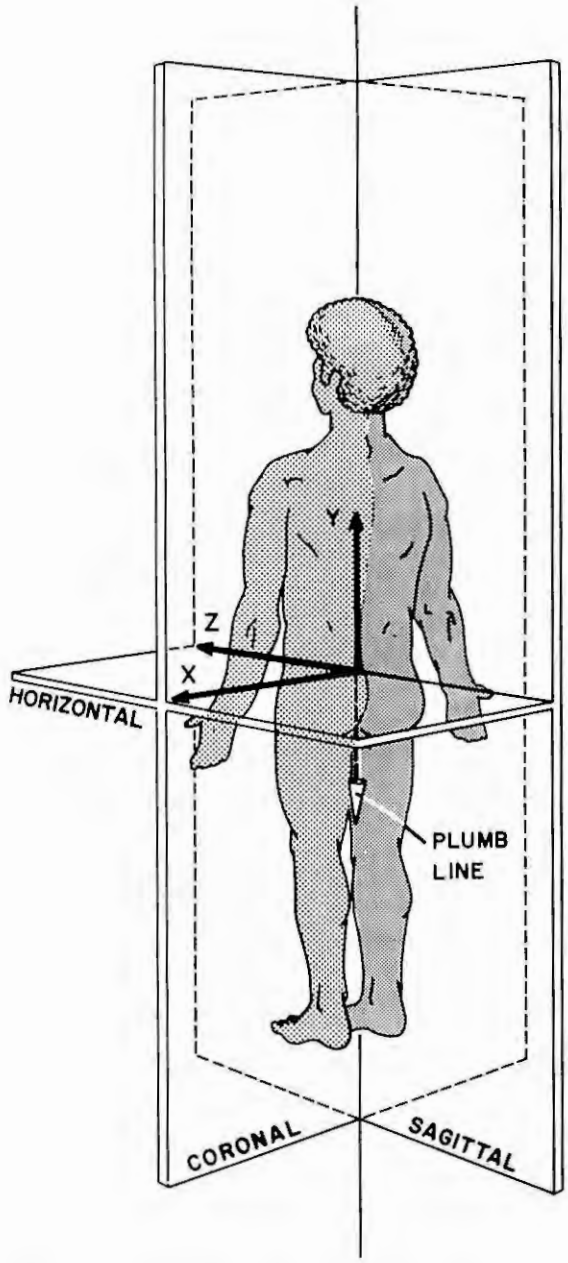


Fig. 1. The suggested central coordinate system with its origin between the cornua of the sacrum is shown. Its orientation is such that negative y-axis is described by the plumb line dropped from the origin, the positive x-axis points to the left, and the positive z-axis points forward. The human body is shown in the anatomic position.

Range of Motion

An indication of the two points at the extremes of the physiological range of translation and rotation of a vertebra for each of the six degrees of freedom.

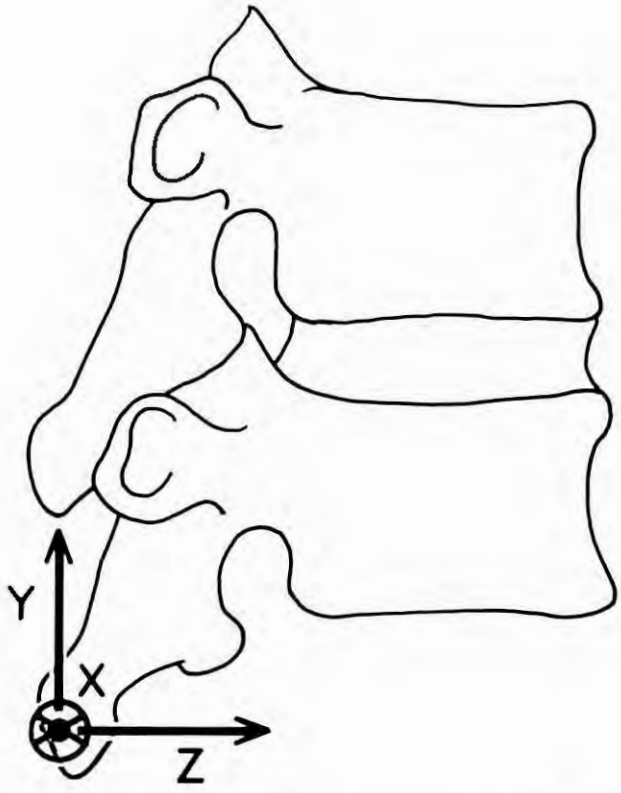


Fig. 2. Here a motion segment is shown with the reference point at the tip of the spinous process of the subadjacent vertebra. The orthogonal system is presented in the y, z or sagittal plane. The x axis is thus indicated by convention showing the cross to represent the base of the arrow.

Coupling

Coupling is applied to motion in which rotation or translation of a rigid body about one axis is consistently associated with rotation or translation of that same rigid body about another axis. Coupling is diagrammed in figure 4.

Pattern of Motion

This is defined by the configuration of a line that the centroid of a body in motion forms as it moves from one point to another.

Instantaneous Axes of Rotations (IAR)

At every instant, for a rigid body in plane motion there is a line in the body or a hypothetical extension of it which does not move. This line is the instantaneous axis of rotation. Plane motion is fully defined by the position of the IAR and the magnitude of the rotation about it. (See figure 5.)

Helical Axis of Motion (HAM)

The instantaneous motion of a rigid body in three-dimensional space can be analyzed as a

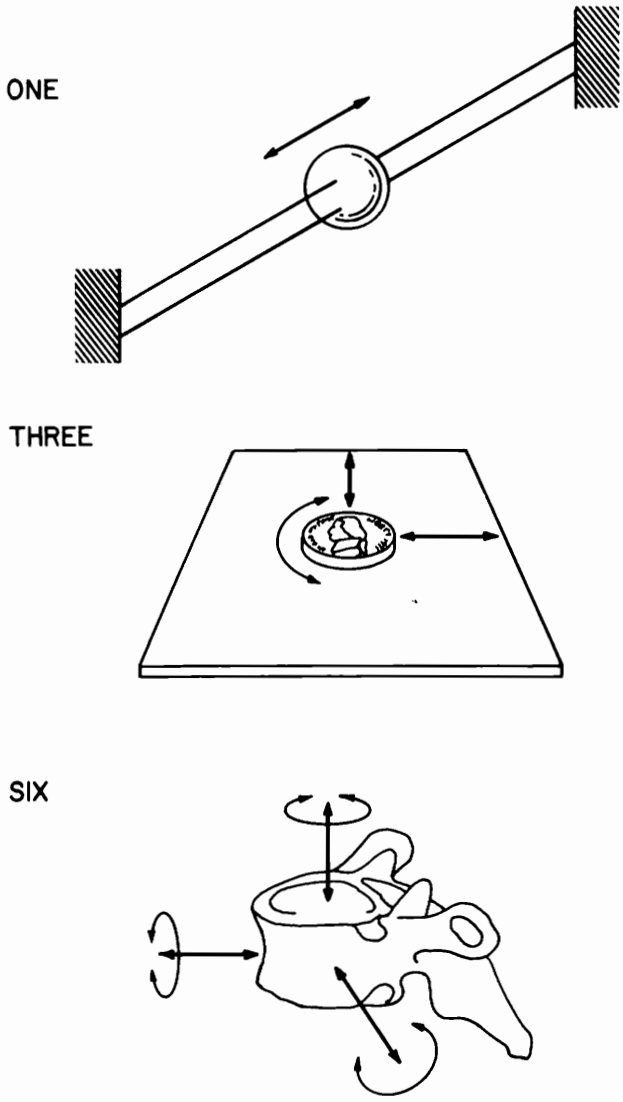


Fig. 3. The concept of degrees of freedom involved in the engineering analysis in kinematics is shown diagrammatically.

simple screw motion. The screw motion is a superposition of rotation and translation about and along the same axis. This axis has the same direction as the resultant of the three rotation components about the x, y, z axes. For a given moving rigid body in space the location of this axis and the designation of the quantity of its rotation and translation constitute a complete, precise, three-dimensional description of the motion. (See figure 6.)

A COMPREHENSIVE DESCRIPTION OF SPINAL KINEMATICS

One of the goals of this presentation is to present what is known, what is not known, and what would be useful to know about spinal kinematics. Table I outlines information which, if available, would approach an idealized comprehensive description of the important basic and clinical aspects of spinal kinematics. The complete description would include the information in table I presented for each motion segment from occiput to sacrum using the orthogonal coordinate system or some other reliable system. If one accepts this table as a comprehensive outline, there are a large number of blanks to be filled in. Rather than concentrate on what would be necessary to fill the empty portion of the "glass," the authors have chosen to present their own brief summary of that which is currently in the "glass."

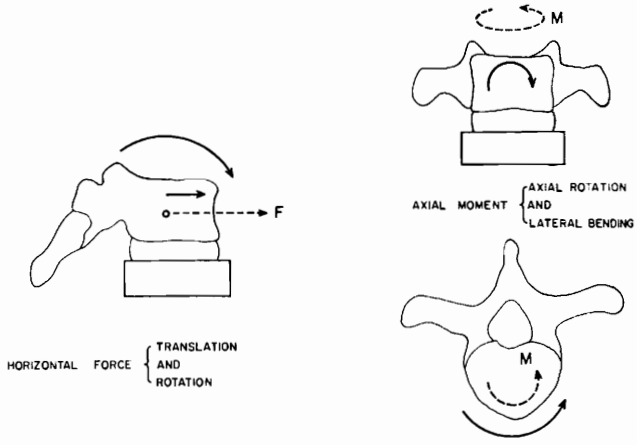


Fig. 4. Diagrammatic representation of some important forms of coupling involved in spinal kinematics. The loads employed to effect the various movements are shown by the dotted line.

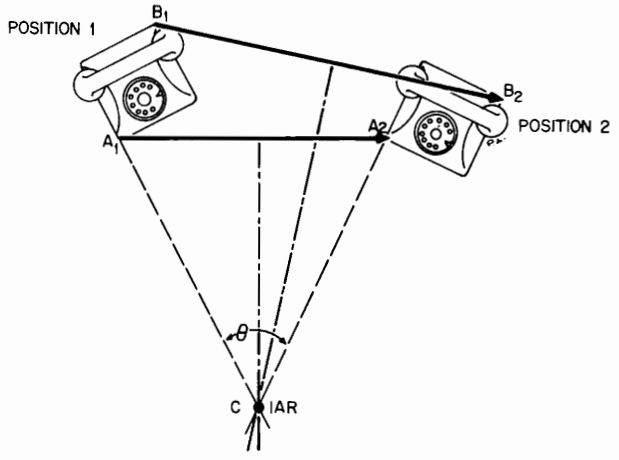


Fig. 5. This shows a concept and the actual method of determining the instantaneous axis of rotation in uniplanar motion. The telephone moves from position 1 to position 2. Two lines are drawn from the two points in the original position 1 to the same points in position 2. These are indicated by A_1, A_2 and B_1, B_2. Perpendicular bisectors are then erected upon these two lines. The point at which they meet is the IAR.

TABLE I

A Comprehensive Description of Spinal Kinematics

1. Range of motion; for all six degrees of freedom.
   a) Translation
   b) Rotation
2. All coupling characteristics and their ratios.
3. Patterns of motion in the traditional physiological patterns.
   a) Flexion Extension
   b) Lateral Bending
   c) Axial Rotation
4. IAR's located for motion segment in the traditional planes.
   a) Sagittal: *(y, z)* plane
   b) Coronal: *(y, x)* plane
   c) Horizontal: *(x, z)* plane
5. HAM's located throughout the range for each motion segment in the traditional planes.
6. Analysis and comparison of the regional variations for all the above.
   a) Cervical
   b) Thoracic
   c) Lumbar
7. Analysis of the functions of the various anatomic elements in spinal kinematics.
   a) What are the roles played by the various anatomic elements in determining 1–6?
   b) What happens to 1–6, if any particular combination of anatomic elements are destroyed or unable to function?

CURRENT KNOWLEDGE OF SPINAL KINEMATICS

This section of the presentation seeks to report on the major aspects of contemporary spinal kinematics. For a detailed review of the literature and an exhaustive bibliography, the reader is referred to other references.[3,4,5,6]

Range of Motion

Few studies have been carried out to carefully analyze, quantitate and report translation ranges in spinal kinematics. The information was generated in several theses done in Professor Hirsch's laboratories in Sweden.[4,5,6] However, in the reporting of these works, most of the attention was focused on the rotary aspects of the motion. Values for translation in all planes, especially in the sagittal (*y*, *z*) plane are of practical importance in

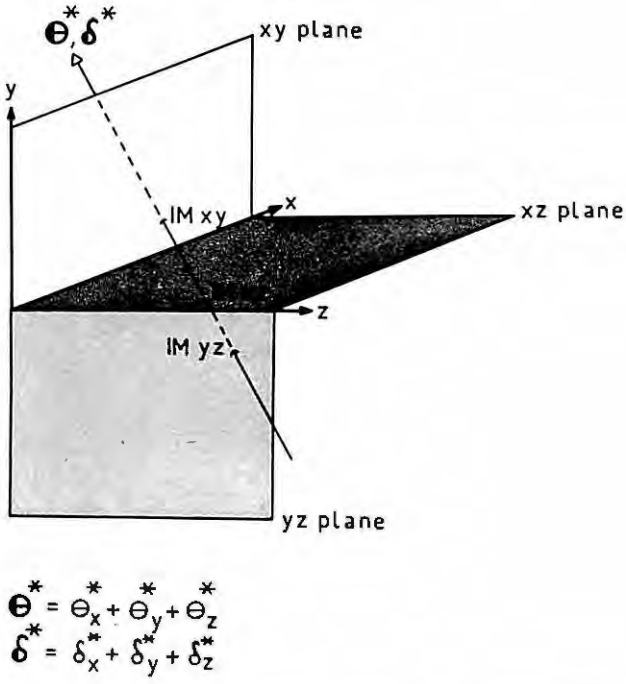


Fig. 6. Helical axis of motion. The screw motion is a superposition of rotation and translation about and along the same axis. This axis has the same direction as the results of the three rotation components about the *x*, *y* and *z* axes. Vector quantities θ^* for rotation and δ for translation lie on this axis. The above described axis is referred to as the helical axis of motion—*HAM*. (Note this diagram has not used the recommended orientation of the orthogonal coordinate system.)

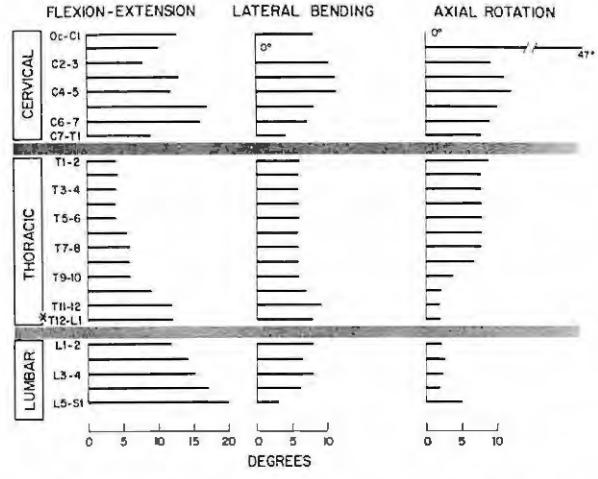


Fig. 7. This is a composite of rotation in the traditional modalities and the traditional planes of movement for the different regions of the spine. See text for explanation of how these figures were derived.

clinical evaluations of traumatized spines. There is information in the radiological and orthopedic literature that offers some guidelines about the upper limits of certain translatory displacements. However, we are not aware of studies that report the ranges in all the traditional planes.

To follow are a graph and a table which summarize the current information on the rotary ranges and representative figures for the human spine. These figures represent the best opinion of the authors based on a review of the literature[3,4,5,6,7,8] and their own analysis. The "representative values" are neither true mediums nor averages but are derived from a weighing which includes a consideration of the experiments that generated them as well as their "fit" with our overall knowledge of the subject. The reader is reminded that in the literature these figures encompass a broad range. This is due to large differences in experimental techniques as well as sizable individual biological variation. For spinal kinematics, instead of a "bell-shaped curve" there is a "cymbal-shaped curve." The figures are submitted with these qualifications and the belief that they represent a pattern characteristic of most human spines. See figure 7 and table II.

TABLE II

Estimated Range and Representative Degrees of Rotation Based on Review of Literature and Authors' Analysis

	Flexion-extension, *x*-axis rotation		Lateral bending, *z*-axis rotation		Axial rotation, *y*-axis rotation	
	Compiled range	Representative angle	Compiled range	Representative angle	Compiled range	Representative angle
Occiput–C_1	4–33	13	4–14	8	0	0
C_1–C_2	2–21	10	0	0	22–58	47
C_2–C_3	5–23	8	11–20	10	6–28	9
C_3–C_4	7–38	13	9–15	11	10–28	11
C_4–C_5	8–39	12	0–16	11	10–26	12
C_5–C_6	4–34	17	0–16	8	8–34	10
C_6–C_7	1–29	16	0–17	7	6–15	9
C_7–T_1	4–17	9	0–17	4	5–13	8
T_1–T_2	3–5	4	6	6	14	9
T_2–T_3	3–5	4	5–7	6	4–12	8
T_3–T_4	2–5	4	3–7	6	5–11	8
T_4–T_5	2–5	4	5–6	6	4–11	8
T_5–T_6	3–5	4	5–6	6	5–11	8
T_6–T_7	2–7	5	6	6	4–11	8
T_7–T_8	3–8	6	3–8	6	4–11	8
T_8–T_9	3–8	6	4–7	6	6–7	7
T_9–T_{10}	3–8	6	4–7	6	3–5	4
T_{10}–T_{11}	4–14	9	3–10	7	2–3	2
T_{11}–T_{12}	6–20	12	4–13	9	2–3	2
T_{12}–L_1	6–20	12	5–10	8	2–3	2
L_1–L_2	9–16	12	3–8	6	< 1–3	2
L_2–L_3	11–18	14	3–9	6	< 1–3	2
L_3–L_4	12–18	15	5–10	8	< 1–3	2
L_4–L_5	14–21	17	5–7	6	< 1–3	2
L_5–S_1	18–22	20	2–3	3	< 1–3	5

Coupling Characteristics and Their Ratios

The coupling patterns of C_1–C_2 have been described by Werne[3] and others.[8,9] The pattern is one of a coupling of lateral bending with axial rotation. This pattern was well described by Lysell[5] in the lower cervical spine where he calculated the ratios of coupling for all of the motion segments below C_1. The ratio of coupling is the proportionality between the amount of one motion and the amount of its associated motion. In this case the ratio of the rotations about the *z*- and *y*-axes. Details are available in Lysell's thesis. The directions of this coupling will be described here. The relationship is such that if one laterally bends one's head to the left, the axial rotation in the cervical spine is such that the spinous processes tend to go to the right. On bending the head to the right the spinous processes would of course go to the left. In the coordinate system the description is as follows: A positive *z*-axis rotation is coupled with a negative *y*-axis rotation and vice versa.

In the thoracic spine the coupling is the same as in the cervical spine but not quite as strong.[6] That is, each degree of lateral bending gets somewhat less axial rotation than it did in the cervical spine. In the middle and lower portion of the thoracic spine this same pattern still exists. However, in these areas, it is neither as marked nor as consistently present. Moreover, in these regions the direction of the coupling in some motion segments was actually reversed in direction. That is, a negative *z*-axis rotation was associated with a negative *y*-axis rotation. This observation led to speculation about some relevance to idiopathic scoliosis.[10] Figure 8 demonstrates the thoracic spine coupling and its possible relation to scoliosis.

We are not aware of data on the coupling pattern that exist in the lumbar spine. Studies to provide this information are now in progress in the Engineering Laboratory for Musculoskeletal Diseases Section of Orthopaedic Surgery, Yale University School of Medicine.

There is another salient coupling pattern that exists in all three regions of the spine. This is the coupling of *z*-axis translation with *x*-axis rotation during flexion and extension. (See fig. 4.)

Patterns of Motion in the Traditional Physiological Ranges

Fielding has carried out cineroentgenographic studies of motion in the cervical spine. This technique is the most dramatic and effective way to get a realistic feel for motion patterns, which can be difficult to vividly describe. The technique developed by Lysell for kinematic studies of the cervical spine included an effective method and manner of presentation of patterns of motion. Detailed descriptions are included in his thesis. The method was adopted and employed to analyze patterns of motion in the thoracic spine.[6] Figure 9 shows an example of thoracic spine motion patterns demonstrated through this method. The

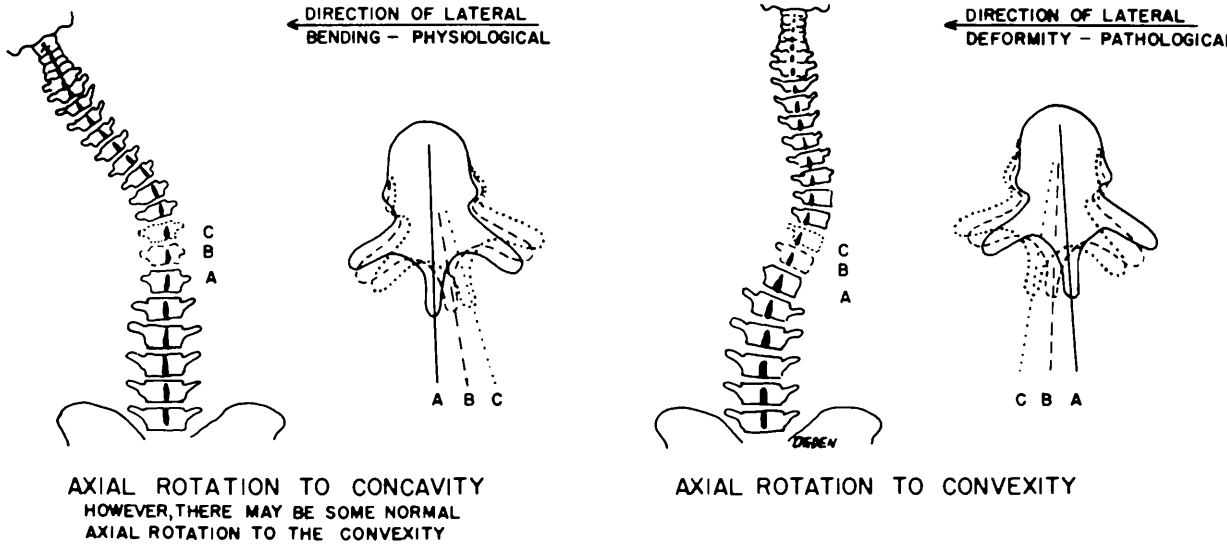


Fig. 8. Diagrammatic representation of the axial rotation coupled with physiological lateral bending is shown on the left. A comparative diagram of the same characteristics in the diseased scoliotic lateral curve is shown on the right.

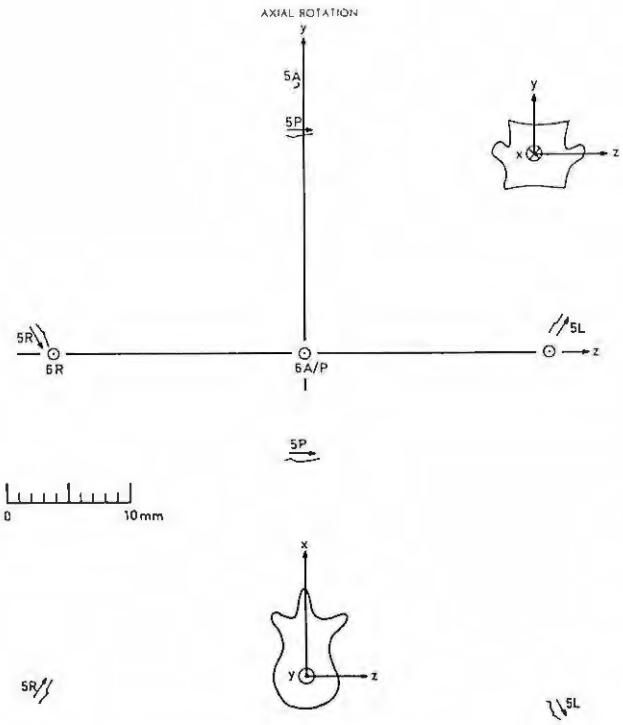


Fig. 9. This is a diagrammatic representation of the pattern of motion in a representative thoracic vertebra (T_5). Points $5R$ and $5L$ are located to the right and left respectively. Points $5A$ and $5P$ represent points in the midsagittal plane anteriorly and posteriorly.

lines show the movement of certain points of the vertebrae in space. The upper figure shows the reference subadjacent vertebra T_6 with the movement of T_5 in axial rotation as seen from the front in the coronal plane. The lower figure shows that same motion of T_5 in the horizontal plane as seen from above. Note that this coordinate system in the figure is orientated differently than the one suggested in figure 1. These points do not represent the centroid, but they serve to depict the patterns more readily as they assist in anatomic orientation. These diagrams show the previously described coupling pattern of the upper thoracic and the cervical spine quite clearly. They also depict paths of motion that are not completely smooth. However, there is wide variation among motion segments from different individual subjects as regards the smoothness or jaggedness of the paths. This is an example of one motion segment of a normal spine. Presumably a motion segment with degenerative changes will show a more jagged pattern of motion. Finally, it should be pointed out that point 5A which is located in the anterior inferior midportion of the lower

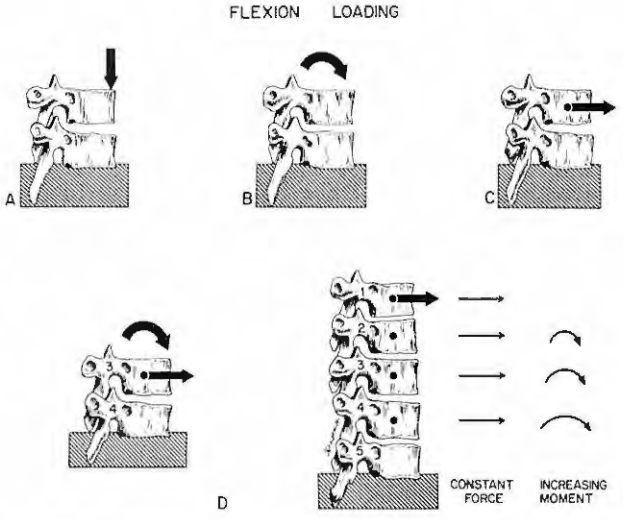


Fig. 10. Demonstrates diagrammatically various methods of loading employed in some experimental studies to determine the instantaneous axis of rotation during flexion.

rim of the vertebra moves very little. This will become important when IAR's are discussed.

Current Considerations Regarding IAR

The instantaneous axis of rotation is a unique characteristic of plane motion. It has theoretical as well as clinical implications. When combined with the angle of rotation it gives a complete description of the plane motion.

A given IAR depends upon the structure as well as to the type of loading. In other words, it is not sufficient to say that, for example, vertebra T_3 has its IAR, with respect to its adjacent fellow, located 3 millimeters anteriorly and 23 millimeters caudal to the anatomic center of its body. In addition, we must also specify the type of loading, which in itself is an ambiguous term. Let us take, for example, flexion loading. Although we all understand what we mean by flexion, i.e. forward bending, when it comes to simulating this loading in cadaver experiments, different researchers use different loadings. Figure 10 shows some versions of flexion loading. A compressive force anteriorly placed, shown in figure 10(A), was used by Rolander[4] when he studied the lumbar spine and White[6] who studied the thoracic spine. Flexion can also be created by applying a moment about a horizontal axis, see figure 10(B), as was done by Markolf[11] or by applying a horizontal force to the center of the body directed forward, Panjabi, Brand, and White[12] as shown in figure 10(C).

We may also take multiple segments of spines and fix its lowest vertebra and apply a transverse

load to the top vertebra. In this flexion loading we have a situation where all intermediate vertebrae are subjected to varying amounts of loads. (See fig. 10D.) This load type was used by Lysell[5] and White[6] in the three dimensional kinematic studies of the spine. The top vertebra is loaded through a pure transverse force, just as in figure 10(C), while all the intermediate vertebras are loaded with a combination of the transverse force which is the same at all levels, and the bending moment which is increasing in magnitude in a cephalocaudal direction. As the IAR is a function of the load applied, it is clear that for the loading situation depicted in 10(D) the IAR calculated for the various vertebras are going to differ as a result of the different combinations of force and moment at different levels. The above discussion applies equally to extension and lateral bending although axial rotation has been treated quite consistently in the literature. This implies that one has to be careful in comparing and interpreting the results of different workers in the field.

In the composite, figure 11, we have attempted to present the collective knowledge available on the IAR's in the three regions of the spine involved in the three traditional motions. There is, to our knowledge, only one study which gives indications of the location of IAR's in the cervical region.[5] The locations were not quantitatively determined but are based on judgment from observations of the patterns of motion (Fig. 11). The thoracic spine has been studied by White.[6] The results have been averaged for the different levels and are presented in the middle of figure 11. Data on the IAR's in the lumbar region is included in the work of Rolander[4] and Cossette et al.[13] Their findings are summarized in the lower portion of figure 11.

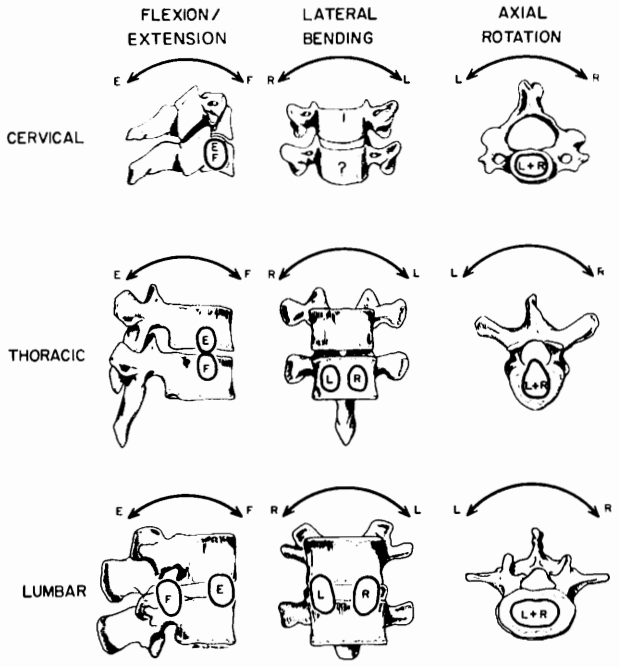


Fig. 11. Shows diagrammatically the approximate locations of instantaneous axis of rotation in the three regions of the spine undergoing rotation in the three traditional planes. *E* – shows approximate location of IAR's in extending from neutral position. *F* – shows them in flexion from neutral position. *L* – shows them in left lateral bending or left axial rotation, and *R* – shows them in right lateral bending or right axial rotation.

Current Status of Helical Axis of Motion

This valuable concept has to our knowledge been used in only one instance in spinal kinematics. In this instance, examples of thoracic spine motion were described with the HAM primarily to introduce the use of the concept to depict spinal kinematics.[6]

Analysis and Comparison of Regional Variations

Translation: The translation data have not been systematically presented. It is available in only a few of the numerous considerations. In the sagittal plane the *z*-axis translation occurs in decreasing magnitude in comparing the atlanto-axial joint, the lower cervical spine and the thoracic spine. Axial or *y*-axis translation occurs in decreasing order in the cervical, thoracic, and lumbar spine. This is based on impressions from the literature and the author's experience, not on specific controlled experiments. There are not enough data on *x*-axis translation for even an impression.

Rotation: Figure 7 is designed to point up regional variations in rotation in the traditional physiological motions.

There is no axial (*y*-axis) rotation between occiput and C_1. There is no lateral bending between C_1 and C_2 (*z*-axis rotation). About 50 percent of head-and-neck axial rotation occurs at the occipito-atlanto-axial complex. The remaining one-half occurs between C_2 and T_1. Most of the movement considering the three modalities, flexion extension, lateral bending, and axial rotation, occurs in the cervical spine.[3,9]

In the thoracic spine, the amount of flexion extension at each motion segment increases in the cephalocaudad direction, while the axial rotation decreases. The lateral bending is about the same

throughout. Though the intersegmental motion in the thoracic spine is relatively modest in most modalities, its total capacity for movement is formidable. The upper thoracic spine tends to behave more like the cervical spine and the lower thoracic spine tends to behave more like the lumbar spine.[6]

In the lumbar spine, flexion extension is the major activity with little axial rotation. There is more axial rotation and probably more flexion at the L_5–S_1 joint than in the other lumbar segments. There may be relatively less lateral bending at that level. Flexion extension is generous here as it is in the cervical spine. There is less lateral bending and axial rotation in the cervical spine and less axial rotation than in the upper thoracic spine.[4]

Analysis of the Functions of the Various Anatomic Elements in Spinal Kinematics

Studies have been carried out on mechanical properties of some of the specific anatomic elements, including the anterior and posterior longitudinal ligaments,[14] the annulus fibrosus,[15,16,17] the yellow ligaments,[18] and the interspinous ligaments.[19] Not much attention, however, has been paid to the role of these elements in determining the characteristic functions of the motion segment.

One approach is to compare the kinematics of a given motion segment under controlled conditions before and after the elimination of anatomic structures. A biomechanical analysis of the translation and rotation in the cervical spines (tested in flexion and extension) as a function of sequential ligament transection including facet ablation has been carried out. These studies showed subtle changes in kinematics up to a point after which dramatic changes were evidenced.[20] It has been shown that removal of posterior elements in thoracic spine motion segments significantly alters the kinematics in extension and axial rotation.[21]

This is an area of spinal kinematics in which a good deal of valuable basic and clinical information can be generated.

DISCUSSION AND CONCLUSION

The preceding is an overview of current spinal kinematics. Large amounts of data have been omitted as have some relevant topics. The latter includes the effects of age, occupation, and disease on spinal kinematics. The important relationship of spinal kinematics to stiffness and damping coefficients and mathematical modeling has not been included.

The outline in table I has been developed in an attempt to focus on what is known and offer some suggestions about future topics for investigation. It is apparent that there is a good deal of research remaining in order to fully describe the kinematics of the human spine.

There are, as with any research, numerous problems involved. The kinematics of interest is that of the living spine. The experimental techniques for precise no-risk in vivo measurement in the human are yet to be developed. The physiological muscle forces have not been simulated. The characteristics of the force vectors that cause in vivo physiological motion are not known. Studies are done to simulate vertebral motion, but it is not known whether the motion experimentally produced is the same as that which is physiologically produced in vivo. The vectors that should represent the existing physiological preloads are not known and at present we are not aware of published studies of kinematics that take them into consideration.

It is possible to fully describe the normal kinematics of the human spine. A good deal of that description is presently available. There is still a considerable amount to be carried out. However, given the available current knowledge, and research techniques, it is no longer necessary to speculate and deliberate about spinal kinematics or alignment. It is possible with defined limits of validity to observe and measure the spatial relationships between vertebras, and in most cases, to determine whether or not those relationships are normal.

REFERENCES

1. Lovett, R. W.: The Mechanism of the Normal Spine and its Relation to Scoliosis. *Boston Med. & Surg. J.*, 153:349, 1905.
2. White, A. A., Panjabi, M. M., and Brand, R. A.: A System for Defining Position and Motion of Human Body Parts. *Journal of the International Federation for Medical and Biological Engineering*, In Press.
3. Werne, S.: Studies in Spontaneous Atlas Dislocation. *Acta Orthop. Scand.*, Supp. No. 23, 1957.
4. Rolander, S. D.: Motion of the Lumbar Spine With Special Reference to Stabilizing Effect of Posterior Fusion (Thesis). *Acta Orthop. Scand.*, Supp. No. 90, 1966.
5. Lysell, E.: Motion in the Cervical Spine (Thesis). *Acta Orthop. Scand.*, Supp. No. 123, 1961.

6. White, A. A.: Analysis of the Mechanics of the Thoracic Spine in Man. An experimental study of autopsy specimens. (Thesis.) *Acta Orthop. Scand.*, Supp. No. 127, 1969.

7. Fielding, J. W.: Normal and Abnormal Motion of the Cervical Spine from C_2 to C_7, Cineroentgenography. *J. of Bone and Joint Surg.*, 46-A, p. 278, 1959.

8. Hohl, M., and Baker, H. R.: The Atlanto-Axial Joint Roentgenographic and Anatomic Study of Normal and Abnormal Motion. *J. of Bone and Joint Surg.*, 46-A, pp. 1739-1752, 1964.

9. Shiparo, R., Youngberg, A. S., and Rothman, S. L. G.: The Differential Diagnosis of Traumatic Lesions of the Occipito-Atlanto-Axial Segment, *Radiolic Clinics of North America*, Vol. XI, pp. 505-526, 1973.

10. White, A. A.: Kinematics of the Normal Spine as Related to Scoliosis. *J. of Biomechanics*, 4:405, 1971.

11. Markolf, K. L.: Stiffness and Damping Characteristics of the Thoracic-Lumbar Spine. *Proc. of Workshop on Bioengineering Approaches to the Problems of the Spine*. NIH, September 1970.

12. Panjabi, M. M., Brand, R. A., and White, A. A.: Three-Dimensional Load Displacement Curves of the Human Thoracic Spine. *J. of Biomechanics*, In Press.

13. Cosette, J. W., Farfan, H. F., Robertson, G. H., and Wells, R. V.: The Instantaneous Center of Rotation of the Third Lumbar Intervertebral Joint. *J. of Biomechanics*, Vol. 4, pp. 149-153, 1971.

14. Tkaczuk, H.: Tensile Properties of Human Lumbar Longitudinal Ligaments. *Acta Orthop. Scand.*, Supp. No. 115, 1968.

15. Galante, J. D.: Tensile Properties of the Human Lumbar Annulus Fibrosus. Thesis. *Acta Orthop. Scand.*, Supp. No. 100, 1967.

16. Horton, W. G.: Further Observations on the Elastic Mechanism of the Intervertebral Disc. *J. of Bone and Joint Surgery*, 40-B, p. 552, 1958.

17. Virgin Experimental Investigations Into the Physical Properties of the Intervertebral Disc. *J. of Bone and Joint Surgery*, 33-B, p. 607, 1951.

18. Nachemson, A., and Evans, J.: Some Mechanical Properties of the Third Lumbar Interlaminar Ligament. *J. of Biomechanics*, Vol. 1, p. 211, 1968.

19. Walters, R., and Morris, J.: An In Vivo Study of Normal and Scoliotic Interspinous Ligaments. *J. of Biomechanics*, Vol. 6, p. 343, 1973.

20. White, A. A., Johnson, R. M., Panjabi, M. M., and Southwick, W. O.: Biomechanical Analysis of Clinical Stability in the Cervical Spine. *Clin. Orthop.*, In Press.

21. White, A. A., and Hirsch, C.: The Significance of the Vertebral Posterior Elements in the Mechanics of the Thoracic Spine. *Clin. Orthop.*, Vol. 81, November-December, 1971.

Biomechanical Aspects of Subluxation

C. H. SUH
Professor and Chairman
Department of Engineering Design
and Economic Evaluation
University of Colorado

1. INTRODUCTION

Next to the word "chiropractic," the term "subluxation," or "spinal subluxation" is perhaps the most important and most frequently used word employed by chiropractors. Some discussion on this term is therefore in order. The definition of "subluxation" varies, depending on whether it is viewed from a chiropractic or medical point of view. For example, Stedman's Medical Dictionary[1] simply defines it as: "Semiluxation; an incomplete luxation or dislocation; though a relationship is altered, contact between joint surfaces remains."

The major difference between the medical and chiropractic viewpoint is that chiropractic includes "neurophysiological disturbances" in the definition of subluxation. This implies that "subluxation" has a living character which includes both biomechanical and neurophysiological abnormalities. Therefore, any conclusion regarding subluxation that is based solely on the study of cadavers is not acceptable.

This paper will concentrate on the biomechanical aspects of subluxation. It will summarize what is known and unknown, indicate necessary areas of research and suggest methods to carry out the research. Research at the University of Colorado pertaining to this field has focused on the following two major areas in the last several years:

1. Precision X-ray method for the visualization of biomechanical aspects of subluxation
2. Computer model of the spine for simulated study of spinal subluxation.

2. PRECISION X-RAY METHOD FOR THE VISUALIZATION OF BIOMECHANICAL ASPECTS OF SUBLUXATION[2,3]

2-1. A Review of X-ray Methods in Biomechanics

Needless to note, the X-ray has become an invaluable means of examination in health care. The graphical output of anatomical or physiological data of the human body produced by X-rays generally serve as "true pictures" of the X-rayed object. However, due to the large amount of distortion in the X-ray image generated by its inherent central projection characteristics and other geometrical errors associated with the X-ray equipment, an accurate measurement from the X-ray image of fundamental biomechanical data, i.e. distance, angle, and relative position, is impossible unless proper scientific techniques are developed and applied.

Techniques known categorically as "photogrammetry," developed mainly for geographical surveys and map production, have been recently extended to X-ray photography. Numerous attempts and contributions in this area appear in various literature.[4-8] In spite of these activities, the techniques are generally not sufficiently formulated for application by practitioners in the health care profession. Recent literature in medical radiology rarely includes use of three-dimensional correction techniques.[9] In 1970, B. H. Dawson and mechanical engineers in Lancashire, England, published an outline of a new method which uses the computer to study the relative positions of the brain and the axes of the operating instrument in stereotactic surgery.[10]

The spine, because of its importance in far-reaching neurological and vascular disturbances, has been of particular concern in the development of accurate X-ray analysis. A review of developments made in the past indicates the seriousness of the efforts in improving accuracy. A special process and apparatus developed by Dr. T. Vladeff for spinal X-ray was patented in 1942 (U.S. Patent No. 2,630,536); another apparatus developed for spinal X-ray by Dr. E. A. Fox was patented in 1956 (U.S. Patent No. 2,774,884). Still another device, known as a "Protractorscope," includes a method for upper cervical X-ray analysis.[11] This apparatus, developed by Dr. J. Kuhn, was patented in 1960 (U.S. Patent No. 2,942,347).

This paper presents a rigorous fundamental analytical method of precision analysis of spinal X-rays, but the method is also applicable to other parts of the body. It is based on the reconstruction of three-dimensional geometry from X-ray films. The analytical method is developed in such a way as to utilize the efficiency and accuracy of digital computers. To extend the scope to practical use, the method is developed in such a way that a typical single X-ray machine can be used.

2-2. Development of Reconstruction of X-ray Geometry

For practical reasons, it is necessary to reconstruct the geometry from the information appearing on the X-rays themselves without the need to record the relative positions of the focus points, the object, and the image planes. To solve this problem, it was necessary to include a reference frame that is X-rayed along with the object.

For example, in cervical X-ray analysis a "Helmet" type reference frame illustrated in figure 1 can be used. It is made of plastic plates with lead wires imbedded in them. When a cervical X-ray is taken with this "Helmet" in place, the lead wires of the "Helmet" are clearly projected and appear around the cervical vertebrae on the X-ray film. Each of these lines on the X-ray film will, in general, appear to be longer than the premeasured true lengths of the lead wires in the "Helmet."

For the three-dimensional analysis, at least two different views are required. In general, orthogonal projections are preferred because of the clear identifications of the images and better intersections of X-ray projection lines in the reconstructed geometry which give higher accuracy.

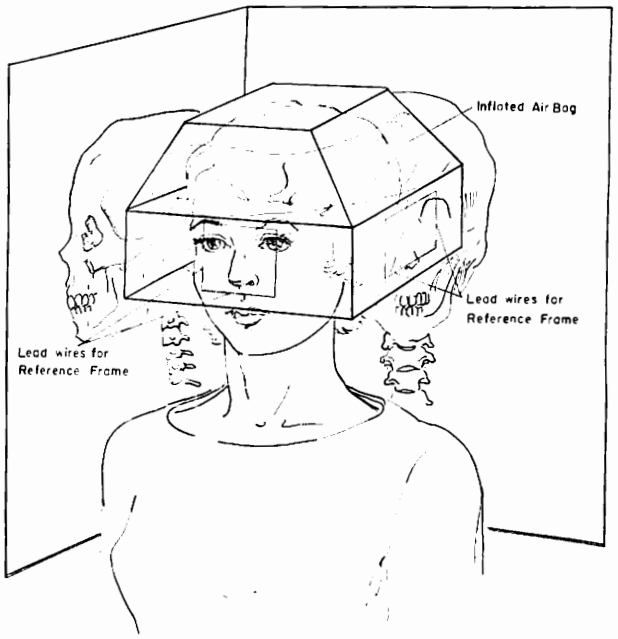


Fig. 1. The helmet for computer aided X-ray analysis.

Figure 2 illustrates the reconstruction and notations used in the computation. The rectangular reference frame has dimensions A, B, C, A_1, B_1, B_2, C_1, ΔX, ΔY_1, ΔY_2, and ΔZ which are constant

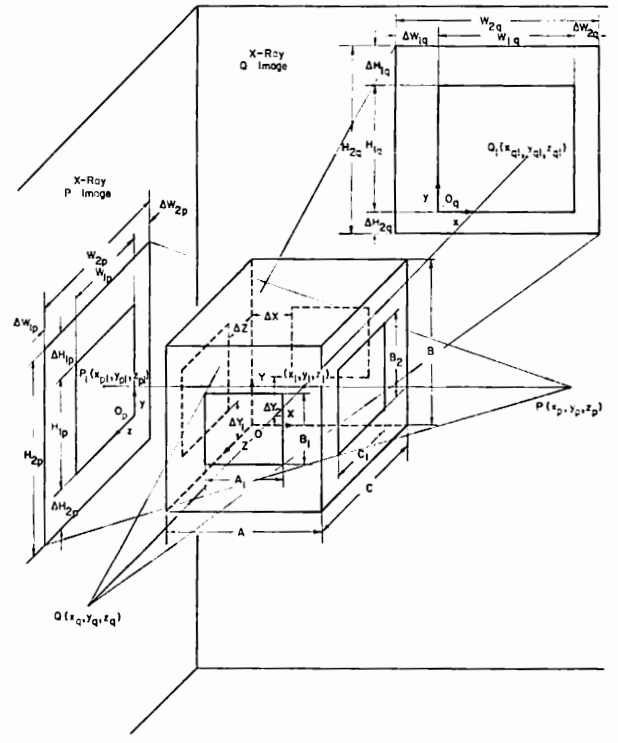


Fig. 2. Geometry and notations for localized computer-aided X-ray analysis.

known lengths measured with extreme accuracy. Two of the opposite side frames are identical. For the sake of simplicity in illustrating the basic method involved in the reconstruction, assume that P and Q image planes are orthogonal and parallel to the corresponding faces BC and AB of the reference frame.

Fixed coordinate axes (X, Y, Z) are established as reference axes in the geometrical system. The "right hand rule" axes (X, Y, Z) are attached to the reference frame in such a way that any point in the reference frame is measured in the positive direction of X, Y, and Z as shown in figure 2. The reconstruction then requires the location of the two focus points P and Q and the image P_1 and Q_1 with respect to the fixed coordinate axes (X, Y, Z).

Since most biomechanical measurements such as distance, angle, and relative position can be calculated by using the coordinates of a series of points in one fixed coordinate system, the process of using only coordinates of points (rather than attempting to measure angles or other geometric features) is sufficient.

To illustrate the procedure, the problem may be stated as follows:

Problem: Let it be required to locate the point (x_1, y_1, z_1) with respect to the localized fixed (X, Y, Z) coordinate axes using the two X-rays; P image and Q image. Besides the two X-ray films, the only known parameters are the reference lengths A, B, C, A_1, B_1, B_2, C_1, ΔX, ΔY_1, ΔY_2, and ΔZ.

Solution of this problem is presented in detail in references 2–3 with all the formula derivations and a numerical example. It consists of eight steps in a sequential method developed with analytical spatial geometry and algebraic manipulations. It gives four different sets of three linear equations which will give four different sets of solutions to x_1, y_1, and z_1 instead of one set.

At this point it was apparent that further analysis was required in the selection.

2–3. The Error Analysis and the Selection Criterion

Let us assume one of the sets of the linear equations as

$$(A)\quad \begin{cases} y_1 = m_1 x_1 + b_1 & \text{(a)} \\ z_1 = m_2 x_1 + b_2 & \text{(b)} \\ y_1 = m_3 x_1 + b_3 & \text{(c)} \end{cases}$$

Obviously, solving this set of three linear equations can be done by solving first for x_1 and y_1 from linear equations (a) and (c) then substituting x_1 in equation (b) to solve for z_1.

For a graphical analysis of this simple algorithm, in figure 3 we see the front (F) and top (T) view of the reference frame with P and Q image X-rays.

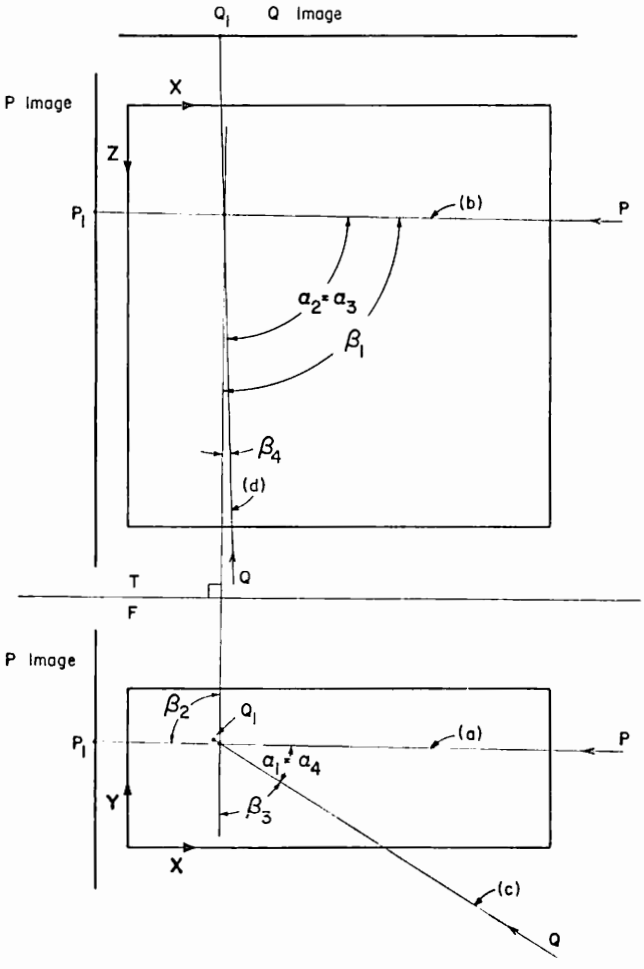


Fig. 3. Intersecting rays.

The first equation of (A) is the x-y view of the ray (a) which comes from the focus point P and similarly the third equation of (A) is the x-y view of the ray (c) which comes from the focus point Q. Solving these two linear equations simul-

597-036 O - 76 - 8

taneously simply means that we are finding the intersection of these two lines. We see this intersection in the F view; then we find x_1 and y_1 by measuring the x-y coordinates in the F view.

The second step of the algorithm is to substitute one of the known values, x_1, into the second equation to solve for z_1.

This process begins, again graphically, by extending a perpendicular line from the intersection point in the front view into the top view. Then, let this line intersect the line (b) that represents the second equation of (A) which shows the x-z view of the ray coming from the focus point P.

Let the first angle of the intersection of the line (a) and (c) be α_1, and the second angle of the intersection of the perpendicular line and (b) be β_1. It is important to notice that a key for the error analysis can be found in these two angles α_1 and β_1.

The "good intersection" which gives an accurate position of the intersection point on a plane is when the angle of the intersection is close to 90°. Figure 4 illustrates a "good intersection" and a "bad intersection."

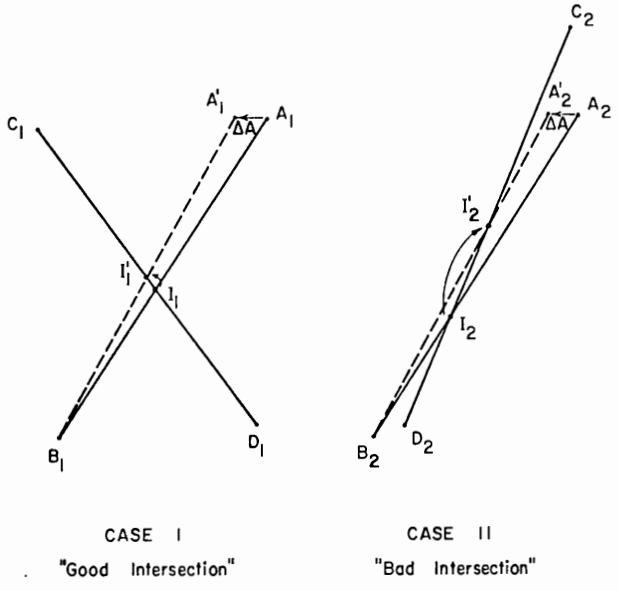


Fig. 4. Good and bad intersections.

Suppose the point A_1 and point A_2, the ends of the lines, have some error, ΔA, in position. The effect of changing the position of the intersection point I due to this error is much more serious in the "bad intersection" case than in the "good intersection" case because it jumps far more from the true intersection point as illustrated in figure 4.

This means, then, if one has a choice in selecting an intersection point from these two cases and if all the end points $A_1, B_1, \ldots, C_2, D_2$ have the same degree of possible error, then one should select case I which gives a more accurate point of intersection.

In selecting one solution out of four, one can use this error analysis as a criterion. To do this one computes α and β angles for each set of four solutions. That solution which gives these angles more nearly 90° than the others is selected. Defining the angles α and β as the first and second intersection angles respectively, one must notice that the angles α and β of a particular solution could be found in the F or T view, but not both in the same view.

2-4. Computer Implementation

The procedure, including error detection and discriminative measure, is a stepwise sequence and thereby renders itself to formulation with a simple computer program. Various computer programs written in BASIC and FORTRAN IV for conversational time-sharing terminals and conventional batch processing, respectively, are available together with appropriate user manuals. The simplicity of the program, as well as the recently improved time-sharing computing network, should make this system practical for any physician or researcher with the mere addition of a single remote console and common telephone.

2-5. Discussion and Further Research Needed

In securing the highest accuracy from the X-ray analysis, the following four points should be added:

(1) Grid size of the reference frame

In general, it may be preferred to reduce the lead wired grid in the frame because of the obvious economy involved in using a smaller X-ray and the better X-ray images of the lead wires themselves due to the wires being closer to the local object focused. However, a clear disadvantage must be noted which will limit the reduction. That is, the shortening of the reference lead wire lengths will naturally make shorter images of these wires on the X-ray, which will in turn reduce the accuracy involved in the entire numerical procedure. Thus, a reasonable optimum size of the wire grid is necessary.

(2) The point identification

Since an X-ray is actually a shadow of the object, there always exists considerable inherent human error in locating the same geometrical points on the bones from different X-rays. This could be largely overcome by the following efforts:

(a) Through years of experience, physicians and radiologists have acquired accurate anatomical insight which should increase the accuracy of locating corresponding points on the X-rays.

(b) Coordinate digitizers are commercially available which can more accurately determine point coordinates on the X-ray film. They also store the data automatically, eliminating the need for hand recording.

(c) Most of the X-ray analysis will involve positions and displacements. The rigid body condition of each bone should be used in the kinematic analysis to correct and/or improve the data points measured and stored.

(3) The structural errors

The proposed method of X-ray analysis is based on correcting the distortion of central projection. However, the X-ray focus is not truly a geometrical point and furthermore the negative X-ray image planes are not truly geometrical planes due to shrinkage and lack of flatness. Another structural error due to opposite sides of the reference frame not being parallel also exists.

In a series of experiments carried out with prototype reference frames for cervical spinal analysis such as shown in figure 5, it was found that effects due to these structural errors are much less serious than the two previously discussed factors. The total of these errors can be estimated by a calibration procedure using a precisely known object and placing it in the reference frame.

Our experience in using this X-ray method in a realistic environment reveals that the problem which is oriented to the point identification is most

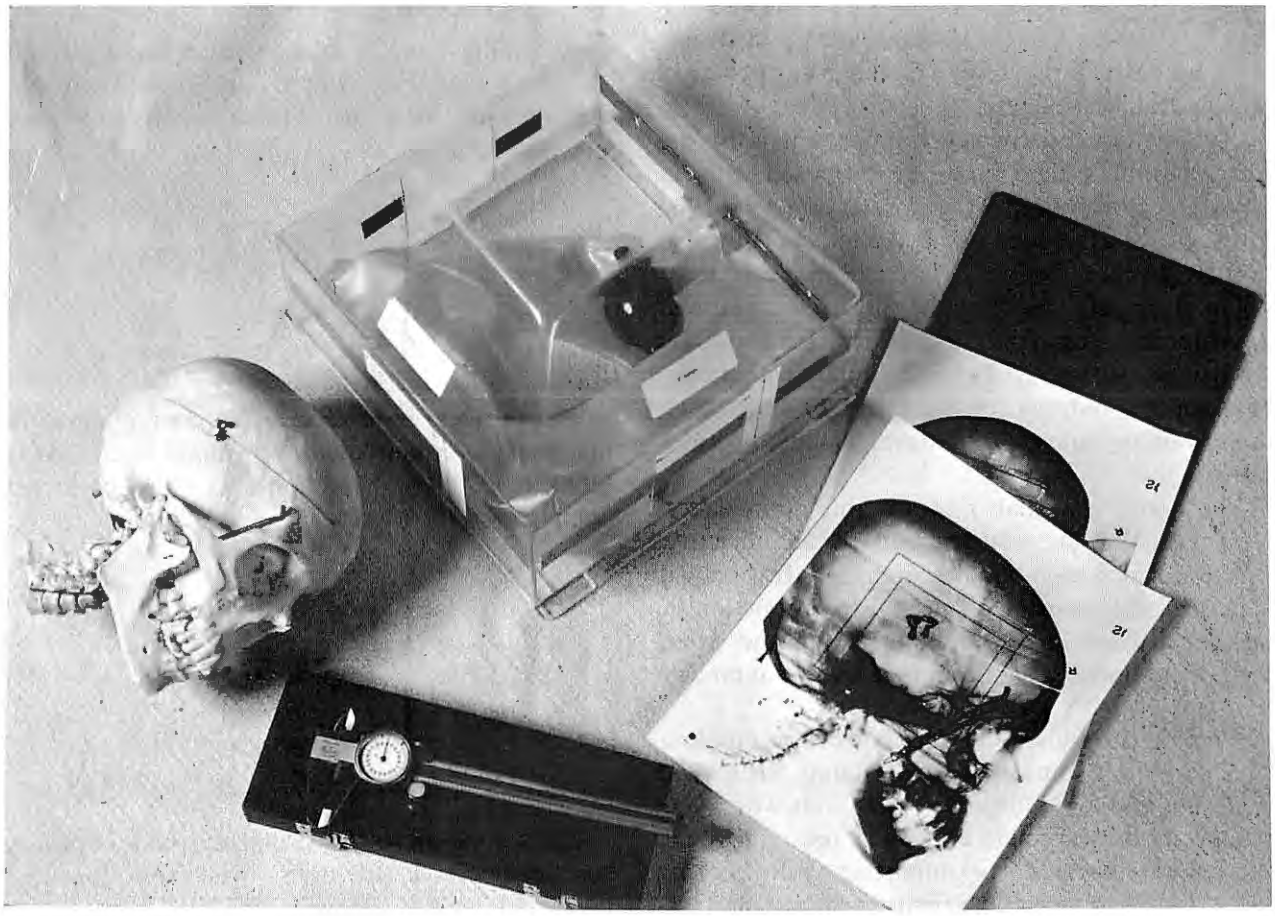


Fig. 5. The experimental "helmet" of reference frame for cervical X-ray analysis.

pressing and needs further research to obtain higher accuracy of this entire X-ray method.

Since a spinal subluxation is associated with the displacement of a vertebra, with respect to other vertebra, theories in kinematics of the rigid body displacement have been used in research since 1973 at the University of Colorado.[12, 13] The X-ray data of spatial coordinates are required for the displacement analysis but unlike other biomechanical analyses, such as distances between two points, angle between two lines, etc., displacement calculations require more accurate data. Therefore, it became necessary to correct the X-ray data before the displacement analysis.

By the definition of a rigid body, any displacement of a rigid body in three-dimensional space should not change its shape. In other words, the distance between any two points in a rigid body should remain constant. Therefore, when the coordinates of the same points on a rigid body are measured at different position and the distances between these points are calculated, they must be the same in any position. Since there is usually inherent measurement error involved, it is not necessarily true that the measured data will give the same lengths with accuracy.

Two methods to correct the data which use the properties of a rigid body are proposed with an objective function in [13] and also the problems involved in the numerical solutions are also discussed.

Unfortunately, most of the facts discussed and developed in the report are difficult to prove by analytical geometry. Instead, numerical checks by computer outputs for the many possible cases are given to support the development by cross checking.

The most essential part on the optimization problem is in the construction of an objective function which then will be minimized. It is known that the establishment of an objective function in analytical form is a difficult matter and often there is no universally systematic way to approach it.

We shall formulate the data correction problem as follows: Assume that three points on a rigid body are measured at the two positions with some amount of measurement error. The reason why the three-point problem is important will become clear from the kinematics. Figure 6 shows the symbols and coordinates for each point; these symbols are used throughout the report.

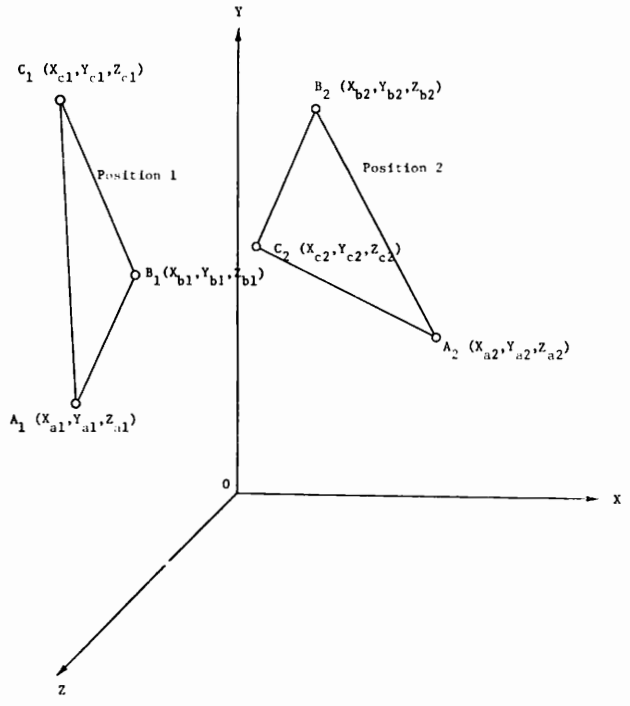


Fig. 6. Coordinates of rigid body in space.

Assuming there is some measurement error in A_1, B_1, C_1, A_2, B_2, and C_2, the calculated distances with these data (between these points) may not be equal, i.e.,

$$\begin{cases} \overline{A_1 B_1} \neq \overline{A_2 B_2} \\ \overline{A_1 C_1} \neq \overline{A_2 C_2} \\ \overline{B_1 C_1} \neq \overline{B_2 C_2} \end{cases} \tag{1}$$

The problem is to change the coordinates such that these constant-length equations hold; that is fulfill rigid body conditions. Let the corrected points be $A'_1, B'_1, C'_1, A'_2, B'_2$ and C'_2, then

$$\begin{cases} \overline{A'_1 B'_1} = \overline{A'_2 B'_2} \\ \overline{A'_1 C'_1} = \overline{A'_2 C'_2} \\ \overline{B'_1 C'_1} = \overline{B'_2 C'_2} \end{cases} \tag{1'}$$

For a solution to this problem, which obviously has innumerable solutions, we know that the corrected points depend on the selection of the objective function. Let us intuitively define the objective function as the sum of the corres-

ponding distances between the original data points (A_1, B_1, C_1, A_2, B_2, and C_2) and the corrected data points (A_1', B_1', C_1', A_2', B_2', C_2'), i.e.,

$$f(x'_{a1}, y'_{a1}, z'_{a1}, x'_{b1}, y'_{b1}, z'_{b1}, x'_{c1}, y'_{c1}, z'_{c1}, x'_{a2}, y'_{a2}, z'_{a2}, x'_{b2}, y'_{b2}, z'_{b2}, x'_{c2}, y'_{c2}, z'_{c2}) = \overline{A_1A'}_1 + \overline{B_1B'}_1 + \overline{C_1C'}_1 + \overline{A_2A'}_2 + \overline{B_2B'}_2 + \overline{C_2C'}_2$$

$$= \sqrt{(\Delta x_{a1})^2 + (\Delta y_{a1})^2 + (\Delta z_{a1})^2} + \sqrt{(\Delta x_{b1})^2 + (\Delta y_{b1})^2 + (\Delta z_{b1})^2} + \sqrt{(\Delta x_{c1})^2 + (\Delta y_{c1})^2 + (\Delta z_{c1})^2} + \sqrt{(\Delta x_{a2})^2 + (\Delta y_{a2})^2 + (\Delta z_{a2})^2} + \sqrt{(x_{b2})^2 + (y_{b2})^2 + (z_{b2})^2} + \sqrt{(\Delta x_{c2})^2 + (\Delta y_{c2})^2 + (\Delta z_{c2})^2} \quad (2)$$

where

$$\Delta x_{a1} = x_{a1} - x'_{a1}$$

$$\Delta y_{a1} = y_{a1} - y'_{a1}$$

$$\Delta z_{a1} = z_{a1} - z'_{a1}, \text{etc.}$$

Now the data correction problem can be stated as an optimization problem as follows: Minimize the objective function f of eq. (2) subject to the constant-length equations of eq. (1)'.

3. COMPUTER MODEL OF THE SPINE FOR SIMULATED STUDY OF SPINAL SUBLUXATION[14-20]

3-1. A Review of Spinal Models

Creating a model of the complicated biomechanics of the spinal column presents a difficult challenge. The osteoid processes of the spinal column exhibit awesome complexity and multiple irregularities in structure. Supportive tissues of the spine express characteristically nonlinear behavior. Even static and dynamic tolerances of the spinal column seem erratic under various measurement attempts. Nevertheless, a model of the spinal column is obligatory for the eventual comprehension of relationships between imposed mechanical force and resulting spinal displacements.

Existing biomechanical data for the spinal column are not very appropriate for modeling attempts. While existing studies may represent creative simulations of automotive or aeronautical eventualities, they do not lend themselves to realistic approaches to most chiropractic disorders. What are required are studies on small, quasistatic displacements of the spinal column, yet such studies are quite rare. It follows that the necessary biomechanical data for a comprehensive model of the spinal column are also rare or nonexistent. Precisely what measurements are required remains uncertain because only a comprehensive model will reveal the gaps in existing biomechanical information on the spine.

A model of the spinal column must accurately conform to mechanical constraints of geometry and force. In addition, such a model must provide some degree of anatomical reality. Recent advances in computer graphics make tenable the satisfaction of these requirements. By comparing the model with the results of a modified cineradiographic procedure used in continuous displacement analysis, it is possible to achieve an accurate, realistic model applicable to clinical as well as research chores.

The spine as a major structure and the column of the body has been a major subject of biomechanics research for many years. To study mechanical and structural behavior of the spinal system there have been numerous attempts to construct a model of the spine. In 1951, Dr. Fred W. Illi, a Doctor of Chiropractic in Geneva, Switzerland, published a book entitled, *The Vertebral Column*. He constructed a hardware model by placing an axis in the vertebral canal and assembling wedged discs connected by means of a wire. He used it to demonstrate among other things that the spinal cord constituted the axis of the torsional movements of the spine itself.

In 1957, S. Werne at the Department of Orthopedic Surgery, University of Lund, Sweden, published a book, *Studies in Spontaneous Atlas Dislocation*. In his work he described a conceptual biomechanical model of the spine's odontoid ligaments in an attempt to illustrate the role of the delicate alar ligaments in the spinal displacement. In 1969, H. P. Kopell, M.D., an orthopedic surgeon, described a spinal model in his book, *Help for Your Aching Back*. He used this model to explain an acute back derangement by illustrating faulty stabilizing systems and muscle spasms. In 1972, K. Markolf, Research Engineer at the

University of California, Berkeley, constructed another hardware model of the spine. This model, made by bending and twisting metal, was made also for those aching backs, as it can aid in the design of braces and other supports in order to immobilize the spine.

These physical and mechanical hardware models are useful in many cases, particularly to demonstrate the basic spinal mechanics to a certain extent, to laymen and researchers, if they are built with enough mobilities. However, modeling is obviously seriously limited in representing the complex spine accurately, mainly because it is practically an impossible task to find or manufacture real artificial materials to duplicate the mechanical properties of the tissues involved, such as spinal discs, ligaments, and muscles. At the same time this type of modeling does not utilize many of the recent scientific and engineering developments made in modeling techniques.

A contribution, perhaps the most important and relevant work to our particular approach, is due to the work of A. B. Schultz at the University of Illinois, at Chicago Circle.[21] Chiropractic problems, such as subluxation analysis were not his concern, but since 1970 he has utilized digital computers and engineering mechanics on spinal problems. Most of the work was closely associated with scoliosis and his attempt at solving spinal movement problems with highly nonlinear relations is still in its initial stages of development. The difficulty becomes obvious when one has to face many highly nonlinear equations to solve simultaneously and repeatedly for continuous motion simulations. At the University of Colorado a highly efficient computer program was developed to attack and overcome this difficulty. At the same time relevant chiropractic analyses were initiated with the development of the nonlinear equation solver.

3-2. Mathematical Modeling of the Spine

Beginning in 1970, Suh and others at the University of Colorado initiated spinal modeling to meet the needs of the biomechanic analysis. Since its inception, it has been based on digital computers for numerical computations and computer graphics output. The results of the developments were continuously presented at the Annual Biomechanics Conferences on the Spine in October 1970, October 1971, November 1972, November 1973, and December 1974. For detailed review on the preliminary research carried out with respect to the spinal model, readers are referred to literature given in references 14, 16, 17, 20.

In developing the three-dimensional computer model of the spine for the biomechanics study involved in chiropractic, the following characteristics and capabilities are required:

(1) The model should be basically elastostatic in nature to be useful at any particular position of equilibrium of the spine or spinal segment, whether the position is within normal or abnormal range.

(2) The model at the same time is to possess the capability of performing voluntary spinal movements for the study of motion patterns and to investigate various kinematic parameters involved in the displacements.

(3) The digital computer model should have the capability of storing the linear and nonlinear characteristics of discs, ligaments, muscles and other tissues involved. All these characteristics of experimental biomechanics data should continuously interact with the geometry involved in the simulated spinal motion.

(4) This model should also be developed simultaneously with computer graphic techniques so that the performing simulations are clearly, accurately, and continuously displayed graphically. Presently, at the University of Colorado most of the essential nonlinear computations involved are programed and executed with the use of the University's dual CDC 6400 system with CDC Computer Graphic system while new equipment such as Evans and Sutherland's Picture System are being proposed to be used mainly for the display of the outputs in graphical forms, taking advantage of the increased line-drawing capacity.

3-3. Computer Graphics of Spinal Biomechanics

Computer graphics, which couples intuitive graphical presentations with high-speed computing, is being utilized in various health care systems and

analyses. These are now major areas of research. Accelerating use of computer graphics is a result of the health crisis and the ever sophisticated techniques applied in diagnosis of diseases and disorders. Physicians are, in general, laymen in understanding computer techniques. Frequently only computer-generated graphics can provide "the picture" necessary for a decision in intricate health care problems.

From a structural point of view, the spine is the main column of the human body. Physiologically, it is the container of the spinal cord which, together with the brain, forms the central nervous system. A spinal subluxation is formed with abnormal displacement of one or more vertebra with respect to others. This causes various direct disturbances such as backache and also is known to lead to other serious diseases as well. Evaluation of these neurophysiological concomitants of mechanical displacements in the spinal nervous system demands accurate analysis of relative position and displacement of the living spinal system.

Recent developments in X-ray research have included various cineradiographic procedures for continuous displacement analysis by Fielding[22] and Howe.[23] Meanwhile, a more realistic and detailed computer simulation model has been developed for the biomechanical study of the spine. The need to animate the spine displacement arose in working with this simulation model.

At the Biomechanics Laboratory at the University of Colorado, Boulder, recent developments of spinal computer graphics are revealed in two major areas: The first deals with three-dimensional computer graphics of the vertebra, and the second with the computer graphics of spinal displacement.

a. Three-dimensional Computer Graphics of the Vertebra[15]

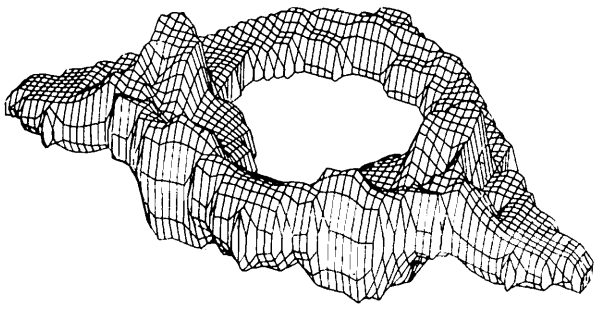


Fig. 7. The three-dimensional perspective view of the atlas (the first cervical vertebra).

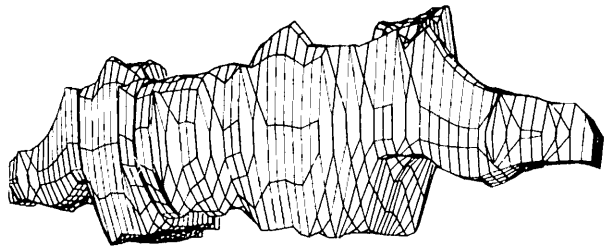


Fig. 8. The three-dimensional perspective view of the atlas (the first cervical vertebra). A different point of view.

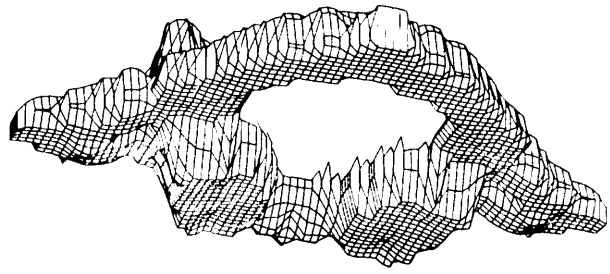


Fig. 9. The three-dimensional perspective view of the atlas. (the first cervical vertebra). Another different point of view.

Investigation of three-dimensional computer graphics for accurate perspective views of the complex, irregular, and anomalously shaped human vertebra was initiated to study the potential as well as the limitations. Figures 7, 8, and 9 are examples of three-dimensional perspective plots of the atlas (the first cervical vertebra) generated by a Tektronix 4010–1 interactive graphics terminal (with Hard Copy Unit Tektronix 4610). These views reflect use of the program PPLOT package based on program PICPER developed by L. D. Matheson of the National Oceanic and Atmospheric Administration of the U.S. Department of Commerce, Boulder Laboratories, and H. Akima's Smooth Curve Fitting Procedures.[24] The data of three-dimensional points are taken from a human atlas with a mechanical XYZ measuring device designed and developed at the Biomechanics Laboratory.

b. Computer Graphics of Spinal Model

The major objective of developing computer graphics routines[18] is to compare the displacements predicted by the mathematical and biomechanical model of the spine with those of a real living human spine revealed by means of the cineradiographs.

To digitize the spine in the sagittal plane, a series of X-rays are taken on a human spine for

the full lateral spine ranging from skull to sacrum. Using these X-ray films and an overlay method, approximately 7,000 data points are digitized by the cursor on the screen of the Tektronix 4610-1. These points are used to generate any segment of the spine in real scale or the full spine with skull in reduced scale as shown in figure 10.

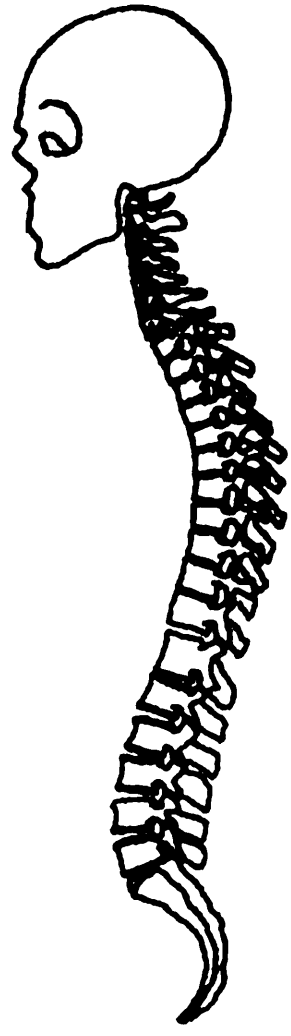


Fig. 10. The computer graphical output of spine from skull to sacrum.

To simulate the displacement all the spinal discs are mathematically modeled[16] using recent measurement data of mechanical properties of discs obtained from a fresh human cadaver.[25] Also, representations of various spinal ligaments and muscles are included in the model. To study the individual effects of some of these ligaments and muscles in the spinal displacement, some of these names are coded by three letters as shown in figure 11, which is also displayed on the computer screen. The modeling of these ligaments and muscles included anatomical studies to determine geometrical shapes and connecting end points for the ligaments, and the origins and insertions for the muscles. Figures 12 and 13 display ligament and muscle models for the cervical spine and lumbar spine, respectively. The computer graphic applications are made both in the spinal curvature study and the natural spinal movement study using this spinal model with the displacement matrix method.[26]

```
LGA=CERVICAL LIGAMENT AND LIGAMENT NUCHAE
DSK=CERVICAL DISK
SCM=STERNOCLEIDOMASTOIDEUS
SCP=SCALENUS POSTERIOR
LOC=LONGUS CAPITIS
RCA=RECTUS CAPITIS ANTERIOR
RCL=RECTUS CAPITIS LATERAL
SCA=SCALENUS ANTERIOR
SME=SCALENUS MEDIUS
LCS=LONGUS COLLI SUPERIOR
LCI=LONGUS COLLI INFERIOR
LCV=LONGUS COLLI VERTICAL

LLG=LUMBAR LIGAMENT
LDS=LUMBAR DISK
PSO=PSOAS
EOB=EXTERNAL OBLIQUE
IOB=INTERNAL OBLIQUE
RAB=RECTUS ABDOMINIS
```

Fig. 11. Coded muscle names and ligaments.

c. Spinal Curvature Study

The curvature of the spine is partitioned into primary and secondary curvatures. Physiologically, the thoracic curve is known to be present during fetal life and is called a primary curve, while the cervical and lumbar curves are due to development after birth under gravitational force and are called compensatory or secondary curvatures. It is also known that various spinal disorders including spinal subluxations are often closely related to the rapid change of these curvatures.

Applying different sets of acting muscular forces which are statically balanced with the gravitational force, the changes of curvatures are observed on the screen and studied along with the change of the center of the curvatures. The computer is programed to draw three circles for each of the major spinal curvatures and to print out the radius and center for each of the curvatures on the screen as shown in figure 14.

d. Spinal Displacement Study

Due to the advent of cineradiography the study

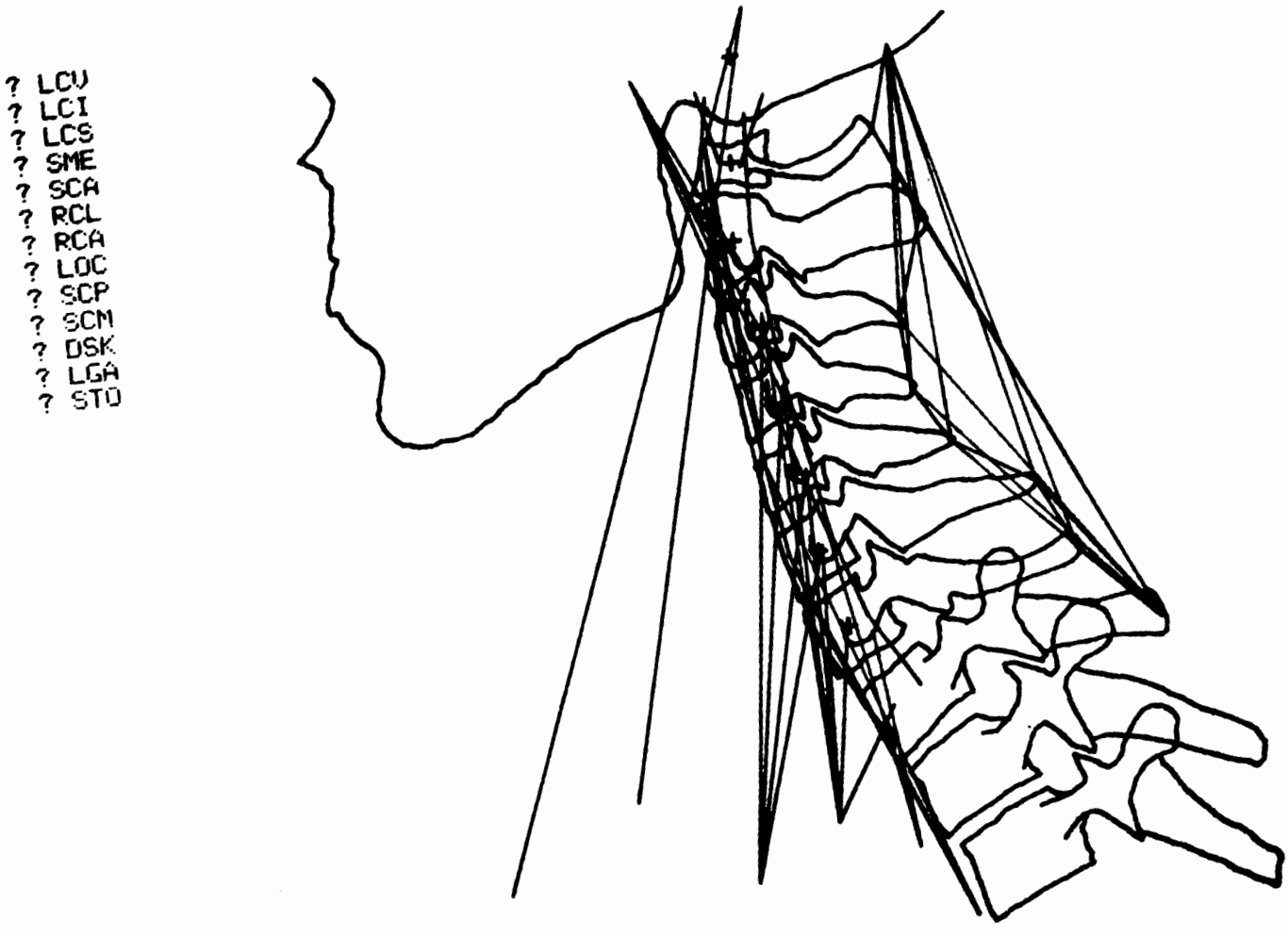


Fig. 12. The biomechanic model of the cervical ligaments and muscles.

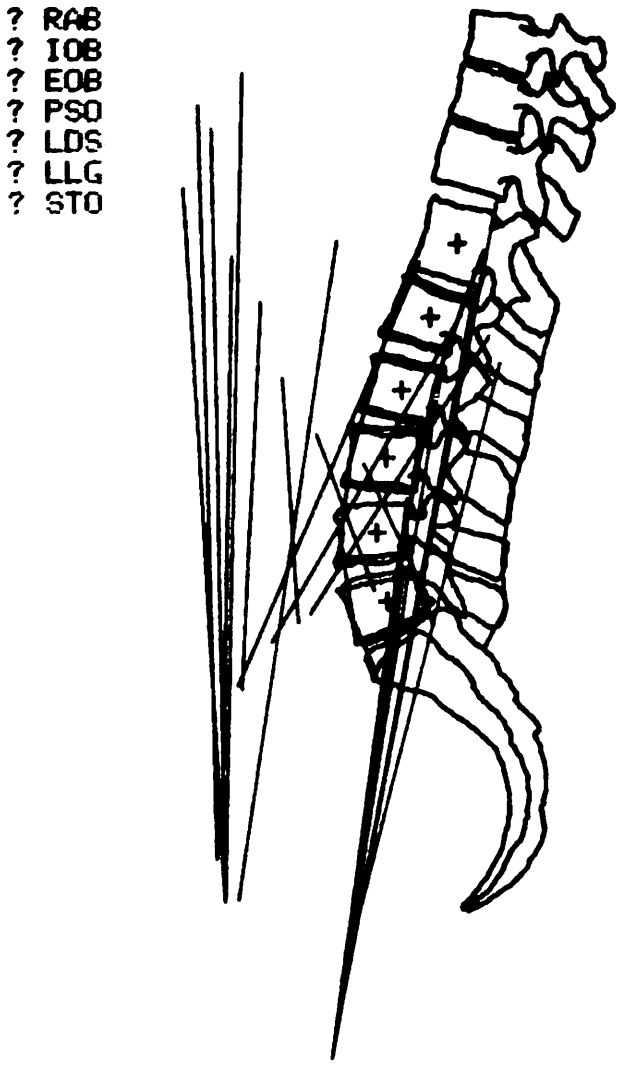


Fig. 13. The biomechanic model of the lumbar ligaments and muscles.

of normal and abnormal spinal motion has progressed significantly. Summaries of such studies[22,23] suggest directly and indirectly that spinal disorders and dysfunctions are closely related to patterns of the voluntary movement of the spine.

The spinal model developed here calculates each vertebral position of the spine under a group of applied muscular forces. The elasto-static equilibrium positions are computed using displacement matrices by either a nonlinear simultaneous equation solver or a nonlinear optimization program, depending on whether the equations are based on a free body force and moment summation type or the principle of minimum potential energy of the spinal system. Figure 15 shows a computer graphics output generated by this system on the Tektronix screen for six different positions of a cervical spine including the skull. Inclusion of the skull enables us to study the motion pattern of the altanto-occipital joint which often becomes the center of study on the upper cervical subluxation complex.

In order to study the path of each vertebra during the flexion of the cervical spine a certain number of positions were superimposed as shown in figure 16. A similar study was done on the lumbar spine with the same computer model and is shown in figure 17 for six different positions and in figure 18 for a superimposed output.

4. CONCLUSION

This paper summarizes the present status of two major aspects of research on biomechanical aspects of subluxation: Development of better methods for visualizing subluxation and development of a computer simulation model of the spine to understand analytically the nature of subluxation.

As in medical research on the spine, there is a large body of descriptive information available in the chiropractic literature on spinal subluxation, but few numerical or analytical relations are known. The computer-aided X-ray method can help doctors of chiropractic obtain accurate numerical data while at the same time supporting the development of the computer model of the spine, which is essential for systematic study of spinal subluxation.

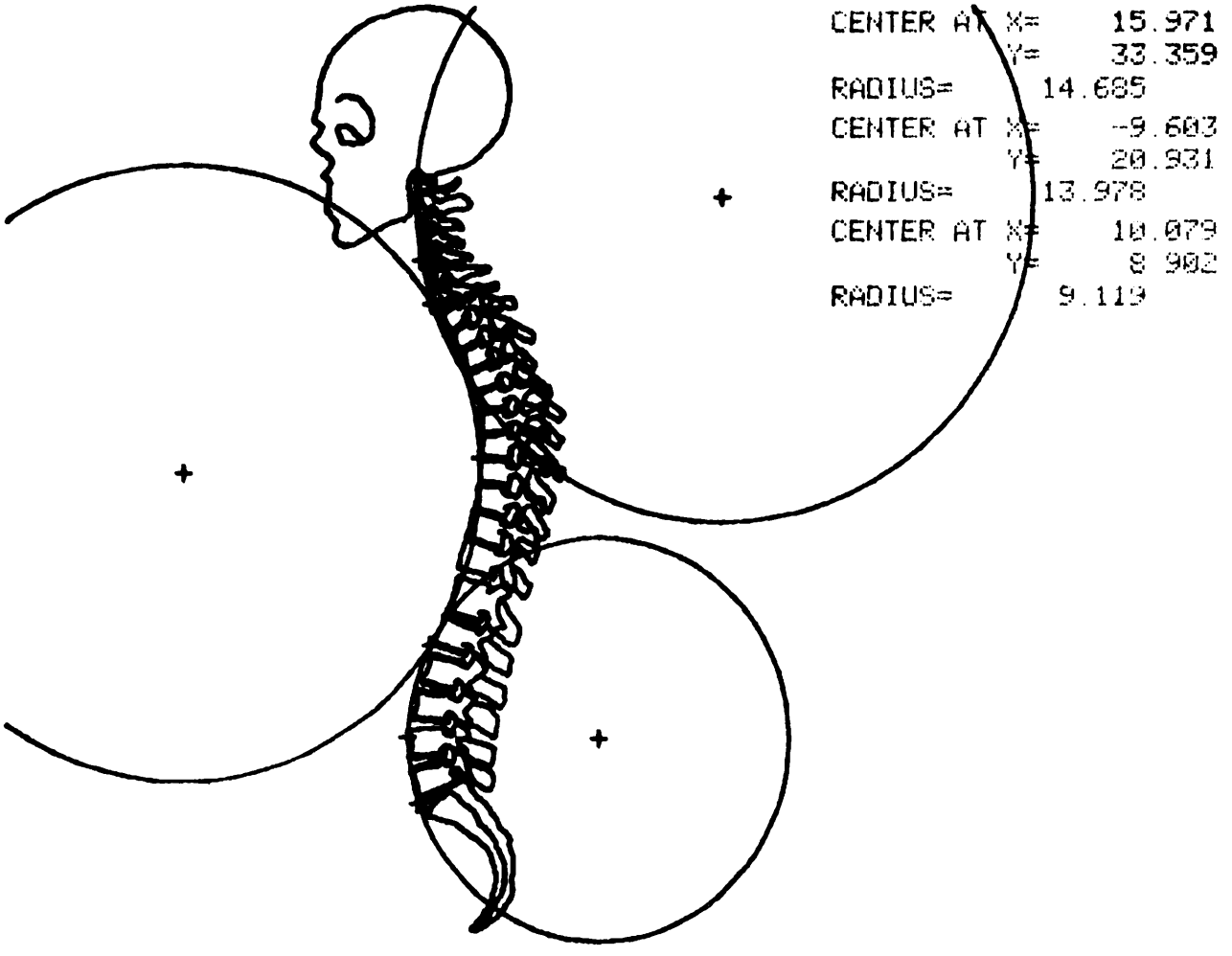


Fig. 14. Curvatures of vertebral column.

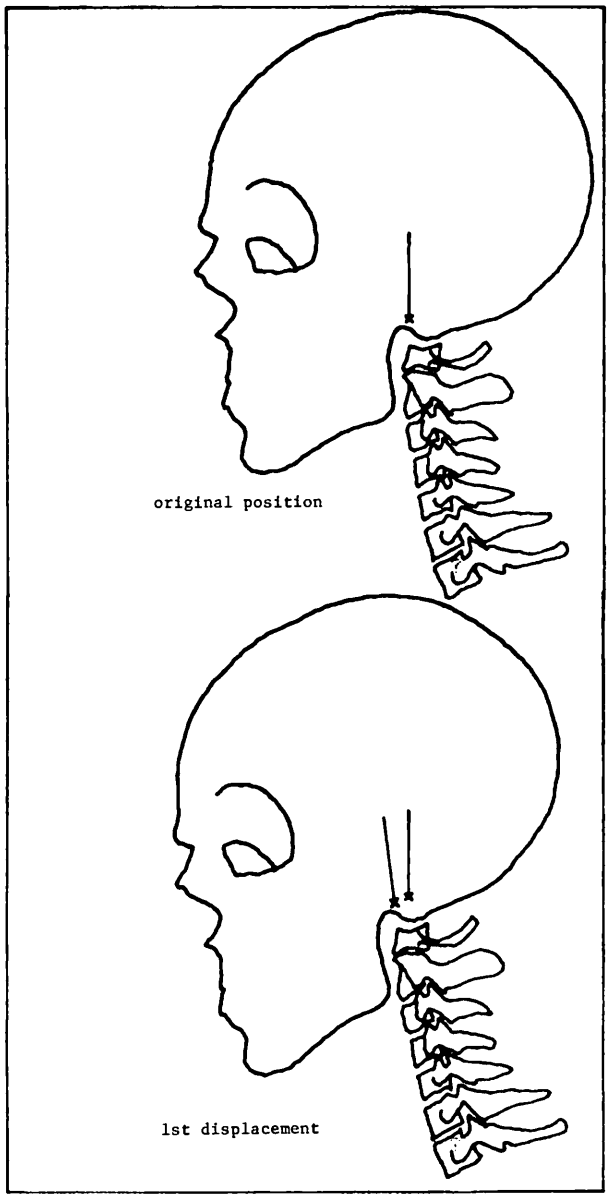


Fig. 15. The displacement analysis of cervical spine.

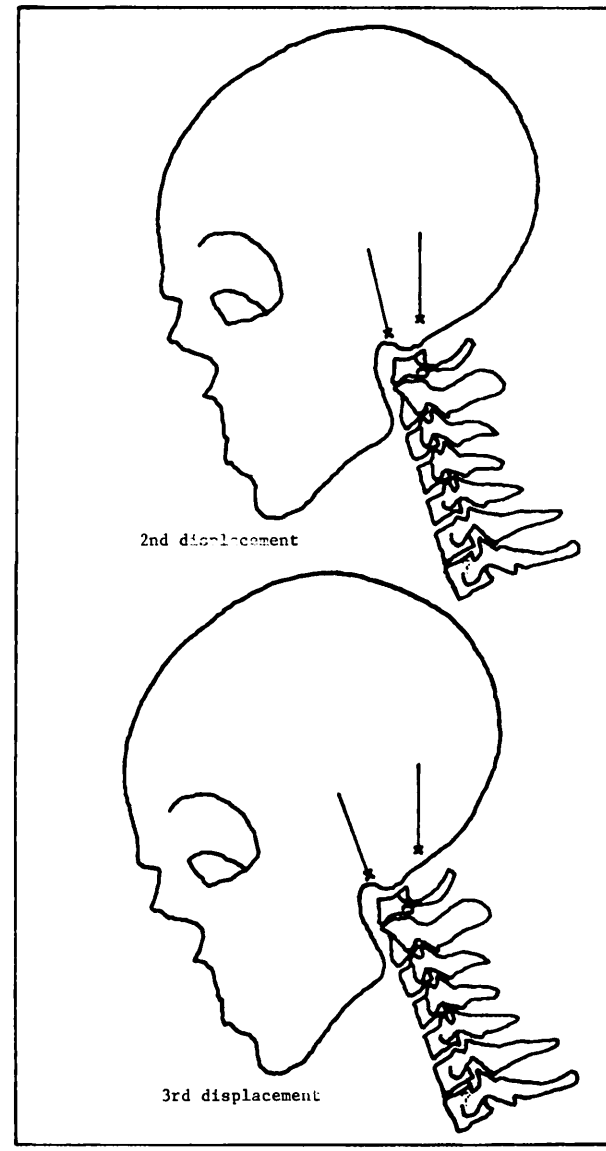


Fig. 15. (Continued)

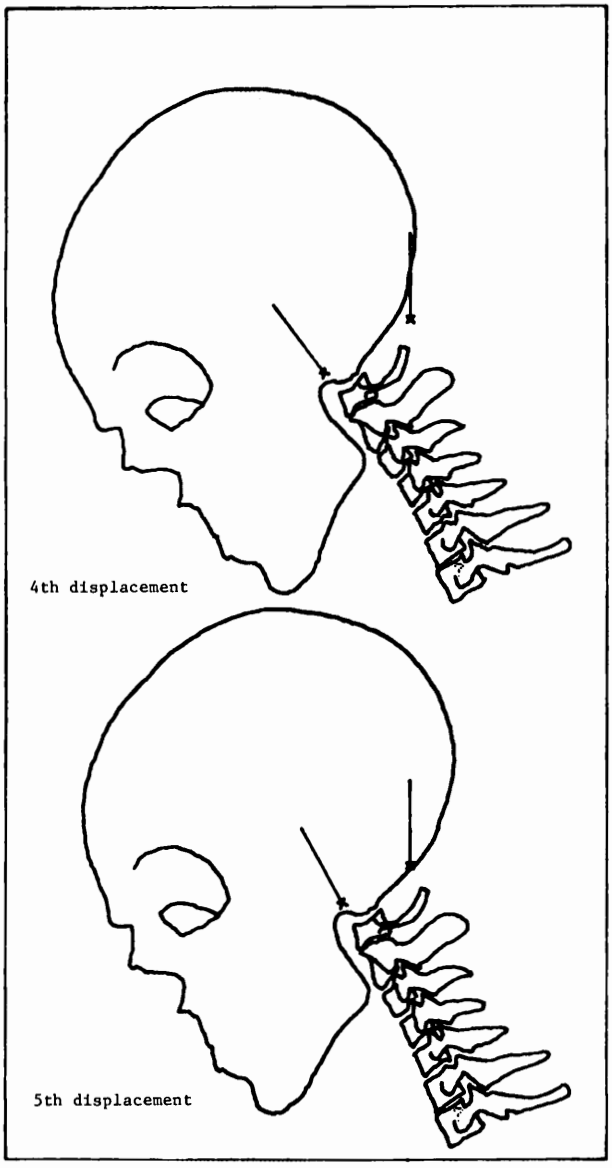


Fig. 15. (Continued)

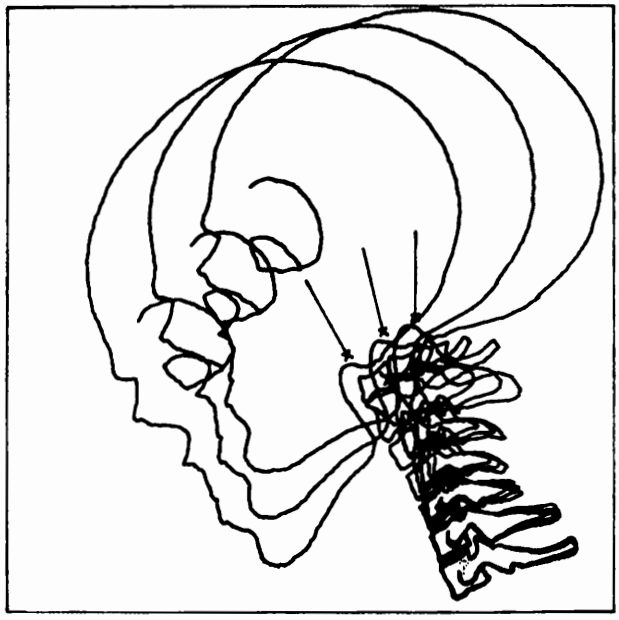


Fig. 16. Superimposed displacements of cervical spine.

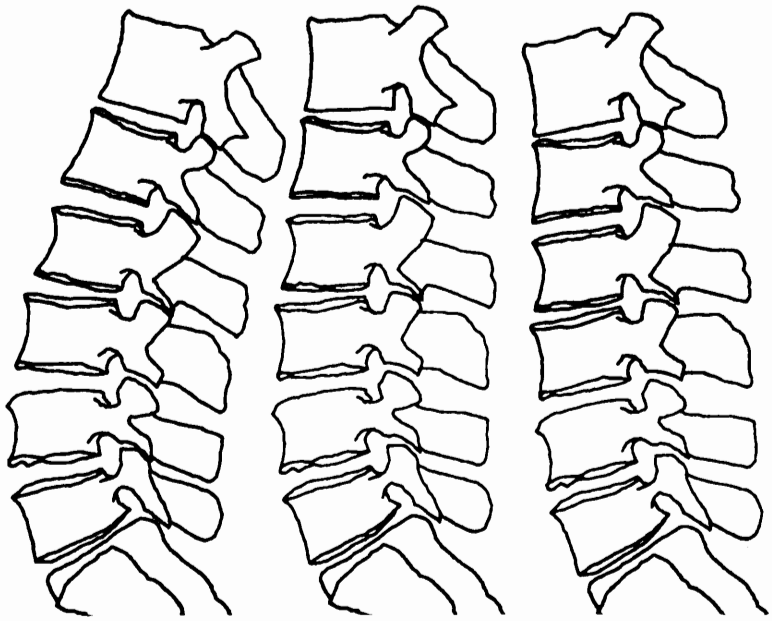


Fig. 17. The displacement analysis of lumbar spine.

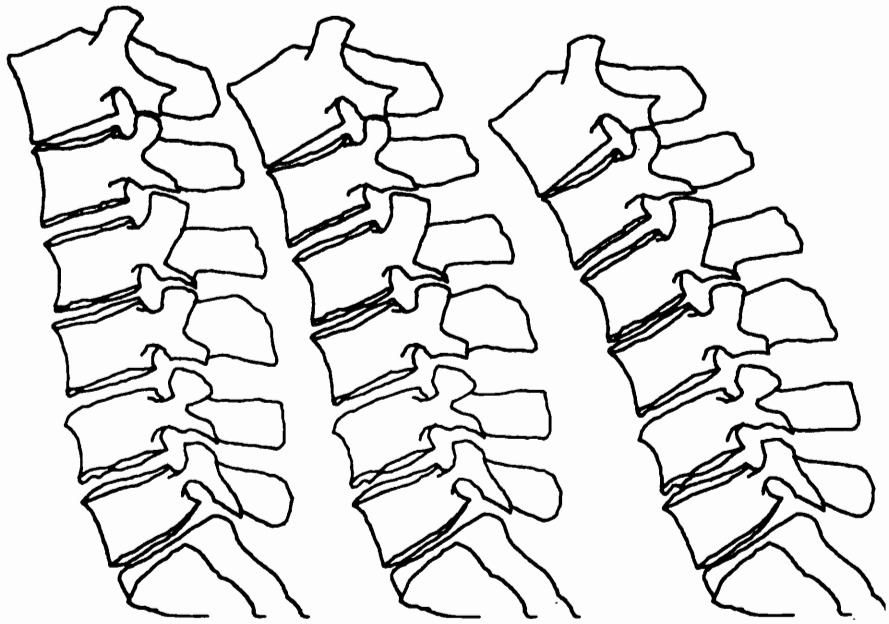


Fig. 17. (Continued)

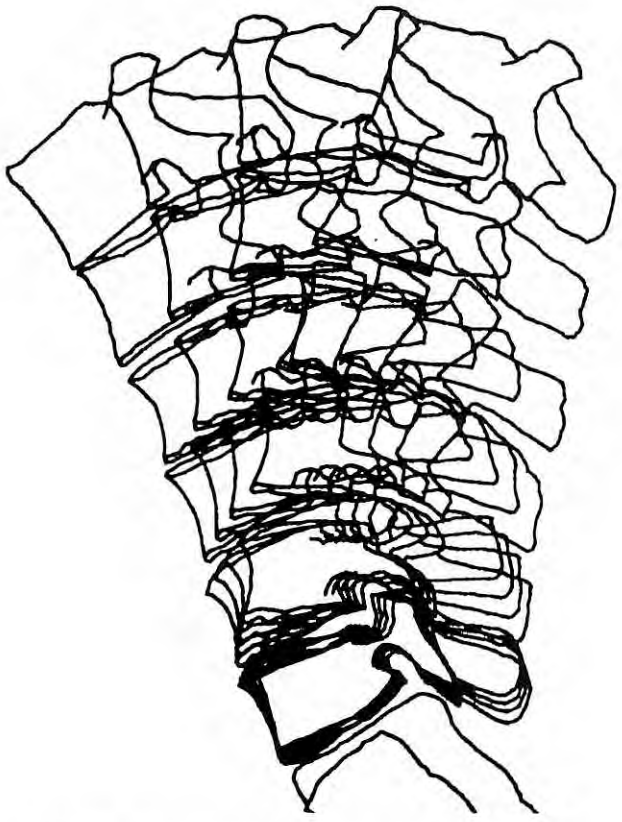


Fig. 18. Superimposed displacements of lumbar spine.

REFERENCES

1. *Stedman's Medical Dictionary*, 22nd Edition, p. 1210, The Williams and Wilkins Co., 1972.
2. Suh, C. H., "Computer-Aided X-ray Analysis of the Spine," Part I and Part II, Biomechanics Lab., University of Colorado, Boulder, Colo., November 1972 (29 pages & 35 pages).
3. Suh, C. H., "The Fundamentals of Computer-Aided X-ray Analysis of the Spine," *Journal of Biomechanics*, 1974, Vol. 7, pp. 161–169, Pergamon Press.
4. Wictorin, L., "Bone Resorption in Cases with Complete Upper Denture. A Quantitative Roentgenographic-Photogrammetric Study," *Acta Radiol.* Suppl. 228: 97 pp., 1964.
5. Wictorin, L., "Measurements on Roentgenographs. A Comparison Between Two Methods," *Acta Odontol. Scand.*, 24(4):517–531, 1966.
6. Hallert, B., "X-Ray Photogrammetry, Basic Geometry and Quality," Elsevier Publishing Co., Amsterdam, London, New York, 1970.
7. Dodge, H. T., Sandler, H., Ballew, D. H., and Lord, T. D., "The Use of Biplane Angiocardiography for the Measurement of Left Ventricular Volume in Man," *Am. Heart J.*, 60:762, 1960.
8. Nelson, C. N., and Lipchik, E. O., "A Computer Method for Calculation of Left Ventricular Volume from Biplane Angiocardiograms," *Investigative Radiology*, Vol. 1, No. 2, March–April 1966, pp. 139–143.
9. Lusted, L. B., and Keats, T. E., "Atlas of Roentgenographic Measurement," Second Edition, Chicago Year Book Medical Publishers Inc., 1967.
10. Dawson, B. H., Dervin E., and Heywood, O. B., "Geometrical Problems in Stereotactic Surgery: A Three Dimensional Analysis for Use With Computer Techniques," *J. of Biomechanics*, Vol. 3, pp. 175–180, 1970.
11. Kuhn, J., "Protractorscopic and Stereoprotractorscopic Spinograph Analysis," The Parthenon Press, Nashville, Tenn., 1956.
12. Suh, C. H., "Displacement Analysis of Spinal System," 21 pages, Proceedings of the 4th Annual Biomechanics Conference, University of Colorado, November 1973.
13. Suh, C. H., and Hong, S. W., "Computer Aided X-Ray Data Correction Method with Use of Rigid Body Conditions," pp. 53–126, Proceedings of the 5th Annual Biomechanics Conference, University of Colorado, December 1974.
14. Dalsania, V., "A Mathematical Model for Elasto-static Systems and Application to Spinal Analysis," Biomechanics Lab., University of Colorado, Boulder, Colo., November 1972 (51 pages).
15. Tsukatani, T., "Computer Graphics of Spine," Biomechanics Lab., University of Colorado, Boulder, Colo., November 1972 (50 pages).
16. Suh, C. H., and Dalsania, V., "Computer Implementation of Spinal Discs in Biomechanical Models," Proceedings of the 4th Annual Biomechanics Conference, University of Colorado, November 1973 (49 pages).
17. Suh, C. H., Dalsania, V., and Hong, S. W., "Program 'Spine' for Solution of Elastostatic System," Proceedings of the 4th Annual Biomechanics Conference, University of Colorado, November 1973 (39 pages).
18. Suh, C. H., and Hong, S. W., "Computer Graphics for Spinal Analysis," Proceedings of the 4th Annual Biomechanics Conference, University of Colorado, November 1973 (39 pages).
19. Suh, C. H., et al., "Researching the Fundamentals of Chiropractic," Proceedings of the 5th Annual Biomechanics Conference, University of Colorado, December 1974, pp. 1–52.
20. Suh, C. H., and Kwak, Y. K., "Initial Investigation for Three-Dimensional Simulation of the Spine as an Elasto-Static System," Proceedings of the 5th Annual Biomechanics Conference, University of Colorado, December 1974, pp. 127–196.
21. Schultz, A. B., "Mechanics of the Human Spine," Applied Mechanics Reviews, ASME, November 1974, pp. 1487–1497.
22. Fielding, J. W., "Cineroentgenography of the normal cervical spine," *Journal of Bone and Joint Surgery*, 39-A:1280–1288, December 1957.
23. Howe, J. W., "Cineradiographic Evaluation of Normal and Abnormal Cervical Spinal Function," *Journal of Clinical Chiropractic*, Archives Edition 2 1972, pp. 76–88.
24. Akima, H., "A New Method of Interpolation and Smooth Curve Fitting Based on Local Procedures," *Journal of Association for Computing Machinery*, Vol. 17, No. 4, October 1970, pp. 589–602.
25. Markolf, K. L., "Deformation of the Thoracolumbar Intervertebral Joints in Response to External Loads," *Journal of Bone and Joint Surgery*, 54-A, No. 3, 511–533, April 1972.
26. Suh, C. H., "Design of Space Mechanisms for Rigid-Body Guidance," *Journal of Engineering for Industry*, TRANS. ASME, Vol. 90, Series B. August 1968, pp. 499–507.

Discussion: Anatomical and Biomechanical Studies

JAMES H. McELHANEY
Professor of Biomechanics
and Director of Graduate Studies
Department of Biomedical Engineering
Duke University School of Engineering

The preceding four papers dealt with the mechanical aspects of the spine from a basic science point of view. This point of view was well characterized by Sir Isaac Newton when he said, "Unless we can assign numbers to the behavior of a system, we know very little about it indeed." Galileo Galilei was also a great proponent of the scientific method when he said, "Measure everything that is measurable and then make measurable some things that are not."

I think that modern science takes a more catholic point of view in recognizing that measurement requires precise definition, that many words are needed to describe complex systems before meaningful measurement can be made. In this context, one should distinguish between the soft sciences where verbal description suffices and the hard sciences where numerical description is a prerequisite.

The basic types of measurement that can be made on the spine may be classified accordingly:

1. size and shape
2. motion
3. structure or load-bearing responses
4. material properties
5. nerve activity
6. chemistry
7. changes in responses and/or properties with time
8. there are, in addition, many types of derived measurements that deal with combinations of these basic categories.

The first two papers presented by Drs. Drum and Weis deal with the first and seventh category – anatomy and geometry over the life of the system. These papers present overviews of the state of the art but are lacking in numbers. However, of all the papers dealing with spinal morphology, only a relatively few offer any measurements. Lanier (1939), Davis (1957), Perez (1957), and Nachemson (1960) offer limited measurements of gross vertebral body geometry, such as heights, diameters and cross-sectional areas. Allbrook (1956) and Roaf (1960) give some information on the increase in vertebral dimensions with age. Schultz (1974), after an exhaustive literature review, concluded that a quantitative characterization of shape does not appear to have been made.

The external geometry of the facet and processes determine to a large extent the ranges of motion. However, descriptions of these geometrics are also largely qualitative and those few measurements that have been made were not complete and were not directed toward qualitative geometric description. It is in this area of quantification that Dr. Suh's report of his work on precision X-ray and computer aided spinal geometrical analysis assumes significance. One reason that much of human anatomy has not been quantitatively described is that this is tedious and uninspiring work. Modern computer aided photogrammetry offers a particularly attractive method for this work. It appears, therefore, that many of the problems associated with complex geometrical descriptions may be solved with the application

597-036 O - 76 - 9

of existing technology. R. E. Herron of the Biostereometrics Laboratory, Baylor College of Medicine, recently (1973) used this method with good fidelity in the Biostereometric Measurement of body form. His results are being used by the Department of Transportation to specify crash test dummy geometry.

The state of the art or, "how full is the glass," of measurements of spinal motion was summarized by Dr. A. A. White. As Dr. White indicated, there have been many studies dealing with so-called spinal kinematics. The majority of these studies assume, based on the relative stiffness of the vertebral discs, ligaments and surrounding soft issue, that the bony elements act as rigid bodies. An additional but frequently tacit assumption is that the properties are time independent. Because of the viscoelastic nature of the tissues involved, this is not true and can yield misleading conclusions. Consider, for example, that the intervertebral discs account for approximately a third of the length of the spinal column. The nucleus pulposus is a gel-like material which creeps or changes shape under load. This can lead to significant changes in facet spacing. Temperature is another important variable that affects the material properties and thus, the kinematics of "free motion."

This leads us to the third and fourth categories, structural and material responses. This type of response, though of vital importance, has been neglected in the discussions so far.

In the engineering disciplines, material properties such as ultimate strength and stress-strain relations are of great importance. A designer starts with a basic building material and shapes it into a structure with specified load and deformation responses. Since the human body exists, its load and deformation responses cannot be changed. Knowledge of the properties of the materials of which the body is composed is only useful insofar as it leads to a better understanding of the structural responses. By definition, structural responses are those load and deformation characteristics that are related to the size, shape, configuration, and material of which a structure is composed. Material properties in contrast are generally represented as being independent of the structure or shape of the material under consideration.

There have been a variety of studies of the load-deformation properties of spinal elements. One of the most complete was reported by Sonada (1962). He provides data on tensil, compression, and torsional loads, stresses and strains of vertebral bodies, intervertebral discs, vertebral trabecular bone, and isolated annulus fibrosus material. He shows that, under static loading, except in the cervical spine, the vertebral body crushes before the disc fails. Figure 1 shows some of his results in terms of stress-strain curves for vertebrae at various levels in the spine.

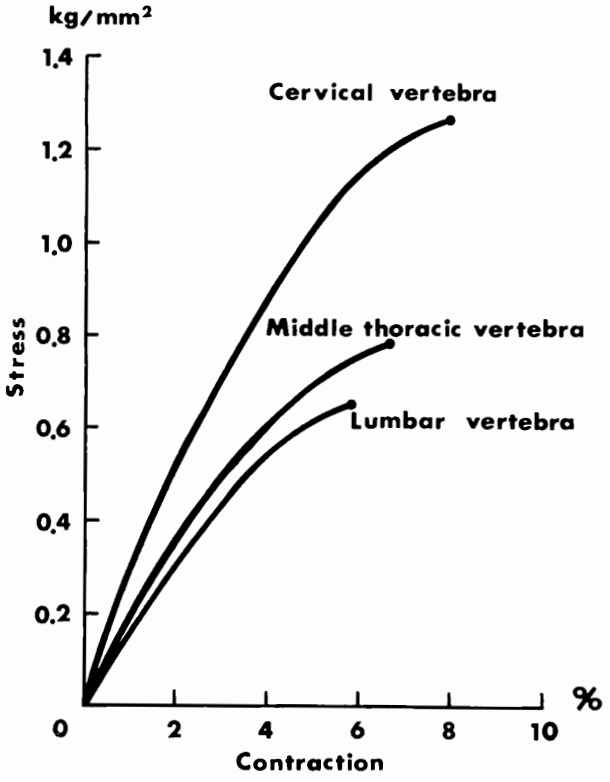


Fig. 1. Stress-strain curves in compression of wet vertebrae of persons 20 to 39 years of age (Sonada).

I would be remiss if I did not indicate also work done by McElhaney (1970).

Based on extensive testing of cuboidal cancellous bone specimens from the femur, vertebral body and the cranium, a model relating the porosity, distribution to the strength and modulus was developed. Figure 2 shows typical stress-strain curves for various bone types in compression. Tests results from 72 donors are given in the following table.

TABLE I

Density #/in³	No. of donors	No. of specimens	Age range	Mean	Standard deviation
Femur	32	160	28–86 yrs.	0.067	0.027
Vertebral Body	72	288	1 mo.–89 yrs.	0.017	0.007
Cranium Full Section	14	240	56–73 yrs.	0.051	0.019
Cranium Compacta	7	27	56–73 yrs.	0.068	0.007
Modulus #/in²					
Femur Long Axis	32	160	28–86 yrs.	1.84×10^6	0.4×10^6
Vertebral Body	72	288	1 mo.–89 yrs.	0.22×10^5	0.17×10^5
Cranium Full Section					
Radial	26	237	39–81 yrs.	3.5×10^5	2.1×10^5
Tangential	14	219	56–73 yrs.	8.1×10^5	4.4×10^5
Cranium Compacta	7	27	56–73 yrs.	1.81×10^6	0.6×10^6
Ultimate Strength #/in²					
Femur Long Axis	32	160	28–86 yrs.	2.6×10^3	6.1×10^3
Vertebral Body	72	288	1 mo.–89 yrs.	0.6×10^3	0.5×10^3
Cranium Full Section					
Radial	26	237	39–81 yrs.	10.7×10^3	5.1×10^3
Tangential	14	210	56–73 yrs.	14×10^3	5.2×10^3
Cranium Compacta	7	27	56–73 yrs.	20.9×10^3	6.8×10^3
Poisson's Ratio					
Femur Long Axis	7	28	37–86 yrs.	0.3	0.06
Vertebral Body	7	28	45–79 yrs.	0.14	0.09
Cranium Full Section					
Radial	14	122	56–73 yrs.	0.19	0.08
Tangential	18	327	56–81 yrs.	0.22	0.11
Cranium Compacta	7	27	56–73 yrs.	0.28	0.04

The vertebral bodies in figure 3 were generally taken at autopsy in the lumbar region at L_2, L_3, L_4, and L_5. A variety of specimen sizes were tested unembalmed, ranging from the whole body with the processes removed down to one-fourth inch cubes.

The vertebral body properties also correlated well with the dry weight density (figure 4). The following linear regression equations were obtained:

$$E\ (3-0.023) \times 10^6;\ C_c\ 0.61$$

$$O\ (300-5.9) \times 10^3;\ C_c\ 0.67$$

$$O\ (3.2 \times 10^{-2})\ E;\ C_c\ 0.71$$

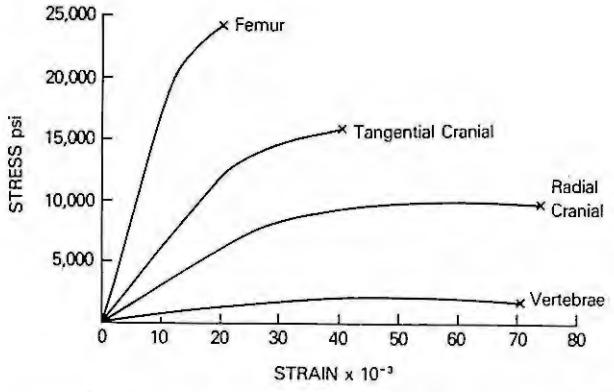


Fig. 2. Typical stress-strain curves for human bone in compression.

Fig. 3. Vertebral body specimen.

where

E = Modulus of elasticity (PSI)
= Density (pound per cubic inch)
= Ultimate Strength (PSI)
C_c = Correlation Coefficient

The vertebral body data also show a strong linear correlation between modulus and strength, indicating that a maximum strain theory of failure applied also to cancellous bone from the vertebrae with a maximum failure strain of 3.2×10^{-2}. These regression equations give approximately valid results for dry weight densities between 0.022 and 0.010 pound per cubic inch.

While no significant correlation of the femoral or cranial bone was found with age, the strength

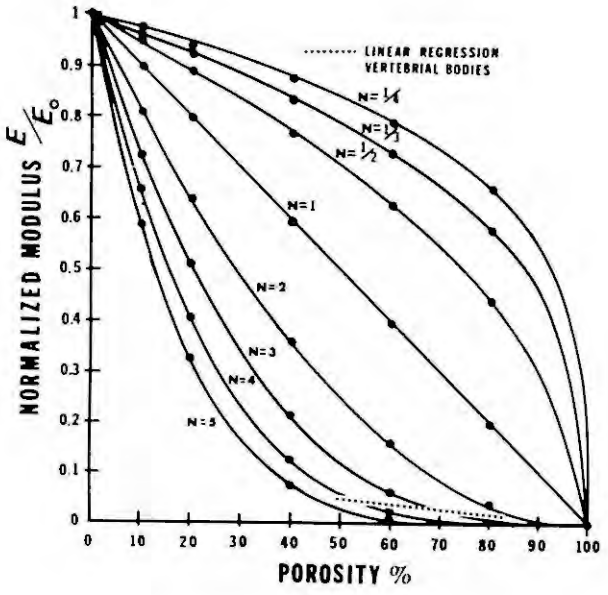


Fig. 4. Porous block model responses.

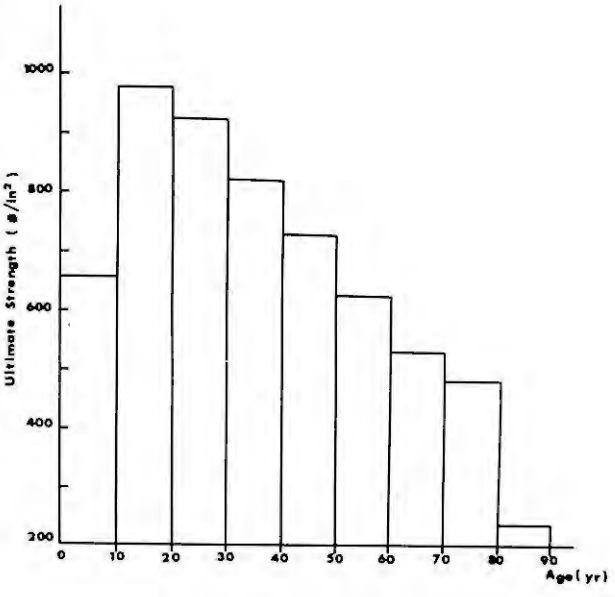


Fig. 5. Strength-aging characteristics of the human vertebral body.

of vertebral bone correlated at the 0.52 level (figure 5).

Many of the mechanical responses of cancellous bone are strongly influenced by the structural arrangement of the trabeculae. Thus, in these tests, properties, such as compressive strength and modulus, are structural properties, and the large values of the standard deviations observed for

these properties are primarily due to variations in the porosity and internal arrangement of the trabeculae. The similarities of the properties and histology of compact bone indicates that a single material porous block model is justified as a first approximation in describing the relation between structure and mechanical response. Relating the value of a property to the density raised to some power n and then determining n empirically provides a means of incorporating in the model many of the structural elements that influence the response but are too complex to be included in detail. The porous block model is, therefore, complex enough to explain much of the property variation observed but much too simple to explain the mechanisms involved. The model shows that the modulus of bone is approximately proportional to the third power of the density. Thus, small porosity changes in bone of low relative density result in only small changes in strength and modulus, while small porosity changes in bone of high relative density result in large changes in strength and modulus. The porosity distribution in a given sample of bone is much more significant in its effect on strength and modulus in bone of low relative density than in bone of high relative density. Of interest is the fact that the homogeneous version of this model fits much of Coble's data (1956) on various porous ceramics, indicating that bone is not unique in its response to porosity variations.

The material properties in the small, i.e., hardness, density, and local compression strength of various bone types, are not significantly different. The amount and distribution of the trabeculae, however, is quite variable, and, therefore, the structural responses, in particular the energy absorption, gross stiffness, and damping characteristics, which are strongly dependent on structure, will vary greatly. The properties of the intervertebral disc have been studied from a variety of viewpoints. Since the disc is an essential element in supporting the body and in permitting the mobility of the bony elements, its mechanical responses are important factors in spinal manipulative therapy. Hakim (1974) showed from in vitro static loading of a spinal element that the disc supported 75 to 80 percent of the load at the L3 level while the articular facet supported 20 to 25 percent of the load. In addition, in his studies on three embalmed cadavers, he indicated that the load sharing was linear in the range tested.

No one has yet assigned numbers to the pressure buildup in the marrow spaces of cancellous bone as it deforms under load but it is known that it happens.

Axial force-deflection properties of intervertebral discs have been reported by several investigators. Virgin (1951) tested single discs attached to thin slices of their inferior and superior vertebrae in compression. He presented load-deflection curves which exhibited various degrees of nonlinear behavior, and reported that the intervertebral discs behave visco-elastically.

Brown (1959) et al., conducted axial compression tests on fresh vertebra-disc-vertebra segments of the lumbrosacral spine with the posterior elements removed.

Rolander (1966) performed a series of mechanical tests on lumbar discs and by macroscopic examination classified the excised discs according to degree of degeneration. A comparison between compression responses of normal and degenerate discs shows a significant difference in behavior: For equal load levels, the degenerate discs deflect much more than the normal discs.

Markolf (1972) conducted axial compression and tension tests, among other tests, on thoracolumbar discs. His results indicate that there are significant differences in behavior between lumbar and thoracic discs. In addition, each disc shows differences in response between tensile and compressive loading. Markolf also reported that intervertebral discs behave visco-elastically.

Galante (1972) experimentally established that the annulus fibrosis is inhomogeneous and anisotropic, and that it exhibits hardening stress-strain characteristics.

Kulak (1974) proposed a nonlinear stress-strain relation representing the behavior of annular material. Incorporation of the relation into an existing finite element model yields results which are in good agreement with available experimental results, such as radial bulging, nuclear pressure, and level-to-level variations.

These studies (Kulak et al. 1974) have shown the importance of including the nonlinear material behavior in the analysis of the mechanical function of soft tissue structures, such as the disc. Although a basic understanding can often be gained with less complex linear models, the predictive value of linear models is severely limited because they are

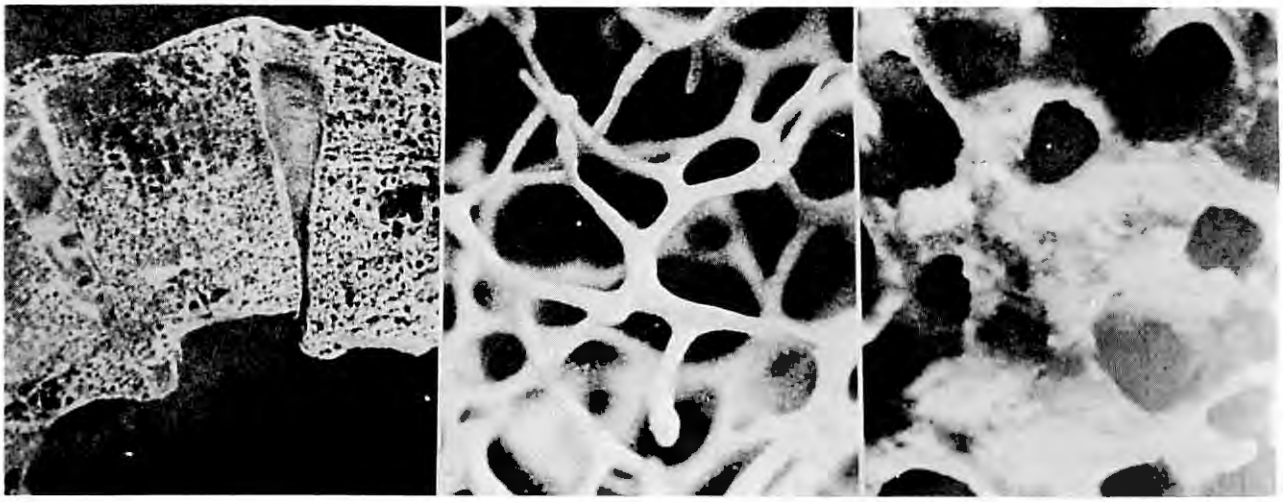


Fig. 6. Bone remodeling in response to load.

restricted to small load ranges. This is illustrated by the marked differences in the response to tension and compression and in the case of a void nucleus. Thus, the analysis of the intervertebral disc over the normal in vivo range of loading requires the inclusion of nonlinear material behavior.

The role of the ligaments in maintaining proper relations between the bony elements of the spine is easily demonstrated but poorly understood. The amount of prestressing on other structural properties is difficult to measure in vitro. The literature in this area is sparse.

Two most important aspects which have received little attention are:

1. The role of load directed growth in the spine
2. The relation between deformation and neurophysiology.

To demonstrate: consider figure 5 from Weinmann and Sicher (1955). They observed that lack of function leads to loss of bone tissue or osteoporosis while increased function leads to the formation of more than the normal amount of bone or osteosclerosis. This can be seen in kyphosis, wherein certain vertebrae are unevenly stressed. One side of the vertebrae may be under increased compression while the other may be relieved of any load and, in fact, may be in tension. In the part of the vertebra which is under increased compression, the number of the trabeculae is increased and each one is thicker than normal. As a consequence, the bone spaces are narrowed. Just the opposite occurs on the other side which is relatively unloaded. The trabeculae are resorbed. They become thinner and fewer and the marrow spaces wider than normal. Thus, the bone tries to minimize the eccentricity by shifting its center of mass toward the load axis through selective deposition and resorption. There are, of course, severe limitations in that the external shape and dimension can be changed very little, so that the remodeling is primarily confined to the interior.

Associated with stresses in bone is an electric field, thought to be piezoelectric in nature. Laboratory experiments demonstrate that electric fields and currents do influence bone growth. However, much research is needed before this phenomenon can be used in therapy.

Perhaps the most important measurements that conceivably could be made involve the last two categories. Very little is known about the effect of load and deformation on nerve function and metabolism. Is increased nervous activity a direct result of pressure or shear, or tension? Or is it related to metabolic changes that occur over time? Are these effects reversible? What is the nature of pain from mechanical trauma? We could fill pages with such questions. Our greatest hope for the answers lies in modern technology which is providing constantly improving measurement techniques.

I would like to close with this metaphor. Birds and bees know well how to fly without knowing anything about aerodynamics or biomechanics, but it took man centuries of careful research, study

and analysis before he too could fly. The basic sciences tell much about manipulative therapy. In particular, they tell us many things it cannot do. It remains for science to document and detail the many things manipulative therapy can do.

REFERENCES

Allbrook, D. (1956), "Changes in lumbar vertebral body height with age," *Amer. J. Phys. Anthrop.* 14, 35–39.

Belytschko, T., Kulak, R. F., Schultz, A. B., and Galante, J. O., "Finite Element Stress Analysis of an Intervertebral Disc." *J. of Biomechanics,* Vol. 7.

Brown, T., Hansen, R. J., and Yorra, A. J., "Some Mechanical Tests on the Lumbo-Sacral Spine with Particular Reference to the Intervertebral Disc." *J. Bone Jt. Surg.* Vol. 39A, No. 5, October 1959, 1135–1164.

Davis, P. (1957), "Studies on the functional anatomy of the human vertebral column with special reference to the thoracic and lumbar regions." Ph. D. thesis, University of London.

Galante, J. O., "Tensile Properties of the Human Lumbar Annulus Fibrosis." *Acta Orthop. Scand.,* Suppl. 100. 1972.

Hakim, N. S., and King, A. I., (1974), "The Load-Bearing Role of Articular Facets During Static Loading of a Spinal Column." *Advances in Bioengineering Pub. ASME.*

Haut, R. C., and Little, R. W., "A Constitutive Equation for Collagen Fibers." *J. Biomechanics,* Vol. 5, No. 5, 1972, 423–430.

Herron, R. E. (1973), "Biostereometric Measurement of Body Form." *J. Amer. Assn. of Physical Anthropology.* Vol. 16.

Kulak, R. F., Belytschko, T. B., Schultz, A. B., and Galante, J. O. (1974), "Nonlinear Behavior of the Human Intervertebral Disc Under Axial Load." *ASME Paper* No. 74–WA/B10–3.

Lanier, R. (1939), "The presacral vertebrae of American White and Negro Males," *Am. J. Phys. Anthrop.* 25, 341–420.

Markolf, K. L., "Deformation of the Thoracolumbar Intervertebral Joints in Response to External Loads," *J. Bone Jt. Surg.*, Vol. 54A, No. 3, April 1972, 511–533.

McElhaney, J. H., Alem, N., and Roberts, V. (1970), "A Porous Block Model for Cancellous Bones." *ASME Paper* No. 70–WA/BHF–2.

Nachemson, A. (1960), "Lumbar Intradiscal Pressure," *Acta Orthrop. Scand.* Suppl. 43.

Perez, O. (1957), "Fracture of the vertebral end-plate in the lumbar spine," *Acta Orthrop. Scand.* Suppl. 25.

Roaf, R. (1960), "Vertebral growth and its mechanical control," *J. of Bone and Joint Surg.* 42B, 40–59.

Rolander, Svante D., "Motion of the Lumbar Spine with Special Reference to the Stabilizing Effect of Posterior Fusion," *Acta Orthop. Scand.,* Suppl. 90.

Schultz, A. B. (1974), "Mechanics of the Human Spine," *Applied Mechanics Reviews,* 1487–1497.

Sonoda, T. (1962), "Studies on the Strength for Compression, Tension and Torsion of the Human Vertebral Column," (in Japanese, with English summary) *J. Kyoto Pref. Med. Univ.* 71, 659–702.

Virgin, W. J., "Experimental Investigations into the Physical Properties of the Intervertebral Disc." *J. Bone Jt. Surg.*, Vol. 33b, No. 4, November 1951, 607–611.

Weinmann, J. R., and Sicher, H. (1955), *Bone and Bones,* C. V. Mosby Co., St. Louis, Mo. Pub.

Anatomical Perivertebral Influences on the Intervertebral Foramen

SYDNEY SUNDERLAND
Department of Experimental Neurology
University of Melbourne, Australia

Of the many anatomical perivertebral influences on the intervertebral foramen, those of particular relevance to the subject under scrutiny concern the joints directly related to the foramina and the nerves in the vicinity. This contribution to the status of spinal manipulative therapy is devoted to a consideration of these matters. While confirming constriction of the foramen as a cause of nerve involvement, attention will be directed to the posterior vertebral joints as a less well-recognized potential site of troublesome pathology involving nerve pathways.

1. THE SPINAL NERVE ROOTS AND SPINAL NERVE IN RELATION TO THE INTERVERTEBRAL FORAMEN

Opposite the foramen each pair of anterior and posterior nerve roots invaginate the dura and arachnoid to form a funnel-shaped depression at the bottom of which each perforates the meninges independently carrying with it, as it does so, an individual and separate bilaminar sleeve of dura and arachnoid (figure 1). The extension of the subarachnoid space formed in this way is continued along the nerve roots usually as far as the ganglion, occasionally to involve its inner pole but never to envelop it completely. The dural layer is continued along the nerve roots for a short distance before finally blending with the anterior root and the ganglion to form an outer fibrous sheath for these structures. This connective tissue is continued outwards to become the strong perineurial sheath of the single bundle of nerve fibers of the spinal nerve formed by the fusion of the two roots. The somewhat condensed layer of epidural tissue on

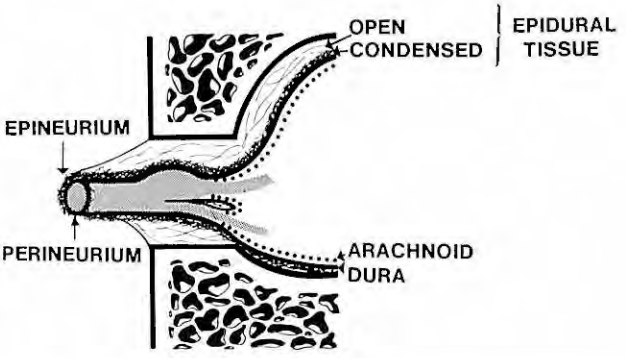


Fig. 1. The nerve complex and its meningeal coverings in the intervertebral foramen. The arrangement is not to scale and has been drawn to accentuate relative relationships.

the surface of the dura is continuous with the epineurium of the spinal nerve. The formation of this perineurial-epineurial sheath adds to the thickness of the spinal nerve so that its cross-sectional area exceeds the combined cross-sectional areas of the nerve roots from which it is formed.

The nerve roots, posterior root ganglion and spinal nerve together with their connective tissue coverings will, henceforth, be referred to as the nerve complex.

(i) *The number and localization of sensory and motor fibers in the nerve complex.* The posterior nerve root is thicker than the corresponding anterior root by a factor of three in the cervical nerve roots, 1.5 in the thoracic and two in the lumbar.[1,2] The two roots remain separate as far as the ganglion and so may be selectively involved in pathological processes central to this level. In the spinal nerve, immediately distal to the ganglion, the nerve fibers are contained in a single funiculus

in which the motor fibers are concentrated anteroinferiorly (figure 2). In the case of the eighth cervical to the second lumbar nerves and the second, third and fourth sacral nerves, these motor fibers include sympathetic (C8–L2) and parasympathetic (S2, 3, 4) elements. Within a few millimeters the single funiculus of nerve fibers divides into several bundles which engage in plexus formations. This effects the first mixing of sensory and motor fibers.

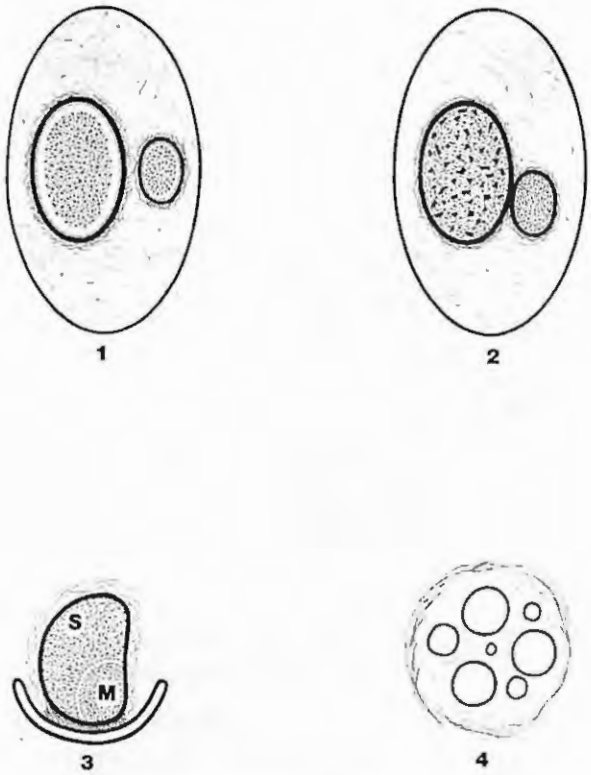


Fig. 2. Transverse sections across the nerve complex, illustrating the transition from nerve roots, enclosed in their separate bilaminar sheaths, to the formation of the single funiculus of the spinal nerve and its division into several funiculi (not to scale). The only attachment of the nerve complex to bone is in the cervical region where the spinal nerve is adherent to the gutter of the transverse process. *S*=sensory fibers; *M*=motor fibers.

(ii) *The cross-sectional area of the foramen occupied by the nerve complex.* Values given in the literature for the space occupied by the nerve vary from 10 to 50 percent;[3–8] though it is not clear from these accounts whether the term "nerve" included or excluded the investing sheath. More recent observations for nerve and sheath gave values of 35 to 50 percent.[9] The significant point is that there is ample space between the complex and the periosteal lining of the foramen. This space is occupied by loose areolar connective and adipose tissue containing the spinal artery, and its anterior and posterior branches, with numerous veins which often surround the complex, lymphatics and the recurrent meningeal nerve.

(iii) *The position of the neural complex in the intervertebral foramen.* The first cervical root is aligned horizontally while the remaining roots are directed downwards with gradually increasing obliquity until the lumbar and sacral roots descend vertically before reaching their respective foramina.

The usual arrangement is for the nerve roots to pass through the dura, ensheathed in their menin-

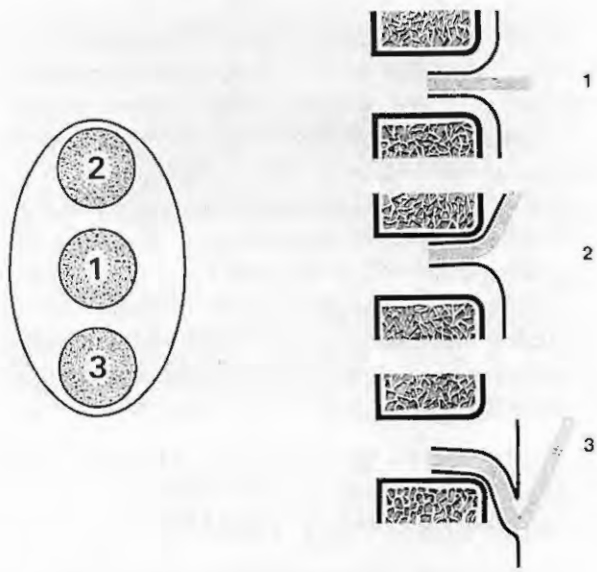


Fig. 3. Diagram illustrating variations in the position occupied by the nerve complex in the foramen. For simplification the complex has been represented as a single bundle.

geal sleeves, opposite the corresponding foramen so that the neural complex occupies a central position on entering the foramen (figure 3). Variations in the level at which this occurs may bring the nerve roots closer to the upper or lower margin of the foramen; in the case of the obliquely and vertically aligned roots an increasingly superior position on entry is favored, the complex becoming increasingly obliquely aligned in the foraminal canal. However, in the lower cervical and upper thoracic regions, it is common for the nerve roots to descend intradurally to a level which may be as much as 8 millimeters below the foramen which they are to enter.[10–21] They then perforate the dura in the

usual way but must ascend acutely, enclosed in their dural sleeves, in order to reach the foramen over the lower margin of which they are again angulated as they pass outwards. Such angulated nerve roots occur in about 40 percent of cases under the age of 25 years; between 25 and 40 the incidence increases to from 71 percent [18] to 76 percent.[21]

Finally the position of the complex in the foramen changes with movements of the head and neck. With ventroflexion the nerve roots are tensed and the complex is drawn inwards and upwards toward the upper margin of the foramen; in dorsal extension the complex is relaxed and returns to its original position.[16, 22–27] The nerve roots maximally involved in this way are the eighth cervical to the fifth thoracic;[28] but ventroflexion of the cervical spine also tenses the lumbar and sacral nerve roots.[24, 29, 30]

2. THE MOBILITY OF THE NERVE COMPLEX IN THE INTERVERTEBRAL FORAMEN

The nerve complex is not attached to the wall of the foramen, the arrangement permitting it to move within and through the foramen.[9] There is abundant evidence, dating from Dana's observations reported in 1882 down to the present time,[32] that traction on peripheral nerves tenses nerve roots. Reference has already been made to the tension developed in the nerve roots and the displacement inwards of the complex with ventroflexion of the head and cervical spine.

The elastic properties of the nerve roots allow them to accommodate to the tension generated in this way though there are limits to this elasticity. It is important that the roots lack the tensile strength of peripheral nerves for it has been shown that under increasing tension nerve roots fail before peripheral nerves.[33] The greater vulnerability of nerve roots to traction deformation is due to structural differences. In nerve roots the nerve fibers are arranged in parallel bundles, each fiber being enveloped in an endoneurial sheath in which the collagen fibers are fewer and finer than those surrounding nerve fibers in peripheral nerve trunks.[34] Nerve roots also lack a protective epineurial and perineurial connective tissue sheath which gives added tensile strength to the peripheral nerve.

The extent to which a nerve root can be stretched before structural failure occurs depends, inter alia, on its initial length, short nerve roots suffering earlier and failing structurally before long roots.[33] Nerve roots range in length from 10 to 168 millimeters, the shortest being the cervical roots and the longest the sacral.[32] In addition, the long roots are slacker in the spinal canal.

Two anatomical features protect nerve roots from being overstretched. One concerns the arrangement of the dura at the entrance to the foramen and the other the attachment of certain of the cervical spinal nerves to the vertebral transverse processes.

(i) *The arrangement of the dura.* Because the dural sheath of connective tissue becomes adherent to and part of the nerve complex laterally, traction on the spinal nerve pulls the entire system outwards so that the dural funnel is also drawn laterally into the foramen and, being cone-shaped, plugs the foramen in such a way as to resist further dislocation of the nerve laterally (figure 4). Thus the

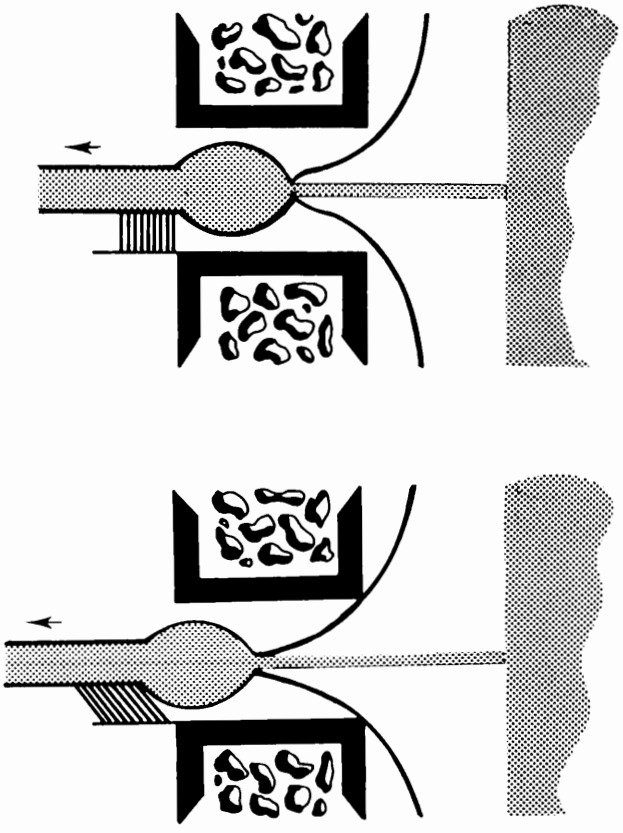


Fig. 4. Diagram showing the manner in which nerve roots are protected from being overstretched during lateral traction on the spinal nerve. Lateral displacement of the nerve complex is limited because of the attachments of the spinal nerve to the cervical transverse process and subsequently, and where no such attachments exist, by the plugging action of the dural funnel as it is drawn into the foramen. For simplification, only one dorsal rootlet is shown.

strength and integrity of the system are due, not to any strong meningeal attachment to the foramen, but to the continuity of the nerve sheath with the dural sac.[9]

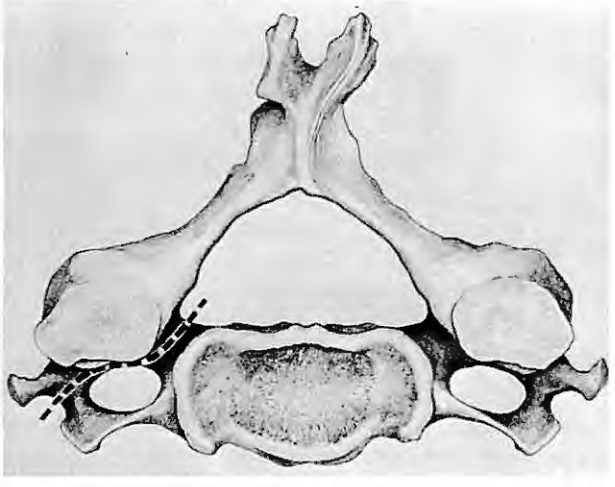
(ii) *Attachments of the spinal nerves.* During the wide range of movements occurring at the cervical spine, shoulder girdle, and shoulder joint, additional strains are generated which fall maximally on the upper spinal nerves forming the brachial plexus which, if transmitted directly to the corresponding short cervical nerve roots, would expose them to traction injury. In order to protect the roots against such injuries, the fourth, fifth, sixth, and seventh cervical nerves are securely attached to the vertebral column. Each, on leaving the foramen, is immediately lodged in the gutter of the transverse process to which it is securely bound by its epineurial sheath, by reflections of the prevertebral fascia, by slips from the musculo-tendinous attachments to the transverse processes and by fibrous slips that descend from the transverse process above to blend with the epineurium of the nerve below (figures 2, 5, 6). The nerve is also held backwards against the posterior bony bar of the transverse process by the vertebral artery whose adventitial coat blends with the sheath of the nerve (figure 7). The full significance of this arrangement emerges when the relative susceptibility to avulsion injury of the several nerve roots contributing to the brachial plexus is examined. Traction injuries, which do not avulse nerve roots, more commonly involve the upper spinal nerves of the plexus whereas the incidence of avulsion injuries is much higher in the case of the lower nerve roots, which, anatomically, are at greater risk.[35]

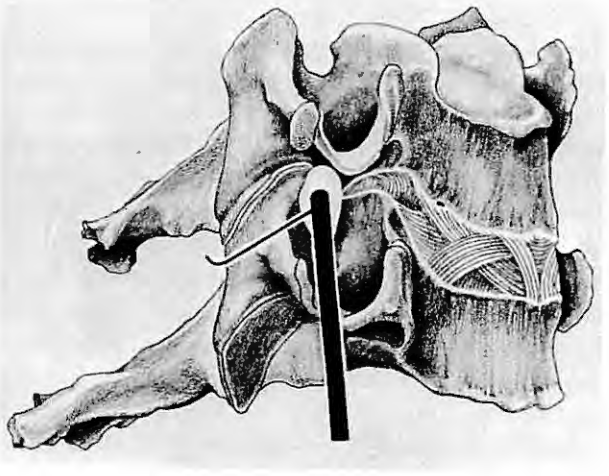
Such protective attachments are not needed for the other spinal nerves.

3. THE NERVE COMPLEX AND THE POSTERIOR VERTEBRAL JOINT SYSTEM

Cervical Region. The first and second cervical spinal nerves are unusual in that they leave the spinal canal *behind* the joint (figure 8). The first occupies a groove on the upper surface of the posterior arch of the atlas where it is behind and a little below the capsule of the atlanto-occipital joint and beneath the vertebral artery. In this situation the nerve and the vessel traverse an osseo-fibrous foramen outlined by the bony arch and the lower border of the posterior atlanto-occipital ligament. The second cervical nerve crosses the arch of the axis immediately behind and in contact with the capsule of the lateral atlanto-axial joint. Here it leaves the spinal canal through a small osseo-fibrous opening formed by a deficiency in the bony attachment of the ligamentum flavum.

In the case of the third to the seventh spinal nerves the nerve complex is lodged in the gutter formed by the articular pillar posteriorly, the pedicle of the subjacent vertebra and the extension upwards of the crested lateral lip (processus uncinatus) of the vertebral body anteriorly (figures





Figs. 5 and 6. Diagrams of cervical vertebrae illustrating the boundaries of the intervertebral foramen, the formation and depth of the gutter of the transverse process and the relationship of the nerve roots and spinal nerve to these anatomical features. The apophyseal joint is above and behind the nerve complex. It can also be seen that any reduction in the distance between adjacent vertebral bodies can not alter the vertical diameter of the foramen but results in tilting which would stretch the joint capsule.

5, 6, 7). The foramen is completed superiorly by the pedicle of the vertebra above. The eighth cervical nerve emerges from the foramen between the last cervical and first thoracic vertebrae and then descends to unite with the first thoracic nerve anterior to the neck of the first rib.

The loose joint capsule of the articular pillar is located postero-superiorly with, further medially, the lateral margin of the ligamentum flavum (figure 6). The intervertebral disc and the neuro-central joint space, if such exists, form the antero-superior boundary of the foramen.

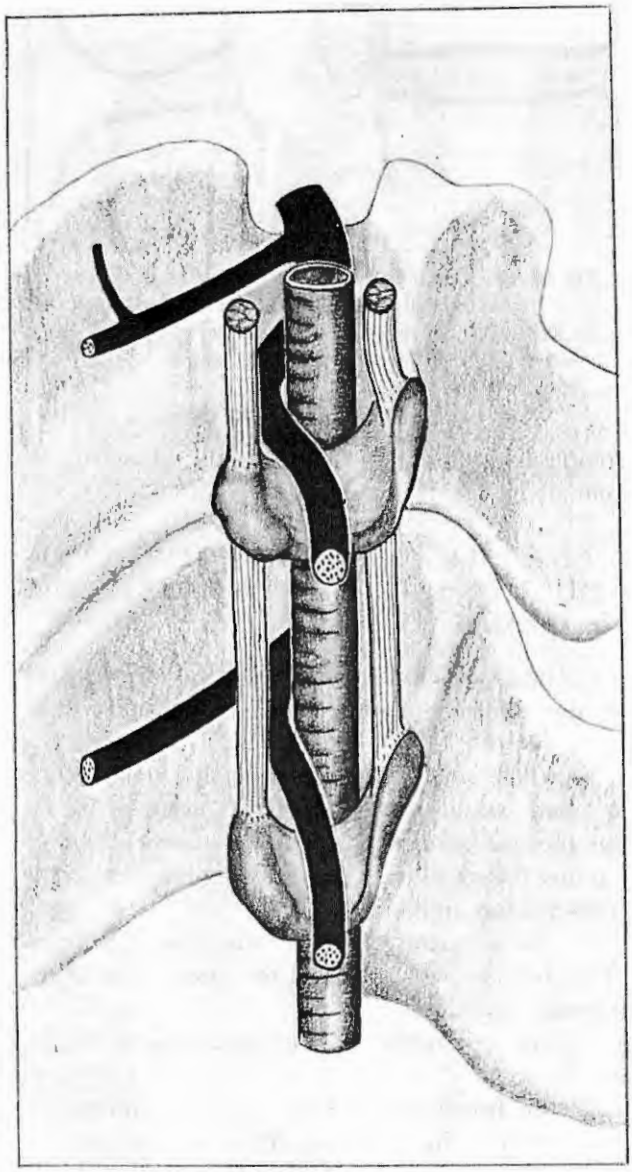


Fig. 7. Diagram to illustrate the manner in which the spinal nerve is forced against the posterior bony bar of the transverse process by the vertebral artery.

Because of the disposition of the vertebral joint, any reduction in the thickness of the disc would have little, if any, effect on the vertical diameter of the foramen. The capsule, however, would be nipped between the superior margin of the articular facet below and the pedicle of the vertebra above while tilting of the vertebral body would stretch the posterior part of the capsule of the joint.

Thoracic Region. Here the loose capsule of the apophyseal joint is behind the nerve complex with, further medially, the lateral margin of the ligamentum flavum. However, the level of the joint with reference to the complex varies. In the upper thoracic region it is situated postero-superiorly as in the cervical region. It then gradually moves down the posterior wall of the foramen until it is finally located postero-inferiorly as in the lumbar region. The intervertebral disc forms the

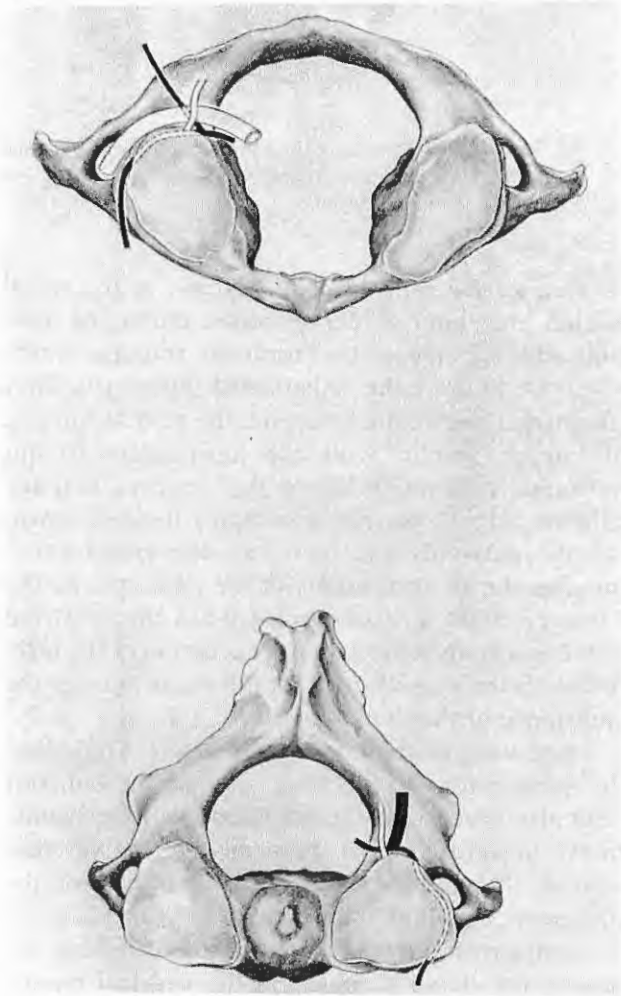


Fig. 8. Diagram illustrating the course of the first and second cervical spinal nerves, and their primary rami, in relation to the atlas and the axis.

antero-inferior margin of the foramen (figure 9). Narrowing of the disc would result in subluxation of the joint and a reduction in the vertical diameter of the foramen.

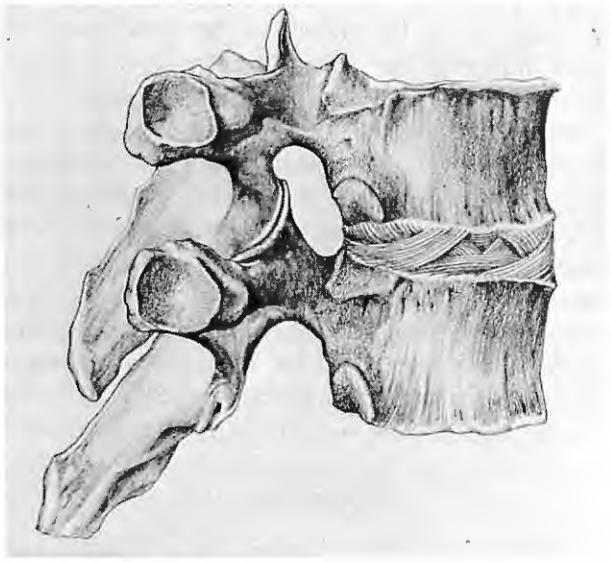


Fig. 9. Diagram illustrating the formation of the boundaries of a typical thoracic intervertebral foramen. The apophyseal joint is behind the nerve complex.

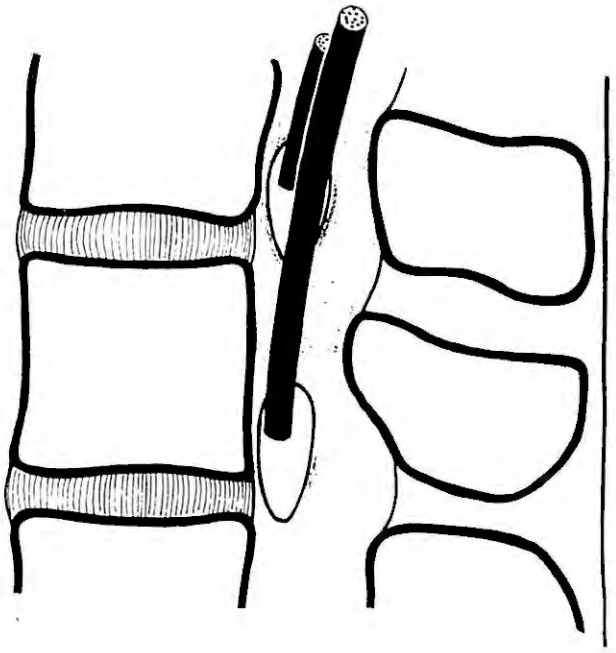


Fig. 10. Diagram illustrating the relationship of nerve roots to the intervertebral discs as they descend in the spinal canal to reach their respective foramina. The nerve leaves the foramen proximal to the disc forming its lower anterior boundary but is directly related to the disc above.

Lumbar Region. As they descend in the spinal canal, the lumbar nerve roots cross the disc immediately above the foramen through which they are to leave the spinal canal (figure 10). They then enter the foramen beneath the pedicle forming its upper margin with the ligamentum flavum posterior and the body of the vertebra anterior (figure 11). As the nerve complex inclines downwards, outwards and forwards, the spinal nerve crosses the anterior aspect of the joint, but, by the time it is at the level of the disc, it has emerged from the foramen to pass, with the exception of the fifth, between the slips of origin of the psoas to enter the substance of that muscle.

Narrowing of the intervertebral disc would lead to subluxation of the joint so that the superior articular process of the subjacent vertebra would move upwards toward the pedicle of the vertebra above, thereby encroaching on that part of the foramen containing the nerve.

Summarizing, the apophyseal joint is behind and above the nerve complex in the cervical region, directly behind it in the mid-thoracic region and behind and below it in the lumbar region. It should also be noted that it is the sensory fibres of the complex which are most intimately related to the joint.

4. CHANGES RESULTING IN A REDUCTION IN THE DIMENSIONS OF THE FORAMEN

1. The foramina are narrowed in dorsal extension of the spine and are widened during ventroflexion.[4–7, 15, 29, 36]

2. While congestion of the many veins in the foramen would reduce the free space available, the profuse venous anastomotic network characterizing the vertebral venous system makes such a complication unlikely.

3. Pathological changes affecting the intervertebral disc which reduce the dimensions of the foramen include:

(i) narrowing of the disc which, in turn,

(a) brings the pedicles together and so reduces the vertical diameter of the foramen;

(b) results in subluxation of the apophyseal joint with stretching of the capsule and other joint changes;

(c) leads to folding of the ligamentum flavum which may bulge sufficiently far forwards to encroach on the entrance to the foramen.

(ii) herniation of the disc and osteophyte formation involving the vertebral rim, both of which encroach on the foramen anteriorly.

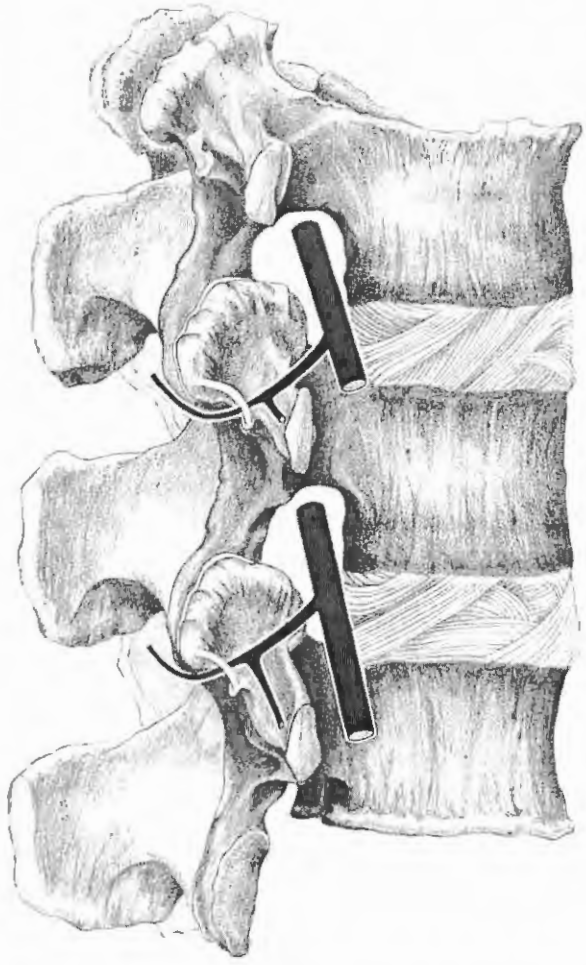


Fig. 11. Diagram illustrating the passage of the lumbar nerve through the upper part of the foramen where it is behind the vertebral body and immediately below its pedicle. The course of the dorsal ramus across the lateral surface of the articular process is also shown along with the passage of the medial branch of the ramus through a tunnel beneath the ligamentous tissue joining the mamillary and accessory processes—only the lateral fibers of this ligamentous arch are shown.

4. Pathological thickening of the ligamentum flavum producing the same effect as in 3(i)c.

5. Pathological changes involving the apophyseal joint which encroach on the foramen from behind are:

(i) joint swelling. Injecting saline into "the joint capsule" narrows the foramen by 2 millimeters.[37]

(ii) osteophyte formation.

6. The anatomy of the foramen and its contents suggests that the reduction in the dimensions of the foramen would need to be considerable before the nerve complex would be compressed. Moreover, nerve tissue tolerates slow compression reasonably well.

The arrangement favors friction fibrosis and the formation of adhesions, induced by the repeated movement of the nerve complex over or against periforaminal pathology of the type outlined above, as a more likely cause of nerve involvement and the development of symptoms.

5. THE BRANCHES OF THE SPINAL NERVES IN RELATION TO THE POSTERIOR VERTEBRAL JOINTS

First and second cervical nerves. Each gives a recurrent meningeal branch for the dura before dividing into ventral and dorsal rami. In each case the ventral ramus, on its way to a prevertebral position (figure 8), curves anteriorly under the overhanging rim of the lateral mass of the vertebra and across the base of its transverse process where it is immediately below the lateral surface of the capsule of the neighbouring joint which it innervates.

The dorsal ramus of the first nerve has no cutaneous distribution but provides a sensory innervation to the atlanto-occipital joint, the ligaments of the region and the suboccipital muscles for which it also provides a motor supply. The dorsal ramus of the second nerve is the largest dorsal ramus of the spinal series. It carries some motor but mostly sensory fibers and divides into a small lateral and large medial branch which, after a circuitous course as the greater occipital nerve, pierces the semispinalis and then the trapezius muscle at its attachment to the skull where it traverses a tendinous tunnel or passes under a tendinous arcade to become cutaneous.

The remaining spinal nerves. Immediately on emerging from the foramen each spinal nerve gives a recurrent meningeal branch which passes back through the foramen to be distributed to the dura, the intervertebral disc and associated structures.

The spinal nerve terminates after a course of a few millimeters by dividing into ventral and dorsal

rami; with the exception of the upper cervical nerves, the ventral are larger than the dorsal.

The ventral rami are directed outwards away from the vertebral column. Juxtavertebral branches are the sympathetic grey rami to each branch and the white sympathetic rami of the eighth cervical to the second lumbar nerves destined for the ganglionated sympathetic chain and those from the second, third, and fourth sacral nerves for parasympathetic ganglia in the pelvis.

On its way to the posterior compartment of the back, each posterior ramus is accompanied by an artery and its associated vein to form a neurovascular bundle. This bundle and the medial and lateral branches into which it divides establish important relations with the apophyseal joint system which merit further attention.

6. THE DORSAL RAMI

The cervical dorsal rami. These diminish rapidly in size from above downwards (figure 12). The first two have already been described. Those of the third to the seventh cervical nerves cross the lateral surface of the articular pillar which is grooved to carry the nerve and an accompanying artery, the groove being converted into an osseo-fibrous tunnel by the posterior inter-transverse ligament and muscle. On emerging from the tunnel the ramus gives branches to the vertebral joint above and below. On its way posteriorly the eighth ramus crosses and grooves the upper surface of the first rib. It then establishes a relation with the vertebral joint which is essentially the same as that to be described for the thoracic nerves.

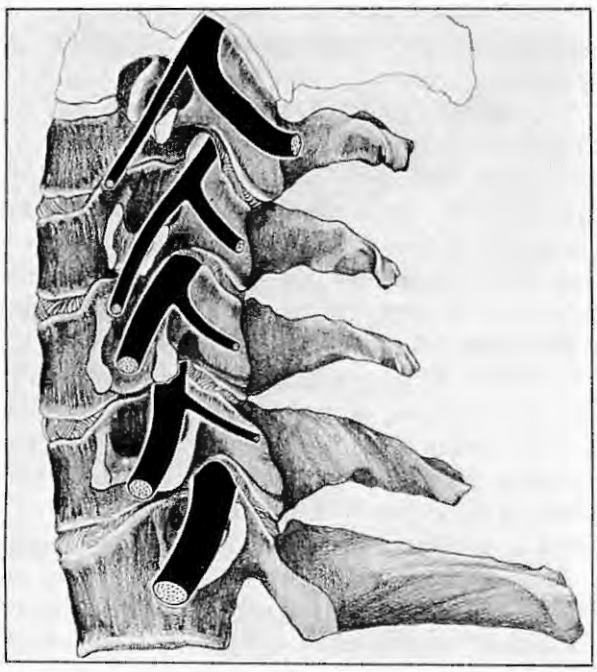


Fig. 12. Diagram illustrating the course of the cervical dorsal rami in relation to the articular pillars. The rami diminish in size from above downwards.

Each ramus terminates behind the articular pillar by dividing into a larger medial and smaller lateral divisions. Both send branches to ligaments and muscles. In addition, the medial provides further twigs to the joint before becoming cutaneous just lateral to the midline to innervate the skin; cutaneous fibers may be absent from the lower cervical rami.

The thoracic dorsal rami. The nerve passes backwards across the apophyseal joint to enter a roomy osseo-fibrous opening formed medially by the apophysis, superiorly by the transverse process, inferiorly by the neck of the rib and the upper border of the subjacent transverse process, and laterally by the superior costo-transverse and intertransverse ligaments and the intertransverse muscle. In its course the nerve is in contact with the joint capsule to which it sends branches.

Posteriorly the nerve curves medially for a short distance before dividing into medial and lateral branches. The medial continues inwards and downwards to give articular, muscular, ligamentous and, in the case of the upper thoracic nerves, cutaneous branches. The lateral branch runs outwards between the layers of the superior costo-transverse ligament to reach and innervate the deep muscles of the back; those of the lower thoracic nerves also have a cutaneous distribution.

The twelfth thoracic and lumbar dorsal rami (figures 11, 13, 14). The dorsal ramus runs downwards and backwards across the lateral surface of the adjacent superior articular process from a point immediately above and anterior to the vertebral joint, to which it gives twigs, to reach and cross the upper surface of the base of the transverse process. The nerve, with an accompanying artery, enters the posterior compartment of the back by passing through a large oval foramen (15 mm x 4 mm) outlined laterally by the curved concave inner edge of the intertransverse ligament and medially by the articular processes. On entering the compartment the ramus divides into medial and lateral divisions as do the accompanying vessels. The site of this branching is readily located on the transverse

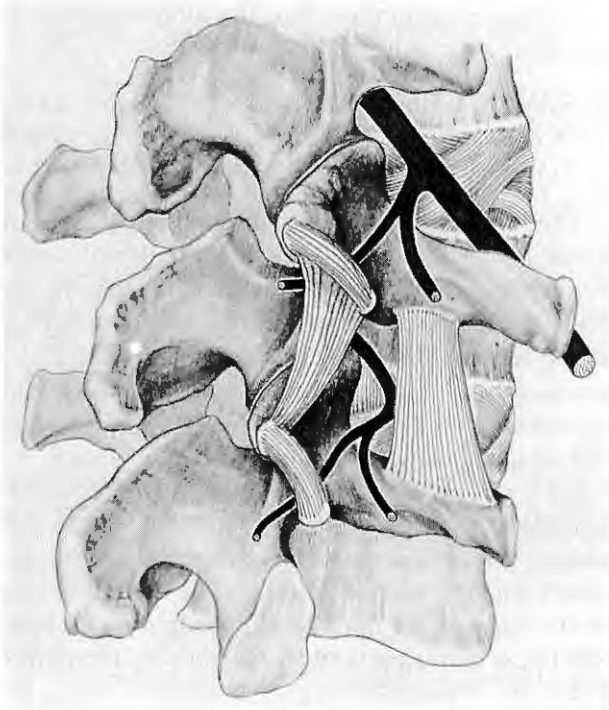


Fig. 13. Diagram illustrating the course of two lumbar dorsal rami and their branches. The medial branch passes through an osseofibrous tunnel outlined by the ligamentous and musculotendinous tissue arching between the mamillary and accessory processes.

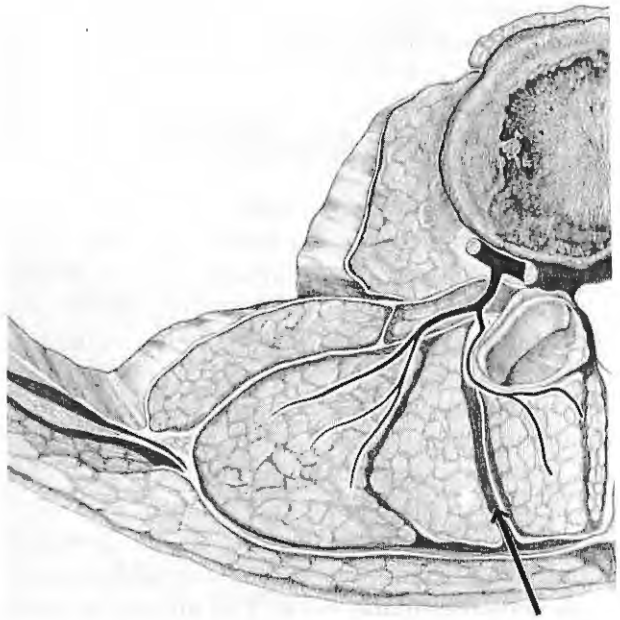


Fig. 14. Diagram illustrating the site of branching of a lumbar dorsal ramus on the transverse process at the bottom of a tissue plane between the multifidus muscle medially and the longissimus muscle laterally. The course of the medial branch is also shown in relation to the vertebral joint. The terminal fibers of the lateral branch are not shown.

process at the bottom of a connective tissue plane between the multifidus muscle medially and the longissimus group laterally (figure 14).

The finer medial branch, somewhat less than 1 millimeter in diameter, continues freely before entering the groove between the accessory and mammillary processes beyond which it curves medially following the lower border of the joint. In the groove and over this infra-articular section, the neurovascular bundle is covered and tightly bound to bone by exceedingly dense tissue formed by fibrous extensions from the capsule of the joint, the intertransverse ligament and the tendinous attachments of the longissimus thoracis, multifidus, rotatores, and medial intertransverse muscles. Pedersen, Blunck and Gardner[38] refer, in passing, to a connective tissue covering for the nerve at this site but make no mention of either its thickness or density or to the nerve being confined in an osseo-fibrous tunnel. After a course of about 1 centimeter in this tunnel, the nerve emerges to pass on to the lamina. It sends branches upwards before turning downwards to descend for some distance, branching as it does so to form an openly arranged plexus along with corresponding branches of neighboring rami. The medial branch innervates ligaments and muscles medial to the line of the vertebral joints, while the joint itself is richly supplied by multiple articular twigs from both the parent ramus and its medial branch. In the curved transverse section of its course the medial branch also sends twigs downwards to the capsule of the joint below.

The larger lateral branch, 1.5 millimeter in diameter, passes outwards and downwards to innervate muscles and ligaments lateral to the line of the vertebral joints. In the case of the 12th thoracic and upper three lumbar nerves the lateral branch descends through muscle to pierce the lumbo-dorsal fascia just above the iliac crest before descending vertically across the crest to innervate the skin of the buttock as far down as the greater trochanter.

The fifth lumbar ramus crosses the sacrum immediately lateral to the lumbosacral joint where it divides into medial and lateral branches (figure 15). The former turns inwards below the joint to supply it before continuing on to terminate in the multifidus. The lateral is lost in the sacroiliac ligament.

The sacral dorsal rami and the plexus on the dorsal surface of the sacrum (figure 15). On emerging from the posterior sacral foramina each dorsal

597-036 O - 76 - 10

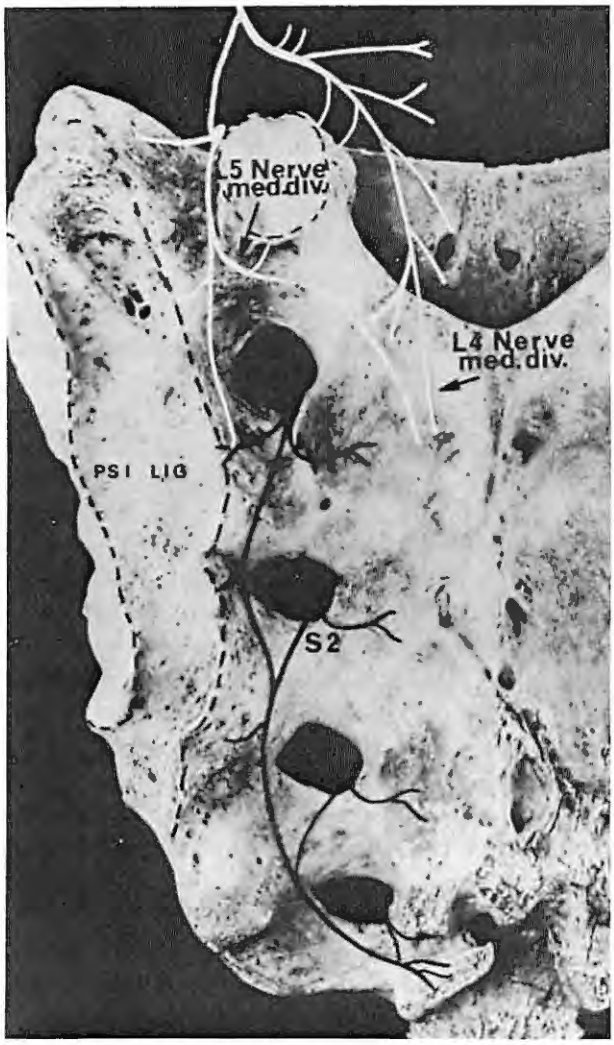


Fig. 15. The nerve plexus formed on the dorsum of the sacrum by the dorsal rami of the lower lumbar and sacral nerves.[39] The overlap of nerve branches from different cord segments is shown (by kind permission of Dr. K. C. Bradley).

ramus divides into a finer medial and a larger lateral branch. These are in turn linked by a complicated system of overlapping branchings and anastomotic loopings to form a plexus which is deeply situated on bone and ligamentous tissue beneath the multifidus and sacrospinalis muscles with extensions between the deep and superficial parts of the posterior sacroiliac ligament. The plexus is joined by the descending medial divisions of the fourth and fifth lumbar rami which reach as far distally as at least the second sacral segment. Terminal medial branches of the system innervate muscles while the lateral are distributed to muscles along with the sacroiliac, sacrotuberous, sacrospinous and iliolumbar ligaments; in addition, those branches derived from the first three sacral nerves send cutaneous branches through the gluteus maximus muscles to the skin.

7. SOME GENERALIZATIONS ON THE TERMINAL DISTRIBUTION OF THE DORSAL RAMI

The line of the posterior vertebral joints marks a tissue plane at the base of which the dorsal ramus divides into medial and lateral branches. This tissue plane represents a watershed for the distribution of the terminal branches of the two divisions of the ramus. Muscles and ligaments medial to this plane are innervated by the medial branch and muscles and ligaments lateral to it by the lateral branch.

Each vertebral joint is supplied by several fine branches from the rami of at least two spinal nerves; these come from the main trunk of the ramus and its medial branch. The inferior parts of the capsule are innervated by the ramus below and the superior parts of the capsule by the ramus above.

In the case of the cervical and upper seven thoracic nerves, the medial branch is the larger of the two and, additionally, contains cutaneous fibers. Below this level the reverse arrangement obtains, the lateral being the larger and containing the cutaneous fibers.

With the exception of the greater occipital (C2) and third occipital (C3) nerves all the cutaneous branches become subcutaneous at a lower level than the origin of their parent ramus, the distance between these two levels increasing from above downwards.

The cutaneous sensory territory innervated by the dorsal rami covers an extensive area extending from the vertex above to the coccyx below. Laterally it extends to a line which follows the outer border of the trapezius to the acromion, then to the lower angle of the scapula from which it continues vertically downwards along the posterior axillary line to the greater trochanter before turning medially to the coccyx.

On their way to the skin, the cutaneous branches pass through muscles before finally emerging through the tough fascial tissue coating the superficial muscles of the back. The greater occipital nerve emerges beneath a tendinous arcade at the attachment of the trapezius to the skull while the lateral branches of the first three lumbar rami are contained in tunnels of the lumbodorsal fascia where they pierce this sheet to descend across the iliac crest.

Both the medial and lateral divisions of each dorsal ramus achieve, by a plexus pattern formed by long ascending and descending branches, a considerable degree of overlap with the corresponding branches of neighboring rami. This overlap may extend over several segments. Thus fibers from the fourth lumbar ramus reach at least as far distally as the second piece of the sacrum.

CLOSING COMMENT

Recapitulating, anatomical features worthy of special attention as potential sites of nerve involvement are:

1. The intervertebral foramen. Normally, the dimensions of the foramen in relation to the cross-sectional area occupied by its neural contents leave ample room for the latter. The narrowing of the foramina which occurs during extension of the vertebral column does not embarrass its contents. The nerve complex is free to move in the foramen which allows it to adjust throughout the normal range of vertebral movements. While pathological changes in and about the foramen may reduce its dimensions and lead to nerve compression, more likely causes of nerve involvement at this site are friction over osseo-fibrous irregularities or traction on a nerve or nerve roots fixed in the foramen by adhesions.

2. The passage of the medial branch of the lumbar dorsal ramus and its accompanying vessels through an osseo-fibrous tunnel and the intimate relationship of this neurovascular bundle to the capsule of the apophyseal joint represents a potential site of fixation and entrapment following pathological changes involving the joint.

3. The passage of the cutaneous branches through the muscles and fascia of the back should not be overlooked as potential sites of entrapment. This applies particularly to the greater occipital nerve and the cutaneous branches of the dorsal rami of the upper three lumbar nerves.

When investigating the problem of back pain and the status of spinal manipulative therapy, one cautions against concentrating exclusively on the intervertebral foramen as the site of the offending lesion lest this obscure the significance of etiological factors originating in the posterior vertebral joints and ligaments, and in the osseo-fibrous canal where a neurovascular bundle could be entrapped.

REFERENCES

1. Soulié, A. *Nerfs rachidiens*. P. Poirier and A. Charpy. Traité d'anatomie humaine. Paris. Masson et Cie. 1899, Vol. 3, Fasc. 3, 2nd ed, pp. 923–944.
2. Hovelacque, A. *Anatomie des nerfs craniens et rachidiens et du système grand sympathique chez l'homme*. Paris. Gaston Doin et Cie. 1927, p. 300.
3. Swanberg, H. The intervertebral foramina in man. *Med. Rec.* 87, 176–180, 1915.
4. Hadley, L. A. Anatomicoradiographic studies of the spine. *N.Y. State J. Med.*, 39, 969–974, 1939.
5. Hadley, L. A. Roentgenographic studies of the cervical spine. *Amer. J. Roentgenol.*, 52, 173–195, 1944.
6. Hadley, L. A. Constriction of the intervertebral foramen. *J. Am. Med. Assoc.*, 140, 473–476, 1949.
7. Payne, E. E., and Spillane, J. D. The cervical spine. Anatomico-pathological study of 70 specimens (using a special technique) with particular reference to the problem of cervical spondylosis. *Brain*, 80, 571–596, 1957.
8. Cyriax, J. *Cervical Spondylosis*. London. Butterworths, 1971, p. 50.
9. Sunderland, S. Meningeal-neural relations in the intervertebral foramen. *J. Neurosurg.*, 40, 756–763, 1974.
10. Pfitzner, W. Über Wachstumsbeziehungen zwischen Rückenmark und Wirbelkanal. *Morph. Jb.*, 9, 99–116, 1884.
11. DuVal, P., and Guillain, G. Pathogénie des accidents nerveux consécutifs aux luxations et traumatismes de l'épaule. Paralysies radiculaires traumatiques du plexus brachial. *Arch. Gén. Méd.*, 2, 143–191, 1898.
12. Baldwin, W. M. The topography of spinal nerve roots. *Anat. Rec.*, 2, 155–156, 1908.
13. Cordier, Coulouma, and Van Varseveld. L'anatomie et l'importance clinique du nerf ganglio-radiculaire. *Encéphale*, 31, 139–158, 1936.
14. Frykholm, R. Deformities of dural pouches and strictures of dural sheaths in the cervical region producing nerve root compression. *J. Neurosurg.*, 4, 403–413, 1947.
15. Frykholm, R. Cervical nerve root compression resulting from disc degeneration and root-sleeve fibrosis. A clinical investigation. *Acta Chir. Scand.*, suppl. 160, 1951.
16. Frykholm, R. Lower cervical nerve roots and their investments. *Acta Chir. Scand.*, 101, 457–471, 1951.
17. Reid, J. D. Ascending nerve roots and tightness of dura mater. *N. Z. Med. J.*, 57, 16–26, 1958.
18. Reid, J. D. Ascending nerve roots. *J. Neurol. Neurosurg. Psychiat.*, 23, 148–155, 1960.
19. Roth, M. Caudal end of the spinal cord. I. Normal pneumographic features. *Acta Radiol.* (Diag.), 3, 177–188, 1965.
20. Shapiro, R. *Myelography*. Chicago. Year Book Medical Publishers, Inc., 1968.
21. Nathan, H., and Feuerstein, M. Angulated course of spinal nerve roots. *J. Neurosurg.*, 32, 349–352, 1970.
22. Inman, V. T., and de C. M. Saunders, J. B. The clinico-anatomical aspects of the lumbosacral region. *Radiology*, 38, 669–678, 1942.
23. O'Connell, J. E. A. Sciatica and the mechanism of the production of the clinical syndrome in protrusions of the lumbar intervertebral discs. *Brit. J. Surg.*, 30, 315–327, 1943.
24. O'Connell, J. E. A. Protrusions of the lumbar intervertebral discs. A clinical review based on five hundred cases treated by excision of the protrusion. *J. Bone Jt. Surg.*, 33B, 8–30, 1951.

25. O'Connell, J. E. A. Discussion on cervical spondylosis. *Proc. Roy. Soc. Med.* (Section of Medicine), 49, 202–208, 1956.
26. Frykholm, R. The mechanism of cervical radicular lesions resulting from friction or forceful traction. *Acta Chir. Scand.*, 102, 93–98, 1952.
27. Cave, A. J. E., Griffiths, J. D., and Whiteley, M. M., Osteoarthritis deformans of the Luschka joints. *Lancet.*, 1, 176–179, 1955.
28. Reid, J. D. Effects of flexion-extension movements of the head and spine upon the spinal cord and nerve roots. *J. Neurol. Neurosurg. Psychiatry*, 23, 214–221, 1960.
29. Breig, A. *Biomechanics of the Central Nervous System.* Stockholm. Almqvist and Wiksell, 1960, pp. 22, 23, 98.
30. Breig, A., and Marions, O. Biomechanics of the lumbosacral nerve roots. *Acta Radiol.* (Diag.), 1, 1141–1160, 1963.
31. Dana, C. L. The mechanical effect of nerve-stretching upon the spinal cord. *Med. Rec.*, 22, 113–115, 1882.
32. Sunderland, S. Avulsion of nerve roots. R. Braakman. *Handbook of Clinical Neurology.* Amsterdam. North-Holland Publishing Co., Vol. 25, Part I: Injuries of the Spinal Cord and Column. In the press.
33. Sunderland, S., and Bradley, K. C. Stress-strain phenomena in human spinal nerve roots. *Brain*, 84, 120–124, 1961.
34. Gamble, H. J. Comparative electron-microscopic observations on the connective tissues of a peripheral nerve and a spinal nerve root in the rat. *J. Anat.* (Lond.), 98, 17–25, 1964.
35. Sunderland, S. Mechanisms of cervical nerve root avulsion in injuries of the neck and shoulder. *J. Neurosurg.* 41, 705–714, 1974.
36. Turner, E. L., and Oppenheimer, A. A common lesion of the cervical spine responsible for segmental neuritis. *Ann. Int. Med.*, 10, 427–440, 1936.
37. Magnuson, P. B., Differential diagnosis of causes of pain in the lower back accompanied by sciatic pain. *Ann. Surg.*, 119, 878–891, 1944.
38. Pedersen, H. E., Blunck, C. F. J., and Gardner, E. The anatomy of lumbosacral posterior rami and meningeal branches of spinal nerves (Sinu-vertebral nerves). *J. Bone Jt. Surg.*, 38A, 377–391, 1956.
39. Bradley, K. C. The anatomy of backache. *Aust. N.Z. J. Surg.*, 44, 227–232, 1974.

Pathology of Spinal Root Compression

H. H. SCHAUMBURG
Associate Professor of Neurology
P. S. SPENCER
Assistant Professor of Pathology and Neuroscience
Albert Einstein College of Medicine

I. INTRODUCTION

Disease of the human spine with compromise of nervous tissue is of great importance in manipulative medicine. The relationship of the cervical spinal cord, spinal roots and meninges to the corresponding vertebral body is illustrated in figure 1. Both the cord and the roots are well protected from outside trauma by the vertebral body, neural arch and a tough, fibrous dura. However, when distorted by degenerative bone and joint disease or a variety of space-occupying lesions, these same protective layers may damage the delicate neural structures. Thus, in the lumbar region, vertebral body and intravertebral disc disease are potential sources of compression of the entire cauda equina and of the individual nerve roots (figure 2). Cervical and thoracic vertebral body and disc disease may compress the spinal cord in addition to the spinal roots. Because of the seg-

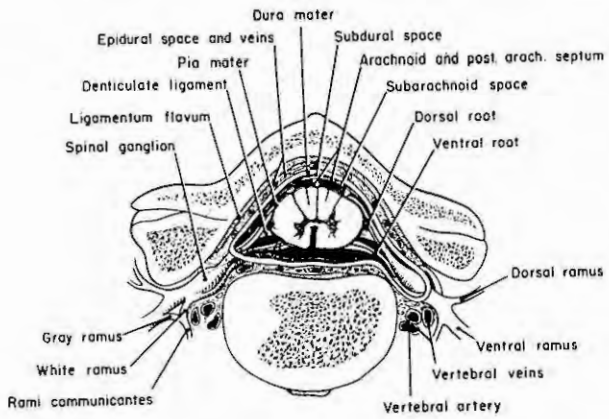


Fig. 1. Diagram of the spinal cord, nerve roots, and meninges by H. K. Corning (from Truex and Carpenter, *Human Neuroanatomy*, 1969; courtesy of the Williams and Wilkins Co.).

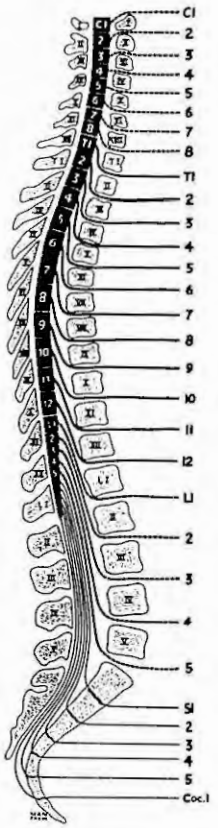


Fig. 2. Diagram of the position of the spinal cord segments with reference to the bodies and spinous processes of the vertebrae (from Haymaker and Woodhall, *Peripheral Nerve Injuries*, 1945; courtesy of the W. B. Saunders Co.).

mental variation in pathology, a knowledge of the anatomy of the spinal roots, their normal relationship to the vertebral body and to the intervertebral foramina, is essential to an understanding of spinal root neuropathology.

II. ANATOMICAL FACTORS

Each dorsal root is composed of myelinated (A) and unmyelinated (c) nerve fibers. Their axons are the centrally projecting processes of the dorsal root ganglion neurons, which are located within the intervertebral foramina. The large myelinated fibers conduct rapidly and transmit impulses from elaborate special receptors in the periphery; the smaller myelinated fibers and the very small unmyelinated fibers conduct slowly and transmit impulses from less specialized receptors in the periphery. Classically, the ventral roots have been held to be composed only of myelinated fibers. Recent work has determined that there are also many unmyelinated axons in the mammalian ventral root.[1] The myelinated fibers in ventral roots are: large myelinated A-alpha fibers carrying motor impulses to extrafusal muscle fibers, small gamma efferent motor fibers innervating the intrafusal fibers of muscle spindles and, in segments T_1 through L_2, preganglionic, sympathetic efferent, B fibers. Preganglionic, visceral efferent fibers of the parasympathetic system are also present in the ventral roots of sacral nerves 2, 3, and 4.[2]

Nerve root axons are ensheathed by Schwann cells. In the case of myelinated fibers, Schwann cells form regularly repeating lengths of myelin throughout the entire length of the roots. The Schwann cells are present up to the junction zones between the roots and the spinal cord. After entry into the spinal cord, the myelinating function of the Schwann cell is undertaken by the oligodendrogial cell. Some dorsal root fibers continue without synapse as spinal tracts extending up to the level of the cervical-medullary junction. Each bundle of root fibers is encased within a thin sheath, the outer part of which is continuous with the pia mater surrounding the spinal cord.[3] In contrast to peripheral nerves (figure 3), nerve roots have a sparse endoneurial connective tissue, a very thin root sheath and no epineurium (figure 4). Furthermore, root fibers run in parallel non-plexiform strands. All of the above factors probably make

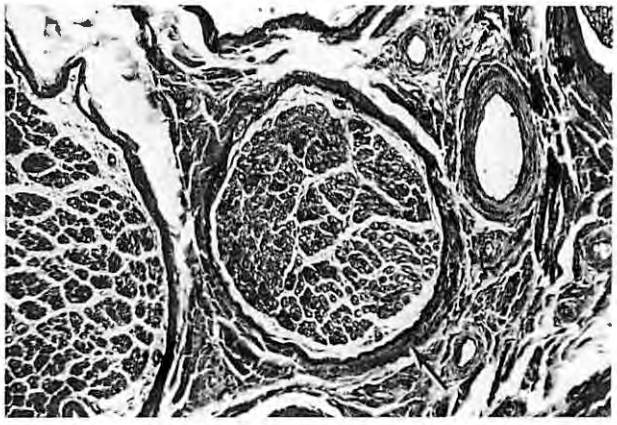


Fig. 3. Cross section of a portion of a normal human sciatic nerve. The arrow indicates the prominent perineurial sheath around a fascicle of nerve fibers. Blood vessels are prominent in the epineurial connective tissue (phosphotungstic acid-hematoxylin, X40).

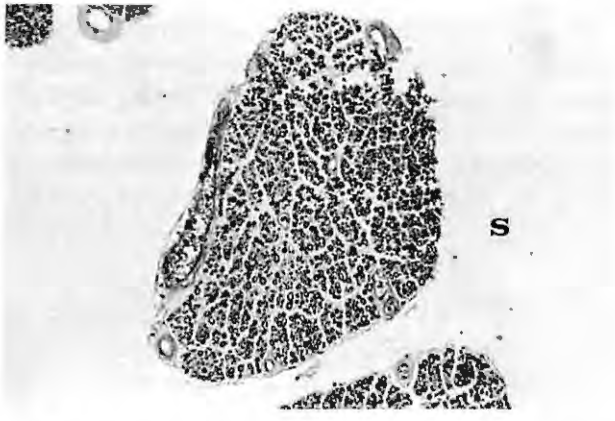


Fig. 4. Cross section of a bundle of normal human lumbar spinal root fibers in the subarachnoid space (*S*). The connective tissue sheath around the bundle is sparse when contrasted with the perineurial and epineurial sheaths around the peripheral nerve fascicle in Figure 4 (phosphotungstic acid-hematoxylin, X40).

the roots very vulnerable to pressure and to stretch.[4]

The spinal ganglion is located within a focally enlarged portion of the dorsal root which is sited at the intervertebral foramen. Distal to the ganglion, dorsal and ventral roots join to form the spinal nerve and the dura appears to be continuous with the perineurium and the epineurium of the peripheral nerve.[5] Dorsal roots (except for C_1) are approximately three times larger than the corresponding ventral roots. There is also a regional variation in root size; the lumbar and cervical roots

are the largest, since they contain nerve fibers which supply the limbs. Both the attachment of the radicular complex at the intervertebral foramen and the relationship of root size to foramen size probably are of critical importance in the production of compression syndromes. Sunderland has recently determined that the human cervical roots and dural pouch are only loosely attached at the foramen and that they account for 35 to 50 percent of the cross-sectional area of the foramen.[5] Since the lumbar and sacral roots are longer than the cervical and thoracic, and pursue a long subdural course before exiting from the spine through their intervertebral foramen, they may be compressed by vertebral degeneration at several levels. By contrast, the shorter cervical and thoracic roots are usually compressed only at the level of their respective intervertebral foramen. Each cervical root exits immediately above the body of the segmentally corresponding vertebra while the thoracic, lumbar, and sacral nerves exit in foramina below their corresponding vertebra.

III. PATHOLOGY

Since this symposium is concerned primarily with spinal misalignment, degenerative disease of the intravertebral disc and arthritic changes in the intervertebral joints, the present paper will be confined largely to the effects on the spinal roots. However, it is important to realize that there are other sources of progressive spinal root compression which are by no means rare. Prominent among these conditions are malignant neoplasms within the subarachnoid space. Metastases affecting either the vertebral body or the epidural space are also sources of root and spinal cord compression, and slowly growing lymphomas in the epidural space clinically may mimic disc disease. Benign neoplasms (meningiomas, neurofibromas, and schwannomas) and the effects of many non-neoplastic conditions (tuberculosis, arachnoid cysts, congenital bony and vascular anomalies) may also result in spinal root compression.[6,7]

a. Pathology of the Spine Relevant to Nerve Root Compression

There are two basically different pathologic reactions of the intervertebral articulations which may affect nerve roots.

1. The first is an acute herniation of the intervertebral disc into the epidural space. There is either a bulging of the nucleus pulposis and surrounding fibrous annulus, or a frank rupture of the annulus with extrusion of material around the often damaged posterior longitudinal ligament. These herniations are most common in the lumbar spine, affect young or middle-aged adults, often are related to exertion and usually occur in a dorsolateral direction, with compression of nerve roots within, or adjacent to the foramina. This type of intervertebral disc disease is responsible for many cases of the sciatic pain syndrome.[8]

2. The second pathologic reaction of the intervertebral articulation occurs with aging or with repeated trauma, is secondary to the gradual dessication of the nucleus pulposis, and is accompanied by degeneration of the disc and of the articular cartilages of the vertebral bodies adjacent to the bulging annulus. This newly-formed bony spur is referred to as an osteophyte and, in combination with the bulging disc, eventually may form an epidural ridge extending across much of the spinal canal. Commonly, osteophytes also form at the uncinate processes (Luschka joints) on the dorsal-lateral margins of the vertebral body, and indent the ventral portion of the intervertebral foramina, thereby impinging on the roots (figure 5).

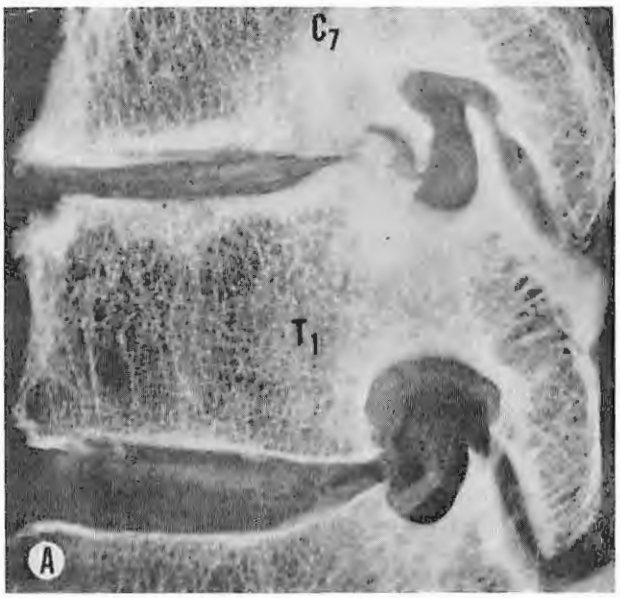
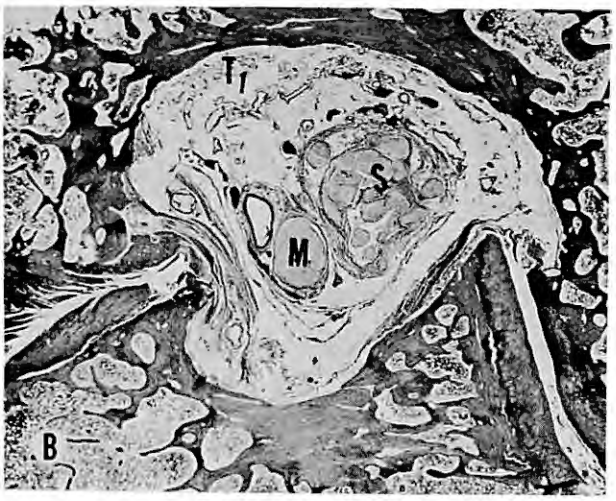
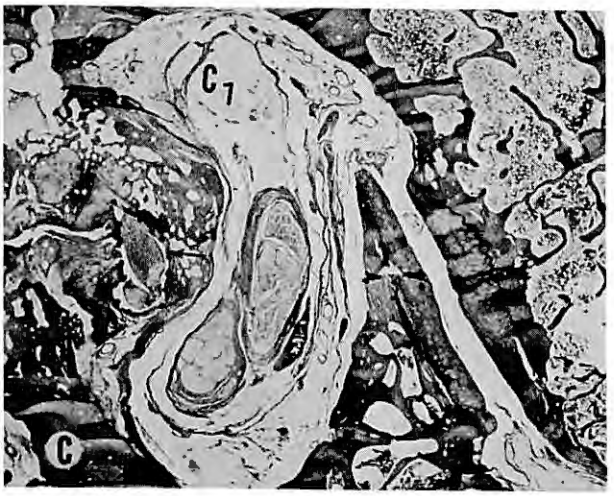


Fig. 5. A. Radiograph of a ¼-inch section at the cervicodorsal level of an 85-year-old male. The first thoracic foramen is essentially normal. At the C_7 level, however, a pair of osteophytes encroach upon the intervertebral foramen. There is also some telescoping of the posterior (diarthrodial) joint.



B. Section of the T_1 foramen (T_1), between the T_1 and T_2 vertebra. This foramen contains the T_1 roots. The dorsal sensory (S) root is larger than the ventral motor (M) root. A small ventral osteophyte (left) projects into the foramen. Compare with radiograph A.



C. Section of the C_7 foramen (C_7), between the C_7 and T_1 vertebrae. This foramen contains the C_8 roots. There is marked encroachment of the ventral portion of the foramen by osteophytes (left) and thickening of the root sheaths in contrast to the section illustrated in B (from W. K. Hadley, *Anatomico-Roentgenographic Studies of the Spine*, 1969; courtesy of the Charles C Thomas Co.).

Intervertebral disc disease may be concurrent with degeneration of the interpedicular (diarthrodial) joints. The diarthrodial joints form the posterior and lateral boundaries of the intervertebral foramina. When osteophytes develop on the margins of these joints, the dorsal portion of the foramen becomes indented. Therefore, if interpedicular joint and disc degeneration both occur at the same level, the intraforaminal root may be compressed from two directions. This constellation of vertebral joint changes is called spondylosis. It is especially common in relation to the mid-lower cervical vertebrae, has a gradual onset, may be asymptomatic or may lead to devastating compression of root and spinal cord, and is present to some degree in many elderly individuals.[9] Thickening of the dural root sleeve is also a normal accompaniment of old age and, in time, the dural sheath becomes firmly fixed in the intervertebral foramen. Most investigators believe this renders the root more vulnerable to osteophyte compression. Direct compression of the root by the thickened dura, with encroachment on the dural tunnel, was implicated by Frykholm as a principal cause of nerve root compression,[10] but this now seems less likely.[9]

b. Pathology of the compressed spinal root

Surprisingly few clinical or experimental pathological studies of nerve root compression have been reported. By comparison with the large volume of information on the pathology of peripheral nerve compression, data on root compression seem almost non-existent. No surgical biopsies of compressed roots are performed since the surgeon's purpose is to remove the source of compression and thereby to free the root. In many procedures designed to relieve bony intervertebral disc compression, care is taken not to incise the dura so the root often is not even visualized. Post-mortem descriptions of root compression are rare. In his monograph on the pathology of the spinal cord, Trevor Hughes was able to report only six cases of cervical radiculopathy that were discovered in a prospective search of a series of 200 adult necropsies.[11] The most extensive reports on root pathology are those of Hadley[12] and Lindblom and Rexed.[13] Unfortunately, no study has utilized teased nerve fiber or ultrastructurally-fixed preparations of nerve roots, and thus the relative amounts of damage to the myelin and to the axon are not known.

Some surgeons have observed spinal roots to be flattened at the site of acute disc herniation while other reports describe grossly edematous, hyperemic roots in the same condition. As previously stated, we have discovered no histopathological descriptions of acutely compressed spinal roots.

At post-mortem, roots compressed by chronic intervertebral disc herniation have been described as flattened and, in a few cases, almost fenestrated. In many instances, the spinal ganglion and corresponding nerve had adhered to the protruding disc. In the lumbar spine, Lindblom and Rexed found that most nerve root compressions were secondary to dorsolateral disc protrusion and only rarely due to compression from osteophytes of the dorsal interpedicular joints. The point of pressure proved to be between the distal half of the spinal ganglion and the first portion of the spinal nerve. The deformed section displayed either a slight flattening at a circumscribed area or an indentation of longer stretches of the nerve. In cases of slight pressure, the ventral root bundles were less deformed than the ganglia; however, with increasing degrees of compression, both ventral root and ganglion were flattened. Microscopically, the ventral roots appeared most obviously damaged although Lindblom and Rexed suggested this may have been an illusion, since the degeneration of large myelinated fibers was most easily seen in the ventral roots. Due to the limitations of the histological technique, no statement of the relative destruction of large myelinated fibers, compared to small myelinated or to unmyelinated fibers, was possible in their study. The dorsal root ganglia often were depressed so that their normal circular appearance in section was distorted to a crescentic or falcate shape. The internal organization of the connective tissue of the ganglion was disturbed; connective tissue was much increased and formed thick, concave septa parallel to the margin of compression. Near the compressed margin of the ganglion, some neurons appeared flattened, atrophied, and stained abnormally. However, the majority of the neurons appeared normal. That dorsal roots were largely spared was confirmed by the relative absence of nerve fiber pathology in the dorsal roots, root-entry zone, and the sparing of the lumbar and thoracic segments of the spinal cord. If significant axonal degeneration or neuronal loss occurs in the dorsal roots, their central projections in the dorsal columns also degenerate. An example of such a phenomenon is illustrated in figure 6, taken from a case with extensive dorsal root ganglion destruction produced by an inflammatory process. In lumbar disc herniations, it has been suggested[13] that the root damage does not follow a massive single trauma but results from many

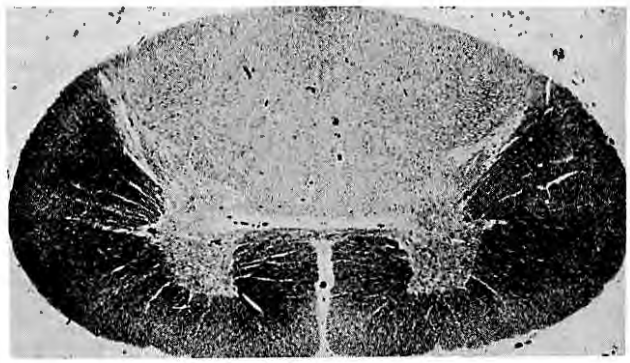


Fig. 6. A section of the entire lumbar spinal cord with degeneration of the posterior columns. These posterior column changes were secondary to the extensive degeneration in the dorsal roots present in a 57-year-old male with carcinomatous neuropathy (Loyez stain, X10).

repeated small blows. Each injury was not restricted to a sharply localized site but seemed to exert an effect over a relatively large area. As a result, degenerating fibers were distributed diffusely over the entire root, and their number was proportional to the severity and duration of the compression. Furthermore, in any one compressed root, they found evidence of both recent and old injuries. These repeated injuries gave rise to the most characteristic histopathologic feature of root compression—the presence of regenerating axons coexistent with actively degenerating fibers. Various stages of regeneration were present proportional to the time delay between fiber injury and investigator observation. The resulting picture was a mixture of preserved, degenerating, and regenerating fibers. In nerves with active degeneration, regenerating fibers appeared in clusters associated with proliferating Schwann cells. Sometimes the Schwann cells formed hypertrophic cellular whorls around the nerve fibers. There were retrograde degenerative changes extending only 1–2 centimeters into the lumbar ventral roots but, surprisingly, there was none in the dorsal roots.

In cervical spondylosis, spinal roots severely damaged by protruding osteophytes have been described.[6] These roots contained regenerating axons proximal to the level of damage which formed a neuroma at the entrance of the intervertebral foramen or, more commonly, gave rise to groups of myelinated fibers coursing through the meninges or passing along blood vessels. Such regenerative phenomena were very common in cases of chronic cervical spondylosis. Cuneiform

areas of degeneration in the dorsal columns of the spinal cord were also observed. The spinal cord change was interpreted to be secondary to damage in the dorsal roots (*vide supra*).

c. Pathology of Peripheral Nerves Relevant to Spinal Root Compression

Although little is known about the pathophysiology of nerve root compression, it seems reasonable to draw some inferences from clinical and experimental neuropathologic reports of mechanically-injured peripheral nerves,[14] since nerves and roots structurally are similar. Such studies have demonstrated the importance of defining the type of mechanical damage and the degree of disruption sustained by the nerve trunk. In general, damage to a nerve fiber appears to increase in proportion to the velocity, force, and duration of the traumatic agent, with the additional factors of traction and friction exaggerating the degree of injury.[15]

Experimental studies have indicated that peripheral nerve fibers (especially those of large diameter) are exquisitely vulnerable to localized pressure differences – the greater the pressure difference exerted on a nerve fiber, the more severe the resulting injury. Nerve root fibers probably behave similarly although the thin root sheath and sparse endoneurium may modify the injury response. In general, however, a peripheral nerve fiber may respond to trauma in one of two ways: focal demyelination and remyelination in a mild injury, and degeneration and regeneration in a more severe injury.

Demyelination: Mild nerve injuries frequently result in the focal loss of myelin sheaths (demyelination), leaving denuded lengths of axon which are subsequently repaired by remyelination. This type of damage probably produces a localized conduction block over the demyelinated region, with removal of the physiological block accompanying remyelination. In experimental animals, focal demyelination and conduction block may be produced beneath the edges of a moderately-inflated pneumatic tourniquet applied around a limb for a short period of time.[16, 17] Pressure differences at the edges of the cuff appear to cause intususseption of adjacent myelin sheaths, focal demyelination and subsequent remyelination.[18] This type of "mechanical neurapraxia"[19] may be analagous to the "Saturday night paralysis" syndrome which follows external compression of the radial nerve and in which function is restored after several days. It is conceivable that the transient clinical phenomena associated with some types of rapidly-remitting root compression syndromes (e.g. from a transiently herniated disc) are also associated with this type of injury.

A second type of experimental nerve injury, associated with segmental demyelination and remyelination, follows the focal surgical release of the perineurium which surrounds each peripheral nerve fasicle.[20] Immediately after intervention of the perineurium, the endoneurium and associated nerve fibers herniate through the opening.[21] Demyelination of the affected myelinated fibers occurs after 3 days and remyelination is evident by 2 weeks after injury. This experiment highlights the importance of localized pressure differences in the production of nerve injury.

The lesion of segmental demyelination is also a feature under the sites of chronic, progressive experimental[22, 23] and clinical compression lesions (e.g. the entrapment of the median nerve in an attenuated carpal tunnel). The nerve roots probably sustain this type of injury during chronic invasion of the intervertebral foramina by progressively enlarging osteophytes or a chronically herniated disc. If the entrapment is severe, and if friction and traction are also present, some fibers will undergo demyelination but more severely damaged fibers will suffer total axonal interruption. If the lesion is repetitive in nature, repeated demyelination and remyelination may occur. Experimentally[24], this is associated with repetitive Schwann cell proliferation and the formation of crescentric cellular processes (hypertrophic "onion-bulbs") around attenuated fibers. Such a combination of repeated trauma, traction, and friction, probably occurs in spondylosis and would account for the analogous pathological descriptions of human lumbar nerve roots affected in this condition.[13]

Axonal Degeneration: When a peripheral nerve fiber is subjected to a severe, focal pressure differential, interruption of axonal fiber continuity ensues (axonotmesis).[19] Experimentally, this lesion may be produced in some fibers by partially tightening a thread around a nerve, or in the entire nerve fiber population by locally crushing the nerve with forceps. It is most unlikely that peripheral nerve fibers located in nerve roots would respond differently to similar mechanical damage;

indeed, the sparse endoneurial connective tissue and thin root sheath may render nerve roots especially vulnerable. Clinically, severe, acute nerve root compression, caused by collision with a herniated disc or a protruding osteophyte during movement, presumably produces interruption of a variable number of nerve fibers beneath the site of injury.

In axonotmesis, interrupted nerve fibers recoil from the site of injury within the confines of their endoneurial tubes. Each distal stump, separated from its source of nourishment in the neuron cell body, undergoes wallerian degeneration. In this process, individual degenerating nerve fibers are replaced by columns of remodeled Schwann cell cytoplasmic processes confined to the original endoneurial tubes. The proximal stump undergoes a traumatic degeneration close to the site of injury. In time, regenerating axon sprouts emerge and grow along the columns of Schwann cell cytoplasm in the distal stump. Restoration of function is delayed since this is dependent on reconnection of the axon with the target organ and restoration of a myelin ensheathment.

Although reestablishment of function after simple focal crush injury is slow, a good repair is usually effected with time. However, in an abnormal foraminal canal, attenuated by an osteophyte, regenerating axons may encounter an area of nerve root entrapment as they grow distally. Data obtained from analogous experimental models by Weiss and Hiscoe[25] and Spencer[26] have demonstrated that the regenerating axons will enlarge proximal to the constriction. Surviving axons are unaffected by the constriction although they may become focally demyelinated. Persistence of the chronic constriction will induce the impeded regenerating axons to seek alternative routes for their growth. One possible pathway, demonstrated by experimental studies, is the perivascular connective tissue of the numerous vessels which grow into the proximal pretraumatic zone shortly after injury. Other regenerating nerve fibers may change direction and grow between perineurial lamellae. By employing these conduits, some regenerating fibers may escape the confines of the nerve trunk to form a lateral neuroma composed of miniature fascicles of misrouted regenerating fibers.[27] This phenomenon may account for the groups of aberrant myelinated nerve fibers in the meninges and, adjacent to the blood vessels present after ventral root damage by osteophytes.[6]

Finally, there are peripheral nerve lesions in which the supporting structures of nerve trunks are injured in addition to nerve fiber interruption (neurotmesis). It is conceivable that some disc herniations might disrupt the continuity of the endoneurial tubes, the root sheath, or sever the root completely. Analogous injuries to peripheral nerves have been classified by Sunderland as types 3, 4, and 5 respectively.[28] Nerve root fiber degeneration and regeneration will occur as described previously, but the disruption of the connective tissue framework of the nerve root will facilitate misrouting of regenerating nerve fibers and permit formation of the traumatic foraminal neuroma described by Greenfield.[6]

REFERENCES

1. Coggeshall, R. E., Coulter, J. D., and Willis, W. D. Unmyelinated axons in the ventral roots of the cat lumbosacral enlargement. *J. Comp. Neurol.* 153, 39–58, 1974.
2. Truex, R. C., and Carpenter, M. B. *Human Neuroanatomy*, sixth edition, Baltimore, Williams & Wilkins Co., 1969.
3. Haller F. R., Haller, A. C., and Low, F. N. The fine structure of cellular layers and connective tissue space at spinal nerve root attachments in the rat. *Am. J. Anat.* 183, 109–123, 1972.
4. Sunderland, S., and Bradley, K. C. Stress-strain phenomena in human spinal nerve roots. *Brain* 84, 120–124, 1961.
5. Sunderland, S. Meningeal-neural relations in the intervertebral foramen. *J. Neurosurg.* 40, 756–763, 1974.
6. Greenfield, J. G. *Neuropathology*, second edition, Baltimore, Williams & Wilkins Co., 1963.
7. Russell, D. S., and Rubenstein, L. J. *Pathology of Tumors of the Nervous System*, third edition, London, Edward Arnold, 1971.
8. Merritt, H. H. *A Textbook of Neurology*, fifth edition, Philadelphia, Lea & Febiger, 1973.
9. Brain, L., and Wilkinson, M. *Cervical Spondylosis and Other Disorders of the Cervical Spine*, Philadelphia, W. B. Saunders, 1967.
10. Frykholm, R. Cervical nerve root compression resulting from disc degeneration and root sleeve fibrosis. *Acta Chir. Scand*, Suppl. 160, 1951.
11. Hughes, J. T. *Pathology of the Spinal Cord*, Chicago, Year Book Medical Publ., 1966.
12. Hadley, L. A. *Anatomico Roentgenographic Studies of the Spine*, Springfield, Ill., Charles C Thomas, 1969.
13. Lindblom, K., and Rexed, B. Spinal nerve injury in dorsolateral protrusions of lumbar discs. *J. Neurosurg.* 5, 413–432, 1948.
14. Thomas, P. K. Nerve Injury In: *Essays on the Nervous System* (eds. Bellairs, R., and Gray, E. G.) Oxford, Clarendon Press, 1974.

15. Spinner, M. S., and Spencer, P. S. Nerve compression lesions of the upper extremity—a clinical and experimental review. *Clin. Orthop.* 104, 46–67, 1974.
16. Denny-Brown, D., and Brenner, C. Paralysis of nerve induced by direct pressure and by tourniquet. *Arch. Neurol. Psychiat.* 51:1–26, 1944.
17. Fowler, T. J., Danta, J., and Gilliatt, R. W. Recovery of nerve conduction after a pneumatic tourniquet. Observations on the hindlimb of the baboon. *J. Neurol. Neurosurg. Psychiat.* 35:638–647, 1972.
18. Ochoa, J., Fowler, T. J., and Gilliatt, R. W. Anatomical changes in peripheral nerves compressed by pneumatic tourniquet. *J. Anat.* 113:433–455, 1972.
19. Seddon, H. J. Three types of nerve injury. *Brain* 66:273–288, 1943.
20. Spencer, P. S. Experimental nerve injury and repair. In: *Microsurgery Workshop Symposium*, C. V. Mosby, St. Louis, 1975.
21. Sunderland, S. The effect of rupture of the perineurium on the contained nerve fibers. *Brain* 69:149–152, 1940.
22. Aguayo, A., Nair, C. P. V., and Midgley, R. Experimental progressive compression neuropathy in the rabbit. *Arch. Neurol.* 24:358–364, 1971.
23. Duncan, D. Alteration in the structure of nerves caused by restricting their growth by ligatures. *J. Neuropath. Exp. Neurol.* 7:261–273, 1968.
24. Dyck, P. J.: Experimental hypertrophic neuropathy. Pathogenesis of onion-bulb formations produced by repeated tourniquet applications. *Arch. Neurol.* 21:73–95, 1969.
25. Weiss, P., and Hiscoe, H. B. Experiments on the mechanism of nerve growth. *J. Exp. Zool.* 107:315–395, 1948.
26. Spencer, P. S. Reappraisal of the model for "bulk axoplasmic flow". *Nature New Biol.* 240:283–285, 1972.
27. Spencer, P. S. The traumatic neuroma and proximal stump. *Bull. Hosp. Joint Dis.* 35:85–102, 1974.
28. Sunderland, S. *Nerve and Nerve Injuries.* Edinburgh, E. & S. Livingstone, 1968.

Chairman's Summary: Anatomical and Biomechanical Studies

PHILIP GREENMAN
Professor and Chairman
Department of Biomechanics
College of Osteopathic Medicine
Michigan State University

This session was devoted to presentations focusing upon the known anatomical features of spinal biomechanics and particularly of the intervertebral foramina, and the effects of this anatomy on the emergent nerves and nerve roots at the intervertebral foraminal level.

The first presentation by David C. Drum focused on the *vertebral unit and the intervertebral foramina.* This paper focused on the vertebral motor unit as the conceptual model made popular by Junghanns. The vertebral motor unit, according to this concept, is composed of an intervertebral disc, neurovascular elements concentrated within the neural foramina and intervertebral foramina, posterior spinal joints, and all the connective and muscular tissues supporting and limiting intersegmental motion. This concept was developed in this presentation and the role of soft tissue structures was clearly delineated. A description of the 23 typical motor units was made together with the atypical motor units of the occipital-atlantal and atlanto-axial levels. Additionally, the concept of the pelvic motor unit including the sacroiliac joints and the pubic symphysis was noted. The need for research into the muscles limiting and coordinating motion of the individual or multiple motor units was indicated. A need for study into the mechanical response of the whole body was also noted. It was concluded by this paper that Junghanns' concept might be useful as a focus for continuation of interdisciplinary dialogue and research.

Edmund B. Weis, Jr., presented a paper on *Spinal Geometry: Normal and Abnormal,* and stated that the analysis must include consideration of anatomical, physiological and mechanical variables. He presented a format of looking at spinal geometry from the basis of normal and abnormal anatomy. Each of these areas was then addressed in terms of large and small deviations. Each deviation was further divided into static and dynamic subsets. The presentation was supported extensively by radiographs of the spine demonstrating the multiple possibilities of altered spinal geometry. Included were scoliosis, degenerative joint disease, congenital vertebral abnormalities, fractures and dislocations, spondylolisthesis and sacralization, herniated nucleus pulposus, and numerous others. Dr. Weis concluded that the highest incidence of problems were in the area of normal anatomy, small deviations, and dynamic state. The same small deviations and dynamic loading on abnormal anatomy appeared to be produced by degenerative processes. He concluded that prospective studies on the efficacy of manipulative therapy, as well as other forms of therapy, should be conducted to answer questions about effectiveness in patient care.

Spinal Kinematics, by Augustus A. White III, was presented and was defined as "that phase of mechanics concerned with the study of motion of particles and rigid bodies with no consideration of the forces involved." He developed the analogy of a train first used by Lovett to apply to the

vertebral unit. A series of terms and definitions of possible vertebral motion was developed and provided a basis upon which a comprehensive description of spinal kinematics was made. The author points out that few studies have been carried out to analyze and quantitate the ranges of spinal kinematics. He presented the ranges of motion in flexion-extension, lateral bending, and axial rotation based upon the review of the literature and experience of the author. A concise overview of the work being done by the author and others in the area of spinal kinematics was made. Some of the problems in this area are based on the fact that most of the work has been done on autopsy material and there is a need to study kinematics in the living spine. The development of measurements for this type of work are needed.

The fourth paper addressed *Biomechanical Aspects of Subluxation*, by Chung Ha Suh, who presented two areas of work in which an attempt has been made to study and define the term of subluxation. He focused on 1) precision x-ray method for the visualization of biomechanical aspects of subluxation, and 2) computer model of the spine for simulated study of spinal subluxation. The former dealt with a review of the x-ray analysis of the spine and the problem of image geometry associated with this x-ray procedure. Methodology was developed to correct for the distortion, as well as single plane aspects, of spinal radiology. It was unfortunate that the author was unable to further develop this work with examples of practical application. The second portion of the presentation dealt with a computer model for the spine. The development of a three-dimensional computer model of the spine was deemed useful for further work in the area of "subluxation." The two methodologies addressed by this paper point out the need for numerical-analytical data to support the large amount of literature based on subjective clinical observations. Such methodologies appeared to the author to be essential for systematic study of the spinal subluxation.

In the discussion of the foregoing four papers, James McElhanney provided a brief review of the state of the art in the anatomical and biomechanical studies of the spine.

Two additional papers were presented in this session. The first by Sidney Sunderland focused on *Anatomical Perivertebral Influences on the Intervertebral Foramina*. Sunderland presented the anatomical and physiological properties of the intervertebral foramina and the relationship of the contents. Changes in the dimensions of the foramina were detailed. The author concluded by noting that the dimensions of the foramina provided ample room for the neural contents. He cautioned "against concentrating exclusively on the intervertebral foramina as the sight of the offending lesion." There appear to be other explanations for the clinically observable phenomena.

The *Pathology of Spinal Root Compression*, by Schaumburg, then followed. Building upon the preceding presentation by Sunderland, he developed the pathologies of the spine in intervertebral foramina and what is known about nerve root compression. Pathologies relevant to spinal root compression included herniation of the intervertebral disc, pathology of intervertebral articulation with aging and trauma, particularly at the arthrodial joint and at the joint of Luschka. He points out that few experimental pathology studies of nerve root compression had been reported. Most of the work in the area has been done with peripheral nerve. He described the pathologies which occurred in the nerve including demyelination, axonal degeneration and their varieties. Possible varieties of repair to degeneration and subsequent regeneration were presented.

In open discussion following these presentations, Gurjian reported on studies involving acceleration-deceleration injuries to the neck; these studies were supported by high speed movies of individuals being subjected to simulated injuries at the level of 10 miles per hour and 15 miles per hour. The varieties of response of the head and neck to the position of the patient were clearly delineated. The role of muscle spasm and tone was noted.

The general discussion which followed demonstrated varying opinions of the participants relative to the role of the various anatomical structures described in the papers and observations of the practitioners of manipulative therapy. It appeared that there was consensus on the need for better techniques and methodologies to measure those abnormalities of spinal biomechanics and the associated changes described by the manipulative practitioner. It was apparent that the neural

compression theory at the intervertebral foramina did not satisfactorily explain all of the phenomena clinically observed. The need for methodologies to study the biomechanics of the spine and its component parts on living subjects was noted. Attention must also be given to the soft tissues in and about the vertebral segments, in which total emphasis on the bony relationships appears to be inappropriate.

This session demonstrated the need for additional collaborative research studies in the area of the biomechanics of the spine and the associated structures. Emphasis on a single structure or concept appears to be less than appropriate.

Chapter IV.

What Do the Basic Sciences Tell Us About Manipulative Therapy? (Neuroscience Studies)

Horace W. Magoun, *Chairman*

597-036 O - 76 - 11

Susceptibility of Spinal Roots to Compression Block

SETH K. SHARPLESS
Professor of Psychology
Department of Psychology
University of Colorado

Fontana, as early as 1797, observed that pressure on a nerve blocks conduction without causing stimulation (cited by Meek and Leaper, 1911). The effect of local compression on conduction has been the subject of extensive, if desultory, investigation since. In the last few years, interest in nerve compression has revived, owing partly to the attempt of bioengineers employing computer simulation techniques to develop a realistic model of the spine as a mechanical system. It is hoped that such efforts will ultimately yield estimates of the forces brought to bear on structures of the spine, including spinal nerves and roots, under various conditions of loading. Needless to say, this objective is a long way from realization, but if it is ever to be achieved and its clinical potential is to be exploited, detailed information will be required on the susceptibility of nerve to tensile and compressive forces.

Given the long history of study of the effect of compression on nerve and the relatively simple technical requirements of such studies, it might be supposed that such information would be found in the literature. This appears not to be the case for several reasons: First, most previous work has been on peripheral nerve, and it may well be that spinal nerve and roots are more vulnerable to mechanical stress than peripheral nerve. Thus, according to Sunderland and Bradley (1961), spinal roots yield more readily to tensile stress owing to the fact that they lack the perineurium and funicular plexus formations of peripheral nerve. Gelfan and Tarlov (1956), in one of the few physiological studies on the effects of compression on spinal nerves and roots, reported their impression that the spinal roots were also more susceptible to compressive forces than peripheral nerve. They did not, however, vary pressure systematically, nor did their compression apparatus permit a direct comparison of spinal and peripheral nerve. Even in the studies of peripheral nerve, it is difficult to find useful values for the minimal pressure increments required to produce significant nerve dysfunction. Indeed, in only a few studies was the compression apparatus calibrated in such a way as to permit translation of values obtained to standard measures of pressure (e.g., mm. Hg.). In most instances, too, the investigators were interested chiefly in pressures required to produce complete block (at least of the rapidly conducting fibers) rather than minimal impairment of conduction which would be of greater clinical interest. Even these values vary greatly in different experiments, as indicated in table I.

TABLE I

Authors	Pressure to block (mm Hg)	Time to block	Recovery time	Nerve
Mitchell, Weir, 1872	508	10–12 secs.	10–12 secs.	Rabbit sciatic
Meek and Leaper 1911	1034	19 mins.		Rat gastroc.

TABLE I—continued

Authors	Pressure to block (mm Hg)	Time to block	Recovery time	Nerve
Edwards and Cattell 1928	1200	10 mins.	--	Frog sciatic
Bentley and Schlapp 1943	130	2.5–3 hrs.	No recovery	Cat popliteal
Denny-Brown and Brenner 1944	762	2 mins.	6 mins.	Cat sciatic
Causey and Palmer 1949	150–400	20 mins.	5–65 secs.	Rabbit gastroc.

Table I gives a fair idea of the kind of quantitative data available on blocking pressures. In most cases, the compressed area was a short segment of nerve, less than one centimeter in length (in the experiments of Bentley and Schlapp, 2–4 cm. segments were compressed). There is little useful information in the table, other than that compression does produce conduction block, and that the block may become progressively more severe if the pressure is maintained. Of the investigators mentioned, Causey and Palmer (1949) used the most novel and probably the most accurate technique for measuring the compressive force. They actually immersed a segment of the nerve in a bath of mercury attached to a mercury column. The minimum pressures required to block rabbit gastrocnemius in their experiments (150 mm Hg) agree closely with the values we have obtained for rat and cat sciatic nerve.

Methods and Apparatus

An attempt was made to devise a compression apparatus which would be simple, accurate, suitable for use in small animals, and, so far as possible, consistent with these requirements, one that would mimic the situation in which nerves are compressed against bony facets by soft tissues in confined spaces. According to Sunderland (1968), it is this kind of situation that most often gives rise to idiopathic peripheral nerve compression syndromes, as in the carpal tunnel.

The apparatus is shown in figure 1. In fact, it is a modification of the technique originally used for this purpose by Weir Mitchell, who allowed a chamois leather bag filled with mercury to rest on the nerve. A miniature rubber balloon (2.5–5 mm. in diameter) filled with fluid is lowered by a manipulator onto the nerve which rests on a plastic shelf with rounded edges; the shelf is 4.5 mm. in width. The *increment* in pressure produced by contact with the nerve is taken as the pressure applied to the nerve. Owing to the fact that the contact area increases as the balloon is lowered onto the nerve, there is some confounding of length of nerve compressed with pressure applied. Thus, the length of nerve compressed increases from 2.5 mm. at about 10 mm. Hg. to a maximum of 4.5 mm. at about 50 mm. Hg.

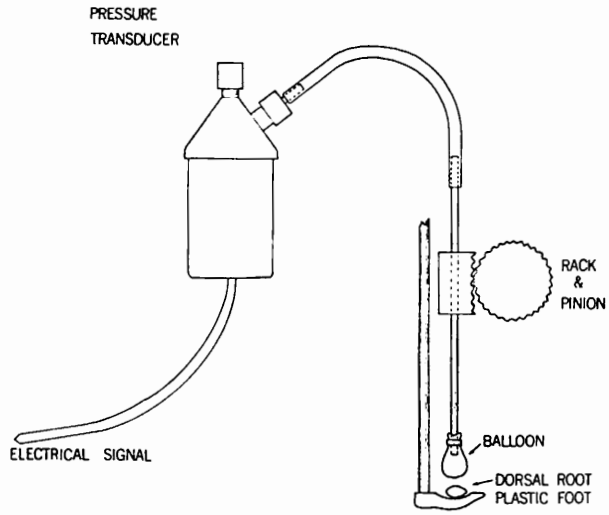


Fig. 1. Compression apparatus. The increment in pressure as the balloon makes contact with the nerve is taken as the pressure applied to the nerve.

Experiments were performed on cat and rat sciatic nerves and dorsal roots, L4–L7. The nerves were dissected free of surrounding tissue on the recording (or proximal) side of the compression shelf and covered with mineral oil which had been agitated with room air and saline. The same procedure was used with dorsal roots, except that the dura was opened, and the roots were freed of their attachments for not more than 2–3 centimeters from their entry to the cord. In both cases, an effort was made to keep the blood supply intact to within a few millimeters of the plastic shelf on the distal side of the compression apparatus (the side of stimulation). Compound action potentials were recorded relative to the killed

ends, digitalized, and led directly to a computer for storage and analysis. Only the A components of the action potentials were examined. Cat and rat nerves and roots appeared identical with respect to blocking pressures.

Results

The single most important finding to emerge from our re-examination of this old problem is the astonishing sensitivity of spinal roots to compression. This is shown in figure 2. In the

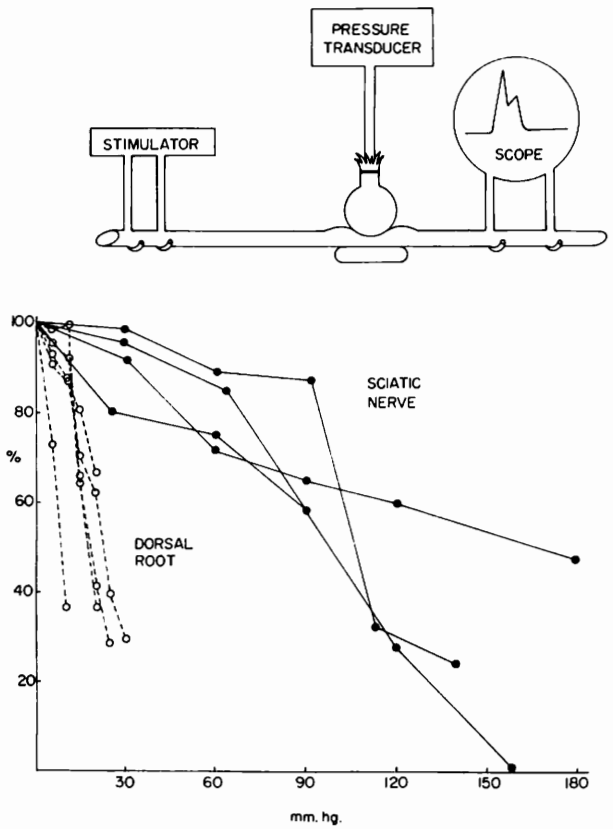


Fig. 2. Susceptibility of spinal roots to compression block. Compound action potentials integrated and expressed as percent of initial value. Pressures applied for 3 minutes only.

experiments depicted here, the pressure was applied for 3 minutes, relieved for 3 minutes, and then a higher pressure was applied, this procedure being continued until a substantial conduction block was evident. Although the three-minute periods were not sufficient to allow full recovery with the higher values of pressure, so that cumulative effects tend to be confounded with the effects of pressure per se, the design is adequate to reveal the remarkable difference between spinal roots and peripheral nerve. Rat and cat preparations are included in the results and the curves are indistinguishable both in the case of the roots and peripheral nerve. This indicates, incidentally, that nerve diameter is probably not a crucial factor in determining resistance to pressure.

With the procedure used, a complete conduction block in the sciatic nerve could not be achieved with pressures of less than 150 mm Hg. This is consistent with the results of Causey and Palmer (1949), mentioned previously. In our experiments, the area (mv.–msec.) of the A components of the compound action potential were reduced to about half-value at 100 mm. Hg. Compressive forces of less than 50 mm. Hg. blocked only a small number of fibers, even when the forces were long maintained. In contrast, dorsal roots were able to withstand only minute pressures, the potentials being reduced to about half their initial values by pressures of 20–25 mm. Hg.

A spectacular demonstration of the sensitivity of spinal roots to compression is shown in figure 3, in this case from a rat, but cat roots have yielded

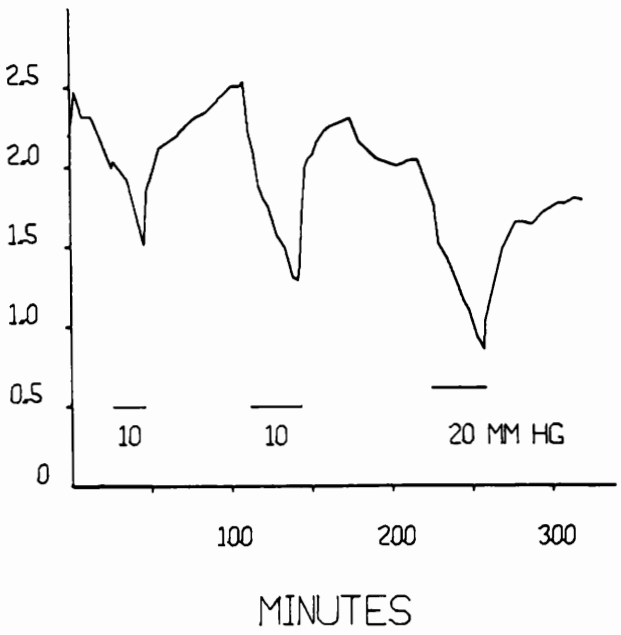


Fig. 3. Effects of sustained pressure. Computer-generated plot of average areas (mv.–msecs.) in *A* components of action potentials from rat dorsal root during application of very small pressures. Note progressive character of conduction block and recovery.

similar results. In this experiment, the sciatic nerve was stimulated 1/sec. for 5 hours and the compressor was applied periodically to a dorsal root. In the computer-generated plot, each point repre-

sents the average area (mv.–msecs.) of 10 successive potentials led from the cut end of the root. A pressure of only 10 mm. Hg. produced a significant conduction block, the potential falling to about 60 percent of its initial value in 15 minutes and to half its initial value in 30 minutes. After such a small compressive force is removed, nearly complete recovery occurs in 15 to 30 minutes. With higher levels of pressure, we have often observed incomplete recovery after many hours of recording.

It is difficult to appreciate the significance of the minute pressures capable of affecting root conduction. It seems doubtful that the most skillful and deft surgeon could touch a spinal root or the balloon of our compression apparatus with his gloved forefinger without producing a pressure increment of at least 5 mm. Hg. One may well consider what happens to the spinal roots when they are manipulated by the far less dextrous electrophysiologist.

Figure 3 also brings out the importance of another parameter, time. One thing that is amply documented in the old literature is the importance of the duration of compression. A few fibers only may be blocked by a transient increment in pressure, whereas the same pressure maintained for a long time may produce a substantial conduction block. What is the cause of gradual failure of nerve fibers during local compression?

In the older literature, there was much controversy regarding the relative roles of ischemia and mechanical deformation in compression block. Ischemia might account for the progressive character of compression block, but it is my impression that the roles of ischemia and mechanical deformation have not yet been entirely disentangled. Thus, Bentley and Schlapp (1943) were able to show that a 4 cm. segment of peripheral nerve could be rendered completely ischemic by compressing it between rubber cushions to a pressure of 60 mm. Hg., and yet maintain conduction for hours, owing to the diffusion of oxygen from the ends of the compressed region. On the other hand, Causey and Palmer (1949) described one experiment in which they had unsuccessfully attempted to achieve conduction block by air pressure alone, suggesting that ischemia may have contributed to conduction block in other instances in which the nerve was compressed by the weight of mercury. Gelfan and Tarlov (1956), in their work on spinal nerve and roots, concluded that mechanical deformation rather than anoxia was responsible for compression block, since compression affected large fibers first, whereas with anoxia the smaller fibers were more vulnerable.

If the observations of Gelfan and Tarlov (1956) on the effects of anoxia are valid, it would appear that even the small pressures employed in our experiments produced conduction block in dorsal roots by mechanical deformation rather than hypoxia. Figure 4 shows the effects of small

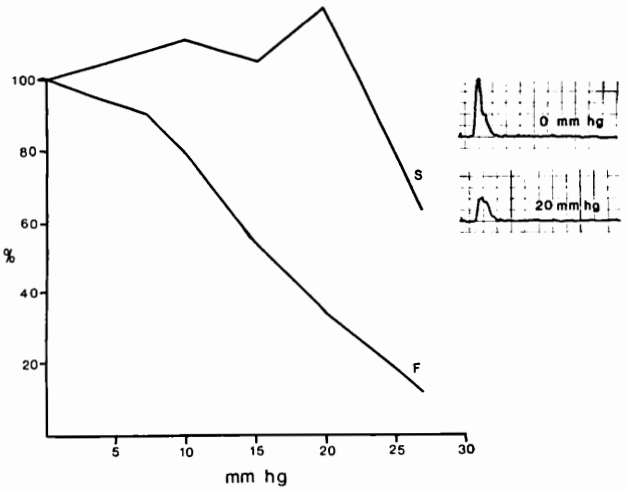


Fig. 4. Curves representing changes in the fast (*F*) and slow (*S*) components of the *A* potentials as a function of pressure. There is some slowing of the fast unit discharges, indicated by the increase in the "slow" components as pressure is increased. However, the fast components are generally more susceptible to block even at pressures of 20 mm. Hg. or less. Dorsal root from rat. Inset: computer-writeout of average potentials.

increments in pressure on the fast and slow components of the action potential led from a dorsal root. It is evident that pressures of as little as 20 mm. Hg. affect primarily the rapidly conducting fibers. The differentiation between slow and fast fibers was especially clear in this experiment: more commonly the differentiation was less conspicuous; however, rapidly conducting elements were always somewhat more susceptible, which makes it unlikely that the block could be ascribed to hypoxia.

The susceptibility of large diameter fibers to compression block, first observed in peripheral nerve by Gasser and Erlanger (1929), provides a clue to the mechanism of compression block. Assuming that a nerve fiber is a pressure vessel, something like a sausage, then the contents would tend to be squeezed out of a compressed zone, the pressure exerted by the displaced fluid

being balanced by hoop stresses in the surrounding membrane. A simplified model (figure 5; MacGregor et al., 1975) can be used to show that the larger fibers will require larger hoop stresses to match a given internal pressure and will undergo a greater percentage decrease in cross-sectional area.

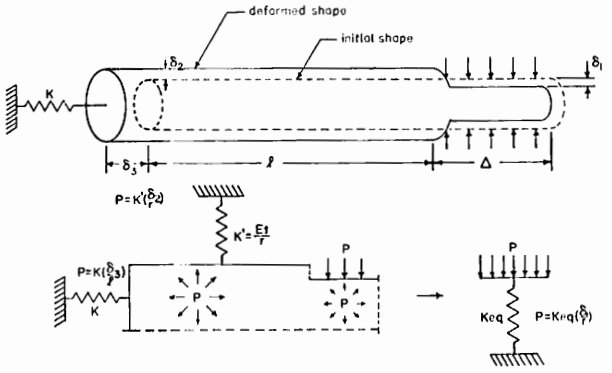


Fig. 5. Pressure vessel ("sausage") model of compressed nerve fiber. Pressure applied in delta region displaces contents to other regions where the pressure is opposed by hoop stresses. Elastic properties of membrane represented by springs. This model predicts that larger fibers will undergo greater percentage deformation than small fibers. From MacGregor, Sharpless and Luttges (1975).

If the safety factor for the longitudinal internal resistance is approximately the same in large and small fibers, the conduction of the large fibers being compensated by greater internodal distances, etc., the larger proportional deformation of the large fibers might account for their susceptibility to compression block. Of course, other factors may be involved, including invagination of the myelin (Causey, 1948; Ochoa, et al., 1972).

This analysis would suggest that the slow onset of compression block is due to the viscosity of the displaced fluid. Whatever the mechanism responsible for the gradual onset of compression block, it may have adaptive value, for it would tend to protect nerves against transient fluctuations in local pressures. Although we do not have data on the increments in pressure in the intervertebral foramina during motion of the spine, there must surely be considerable fluctuations of pressure, particularly during extremes of motion. In other confined spaces through which nerves pass, there are such fluctuations in pressure. For example, the pressure in the carpal tunnel is increased during both acute flexion and hyperextension of the wrist (Tanzer, 1959; Robbins, 1963). Given the slow onset of compression block, a transient increase in pressure would have little effect. It is only when the joint is fixed in a position yielding a significant increase in pressure that one would expect compression block to develop. This consideration is relevant to the chiropractic definition of a "subluxation" as "a fixation of a joint within its normal range of movement, usually at the extremity of that range" (Weiant, cited by Harper, 1964). It may also account for the observation that many instances of idiopathic compression syndrome are associated with occupations in which an awkard position must be maintained for some time (See Sunderland, 1968, for references).

The spinal roots, floating in their bony canal, are well protected from local fluctuations in pressure, and it is for this reason, perhaps, that they have not developed other mechanisms to protect them from compressive forces. But it is scarcely credible that spinal nerves within the intervertebral foramina, where considerable pressure fluctuations must occur, could be so vulnerable. Somewhere along their course from the cord to the periphery, nerves acquire a resistance to compressive forces. We have carried out some experiments to determine *where* along the course of the spinal nerves these protective features become manifest. In one series, cat spinal nerves, still encased in their sheaths, were compressed just proximal to the dorsal root ganglia—i.e., within the spinal canal before the nerves enter the foramina. For this purpose, the plastic shelf was removed from the compression apparatus and the nerves were compressed against the floor of the spinal canal. Records were taken from the corresponding dorsal roots, exposed near their entry to the cord. Figure 6 is a computer-generated plot of the results from three L6 and L7 nerves. The nerves were relatively resistant to pressure, a force of approximately 90 mm. Hg. being required to reduce the response to half-value.

To determine whether the sheath surrounding the nerves at this point afforded significant protection, the sheath was slit and partially dissected away so that the balloon made direct contact with the nerve. This had no effect on resistance to pressure, notwithstanding the fact that the dissection damaged some of the fibers. Thus, the dissected nerves yielded initial responses smaller than the intact nerves, but a plot of the magnitude of the response relative to its initial value as a function of pressure (fig. 6—see Xs) corresponded exactly with a similar plot from intact, sheathed nerve,

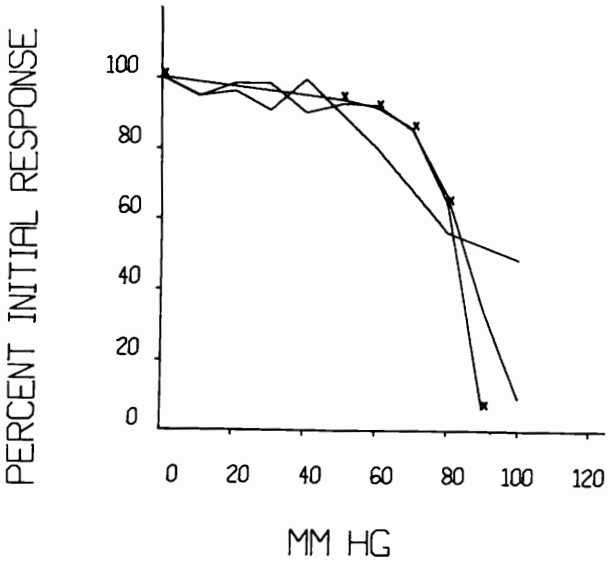


Fig. 6. Effects of compressing spinal nerves just proximal to ganglion. Nerves compressed against floor of spinal canal. Partially desheathed nerve identified by *X*'s shows same vulnerability to pressure as intact nerve. Note that nerves have become resistant to pressure before they enter the foramina.

the response being reduced to half with about 90 mm. Hg.

Thus, well before they enter the intervertebral foramina where they might be expected to experience significant fluctuations in pressure, spinal nerves have acquired some structural feature which protects them against compressive forces. Sunderland and Bradley (1961) have suggested that the funicular plexus formations of peripheral nerve trunks protect the nerve fibers from tensile stresses, the load being resisted by the perineurium associated with the undulating nerve fibers of the funiculus. Spinal roots lack this feature and accordingly are more vulnerable to stretch. It is not clear, however, why this feature should also protect against compressive forces. In fact, we do not know what constitutional specialization is responsible for protecting nerve fibers within peripheral nerve trunks against compression, nor how such a feature may be altered by disease or aging.

Fontana, it will be recalled, made two observations in 1797: that compression blocks conduction and that it does not cause nerve stimulation. Patrick Wall and his associates (1975) have just published a paper confirming the second observation, that compression injury produces only a transient burst of impulses before conduction block sets in. Here, we have been concerned with the nature of the conduction block itself. It is curious that in the latter part of the 20th century, we should still be confirming Fontana's observation, and that we still know so little about the pathophysiology of compression syndromes.

CONCLUSIONS

1. Dorsal roots are far more susceptible to compression block than peripheral (sciatic) nerve. When pressure is applied for 3 minutes followed by 3-minute recovery periods, 100 mm. Hg. must be applied to sciatic nerve to achieve the same conduction block that can be produced in spinal roots by 20 mm. Hg.

2. As little as 10 mm. Hg. pressure, maintained for 15–30 minutes, reduces the compound action potentials of dorsal roots to about half of their initial values. With such small pressures, nearly complete recovery occurs in about 30 minutes.

3. It is probable that the compression block produced even by such small pressures is due to mechanical deformation rather than ischemia, since the larger fibers are blocked first, whereas anoxia is believed to affect small fibers first.

4. It has been shown elsewhere (MacGregor et al., 1975) that a pressure vessel model of nerve predicts that large fibers would be most compressed, which may account for their susceptibility to block. The pressure vessel model might also account for the progressive character of compression block, assuming a viscous flow of the fiber contents.

5. The slow onset of compression block would have adaptive value, since transient increments of pressure which occur in confined spaces during extremes of motion would have little effect.

6. Spinal nerves acquire a structural feature which protects them from compression block before they enter the intervertebral foramina. The sheath does not appear to play an important role. The nature of this protective feature is still unknown.

REFERENCES

1. Bentley, F. H. and Schlapp, W. (1943) The effects of pressure on conduction in peripheral nerves. *J. Physiol.*, 102, 72–82.
2. Denny-Brown, D. and Brenner, C. (1944) Paralysis of nerve induced by direct pressure and by tourniquet. *Arch. Neurol. Psychiat.*, Chicago, 51, 1–26.
3. Causey, G. (1948) The effect of pressure on nerve fibers. *J. Anat.*, 82, 262–270.

4. Causey, G. and Palmer, E. (1949) The effect of pressure on nerve conduction and nerve fiber size. *J. Physiol.*, 109, 220–231.
5. Edwards, D. J. and Cattell, M. (1928) Further observations on the decrement in nerve conduction. *Amer. J. Physiol.*, 114, 359–367.
6. Gasser, H. S. and Erlanger, J. (1929) The role of fiber size in the establishment of a nerve block by pressure or cocaine. *Amer. J. Physiol.*, 88, 581–591.
7. Gelfan, S. and Tarlov, I. M. (1956) Physiology of spinal cord, nerve root and peripheral nerve compression. *Amer. J. Physiol.*, 185, 217–229.
8. Harper, W. D. *Anything Can Cause Anything*. San Antonio, Texas: By the author.
9. MacGregor, R., Sharpless, S. and Luttges, M. (1975) A pressure vessel model for nerve compression. *J. Neurol. Sciences* (In press).
10. Meek, W. J. and Leaper, W. E. (1911) The effect of pressure on conductivity of nerve and muscle. *Amer. J. Physiol.*, 27, 308–322.
11. Mitchell, W. (1872) *Injuries of Nerves and Their Consequences*, Philadelphia, J. B. Lippincott Company.
12. Ochoa, J., Fowler, T. and Gilliatt, R. (1972) Anatomical changes in peripheral nerves compressed by a pneumatic tourniquet. *J. Anat.*, 113:3, 433–455.
13. Robbins, H. (1963) Anatomical study of the median nerve in the carpal tunnel and etiologies of the carpal-tunnel syndrome. *J. Bone Jt. Surg.*, 45–A, 953.
14. Sunderland, S. (1968) *Nerves and Nerve Injuries*, Edinburgh, Churchill Livingstone.
15. Sunderland, S. and Bradley, K. (1961) Stress-strain phenomena in human spinal roots. *Brain*, 84, 121–124.
16. Tanzer, R. C. (1959) The carpal-tunnel syndrome: A clinical and anatomical study. *J. Bone Jt. Surg.*, 41–A, 626.
17. Wall, P., Waxman, S., and Basbaum, A. (1975) Ongoing activity in peripheral nerve: injury discharge. *Exp. Neurol.*, 45, 576–589.

I should like to thank the American Chiropractic Association and the International Chiropractic Society for supporting this research. I should also like to thank Susan Cobb, Peter Lund, and Stephen Young for their participation.

The Somatosympathetic Reflexes: Their Physiological and Clinical Significance

AKIO SATO
2nd Department of Physiology
Tokyo Metropolitan Institute of Gerontology
Tokyo, Japan

1. INTRODUCTION

Some of the important functional aspects of somatosympathetic reflexes may be summarized in this quote from A. Kuntz:[1] "Reflex responses of the viscera, including the splanchnic blood vessels, elicited by stimulation of localized cutaneous areas with the same segmental innervation as the viscera in question are common physiologic phenomena. The efficacy of cold and warm applications in the treatment of visceral disease undoubtedly depends upon reflex responses elicited by the localized cutaneous stimulation. The segmental character of the reflex mechanisms employed appears to be obvious, but the distribution of the autonomic innervation of the viscera, particularly the gastrointestinal tract, is less strictly segmental than that of the sensory innervation of the skin. Localized cutaneous stimulation, like painful stimulation of other somatic tissues, may also elicit intersegmental and suprasegmental reflexes through autonomic nerves."

Detailed electrophysiological analysis of these reflexes has long been delayed, and apart from rare exceptions, has largely been restricted to the study of changes in effector organs. Several factors seem to be responsible for this situation, which is so much in contrast to the much faster progress made in the analysis of the sensory and motor functions of the nervous system: For example, it is certainly much more difficult to record action potentials in the thin myelinated and unmyelinated nerves than those in motor axons. Further, the ongoing tonic activity of sympathetic neurons, which is continuously modulated by influences related to the respiratory and cardiac cycles, impedes the quantitative measurement of excitatory or inhibitory effects of afferent volleys on the sympathetic nervous system. Fortunately, modern electrophysiological techniques have largely overcome these obstacles. In recent years progress has been made in the analysis of the effects of somatosensory afferent activity on the sympathetic nervous system as well as in the analysis of the central reflex pathways of somatosympathetic reflexes.

A detailed history of these studies was recently published by Sato and Schmidt (1973)[2] in which the authors presented a summary of these new findings obtained by recording nervous activity and also attempted to correlate these findings with each other and with the results obtained by studying the effector organs. At first I will give a brief summary of this review article, and then I will discuss recent experimental results in the cutaneo-visceral reflexes. Those results are important for clinical application of the basic somatosympathetic reflex system in the future.

There are various reflex responses of the sympathetic nervous system produced by somatic afferents. These include reflex responses in the cardiovascular system, the gastrointestinal tract, micturition, the galvanic skin reflex, etc. However, let me begin with a brief history of the somatosympathetic reflexes in relation to the cardiovascular system only, since the nervous control of the

cardiovascular organs has been investigated in more detail than that of other effector organs under the control of autonomic nerves.

About a century ago Carl Ludwig and his coworkers recorded blood pressure changes after electric stimulation of limb nerves and studied the modification of these responses on transections of the brain stem. They concluded from their results that the pathways of somatosympathetic reflexes run from the spinal cord up to the medulla oblongata and back to the preganglionic neurons. In 1916 Ranson and Billingsley[3] reinvestigated the problem and with weak electrical stimuli examined the medulla oblongata. They introduced the concept of the pressor and depressor centers. Corrections and additions were made later by others: Sherrington in 1906[4] and Brooks in 1933[5] described a spinal pathway for the somatosympathetic reflex. Alexander (1946)[6] seems to be the first person who recorded somatically evoked sympathetic reflex discharges directly from sympathetic nerves. It was soon recognized that a single electrical stimulus to a somatic nerve can provoke a reflex discharge in sympathetic nerves, whereas a single shock usually cannot produce a detectable response in cardiovascular effectors. From 1952 on Schaefer and his colleagues studied various aspects of the somatosympathetic reflex connections. They confirmed Alexander's results and, in addition, reported that, according to their findings, somatic afferent inputs usually elicit generalized massive reflex discharges in all types of sympathetic efferents followed by a generalized postexcitatory depression.

In the investigation of somatosympathetic reflexes, direct recording of the action potentials from sympathetic nerves has advantages over recording from effector organs, particularly when studying the central organization and the mode of operation of these reflexes. In addition, one avoids the blurring of time relationships introduced by the great inertia of the neuroeffector transmission. It is surprising, therefore, that almost 20 years elapsed between the pioneering work of Alexander and the beginning of a detailed and rigorous investigation of the somatosympathetic reflexes.

II. SOMATIC AFFERENT SYSTEMS PRODUCING SOMATOSYMPATHETIC REFLEXES

Myelinated somatic afferents. Most of the physiological experiments on somatosympathetic reflexes have been performed on cats with chloralose or a mixture of chloralose and urethane. The first detailed evaluations of the potency of the various groups of myelinated somatic afferents in evoking sympathetic reflexes were initiated by Sato and Schmidt (1966)[7] and investigated in more detail by Schmidt and Schönfuss (1970)[8] and Sato and Schmidt (1971).[9] It was always seen that low threshold (< 2T) cutaneous nerve stimulation evoked sympathetic reflex discharges which reached their maximal size at about 5T. At this stimulus strength, all cutaneous group II afferent fibers were included in the volley. There was always a further but small increase when the stimulus was increased to include the group III afferents in the volley. The effects of muscle nerve stimulation were distinctly different from those obtained with cutaneous nerve stimulation. In muscle nerve stimulation, the sympathetic reflex never appeared at stimulus strength in the 2T range, i.e., at stimuli that excited practically all group Ia and Ib fibers from the primary muscle spindle receptors and the Golgi tendon organs, respectively. Only when the stimulus strength included all group II fibers (8–10T) and extended into the group III range (< 10T) was a large reflex observed. The effects of the various somatic afferent fiber groups on the reflex activity of single sympathetic nerve units have been studied meanwhile on the preganglionic site by Jänig and Schmidt (1970)[10] and Sato (1972)[11] as well as on postganglionic units in cutaneous (Jänig, Sato and Schmidt 1972)[12] and muscle nerves (Koizumi and Sato 1972).[13]

Unmyelinated somatic afferents. It has been known that high-threshold electrical stimulation of somatic nerves produces increases in blood pressure (see Ranson's Review 1921[14]). With modern electrophysiological techniques it was shown by Laporte and Montastruc (1957)[15] and Laporte, Bessou, and Bouisset (1960)[16] that these pressor effects were clearly related to the activation of unmyelinated afferent fibers. More recently there have been demonstrations of mass reflex discharges in sympathetic nerves induced by group IV somatic afferent volleys (Fedina, Katunskii, Khayutin, and Mitsányi 1966,[17] Coote and Perez-Gonzalez 1970,[18] Khayutin and Lukoshkova 1970,[19] Koizumi, Collin, Kaufman, and Brooks 1970,[20] Schmidt and Weller 1970[21]). The group IV reflex, as it may be called, usually requires a temporal facilitation of afferent volleys.

The much longer latency of group IV reflexes relative to those induced by group II and III volleys is practically entirely due to the low conduction velocity of the unmyelinated afferents. Analysis of the influence of somatic group IV afferents on single postganglionic units on cutaneous nerves was covered by Jänig, Sato, and Schmidt (1972)[12] on muscle by Koizumi and Sato (1972).[13]

III. CENTRAL PATHWAYS OF SOMATOSYMPATHETIC REFLEXES

In this section the evidence regarding the various central pathways of the various somatosympathetic reflex components will first be taken from experiments where the recording was done from the lumbar sympathetic trunk and from lumbar white rami, because, at these recording sites, the latency differences between the various components are most pronounced. The reflexes evoked by myelinated somatic afferents (group II and III fibers) have reflex pathways at three different levels of the central nervous system; the spinal, medullary, supramedullary. On the other hand those reflexes evoked by unmyelinated somatic afferents (group IV fibers) have only two pathways; spinal and medullary.

Central pathways of reflexes induced by myelinated somatic afferents. In cats anesthetized with chloralose, single-shock electrostimulation of the sciatic nerve produced two mass reflex discharges in the lumbar sympathetic trunk with latencies of 25–50 and 80–120 msec, respectively, termed the "early" and "late" reflex potentials (Sato, Tsushima and Fujimori 1965[22]). It was shown that the early response was transmitted via spinal pathways, the late one via the medulla oblongata. In mass recordings from lumbar white rami it was seen that the late reflex was evoked by low- and high-threshold myelinated afferents (group II and III afferents), whereas the early component appeared only when the high-threshold afferents group III were also included in the volley (Sato, Kaufman, Koizumi, and Brooks 1969).[23] In chronic spinal cats the low-threshold (group II) myelinated afferents also evoked the early reflex but the late component never reappeared (Koizumi, Sato, Kaufman, and Brooks 1968[24]) even if the spinalized animal was kept for 3 months or longer after the operation (Sato 1973[25]).

The size of the early reflex component seems to depend to a great extent on the segmental level of afferent input relative to the segmental level of the white ramus from which it is recorded: Its amplitude was largest when the afferent volley entered the spinal cord at the same or adjacent segment of the white ramus under observation. In contrast, the size of the late reflex was rather independent of the segmental level of afferent input (Sato and Schmidt 1971,[26] Sato 1972[27]).

Spinal reflex potentials in nonanesthetized spinal cats were first reported by Beacham and Perl in 1964,[28,29] who showed that the potentials have a polysynaptic pathway by measuring their central reflex time.

The results reviewed so far can be summarized by saying that afferent volleys in myelinated somatic afferents have a twofold action on the sympathetic nervous system: A more generalized action via the supraspinal sympathetic reflex centers and a more circumscribed action on the preganglionic neurons at the segmental level. This generalization is in line with the results and conclusions of a variety of investigations where the somatically evoked reflexes were recorded from more peripheral sites of the sympathetic nervous system, such as the renal and cardiac sympathetic nerves (Sell, Erdelyi, and Schaefer 1958,[30] Weidinger, Fedina, and Kehrel 1963,[31] Coote and Downman 1966,[32] Katunsky and Khayutin 1968,[33] Kirchner, Sato, and Weidinger 1971[34]). Usually in these experiments peripheral limb nerves were stimulated, so the reflexes were more or less of the medullary variety only.

In addition to the early spinal and late supraspinal reflex components induced by myelinated somatic afferents, Sato (1972)[35] has recently reported the existence of a "very late" reflex discharge with a latency of 300–350 msec when recorded from lumbar white rami. This late reflex is seen only in animals under light chloralose anesthesia, which may be the main reason why it has not been previously recognized. It was concluded that the very late reflex discharge has a suprapontine reflex pathway.

Central pathways of reflexes induced by unmyelinated somatic afferents. The various and often conflicting reports on somatosympathetic reflexes evoked by unmyelinated somatic afferents (group IV reflexes) have been reviewed in some detail by Schmidt and Weller (1970).[21] More recently Sato in 1973[25] in a series of experiments on intact, acute, and chronic spinal cats was able to resolve most of the existing discrepancies and

to present a coherent picture of the group IV reflex pathways: (1) Afferent inputs entering the spinal cord at the same or adjacent segment of the sympathetic outflow under observation evoke group IV reflexes. The group IV reflexes are seen after single volleys and do not need temporal facilitation. The pathway for these reflexes is complete at the spinal level. (2) Afferent inputs entering the spinal cord at segments different from those in which the sympathetic outflow is measured evoke the group IV reflexes in the anesthetized animal with intact neuraxis. Temporal facilitation is needed to obtain maximum reflex effects. This reflex disappears after spinal transection; its pathway is presumably complete at the medullary level. (3) In well-kept chronic spinal cats, about 3 months after operation, afferent inputs entering the spinal cord at segments different from those from which the sympathetic outflow is measured elicit the group IV reflexes that are not seen in the intact anesthetized animal and do not need temporal facilitation for maximum reflex effects.

Convergence of various reflex pathways on preganglionic and postganglionic neurons. By testing the discharge properties of single preganglionic neurons in lumbar white rami it is possible to determine to what extent the preganglionic neurons serve or do not serve as a final common path for the different reflex pathways (Kaufman and Koizumi, 1971[36]). So far, Sato (1972)[11] tested 76, sympathetic single units for their excitation by the four major reflex pathways – the early, late, and very late group II and III afferent reflex pathways and the group IV reflex pathway (figure 1). All 16 combinations of response behavior have been found, and surprisingly, there were as many neurons showing a high degree of specificity (22 neurons were excited via one pathway only) as neurons having a high degree of convergence (36 had convergence from all 4, 24 from 3 pathways). At present it is an open question as to what extent the great variety of reflex response patterns of preganglionic units reflects different functional roles and/or destinations in different effector organs.

The various central pathways of somato-sympathetic reflexes ending in preganglionic units are outlined in figure 1, which contains all the information presently available in regard to the convergence of the central excitatory and inhibitory effects of somatic nerve activity on the preganglionic side of the sympathetic nervous system.

In the postganglionic fibers of the cutaneous nerves of the hindlimb, stimulation of hindlimb nerves evokes the late medullary reflex in the majority of nerve units, whereas the group IV reflex is elicited in a much smaller percentage of units (Jänig, Sato, and Schmidt 1972[12]). In muscle nerves of the hindlimb the situation is reversed: More units show group IV reflexes than medullary reflexes (Koizumi and Sato 1972[13]). All these units, both in muscle and cutaneous nerves, are

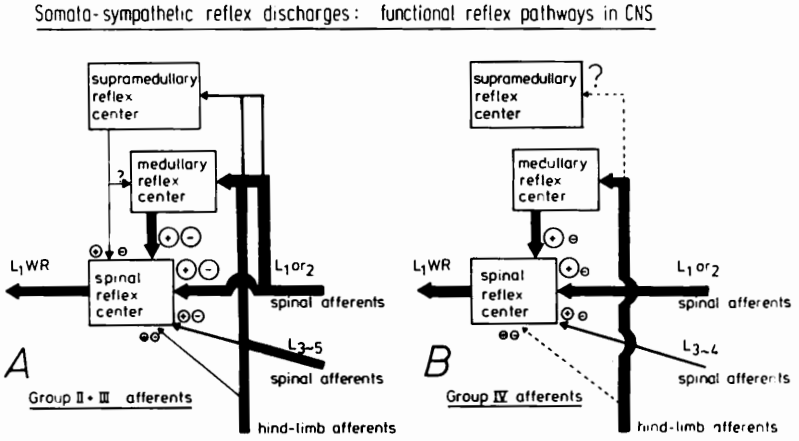


Fig. 1. Central reflex pathways of somato-sympathetic reflexes. *A:* Central pathways of those reflexes induced by myelinated somatic afferents. *B:* Corresponding pathways for unmyelinated afferents. Thicknesses of various pathways are a measure of their potency. Excitatory or inhibitory effects are indicated by + and − signs, respectively. Sizes of signs provide a rough measure of effectiveness of excitatory or inhibitory action.

considered to be vasoconstrictors, the difference in their response behavior indicating their different functions.

IV. RECENT STUDIES ON THE CUTANEO-VISCERAL REFLEXES

As noted above some of the most interesting and important recent findings in the field of neurophysiological studies seem to be the establishment of propriospinal as well as supraspinal components in the somato-sympathetic reflexes and the analysis of the segmental organization of the propriospinal reflex. The question then arises: Can the results of this recent neurophysiological analysis of the somato-sympathetic reflexes be actually applied to the reflex responses of the visceral organs in response to somatic afferent excitation? Little is known concerning the somato-visceral reflex responses, and there is a great gap between the results of recent studies at the nerve level and those at the effector organ level. For the last year our laboratory has been carrying on a systematic series of experiments of the somato-visceral reflexes both at the level of visceral organs such as the heart, gastro-intestinal tract and urinary bladder and at the level of the autonomic nerve fibers. The purpose of this series of the experiments is first to determine the existence of somato-visceral reflex responses and second, if there is any reflex response, to analyze the neural control mechanism of the reflex. I would like to introduce some of the results of our recent series of experiments on the cutaneo-visceral reflexes in rats (Sato, Sato, Shimada, and Torigata 1975[37,38,39]), since I believe these results are important not only in the field of basic research of the somato-sympathetic reflexes, but also for developing clinical applications of these reflexes.

Cutaneo-gastric reflexes. When the intra-pyloric balloon pressure was increased from 0 to about the 100 to 130 mm H_2O levels, pyloric contractions

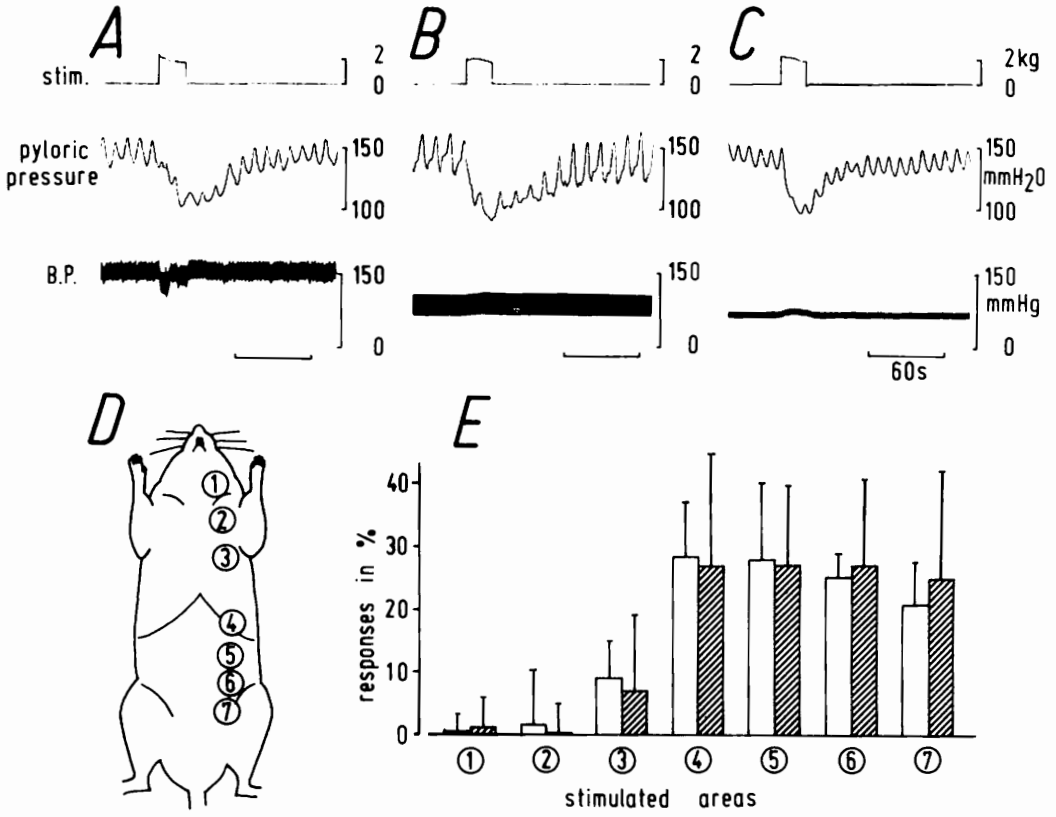


Fig. 2. Effect of cutaneous nociceptive stimulation on the gastric movement. Examples of changes in pyloric pressure (middle traces) produced by nociceptive stimulation of the abdominal skin (as indicated in top traces) are shown in three different rats (*A*, *B*, and *C*) with simultaneous recordings of blood pressure (lower traces). *A:* CNS intact chloralose anesthetized rat. *B:* Decerebrated nonanesthetized rat. *C:* Spinalized anesthetized rat. *D.:* Various areas on the left mammillary line used for nociceptive stimulation. *E:* Histogram summarizing the results of changes in pyloric pressure produced by stimulation of various areas indicated in *D* in the seven rats before spinal transection (white column) and in the seven rats after spinal transection (hatched column) (from Sato et al.[37]).

of five to six per minute were observed and continuously recorded. The pyloric pressure decreased after nociceptive stimulation of the abdominal skin in the CNS intact anesthetized, the decerebrated nonanesthetized and spinalized rats as shown in figure 2(A, B, C). This inhibitory pyloric response was completely abolished after destroying the spinal cord between Th5 to Th11 by passing a small wire cable back and forth in the vertebral canal several times. Therefore the inhibitory pyloric response must have been produced as a result of cutaneo-gastric reflex response. Transection of the bilateral vagal nerves at the cervical level did not abolish the inhibitory cutaneo-gastric response. On the contrary the inhibitory cutaneo-gastric response was abolished completely after (1) cutting the bilateral splanchnic nerves, or (2) crushing bilateral coeliac ganglia in the rat whose vagal nerves were intact. These results indicate that the sympathetic nerve activity is essential for the inhibitory cutaneo-gastric response. Nociceptive stimulation of the abdominal skin produced an increase in the discharge activity of a postganglionic sympathetic nerve branch to the stomach in the CNS intact anesthetized rat and as well as in the same rat after spinal transection as shown in figure 3(A and B). On the contrary, spontaneous nerve activity recorded from gastric vagal nerve branches was not influenced by nociceptive stimulation of abdominal skin as shown in figure 3(C). From the data on denervation and action potentials of the gastric sympathetic as well as parasympathetic nerve branches it was concluded that the reflexly increased discharge activities of the gastric sympathetic nerve branches are responsible for producing the inhibitory cutaneo-gastric reflex responses and that the reflex does occur at the spinal level. The vagal efferent activity does not seem to be directly responsible for producing the cutaneo-gastric reflex. The total results of pyloric pressure response to nociceptive stimulation of various localized skin areas (as shown diagrammatically in figure 2(D) of the 7 CNS intact and 7 spinal rats are presented in a histogram in figure 2(E). This histogram shows that the pyloric pressure was strongly decreased by stimulation of the abdominal or near abdominal skin areas and that stimulation of other areas had little effect. Especially it should be emphasized that the reflex responses in the CNS intact as well as in the spinal rats are almost identical. Those results indicate that the inhibitory cutaneo-gastric

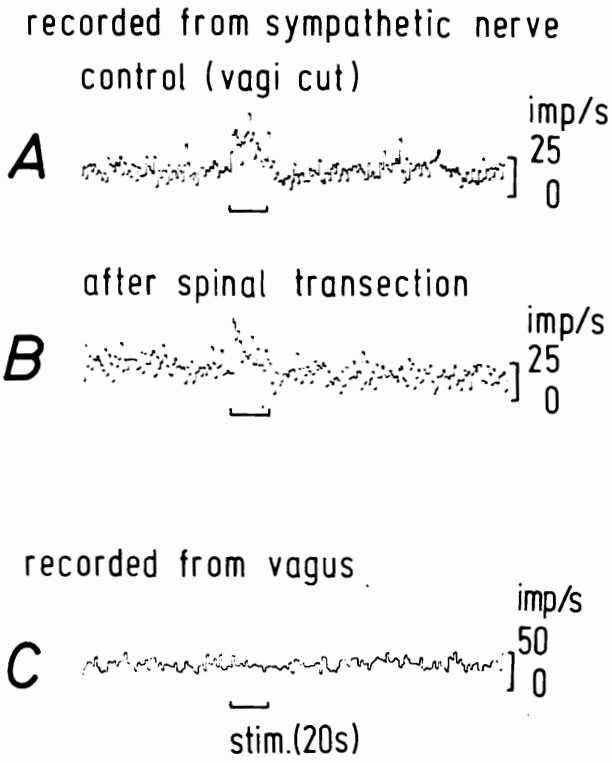


Fig. 3. Effect of cutaneous nociceptive stimulation on discharge activity of the autonomic nerves innervating the stomach. *A* and *B*: Discharge activities recorded from a sympathetic nerve branch going to the stomach, before (*A*) and after (*B*) spinal transection in the same rat. *C*: Recorded from a vagus nerve branch going to the stomach. Nociceptive stimulation was given to the abdominal skin for 20s as indicated by underbars (from Sato et al.[37]).

reflex response is primarily a propriospinal somato-sympathetic reflex.

Cutaneo-cardiac reflexes. In CNS intact chloralose anesthetized rats or in the decerebrated nonanesthetized rats, nociceptive cutaneous stimulation of the various areas on a mammillary line could reflexly produce changes in the heart rate as shown in figure 4(A and B). When the rectal temperature was maintained at around 38.5° C, stimulation of all these skin areas caused an increase in the heart rate. However after spinal transection at the cervical level, a large response in the heart rate could be obtained only by cutaneous stimulation of the chest (see figure 4(C)). Therefore, in this excitatory cutaneo-cardiac reflex, it should be emphasized that the segmental organization of the reflex is clear in the spinalized preparation and that this segmental organization is modified into a generalized reflex response by the existence of the supraspinal structures (see figure 4 D and E). Furthermore, it was shown that the

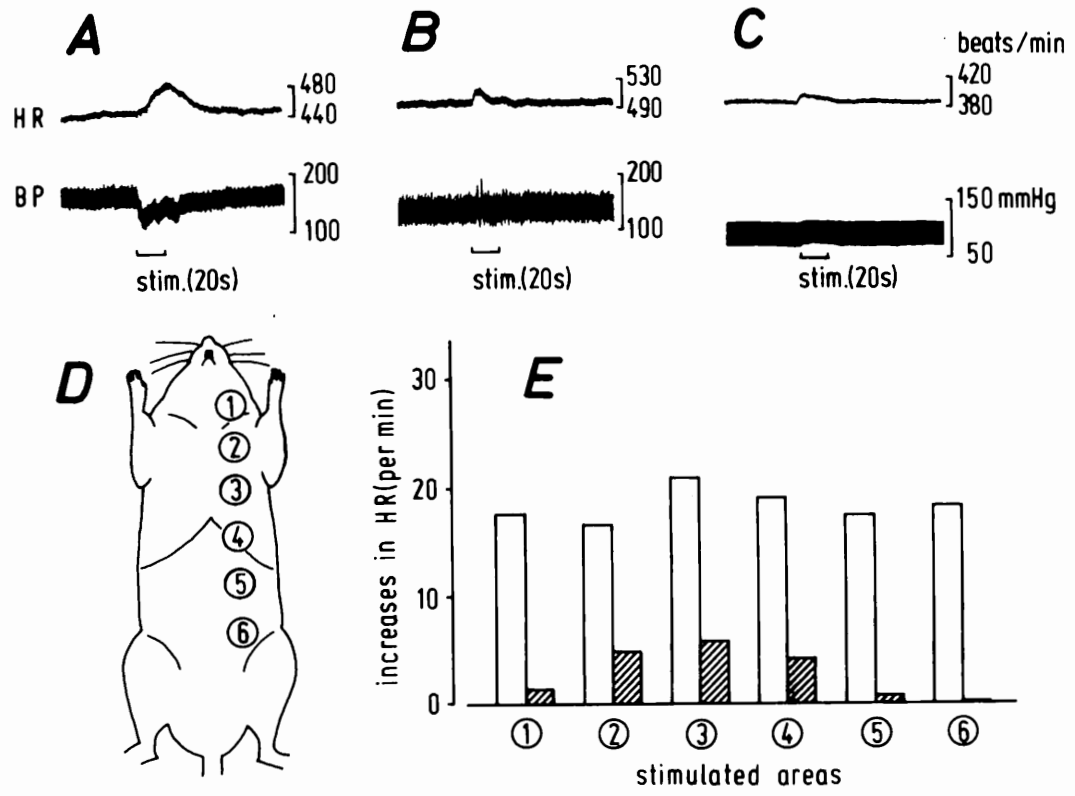


Fig. 4. Effect of cutaneous nociceptive stimulation on the heart rate. Examples of changes in heart rate (upper traces) produced by nociceptive cutaneous stimulation of the chest are shown in three different rats (*A*, *B*, and *C*) with simultaneous recordings of blood pressure (lower traces). *A:* CNS intact chloralose anesthetized rat. *B:* Decerebrated nonanesthetized rat. *C:* Spinalized rat. Stimulations were given for 20s as indicated by bottom bars. *D:* Various areas on the left mammillary line used for nociceptive stimulation. *E:* Histogram summarizing the results of changes in heart rate produced by stimulation of various areas indicated in *D* in the same rat before (white column) and after (hatched column) spinal transection.

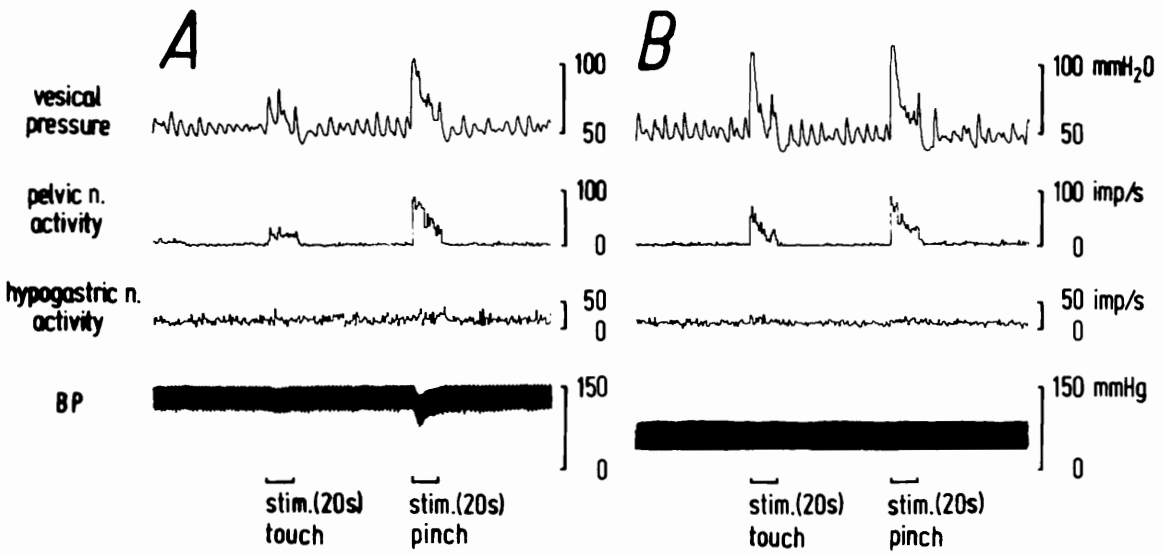


Fig. 5. Effect of perineal cutaneous stimulation by touching and pinching on vesical pressure. Results in *A* and *B* were obtained in the same rat, anesthetized with chloralose-urethane. Results in *A* were taken before spinal transection, and those in *B* were taken after spinal transection at the second cervical level. Intravesical pressure (the top traces in *A* and *B*). Pelvic nerve activity (the second traces from the top). Hypogastric nerve activity (the third traces from the top) and blood pressure (the fourth traces from the top). Each stimulation by touching and pinching was given for 20s as indicated by the bottom bars (from Sato et al.[38]).

597-036 O - 76 - 12

increased activity of the cardiac sympathetic nerve branches is essential for producing such an excitatory cutaneo-cardiac reflex. In other words, the somatosympathetic reflexes are responsible to the excitatory cutaneo-cardiac reflexes.

Cutaneo-vesical reflexes. When the intravesical balloon pressure was slightly increased by injecting water into balloon from 0 to a low intravesical pressure of about 40 ± 25 mm H_2O spontaneous contractions with a low amplitude of about 10 ± 5 mm H_2O could be continuously recorded. These vesical contractions were observed essentially in the same manner in the CNS intact anesthetized, the decerebrated nonanesthetized and the spinalized rats. Touch or nociceptive cutaneous stimulations to the perineal area caused an abrupt increase in the intravesical pressure of about 20–60 mm H_2O as shown in figure 5. Corresponding to increases in the intravesical pressure due to perineal stimulation, there were always marked increases in discharge activity of the pelvic nerve branches to the bladder. Simultaneously, some very slight changes in discharge activity of the hypogastric nerve branches to the bladder were noted. These responses essentially remained the same in the animal after spinal transection at the middle thoracic level. This excitatory cutaneo-vesical reflex was shown to be a propriospinal reflex whose efferent pathways are the pelvic nerve branches. This is the somato-parasympathetic reflex.

When the intravesical pressure was set to about 215 ± 55 mm H_2O by injecting water 0.1 to 0.7 cc into the balloon, fairly large rhythmical contractions, called micturition contractions, with amplitude of 610 ± 150 mm H_2O of a frequency of about 0.6 to 3 per minute were obtained. These micturition contractions were completely abolished either after denervation of the bilateral pelvic nerve branches or after spinal transection at the cervical or at the middle thoracic level. Therefore, these rhythmic micturition contractions must have been activated by pelvic efferent nerve activity due to the existence of the supraspinal structures. Simultaneous recordings of the efferent discharge activity of the pelvic vesical nerve branches showed rhythmic discharges corresponding to the large vesical contractions. Cutaneous nociceptive stimulation localized in the perineal area most often caused an inhibition of the large contractions as well as the disappearance of the rhythmic discharges of the pelvic vesical nerve branches as shown in

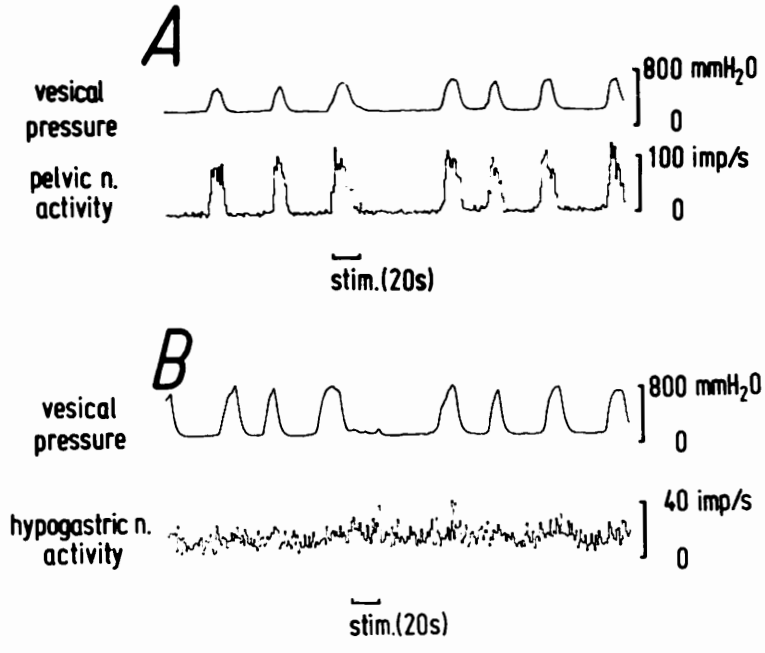


Fig. 6. Effect of cutaneous nociceptive stimulation on micturition contractions of the bladder. Micturition contractions (upper traces in *A* and *B*). Pelvic nerve activity (bottom trace in *A*) and hypogasyric nerve activity (bottom trace in *B*). The results in *A* and *B* were taken from the same CNS intact chloralose anesthetized rat. Nociceptive stimulations were given to the perineal skin for 20s as indicated by the bottom bars in *A* and *B* (from Sato et al.[38]).

figure 6. These results indicate that perineal stimulation can reflexly affect bladder function, in either an excitatory or inhibitory way depending on whether the bladder is or is not resting.

V. CONCLUDING REMARKS

I began with a brief review of neurophysiological studies on the somato-sympathetic reflexes and, in particular, of recent results of somatic afferents producing sympathetic reflexes and the central reflex pathways of these reflexes. Then I discussed recent studies on the cutaneo-visceral reflexes performed in rats. I would like to emphasize again that the functions of the various visceral organs can be influenced by a proper cutaneous stimulation as a result of the somatosympathetic or the somatoparasympathetic reflexes. I trust that such research will be expanded to include a higher species of mammals, i.e. cats and monkeys and that finally, this knowledge of the cutaneo-visceral reflexes will be clinically useful in altering the visceral functions of humans.

REFERENCES

1. Kuntz, A., Anatomic and physiologic properties of cutaneo-visceral vasomotor reflex arcs. *J. Neurophysiol.* 8: 421–430, 1945.
2. Sato, A., and Schmidt, R. F. Somatosympathetic reflexes: Afferent fibers, central pathways, discharge characteristics. *Physiol. Rev.* 53:916–947, 1973.
3. Ranson, S. W., and Billingsley, P. R. Vasomotor reactions from stimulation of the floor of the fourth ventricle. Studies in vasomotor reflex arcs. III. *J. Physiol.* (London) 41:85–90, 1916.
4. Sherrington, C. S. *The Integrative Action of the Nervous System.* New Haven, Conn: Yale Univ. Press, 1906.
5. Brooks, C. M. Reflex activation of the sympathetic system in the spinal cat. *Am. J. Physiol.* 106:251–266, 1933.
6. Alexander, R. S. Tonic and reflex functions of medullary sympathetic cardiovascular centers. *J. Neurophysiol.* 9:205–217, 1946.
7. Sato, A., and Schmidt, R. F. Muscle and cutaneous afferents evoking sympathetic reflexes. *Brain Res.* 2:399–401, 1966.
8. Schmidt, R. R., and Schönfuss, K. An analysis of the reflex activity in the cervical sympathetic trunk induced by myelinated somatic afferents. *Arch. Ges. Physiol.* 314: 175–198, 1970.
9. Sato, A., and Schmidt, R. F. Ganglionic transmission of somatically induced sympathetic reflexes. *Arch. Ges. Physiol.* 326:240–253, 1971.
10. Jänig, W., and Schmidt, R. F. Single unit responses in the cervical sympathetic trunk upon somatic nerve stimulation. *Arch. Ges. Physiol.* 314:199–216, 1970.
11. Sato, A. The relative involvement of different reflex pathways in somato-sympathetic reflexes, analyzed in spontaneously active single preganglionic sympathetic units. *Arch. Ges. Physiol.* 333:70–81, 1972.
12. Jänig, W., Sato, A., and Schmidt, R. F. Reflexes in postganglionic cutaneous fibers by stimulation of group I to group IV somatic afferents. *Arch. Ges. Physiol.* 331: 244–256, 1972.
13. Koizumi, K., and Sato, A. Reflex activity of single sympathetic fibers to skeletal muscle produced by electrical stimulation of somatic and vago-depressor afferent nerves. *Arch. Ges. Physiol.* 332:283–301, 1972.
14. Ranson, S. W. Afferent paths for visceral reflexes. *Physiol. Rev.* 1:477–522, 1921.
15. Laporte, Y., and Montastruc, P. Róle des différents types de fibres afférentes dans les réflexes circulatoires généraux d'origine cutanée, *J. Physiol.* (Paris) 49:1039–1049, 1957.
16. Laporte, Y., Bessou, B., and Bouisset, S. Action réflexe des différents types de fibres afférentes d'origin musculaire sur las pression sanguine. *Arch. Ital. Biol.* 98: 206–221, 1960.
17. Fedina, L., Katunskii, A. Y., Khayutin, V. M., and Mitsányi, A. Response of renal sympathetic nerves to stimulation of afferent A and C fibers of tibial and mesenterial nerves. *Acta Physiol. Acad. Sci.* Hung. 29:157–176, 1966.
18. Coote, J. H., and Perez-Gonzalez, J. F. The response of some sympathetic neurones to volleys in various afferent nerves. *J. Physiol.* (London) 208:261–278, 1970.
19. Khayutin, V. M., and Lukoshkova, E. V. Spinal mediation of vasomotor reflexes in animals with intact brain studied by electrophysiological methods. *Arch. Ges. Physiol.* 321:197–222, 1970.
20. Koizumi, K., Collin, R., Kaufman, A., and Brooks, C. McC. Contribution of unmyelinated afferent excitation to sympathetic reflexes. *Brain Res.* 20:99–106, 1970.
21. Schmidt, R. F., and Weller, E. Reflex activity in the cervical and lumbar sympathetic trunk induced by unmyelinated somatic afferents. *Brain Res.* 24:207–218, 1970.
22. Sato, A., Tsushima, N., and Fujimori, B. Reflex potentials of lumbar sympathetic trunk with sciatic nerve stimulation in cats. *Japan. J. Physiol.* 15:532–539, 1965.
23. Sato, A., Kaufman, A., Koizumi, K., and Brooks, C. McC. Afferent nerve groups and sympathetic reflex pathways. *Brain Res.* 14:575–587, 1969.
24. Koizumi, K., Sato, A., Kaufman, K., and Brooks, C. McC. Studies on sympathetic neuron discharges modified by central and peripheral excitation. *Brain Res.* 11:212–224, 1968.
25. Sato, A. Spinal and medullary reflex components of the somato-sympathetic reflex discharges evoked by stimulation of the group IV somatic afferents. *Brain Res.* 51: 307–318, 1973.
26. Sato, A., and Schmidt, R. F. Spinal and supraspinal components of the reflex discharges into lumbar and thoracic white rami. *J. Physiol.* (London) 212:839–850, 1971.
27. Sato, A. Spinal and supraspinal inhibition of somatosympathetic reflexes by conditioning afferent volleys. *Arch. Ges. Physiol.* 336:121–133, 1972.
28. Beacham, W. S., and Perl, E. R. Background and reflex discharge of sympathetic preganglionic neurones in the spinal cat. *J. Physiol.* (London) 172:400–416, 1964.

29. Beacham, W. S., and Perl, E. R. Characteristics of a spinal sympathetic reflex. *J. Physiol.* (London) 173:431–448, 1964.
30. Sell, R., Erdelyi, A., and Schaefer, H. Untersuchungen über den Einfluβ peripherer Nervenreizung auf die sympathische Aktivität. *Arch. Ges. Physiol.* 267:566–581, 1958.
31. Weidinger, H., Fedina, L., and Kehrel, H. Der Einfluβ von Adrenalin auf die Tätigkeit des "Sympathicus." *Arch. Ges. Physiol.* 278:229–240, 1963.
32. Coote, J. H., and Downman, C. B. B. Central pathways of some autonomic reflex discharges. *J. Physiol.* (London) 183:714–729, 1966.
33. Katunsky, A. Y., and Khayutin, V. M. The reflex latency and the level of mediation of spinal afferent impulses to the cardiovascular sympathetic neurones. *Arch. Ges. Physiol.* 298:294–304, 1968.
34. Kirchner, F., Sato, A., and Weidinger, H. Bulbar inhibition of spinal and supraspinal sympathetic reflex discharges. *Arch. Ges. Physiol.* 326:324–333, 1971.
35. Sato, A. Somato-sympathetic reflex discharges evoked through supramedullary pathways. *Arch. Ges. Physiol.* 332:117–126, 1972.
36. Kaufman, A., and Koizumi, K. Spontaneous and reflex activity of single units in lumbar white rami. In: *Research in Physiology,* A Liber Memorialis in Honor of Prof. Chandler McC. Brooks, edited by F. F. Kao, M. Vasalle, and K. Koizumi. Bologna: Aulo Gaggi Publ., pp. 469–481, 1971.
37. Sato, A., Sato, Y., Shimada, F., and Torigata, Y. Changes in gastric motility produced by nociceptive stimulation of the skin in rats. *Brain Research,* 87:151–159, 1975.
38. Sato, A., Sato, Y., Shimada, F., and Torigata, Y. Changes in vesical function produced by cutaneous stimulation in rats. *Brain Research,* 94:465–474, 1975.
39. Sato, A., Sato, Y., Shimada, F., and Torigata, Y. Varying changes in heart rate produced by cutaneous nociceptive stimulation of the skin in rats at different temperature. *Brain Research* (in press).

Pain: Spinal and Peripheral Nerve Factors

EDWARD R. PERL
Professor
Department of Physiology
University of North Carolina

This discussion is not meant as an extensive survey of the peripheral and spinal cord mechanisms associated with physical pain, but rather as a selective consideration of morphological, physiological and pathophysiological factors of importance to this question. The emphasis has been dictated as much by what is known about pain-related neural mechanisms as by what might be germane to the question of the alteration of pain by manipulation.

Pain as a sensation. It is now generally accepted that pain is a somatic sense, a perceptual experience akin to the appreciation of touch and temperature.[1] It has been argued that pain has special attributes which set it aside from other sensations[2,3]; however, our present understanding suggests that there are more similarities than differences between pain and other sensations.[4]

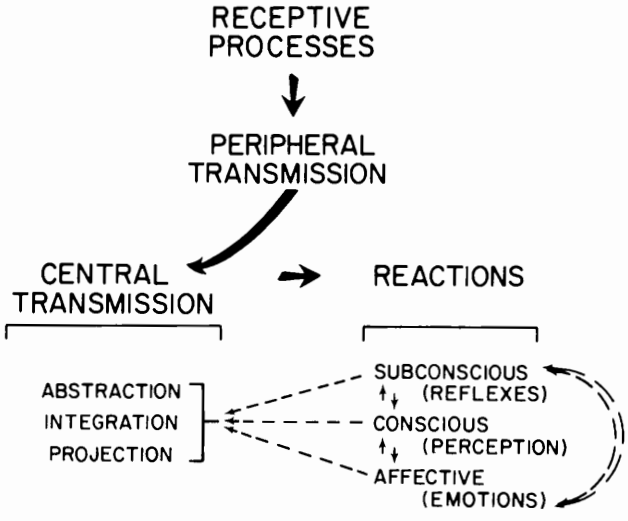


Fig. 1. Schematic representation of neural processes associated with conscious sensation.

Figure 1 provides a simplified, diagrammatic representation of some neural processes or steps that appear common to all sensations, regardless of modality. Sensations begin with a certain state (or change) of the body's internal or external environment. In this regard, the apparent variety and ubiquitous effectiveness of environmental alterations or stimuli responsible for pain puzzled early observers. Sherrington[5] provided insight by pointing out that stimuli threatening the physical integrity of tissue regularly cause pain. On this basis, he suggested labelling stimuli which tend to, or actually damage tissue as noxious, thereby implying a damaging quality. Noxious stimuli can be of any type – mechanical, chemical, radiant; the intensity which becomes noxious can be expected to differ according to the tissue type and its location. Thus, a starting point for considering the sensory mechanisms leading to pain consists of determining the means by which noxious circumstances (stimuli) are detected. In figure 1 this step is indicated as "receptive processes"; the latter are followed by transmission of information centrally. The neural discharges produced by every sensory receptor are projected to more than one nucleus or locus of the central nervous system and in the process are modified or operated upon by other neural mechanisms. Finally, with varying directness, reactions take place, one of which is associated with conscious or perceptual recognition of the stimulus. Figure 1 suggests the existence of considerable interaction between the various concomitants of sensation, a consideration which must be kept in mind whenever modification of an endpoint such as conscious appreciation of pain is considered.

Receptive processes. The events in the detection

of the stimulus by a sense organ deserve brief attention in view of later pathophysiological considerations. The stimulus, in our case a noxious event, causes a change in the tissue surrounding specialized sense cells or a sensory nerve terminal. The results of the changes in the tissue – mechanical distortion, a physiochemical interaction and/or the liberation of a substance – in turn cause a decrease of the transmembrane potential of specialized sense cells or directly of a sensory nerve terminal. Only then are the conducted action potentials initiated. This process involves the transduction of the energy of the stimulus into a series of discontinuous events, action potentials, in the elongated extensions of sensory nerve cells in the dorsal root ganglia which make up the sensory component of the peripheral nerves.

Peripheral transmission. Conveying information by impulses places constraints on the quantity of information which a given channel (nerve fiber) can transmit. It seems apparent that a single kind of nerve element does not have the capacity to provide information necessary to distinguish between the various stimuli man or animals encounter in their natural history and to give data on the intensity of such events. Somatic sense organs (sensory receptors) exhibit specialization in sensitivity to various kinds of stimuli; one type readily excited by a class of events as, for example, a transient mechanical distortion, is poorly excited by others such as temperature changes. Clues to a specialization of sense organs for noxious stimuli and pain came from experiments originally done over 40 years ago, which demonstrated that only part of the afferent fibers making up typical peripheral nerves are critical for the evocation of pain or pain-like reactions. Figure 2, taken from a paper by Heinbecker, Bishop and O'Leary published in 1933[6], shows compound action potentials recorded from a man's peripheral nerve; several deflections appearing after a single electric shock result from the different conduction velocities of impulses in fractions of the population of fibers making up the nerve. By graded stimulation or selective block of the different fibers contributing to components of the compound action potential, it became known that for pain to result, slowly-conducting, fine-calibered afferent fibers must conduct impulses[16].

It has been generally assumed that afferent fibers producing pain, like most afferent fibers, enter the spinal cord through the dorsal spinal roots; however, there is recent evidence that a number of fine diameter (unmyelinated) ventral root fibers are sensory in function[7, 8]. This fact may be significant in pain associated with the back and its alteration by manipulation.

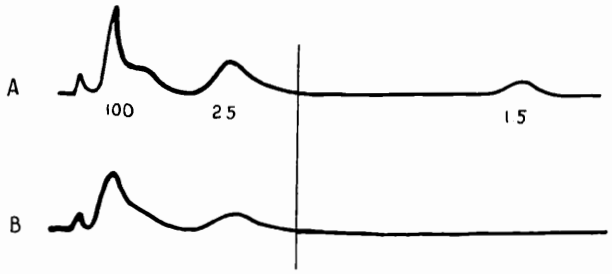


Fig. 2. Compound action potential recorded from a human cutaneous nerve *in vitro*. *A* – response to a supramaximal stimulus. *B* – stimulus intensity evoking report of pain in partially conscious patient before leg amputation. Weaker stimuli, not exciting group conducting at 25 m/sec, did not cause report of pain. Figures are calculated conduction velocity in meters/sec. (From Heinbecker, Bishop and O'Leary, 1933.)

The sense organs or receptors associated with pain. There are many different types of somatic sense organs. It has been postulated from time to time that those associated with other sensations, for example, touch-pressure, respond in some unusual way to noxious stimuli and thereby signal pain-causing circumstances[3]. This possibility has been systematically tested for a number of types of somatic sense receptors by comparing the responses evoked by non-noxious and noxious stimuli.[9, 10, Perl unpublished] An example is illustrated in figure 3, which in graphic form displays the impulse by impulse pattern of activity in a slowly-conducting primary afferent fiber initiated by controlled mechanical stimuli. Figure 3A and B show the pattern of impulses to mechanical deformation of the skin by a blunt probe which did not damage the skin (and was not painful to man.) Figure 3A′ and B′ show the responses initiated by a mechanical movement of the same amplitude and location by a sharp needle which penetrated the skin. The graphs show no systematic difference in the response patterns. Similar results have been obtained whenever somatic sense organs with a low threshold (high sensitivity) to non-noxious stimuli, including those with slowly-conducting peripheral nerve fibers, have been subjected to a deliberate test of differences in response between non-noxious and

noxious stimuli. These data have two implications: (1) ordinary receptors do not provide appropriate signals to allow the central nervous system to distinguish between noxious and non-noxious events and (2) not all primary afferent neurons with slowly-conducting fibers are important for the process of nociception and pain.

more vigorously when a still more damaging stimulus, pinching with a sharp toothed forceps, was used.

Several kinds of high threshold sensory receptors have been identified. In the skin certain high threshold receptors are responsive only to mechanical stimuli, while others are excited by a variety

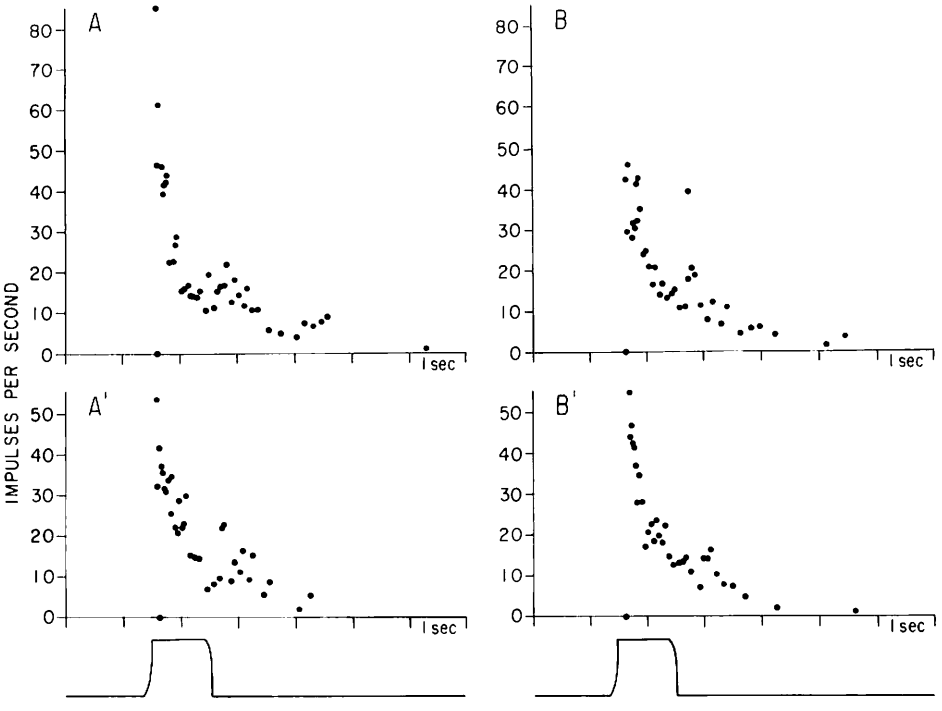


Fig. 3. Plot of responses of a low threshold cutaneous mechanoreceptor with a slowly conducting "C" afferent fiber to non-noxious (*A*, *B*) and noxious (*A'*, *B'*) stimuli. Stimulus timing and form shown in tracing below the graphs. Stimuli were 1200 μ movements by an electromechanically controlled probe with a blunt (1 mm diameter) for *A*, *B* and sharp (needle) tip for *A'*, *B'*. *A* series were the first and *B* series the third of a sequence with each probe. Graph ordinates show "instantaneous frequency," i.e., the reciprocal of the time interval between successive impulses. (From Bessou, Burgess, Perl and Taylor, 1971.)

Such conclusions emphasize the importance of studies which have shown that a fraction of primary afferent neurons with slowly-conducting peripheral afferent fibers have elevated thresholds for all kinds of somatic stimuli.[9, 11, 12] An example is shown in figure 4. In figure 4A, pressure was directed against a spot on the skin by a small disc with forces exceeding 100 gm. and no impulses were evoked in the afferent nerve fiber. In figure 4B, the small disc was replaced with a sharp needle and the same spot on the skin was pressed upon, the needle penetrating the skin at forces exceeding 5 gm.; impulses were regularly initiated by this clearly noxious form of stimulation. Figure 4C shows that this "high threshold" receptor responded even

of noxious events, heat, strong mechanical stimulation and irritant chemicals. High threshold receptors in muscles are excited by intense pressure or other non-physiological muscle distortion and by certain irritant chemical agents.[13, 14] Joint receptors have been identified which discharge only when a joint is extended beyond its normal or physiological range.[15] It is interesting that the unmyelinated afferent fibers recently demonstrated in ventral roots are reported to be largely of the high threshold type; in a sacral root, the majority of unmyelinated afferent fibers were found to innervate visceral structures.[7, 8]

Hyperalgesia and inflammation. Noxious stimulation of the skin and certain other structures is

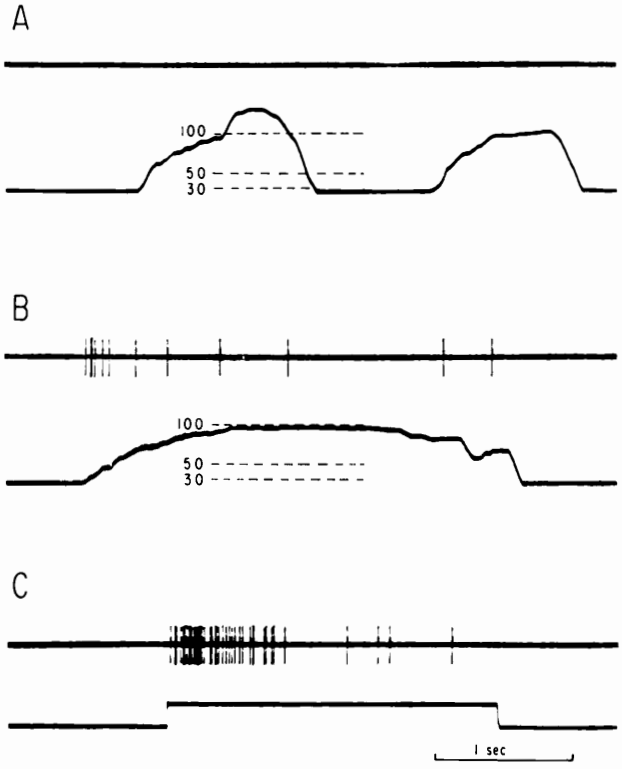


Fig. 4. Responses of a primate cutaneous nociceptor (upper traces) with a slowly conducting myelinated afferent fiber. For *A* and *B*, force of mechanical stimulus indicated by deflection of lower traces (calibration in grams). All stimuli applied to same skin area. *A*—pressure exerted by 2.2 mm diameter probe; *B*—pressure exerted by a needle; *C*—forcible pinch with serrated forceps (at deflection of lower beam). (From Perl, 1968.)

known to be associated with persisting changes in sensibility. This change in sensation takes the form of a lowered threshold for the production of pain (hyperalgesia), not only at the injured site, but also in surrounding tissue.[16] Pain from ordinarily nonpainful stimuli is also a regular concomitant of inflammation practically anywhere in the body. The correlation with inflammation has long been common knowledge and serves as an important diagnositic sign of an inflammatory process. Several hypotheses have been put forth to explain hyperalgesia and the pain of inflammation. One presumes the release of a diffusible substance by injured tissues which serve to lower the threshold of sensory fibers responsible for pain and another suggests the existence of a special peripheral nerve network excited by noxious events.[16]

Sensitization of nociceptors. A remarkable change in responsiveness and the lowering of threshold with repeated stimuli have been observed for nociceptors with unmyelinated afferent fibers. The polymodal nociceptors of the skin, a sensory receptor neuron with an unmyelinated or (C) fiber, respond vigorously to intense mechanical stimuli, noxious heat and irritant chemicals. The response of one such element to a controlled heating of the skin is graphically shown in figure 5A. After the heating phase in which the receptor was activated, the receptor began a low frequency discharge at skin temperatures below its original threshold. Such ongoing or persisting discharge is a regular postactivation feature for this kind of nociceptor and lasts for hours. Figure 5B plots the responses shown for the first 150 sec of figure 5A as a function of temperature. Figure 5C shows the responses to an equivalent 150 sec for a second similar heating cycle which began immediately after that shown in figure 5A. More impulses and a higher frequency of activity were evoked by the second heating (figure 5C) than by the initial phase of stimulation (figure 5B). In addition to the background activity

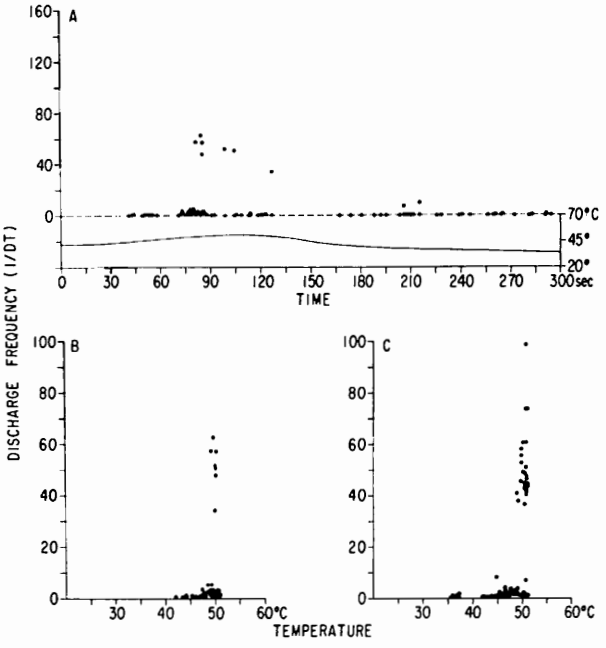


Fig. 5. Responses of a nociceptor ("C" fiber, polymodal type) to skin heating. Filled circles positioned on the ordinate scale according to instantaneous frequency (see Fig. 3). *A*—thermode temperature shown by unbroken line (right scale). *B*—plot of results for initial 150 sec of *A* as function of thermode temperature (abscissa). *C*—plot of initial 150 sec of a second similar heating cycle for the same receptor. (From Perl, in *Cervical Pain*, C. Hirsch & Y. Zotterman, eds., Pergamon Press, New York, 1972.)

and enhanced responses after an initial exposure to damaging stimuli, polymodal nociceptors also often exhibit significant lowering of threshold. The threshold change may be such that innocuous stimuli, previously ineffectual, can provoke a significant amount of response. In light of these observations, background discharge and enhanced responses of nociceptors should be a consideration in analyses of hyperalgesia and the pain of inflammation.

The mechanisms underlying sensitization or enhanced responsiveness of nociceptors are not known, although the question is under active investigation. One hypothesis illustrated by figure 6 proposes that damaged and inflamed tissue produces an unknown substance, "P" [16], which acts upon the nerve terminals to partially depolarize them and thereby lower their threshold for the initiation of action potentials. Figure 7 diagrams a scheme carrying this hypothesis further by suggesting that a noxious stimulus may directly activate some nociceptors and at the same time produce tissue damage which releases an agent causing inflammation. The agent for inflammation then activates nociceptors. The inflammatory process itself may produce other substances also capable of exciting nociceptor terminals. The essential point being proposed is that the system leading to pain may be triggered at a much lower level by stimuli in the presence of inflammation. In particular, subthreshold events for receptors leading to pain in normal circumstances can become effective for exciting such sensory terminals in inflamed or damaged tissue.

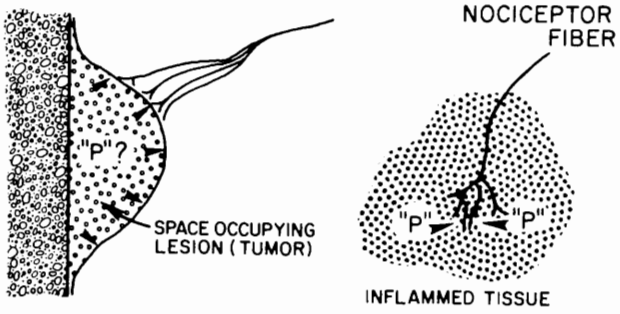


Fig. 6. Diagram showing postulated action of a substance (*P*) on nociceptor terminals leading to sensitization.

Another possible factor is the effect of inflammation directly upon nerve fibers in peripheral

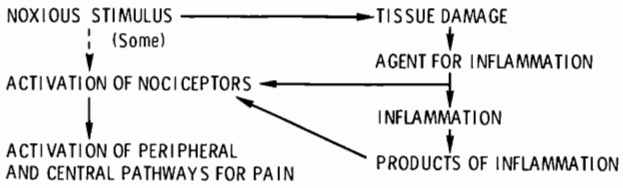


Fig. 7. Representation of possible steps linking nociceptor activation to inflammation from trauma or disease.

nerves or spinal roots. In pathology of the vertebral musculoskeletal system, spinal roots and the associated connective tissue may be subjected to unusual stresses. These unusual stresses could lead to an inflammatory reaction which, in turn, could cause the liberation of substances capable of changing the excitability of nerve fibers in the root or trunk, including those which are central processes of the nociceptive terminals. It is possible that chemical substances related to inflammation can partially depolarize unmyelinated fibers and hence, render such fibers responsive to stimuli which previously could not generate impulses. In this fashion, ordinary mechanical stresses which do not evoke action potentials in root fibers with a normal transmembrane polarization could turn them into effective "receptors". This possibility has been brought to the fore by observations suggesting that low concentrations of certain algogenic substances when applied to nerve trunks can initiate vasomotor reflexes of the type typically associated with pain.[17]

Pathology and disturbed anatomy. Alterations of structure can be expected to alter conditions for activation of nociceptors. When a ligament is torn, an intervertebral disc degenerates or a joint is displaced, major changes take place in the position and stresses upon parts of the musculoskeletal system. Figure 8 diagrams a hypothetical example. Figure 8–I diagrams a normal situation for several vertebrae and ligaments A and B connecting their processes. In figure 8–II an intervertebral disc has partially collapsed (marked by stippling), causing angulation of one vertebra; as a consequence, ligament B is shortened and ligament A lengthened. Any additional tension on ligament A produced by movements associated with ordinary activity could create unusual stresses in it and on its attachments, activating high threshold sensory terminations normally unexcited by the usual range of activity. In time inflammatory changes at the attachments of ligament A as shown in figure

8–III could cause a still further reduction in the amount of movement or stress necessary to excite nociceptors located there.

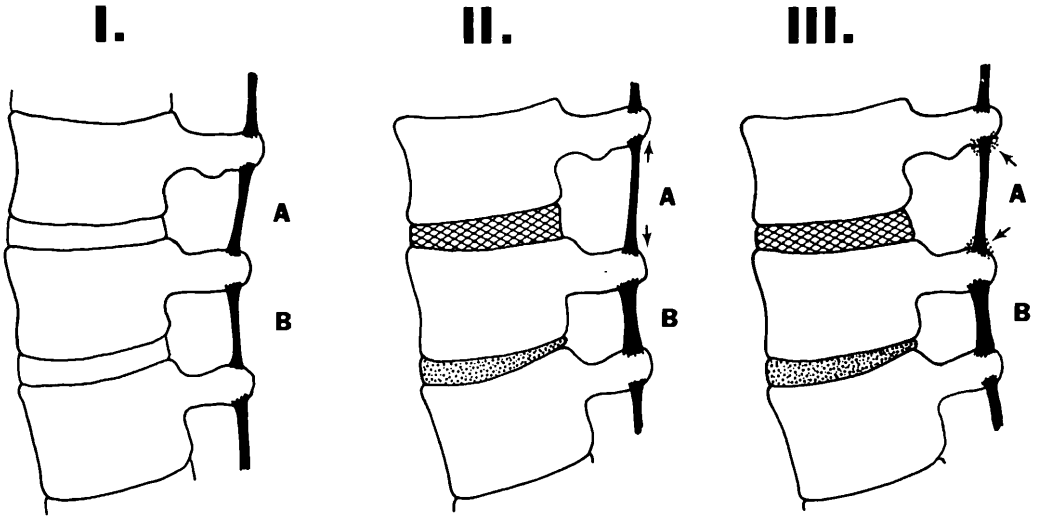


Fig. 8. Diagrammatic representation of the possible place of altered spinal architecture due to disease in activation of nociceptors. See text for additional explanation.

Details of the nature and distribution of nociceptive terminals within the musculoskeletal system are only partially understood. It is known that high threshold sense organs with characteristics appropriate for nociceptors exist in and around joints, within muscles and in tendons and ligaments. Their distribution around the vertebra has not been directly studied. We do not have direct studies of sensory terminations in or around spinal roots, although circumstantial evidence suggests their presence. So far, every tissue studied which is likely to be exposed to unusual stress and noxious circumstances in the ordinary life cycle of an animal has been found to contain sensory terminals with features suggesting a special responsiveness to stimuli intense enough to threaten the integrity of that tissue.

Central mechanisms for pain. For many years a system of nerve fibers running rostrally through the ventrolateral funiculi of the spinal cord (the spinothalamic and spinoreticular tracts) has had accepted importance for pain sensation. It has become increasingly clear over the past 5 years that part of this central projection system originates from neurons of the spinal dorsal horn which receive rather specific excitatory inputs from nociceptors.[18, 19, 20] Many details of the organization of this specific projection system for high threshold sense organs are unknown; however, direct evidence for a specific central pathway for the transmission of information about intense or noxious stimuli is important for explaining the phenomenon of referred pain. Figure 9 diagrams some features of the spinoreticular-spinothalamic system and illustrates one of the hypothesized explanations for referred pain. Pain resulting from pathology or stimuli to one part of the body and referred, in perception, to another body region is known to have a segmental organization; the affected body part and the reference point are both usually innervated by nerve roots entering the spinal cord at the same segmental level. An explanation of referred pain suggested by Ruch[21] implicitly demands a specific projection system. It proposes that under normal circumstances noxious events ordinarily are initiated at the body surface or some other exposed structure. By organization and/or habit, the perceptual reference of neurons carrying nociceptive information centrally is to these structures. Ruch's concept further suggests that some of the same spinal neurons receive a convergent excitatory input from nociceptors of muscle or tendon or other deep structures. When the noxious event takes place in the deep structure, the dorsal horn neuron projecting

centrally over the spinothalamic tract is activated in the same fashion as when a skin nociceptor excites it. The central nervous system interprets the input as if it had come over the more usual channel. This same kind of mechanism could explain the pain of central nervous system origin. Activation of a neuronal system normally excited by the specific input from nociceptors would be interpreted perceptually as if it originated from the usual source. Convergent inputs could come from physically close structures such as parts of the vertebral column and the nearby skin or musculature. In this way a referred locus could, in fact, represent a trigger point since the inputs from two different sources could converge upon one central neuron. A mechanism based upon this organization could represent the underlying cause of persisting back or other musculoskeletal pain long after the initiating event had ameliorated. Convergence of deep (visceral) and cutaneous input upon dorsal horn neurons has been demonstrated, making the convergent hypotheses for the explanation of referred pain all the more attractive.[22, 23]

Mention must also be made of nonspecific central pathways and their possible relation to pain. Some spinal neurons projecting centrally receive excitatory input from nociceptors and from low threshold receptors. It seems probable that such nonspecific central pathways contribute in some way to the overall reaction to a noxious input, including perception. It is possible that in primate and man, some of the nonspecific or convergent pathways also follow the route of the spino-reticular-thalamic system.[24]

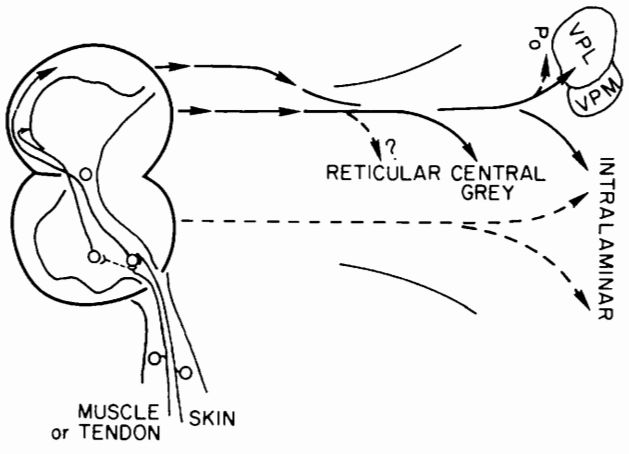


Fig. 9. Schema indicating possible explanation for referred pain on diagram showing the ventrolateral spinal tracts and some of their regions of projection.

The influence of reactions parallel to those causing sensation. Few, if any, primary afferent fibers in their central distribution solely engage a single neural mechanism. It is well established that noxious input or activation of nociceptive neurons evokes a number of different kinds of reflexes. Some are reflexes to skeletal muscle while others involve the autonomic system. Clinical observations have shown that reflex contractions can be persistent and powerful after musculoskeletal injury. These reflexes may have important protective functions in splinting injured parts; however, prolonged muscle contraction in such a reflex can contribute to discomfort, as suggested by figure 10. Limitation of circulation to the contracting muscles and undue stress upon ligaments and other structures by maintained powerful contraction can lead to activation of nociceptors in other structures than the injured part. In addition, voluntary movement in the face of a powerful reflex contraction could cause opposing muscles to generate pathological degrees of stress.

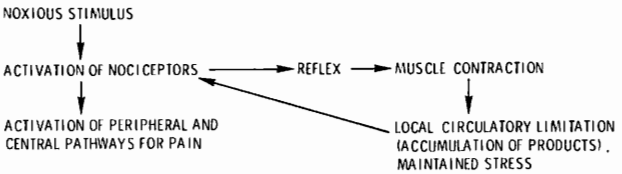


Fig. 10. Representation of possible steps linking concurrent muscular reflexes to reported pain.

A corollary to this concept is that manipulation, which tends to depress reflex contractions related to some continuing inflammatory or nociceptive passes, may alleviate part of the discomfort. One long-established reflex, the lengthening reaction (clasped-knife effect) is initiated by externally imposed lengthening of contracting muscles. At some point in the forced lengthening, inhibition of the motoneurons, causing the muscle to contract, dominates and causes a sudden relaxation of the muscle. The sense organ responsible for the lengthening reaction has been considered to be the Golgi tendon organ of the contracting muscle, although recently there is mounting evidence that the Group II ending of the muscle spindle is the probable source of the inhibitory effect. In any case, the lengthening reaction of muscle deserves special consideration in view of the probable importance

of reflex contraction in the generation of certain kinds of pain.

The modulation of pain mechanisms. Figure 11 diagrams some known and some postulated interactions to a sensory process. It emphasizes the complexity of the interrelations. Given this situation, one should expect that the sensation of pain can be a variable reaction and that many forms of physical and psychological manipulation may alter it, even though there may be highly specific mechanisms for the detection and signalling of strong stimuli.

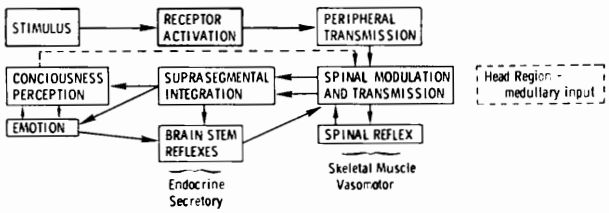


Fig. 11. Schema showing known and hypothesized mechanisms initiated by a somatosensory stimulus and some possible interactions between such processes.

SUMMARY

It has been emphasized that pain is a complex reaction to a definable set of circumstances. It is ordinarily initiated by activation of specific sense organs whose major characteristics include a high threshold to all kinds of stimuli. Some high threshold sense organs show a remarkable enhancement of sensitivity after an exposure to noxious stimuli: this enhancement of sensitivity bears considerable similarity to the hyperalgesia associated with inflammation and tissue damage. Reflex reactions to pain-producing stimuli can contribute to the overall sensory experience and modification of such reflexes may explain the success of some therapeutic maneuvers. The central connections of nociceptors are in part specific, an organization which provides an explanation for referred pain and pain of central origin.

REFERENCES

1. Dallenbach, K. M. Pain: history and present status. *Am. J. Psychol.* 52: 331–347, 1939.
2. Beecher, H. K. *Measurement of Subjective Responses.* Oxford University Press, New York, 1959.
3. Melzack, R. and Wall, P. D. Pain mechanisms: a new theory. *Science* 150: 971–979, 1965.
4. Perl, E. R. Is pain a specific sensation? *J. Psychiat. Res.* 8: 273–287, 1971.
5. Sherrington, C. S. *The Integrative Action of the Nervous System.* New Haven: Yale University Press, 1906.
6. Heinbecker, P., Bishop, G. H., and O'Leary, J. Pain and touch fibers in peripheral nerves. *Arch. Neurol. Psychiat.* (Chic.) 29: 771–789, 1933.
7. Coggeshall, R. E., Coulter, J. D., and Willis, W. D., Jr. Unmyelinated axons in the ventral roots of the cat lumbo-sacral enlargement. *J. Comp. Neurol.* 153: 39–58, 1974.
8. Clifton, G. L., Vance, W. H., Appelbaum, M. L., Coggeshall, R. E., and Willis, W. D. Responses of unmyelinated afferents in the mammalian ventral root. *Brain Res.* 82: 163–167, 1974.
9. Perl, E. R. Myelinated afferent fibers innervating the primate skin and their response to noxious stimuli. *J. Physiol.* 197: 593–615, 1968.
10. Bessou, P., Burgess, P. R., Perl, E. R., and Taylor, C. B. Dynamic properties of mechanoreceptors with unmyelinated (C) fibers. *J. Neurophysiol.* 34: 116–131, 1971.
11. Burgess, P. R., and Perl, E. R. Myelinated afferent fibers responding specifically to noxious stimulation of the skin. *J. Physiol.* 190: 541–562, 1967.
12. Bessou, P., and Perl, E. R. Response of cutaneous sensory units with unmyelinated fibers to noxious stimuli. *J. Neurophysiol.* 32: 1025–1043, 1969.
13. Bessou, P., and Laporte, Y. Étude des récepteurs musculaires innervés par les fibres afférentes du groupe III (fibres myelinisées fines), chez le Chat. *Arch. Ital. Biol.* 99: 293–321, 1961.
14. Paintal, A. S. Functional analysis of group III afferent fibers of mammalian muscles. *J. Physiol.* 152: 250–270, 1960.
15. Burgess, P. R., and Clark, F. J. Characteristics of knee joint receptors in the cat. *J. Physiol.* 203: 317–335, 1969.
16. Lewis, T. *Pain.* New York: MacMillan Company, 1942.
17. Khayutin, V. M., Baraz, L. A., Lukoshkova, E. V., Sonina, R. S. and Chernilovskaya, P. E. *Chemosensitive Spinal Afferents: Thresholds of Specific and Nociceptive Reflexes as Compared With Thresholds of Excitation for Receptors and Axons. Progress in Brain Research,* Iggo, A. and Ilyinsky, O.B. eds. Elsevier Scientific Publishing Company, Amsterdam, 43: 1976.
18. Christensen, B. N., and Perl, E. R. Spinal neurons specifically excited by noxious or thermal stimuli: marginal zone of the dorsal horn. *J. Neurophysiol.* 33: 293–307, 1970.
19. Trevino, D. L., Coulter, J. D., and Willis, W. D., Jr. Location of cells of origin of spinothalamic tract in lumbar enlargement of the monkey. *J. Neurophysiol.* 36: 750–761, 1973.
20. Willis, W. D., Trevino, D. L., Coulter, J. D. and Maniz, R. A. Responses of primate sponothalamic tract neurons to natural stimulation of hindlimb. *J. Neurophysiol.* 37:358–372, 1974.
21. Ruch, T. C. Pathophysiology of pain. Chapter 15 in *Medical Physiology and Biophysics.* T. C. Ruch and J. F. Fulton, eds., Philadelphia: W. B. Saunders, 1960, pp. 363–364.

22. Selzer, M., and Spencer, W. A. Convergence of visceral and cutaneous afferent pathways in the lumbar spinal cord. *Brain Res.* 14: 331–348, 1969.
23. Selzer, M., and Spencer, W. A. Interactions between visceral and cutaneous afferents in the spinal cord: reciprocal primary afferent fiber depolarization. *Brain Res.* 14: 349–366, 1969.
24. Boivie, J., and Perl, E. R. Neural substrates of somatic sensation. Chapter 8 in *Physiology*, Series One, Vol. 3 *Neurophysiology*, MTP International Review of Science, C.C. Hunt ed. London: Butterworths, 1975, pp. 303–411.

Discussion: The Importance of Neurophysiological Research into the Principles of Spinal Manipulation

SCOTT HALDEMAN
Vancouver, B.C., Canada

This conference is extremely privileged to have neurophysiologists of the stature of Drs. Sharpless, Sato, and Perl present papers on the subjects for which they have become internationally renowned. Excellent summaries of the more pertinent experimental results and physiological principles of nerve compression, somatovisceral reflexes, and pain have been presented. It is interesting to note that, with the exception of the work done by Sharpless during the last 2 years, none of the research quoted has been initiated through an interest in the practice of spinal manipulation. Nonetheless, these physiological principles are extremely important in the understanding of the pathophysiology of spinal lesions and probably hold the answer to the mechanism of action of the spinal manipulation.

The most pertinent way of discussing these papers would be to see how the concepts which have been presented have been used by clinicians to explain observed clinical results, how a lack of physiological research has led to errors of excessive claims and unreasonable criticism, and how some of the newer developments in neurophysiology have opened exciting theoretical considerations which may be important in the future practice of spinal manipulation.

NERVE COMPRESSION

One of the earliest explanations given for the results obtained by spinal adjusting was relief of nerve compression at the intervertebral foramen. It is unfortunate that this concept was often incorporated into complex philosophies of health and disease which interfered with the development of scientific research into the effects of the adjustment. However, it is promising that practically all practitioners of spinal manipulation have now passed through this phase of their history and are willing not only to look for alternate explanations for their form of therapy but to finance research by independent basic scientists.

The original concepts of nerve pressure have changed with increasing awareness of the pathophysiology of the spine and nervous system. There is now general agreement that nerve compression can exist at a spinal level but that it is not the primary lesion to which the adjustment or manipulation is directed.

There has already been extensive discussion of nerve compression at this symposium. However, two questions have not yet been answered:

1. Does compression of nerve roots cause pain?

It has often been presumed that pressure on a nerve could stimulate the pain fibers in the nerve or surrounding sheaths thus causing local and referred pain along the nerve. This is often referred to in diagnostic textbooks as the cause of sciatica and other neuralgias of spinal origin. Patrick Wall addressed this question at a recent international symposium on cervical pain.[1] At that time he expressed doubt that such an irritable lesion at the level of dorsal roots could exist. Since then Wall et al.[2] have reproduced the earlier experiments of Adrian.[3] They showed that acute injury to a nerve root by pressure, stretching or bending, results in a volley of impulses being initiated at the time of insult, but that this initial volley is shortlived. It is

possible that the sciatic pain from nerve root compression is not caused by direct injury to the nerve but instead by irritation of the dura, which is pain sensitive, or through stimulation by products of inflammation as suggested by Perl.

2. *What is the significance of the difference in the sensitivity of large and small nerve fibers to pressure?*

The observation by Sharpless that large nerve fibers are more sensitive to compression than small nerve fibers leaves the impression that it may be possible to determine the degree of compression by monitoring the modality of nerve function which has been lost. This assumption is based on the fact that larger nerve fibers have been shown to conduct motor impulses, proprioception and touch sensation, while the smaller nerve fibers carry pain and temperature sensation and sympathetic impulses. Although there is a great deal of confusion in the literature on this topic (reviewed by Sunderland[4]), the majority of researchers seem to agree that motor fibers are more susceptible to compression than sensory fibers, and that sensory loss occurs in the following order: proprioception, touch, temperature, and pain. It is interesting to note that sympathetic fibers are the most resistant to pressure. Therefore, in order to get visceral changes from nerve pressure, there must be sufficient compression to cause motor and sensory paralysis. It must, however, be remembered that this progression in the loss of function of nerve fibers is not absolute and that there is some loss in both fast and slow conducting fibers at all levels of compression.

THE SOMATOVISCERAL REFLEX

Somatovisceral interaction has gradually become one of the major theories currently being used to explain the results on visceral dysfunction which have been claimed by practitioners of spinal manipulation. Once again, however, a lack of research and discussion on the role of this physiological principle has led to its incorporation into a much wider philosophical concept of a so-called "holistic" approach to patient care by certain academics in the field of spinal manipulation.[5,6]

If the theory of abnormal somato-visceral reflexes is to be used to explain the results of spinal manipulation, it is necessary to dissociate this theory from vague philosophical concepts and establish that the spinal lesion does in fact cause visceral dysfunction through this mechanism. For this to be true the following requirements must be met.

1. *The spinal lesion must produce continuous stimulation of a large number of receptors.*

With the exception of the internal lamina and nucleus pulposus of the disc, all structures which make up the functional spinal column are considered to have sensory receptors of one type or another. Unmyelinated fibers have been found in the fascia, ligaments, periosteum, the intervertebral joint capsule and the outer lamina of the intervertebral discs.[7] Gardner[8] feels that the vertebral joints have the same basic position and movement receptors as other peripheral joints and it is likely, but not established, that the paraspinal muscles and tendons have a full complement of muscle spindle, Golgi tendon apparatus, pressure receptors and the group III chemoreceptors which are considered to be responsible for the cardiovascular responses to exercise.[9]

These receptors each have their own characteristics of response to repeated stimulation. Perl has demonstrated how small unmyelinated fibers can become sensitized so that the responsiveness of the receptor increases with repeated stimulation. Other receptors such as the Pacinian corpuscle and primary receptors of the muscle spindle react to repeated stimulation by adapting to extinction. These factors must be taken into account when determining the final "sensory bombardment" of the central nervous system.

The manifestations of the spinal lesion which are the target of manipulation, i.e. pain, muscle spasm, joint malposition, joint fixation, or hypermobility, all have the potential of stimulating these receptors. Nonetheless, with the exception of pain, it remains to be demonstrated that these lesions produce sufficient constant irritation of receptors to cause repeated discharge of sensory neurones.

2. *Stimulation of these particular receptors must result in a measurable somatosympathetic response*

Sato has demonstrated that sympathetic responses do occur on stimulation of cutaneous and deep somatic receptors. However, it does not necessarily follow that the spinal lesion will bring about this type of reflex response. The quality and quantity of the response is dependent upon both the type of receptor affected and the frequency of stimulation. Both these factors are unknown quan-

tities in the spinal lesion. It would appear that an experiment could be designed in which the spinal lesion was reproduced in test animals while recording from sympathetic nerves. Such an experiment would throw some light on these first two requirements of the somatovisceral reflex theory of spinal manipulation.

3. *These reflexes must not habituate to any significant degree.*

Habituation and facilitation in the spinal cord may result in significant alteration of the visceral response to repetitive or continuous stimulation of somatic nerves. For example, Schmidt and Schonfuss [10] found that when the stimulus interval was 0.25 second the somatosympathetic response to group II and III somatic afferents was less than 10 percent of that obtained at a stimulus interval of 10 seconds. On the other hand, consecutive group IV volleys appear to recruit greater numbers of sympathetic units with increasing frequency of stimulation.[11] Since most of these experiments have been carried out on acute animals which are sacrificed after a few hours, some of the results may not be directly transferable to a spinal lesion which theoretically produces these reflexes over a period of days or weeks.

4. *Sympathetic stimulation of the type produced by these reflexes must cause functional disorders in visceral organs.*

This is probably the most controversial issue of the entire theory of "structure-function" relationships. There can no longer be any doubt that direct electrical stimulation to sympathetic nerves can influence blood flow, smooth and cardiac muscle contraction and glandular secretion in many internal organs. Sato has just demonstrated that cutaneogastric, cardiac, and vesical reflexes can be of sufficient strength to produce measureable changes in the function of these organs. It is therefore likely that such changes occur following stimulation of other somatic receptors which have been shown to cause somato-sympathetic discharges. The problem is whether or not these functional changes play any part in the etiology of known visceral pathology and functional diseases. This question can easily be sidestepped in this discussion by stating that it would require another conference of at least this magnitude to begin to cover the literature on the role of the autonomic nervous system in visceral pathology.

5. *Correction of the spinal lesion must result in cessation of the reflex activity and improvement in the visceral dysfunction.*

This requirement is probably the most important from a clinical point of view. However, since it is the subject of another paper at this conference it would be inappropriate to discuss it at this time. Suffice it to say that the general rules of clinical research regarding experimental design and adequate controls must be adhered to if the results are to be considered valid.

PAIN

By far the greatest number of patients attending practitioners of spinal manipulation are doing so in an attempt to obtain relief from pain. Most of the controversy which has taken place between authorities in spinal manipulation has been on locating the irritating lesion. The structures which have received the greatest attention are the posterior joints and the intervertebral discs, with lesser emphasis on paraspinal muscles and ligaments. Detailed histological examinations have revealed that, with the exception of the central portions of the disc, all of these structures are well-endowed with pain fibres and capable of reacting to injury in the manner described by Perl.

A lack of understanding of the physiology of pain, together with inadequate diagnosis, could be one of the reasons for excessive claims by early practitioners of spinal manipulation. Lewis and Kellgren [12] showed that pain from paraspinal structures could mimic the referred pain characteristic of certain visceral diseases. They found that the injection of saline into the interspinous ligaments of the vertebral column could produce subjective signs of pain, superficial and deep tenderness, and muscle rigidity indistinguishable in quality and similar in quantity to that produced by renal colic, angina pectoris, gastric ulcers, cholecystitis and acute appendicitis. Furthermore, relief from these symptoms in other patients was achieved by the injection of novocain into spinal muscles which exhibited deep pain.[13] In addition, Fernstrom [14] described pain in the abdominal-anogenital region caused by intervertebral disc lesions which could be confused with a number of common obstetrical problems. It can be seen that lack of cooperation between practitioners of spinal manipulation and the rest of the health team, who were often more qualified in the diagnosis of these internal disorders,

597-036 O - 76 - 13

could have resulted in claims of cures for visceral disorders when their symptoms were mimicked by spinal lesions.

The mechanism of referred pain has generally been considered one of convergence of nerve fibers from two different areas onto single neurones in the spinal cord. Such convergence has been demonstrated by recording from single neurones in the dorsal horn of the spinal cord while stimulating independently visceral afferent nerves, small myelinated cutaneous nerves and unmyelinated nerves from muscles.[15, 16] The observation that there are considerably more primary afferent pain fibers in the posterior roots than secondary afferents in the spinothalamic tract has been used as further evidence for convergence at the level of the spinal cord.[17]

Recent research into spinal integration of pain impulses has opened some new possible mechanisms by which spinal lesions could cause referred pain and tenderness. The observation by Kibler and Nathan [18] that the pain of nerve root compression could be relieved by anesthetizing distal structures tends to support the suggestion by Wall [1] that the site of noxious stimulation is in the periphery rather than in the root itself. The gate-control theory proposed by Wall and Melzack [19] incorporated a model in which input from large sensory neurones had the ability to inhibit impulses entering the spinal cord along small nerve fibers. If, as has been shown by Sharpless, the large fibers were to be blocked preferentially by nerve pressure, there would be a reduction of this peripheral inhibition with a subsequent hyperalgesia and pain along that dermatome. It is true that the original model of the gate-control theory has not stood up to closer experimental examination. Nonetheless, the basic concept of impulses along one set of nerves inhibiting pain impulses in a second set of nerves has been repeatedly demonstrated.[20, 21] Except when practiced in its purest form by one school of thought in chiropractic, spinal manipulation has had a variety of fellow travelers which are often applied at the same time as the manipulation. These adjunctive procedures have included simple massage, deep friction massage, connective tissue massage, the application of heat, cold, ultrasound, microwave, or galvanic stimuli, the stimulation of trigger points and more recently, acupuncture. Stimulation of peripheral receptors is one of the effects of each of these procedures which could thus be considered counterirritants. Recent research on the gate-control theory has shown that the most effective inhibition of small fiber high-threshold (pain) receptors is achieved by the stimulation of other high-threshold receptors.[21] It is likely, therefore, that the most successful inhibition of pain would be by procedures which cause the greatest degree of stimulation of sensory receptors. It must be remembered, however, that the inhibition of pain by this mechanism, if valid, would last only for short periods of time. The original pain producing lesion must be corrected by some other method such as the manipulation (where applicable) in order to get permanent relief.

Another mechanism through which neurones carrying pain impulses can be inhibited is the stimulation of higher brain centers. Liebeskind et al.[22] have shown that the stimulation of specific areas in the reticular formation such as the dorsal raphe and the periventricular grey matter can cause complete inhibition of those neurones in the spinal cord which carry pain impulses and in this way cause complete analgesia. Similar effects following stimulation of structures in the limbic system [23] together with observed psychological reactions to pain have led to the conclusion that pain may be inhibited completely via psychogenic mechanisms.[24] Clinicians who practice spinal manipulation often become very defensive when their detractors derisively state that all results can be explained on the basis of psychological effects of the manipulation. However, there are very few therapies which have the advantages of laying on of hands, relaxing tense muscles, causing a sensation at the area of pain (the "click" of the adjustment) and a clinician who has confidence in the therapy. It is a pity that this possibility has been considered a criticism of the therapeutic procedure instead of one of its advantages.

Finally, Perl has shown that one of the most important effects of pain impulses is protective reflex contraction of adjacent muscles and suggested that stretch of the contracting muscle was one of the mechanisms by which spinal manipulation and traction might relieve pain and muscle spasm. This may be one reason why the specific adjustive thrust on a deviated or subluxated vertebra from the side of deviation, which is also the side of the muscle contraction, has been considered to be more effective in achieving results in certain patients than the more general manipulative techniques.

CONCLUSION

Although the three concepts under discussion are those used by most practitioners of spinal manipulation to explain their results, there is still insufficient experimental data to state, with any certainty, exactly why the manipulation is an effective therapeutic procedure. It appears, however, that no single theory can be utilized to explain all results of manipulation.

REFERENCES

1. Wall, P. D.: "The mechanisms of pain associated with cervical vertebral disease." In C. Hirsch and Y. Zotterman. (Editors). *Cervical Pain.* Wenner-Gren Centre International Symposia series. Pergamon Press (Oxford), 201–210, 1971.
2. Wall, P. D., Wanman, S., and Basbaum, A. I.: Ongoing activity in peripheral nerve: Injury discharge. *Exp. Neurol.* 45:576–589, 1974.
3. Adrian, E. D.: The effects of injury on mammalian nerve fibers. *Proc. Roy. Soc.* Ser. B. 106:596–618, 1930.
4. Sunderland, S.: *Nerves and Nerve Injuries.* E. and S. Livingstone Ltd. (Edinburgh & London), 1968.
5. Hoag, J. M., Cole, W. V., and Bradford, S. G. (Editors). *Osteopathic Medicine.* McGraw-Hill Book Co. (New York), 1969.
6. Stonebrink, R. D.: Neurological features of the chiropractic philosophy. *J. Am. Chiropractic Assn.* Vol. 11.S:33–40, 1968.
7. Pederson, H. E., Blunchk, C. F. J., and Gardner, E.: The anatomy of lumbosacral posterior rami, meningeal branches of spinal nerves (sinu-vertebral nerves). *J. Bone Jt. Surg.* 38A:377–399, 1956.
8. Gardner, E.: Pathways to the cerebral cortex for nerve impulses from joints. *Acta Anat.* Suppl. 56:1 and 73: 203–216, 1969.
9. Hnik, P., Hudlika, O., Kucera, J., and Payne, R.: Activation of muscle afferents by non proprioceptive stimuli. *Am. J. Physiol.* 217:1451–1457, 1969.
10. Schmidt, R. F., and Schonfuss, K.: An analysis of the reflex activity in the cervical sympathetic trunk induced by myelinated somatic afferents. *Arch. Ges. Physiol.* 314: 175–198, 1970.
11. Schmidt, R. F., and Weller, E.: Reflex activity in the cervical and lumbar sympathetic trunk induced by unmyelinated somatic afferents. *Brain Res.* 24:207–218, 1970.
12. Lewis, T., and Kellgren, J. H.: Observations relating to referred pain, visceromotor reflexes and other associated phenomena. *Clin. Sci.* 4:47–71, 1939.
13. Kellgren, J. H.: Somatic stimulating visceral pain. *Clin. Sci.* 4:303–309, 1940.
14. Fernstrom, U.: Ruptured lumbar discs causing abdominal pain. *Acta Chir. Scand.* Suppl. 357:160–161, 1966.
15. Fields, H. L., Meyer, G. A., and Partridge, L. D., Jr.: Convergence of visceral and somatic input onto cat spinal neurons. *Exp. Neurol.* 26:36–52, 1970.
16. Pomeranz, B., Wall, P. D., and Weber, W. V.: Cord cells responding to fine myelinated afferents from viscera, muscle and skin. *J. Physiol.* 199:511–532, 1968.
17. Ruch, T. C.: Visceral sensation and referred pain. In Fulton J. F. (Ed.) *Howell's Textbook of Physiology.* 15th Edition. Saunders (Philadelphia) 385–401, 1947.
18. Kibler, R. F., and Nathan, P. W.: Relief of pain and paraesthesiae by nerve block distal to the lesion. *J. Neurol. Neurosurg. Psych.* 23:91–98, 1960.
19. Melzack, R., and Wall, P. D.: Pain Mechanism: a new theory. *Science,* 150:971–979, 1965.
20. Price, P. D., and Wagman, I. H.: Relationships between pre- and postsynaptic effects of A and C fiber inputs to dorsal horn of M. Mulatta. *Exp. Neurol.* 40:90–103, 1973.
21. Whitehorn, D., and Burgess, P. R.: Changes in polarization of central branches of myelinated mechanoreceptor and nociceptor fibers during noxious and innocuous stimulation of the skin. *J. Neurophysiol.* 36:226–237, 1973.
22. Liebeskind, J. C., Guilbaud, G., Besson, J. M., and Oliveras, J. L.: Analgesia from electrical stimulation of periaquaductal gray matter in the cat: behavioral observations and inhibitory effects on spinal cord interneurons. *Brain Res.* 50:441–446, 1973.
23. Lico, M. C., Hoffmann, A., and Covian, M. R.: Influence of some limbic structures upon somatic and autonomic manifestations of pain. *Physiol. Behav.* 12:805–811, 1974.
24. Perl, E. R.: "Mode of action of nociceptors." In Hirsch, D., and Zotterman, Y.: (Editors) *Cervical Pain.* Wenner-Gren Centre International Symposium Series. 17:157–164, 1971.

A Brief Review of Material Transport in Nerve Fibers

SIDNEY OCHS

Professor, Department of Physiology
Indiana University School of Medicine

INTRODUCTION

In recent years we have become aware of the relatively high level of protein synthesis present in the neuron cell body [1] with a subsequent movement of proteins and other materials out into the nerve fiber to be carried along by an internal system of transport. Wallerian degeneration, the failure of excitability and disruption of fiber structure seen to occur in nerve fibers a few days after their separation from their cell bodies, long ago suggested the loss of some key substance supplied by the cells to the fibers.[2] Additionally, the failure of neuromuscular transmission and the changed properties of muscle appearing after nerve transection has been ascribed to the loss of materials or "trophic" substance normally conveyed by the nerve fibers.[3] This brief review describes some of the properties of the transport mechanism of mammalian nerve.

Characteristics of transport

Materials moving down the fibers are found to accumulate above a site of ligation or constriction [4] with, in some cases, several peaks of accumulation of materials normally present in the nerve fibers. Monitoring of the flow of labeled proteins subsequent to the incorporation of labeled amino acids by nerve cell bodies suggests two or more rates of downflow.[5-9] A direct measure of the rate and characteristics of downflow was shown by the pattern of labeled activity found in long lengths of nerve present in mammals, e.g. the cat sciatic, after injection of ^{3}H-leucine or ^{3}H-lysine into either the lumbar seventh (L7) dorsal root ganglia or into the motorneuron region of the cord.[10, 11] The characteristic crest found in the sciatic nerve has a linear advance with time at a rate of 410 ± 50 (S.D.) millimeters per day. The general plan of injection and the sampling of activity in the sciatic nerve is shown in figure 1. At a predetermined time after the injection, the animal is sacrificed, the nerve removed, and sectioned as shown. The characteristic outflow is seen as a crest of advancing activity followed by a plateau, typically with less activity in it compared to the crest. Large numbers of labeled components remain in the ganglion cells, part of which are transported later.

The crest advances at a linear rate down the fibers as shown in five nerves taken at different times after injection (figure 2). The rate determined in a large number of such experiments was 410 millimeters per day.[2] The rate is independent of nerve size, or of the diameter of myelinated fibers, and the same rate is also present in nonmyelinated fibers[12, 13] even in the nerves of several non-mammalian species. The garfish olfactory nerve[14, 15] and frog nerve [16] with a temperature correction to 38° C, was used for comparison.

A range of soluble proteins, polypeptides and particulates are carried down at a fast rate,[17, 19] as are glycoproteins, glycolipids and phosphatidylcholine,[20, 22] along with specific components related to transmission, catecholamines[23a] and enzymes related to their synthesis,[23b] acetylcholinesterase (AChE).[24, 25]

The Transport Mechanism—a Model

The similarity of the rate of transport found for a wide range of materials with molecular weights known to extend from small molecules to particles of comparatively large size is of theoretical interest.

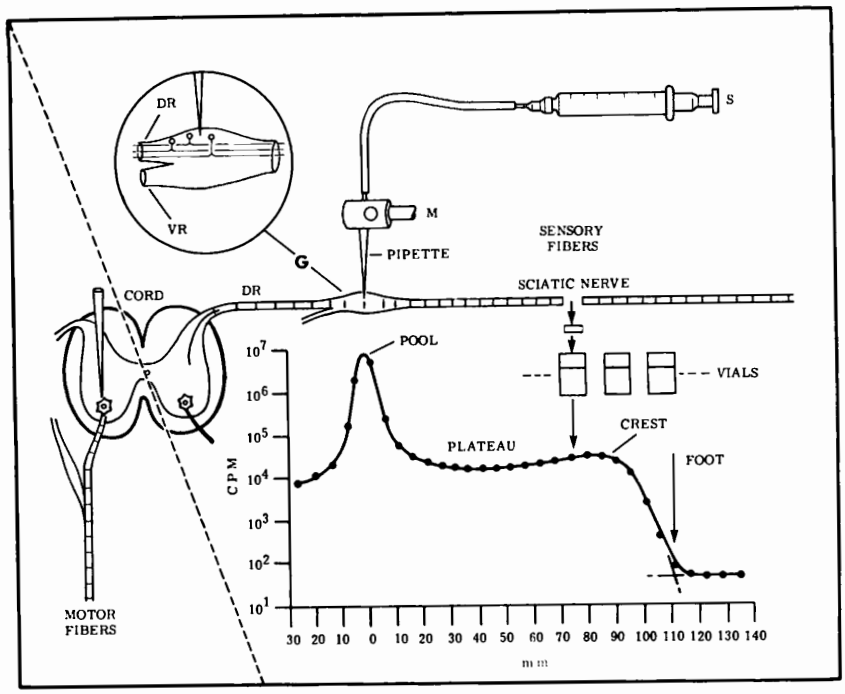


Fig. 1. *Injection and sampling technique showing transport.* The L7 ganglion shown in the insert contains T-shaped neurons with one branch ascending in the dorsal root, the other descending in the sciatic nerve. A pipette containing ^{3}H-leucine, is passed into the ganglion and after its injection and the incorporation of precursor, the downflow of labeled components in the fibers is sampled at various times by sacrificing the animal and sectioning the nerve. Each segment is placed in a vial, solubilized, scintillation fluid added and the activity counted. The outflow pattern is displayed on the ordinate log scale in CPM, the abscissa is in mm, taking the distance from the center of the ganglion as zero. A high level of activity is seen remaining in the ganglion region with more distally, a plateau rising to a crest before abruptly falling at the front of the crest of baseline levels. The left hand side of the cord shows for motoneurons an injection of the precursor into the L7 cell body region followed by removal of ventral root and sciatic nerve at a later time for a similar treatment and display of outflow.

It indicates that a common carrier, a "conveyor belt" type of system, is involved in the mechanism of transport. A hypothesis to account for fast axoplasmic transport was proposed based on the sliding-filament theory of muscle.[26,27] A "transport filament" is considered to bind the various materials moving along the microtubules and/or neurofilaments of the axon by means of cross-bridges (figure 3). The heterogenous range of labeled components are thus moved quickly down the nerve axon at the same fast rate. The plateau behind the crest is made up of materials exported into the fibers at later times from some "compartment" in the cell bodies with a portion of the labeled materials being locally deposited in the fibers. Still later, over a period of hours, days, or weeks, more labeled components come from the cell body at earlier times and more distally later, thereby accounting for the characteristic outflow pattern of slow transport. With this "unitary concept," slow transported materials are not so firmly bound to the transport filaments as are the fast transported components, and so can more readily leave the transport filament to become locally incorporated into various nerve structures;[31] an example is the mitochondrion. It is seen microscopically with Nomarski optics to have fast movement forward or retrograde with a net slow transport.[32-34] This could be due to binding and unbinding from the transport filaments.

Evidence that the microtubules are an integral part of the transport mechanism comes from the effect of the mitotic blocking agents, colchicine and the *vinca* alkaloids. These substances, when injected under the epineurium of a nerve trunk, were shown to block fast axoplasmic transport by the failure of catecholamines[35] and AChE[36] to accumulate at a ligation downstream from the site injected with colchicine or vinblastine. At first it was presumed that this comes about through a disassembly of the microtubules, the basis for their antimitotic action. Electron microscopy of crayfish

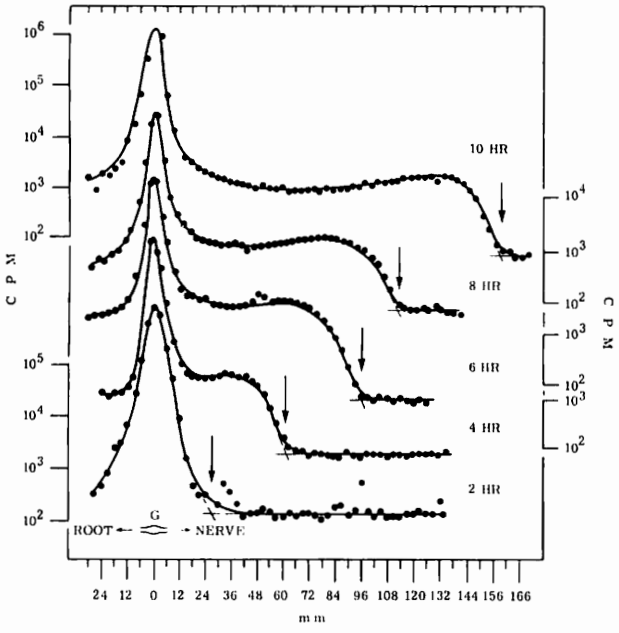


Fig. 2 *Distribution of radioactivity.* Activity present in the dorsal root ganglia and sciatic nerves of five cats taken 2 to 10 hours after injection of ^{3}H-leucine into the L7 ganglia (*G*) is shown. The activity present in 5 mm segments of roots, ganglia, and nerves is given on the ordinate in logarithmic divisions. The ordinate scale for the nerve taken 2 hours after injection is shown at the bottom left with divisions in CPM. At the top left a scale is given for the nerve taken 10 hours after injection. Only partial scales are shown at the right for the nerves taken 4, 6, and 8 hours after injection. Abscissa in mm from the ganglion taken as zero.

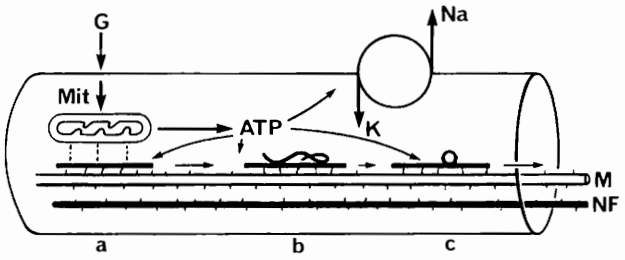


Fig. 3. *Transport filament hypothesis.* Glucose (*G*) enters the fiber and after glycolysis, oxidative phosphorylation in the mitochondrion (*Mit*) gives rise to *ATP*. The ~*P* of *ATP* supplies energy to the sodium pump controlling the level of Na^+ and K^+ in the fiber and also to the cross-bridges activating the transport filaments. These are shown as black bars to which the various components transported are bound and so carried down the fiber by crossbridge activity. The components transported include the mitochondria (*a*) attaching temporarily as indicated by dashed lines to the transport filament this giving rise to either fast forward or retrograde movement (though with a slow net forward movement), soluble protein (*b*) shown as a folded or globular configuration, polypeptides and small particulates (*c*). Simpler molecules are also bound to the transport filaments. Thus, a wide range of components are transported at the same fast rate. The cross-bridges between the transport filament and the microtubules presumably act in similar fashion to the sliding filament theory of muscle and the Mg Ca ATPase found in nerve utilizes *ATP* as the source of energy.

nerve fibers exposed to colchicine and vinblastine in amounts which blocked transport showed, however, that the microtubules still remain intact in some of those fibers.[37,38] This leaves open the possibility that these agents may act on the surface of the microtubules or at the cross-bridges to block transport.[39]

In analogy with the sliding-filament theory of muscle, the cross-bridges invoked in the transport filament hypothesis require energy, which is likely to be supplied by ATP. A close dependence of fast axoplasmic transport on oxidative metabolism was shown in in vitro studies.[40-43] When N_2 anoxia was initiated or azide, CN or DNP added to the nerve in vitro, fast axoplasmic transport was blocked within approximately 15 minutes. At that time, the level of ~ P (the combined concentration of ATP and phosphocreatine), fell by about 0.6 μM/g.[43] Axoplasmic transport failed at a later time when the citric acid cycle was blocked with flouroacetate, or when glycolysis was blocked with IAA [41] and a corresponding fall of ~ P occurred at those later times.[43]

The dependence of transport on oxidative metabolism was shown by the block of transport produced either by stripping the blood vessels or by the use of cuff compressions at pressures above 300 mm. Hg. to block circulation.[43a] A damming of activity was seen above the block and no transport found within the region made anoxic. This was demonstrated in vitro by covering the nerve with petrolatum jelly and parafilm strips so that O_2 cannot diffuse into that region. A block of transport was seen starting just at the edge of the anoxic region by the damming of activity above that site [46] (figure 4). Such studies indicate that ATP is produced all along the length of the nerve fibers.

The utilization of ATP is likely to be accomplished by the MgCa ATPase found present in myelinated nerve,[44] the enzyme having many of the actomysin-like properties previously shown in brain preparations.[45] The ATP is apparently supplied and is required all along the length of the nerve as was indicated when a short region of the nerve was made anoxic and block of transport occurred just at the anoxic region.[46]

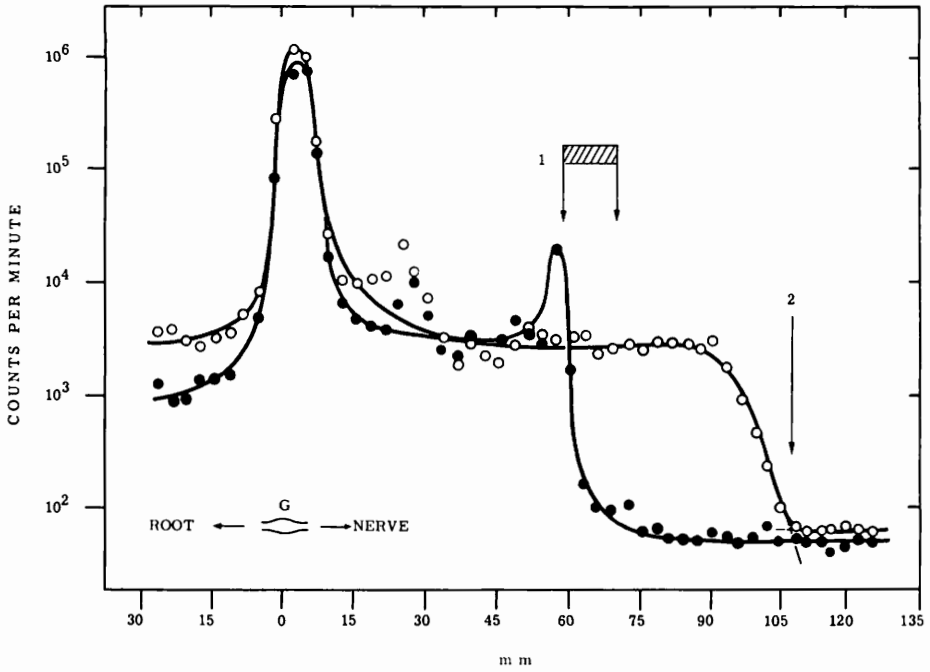


Fig. 4. *Local anoxia and block.* Local anoxia produced by covering part of the nerve with petrolatum and plastic strips at the site indicated by the cross-hatched bar. Downflow occurred in the animal for 3 hours and then 3 hours of *in vitro* transport took place with the local anoxia block present. Arrow 2 indicates downflow at crest of control nerve.

The anoxic block of fast transport in vitro due to N_2 is reversible after approximately 1–1.5 hours.[47] After longer times of anoxia produced in vivo by cuff compression of the upper limb, a partial return of fast axoplasmic transport occurred, though full recovery was found to require more time. This might take several days after a 4- to 5-hour compression. Irreversible block occurs after 6 hours of anoxia at 37° C. A similar time of 7 hours for reversibility was seen using Wallerian degeneration and electrical conduction as criteria.[48] The effect of cuff compression is not due to mechanical changes per se as seen by comparing the effect of compression at much higher pressures.[49]

Retrograde Transport and Chromatolysis

Various hypotheses have been advanced to account for the phenomenon of chromatolysis.[50] Most likely chromatolysis is triggered because of the failure of some "signal" substance, which normally ascends the fibers, and as a result of interruption fails to reach the cell bodies.[51] The increased accumulation of protein in the cells[52, 53] seen after nerve section could thus represent the failure of a normal negative feedback control due to the signal substance which acts to regulate the level of protein synthesis or the amount of protein present in the nerve cell body.[2]

A retrograde transport of material was first shown for AChE by Lubinska and her colleagues by the accumulation of AChE just distal to nerve ligations.[5] More recent studies from her laboratory showed the retrograde AChE movement to have a rate of about 110 millimeters per day.[54] Our studies showed a somewhat higher rate for its retrograde transport, 220 millimeters per day, at about half that of the forward rate of close to 410 millimeters per day.[55] A retrograde transport was also shown for foreign materials, e.g. for horseradish peroxidase after its uptake by the nerve terminals,[56, 57] the substance apparently carried at a fast rate.[58] In the older literature, toxins and viruses have been reported as moving upward in nerve fibers and no doubt will be further investigated in the light of what is now known of axoplasmic transport.[59, 60]

During chromatolysis the rate of axoplasmic transport has been reported as increased, decreased or unchanged, though in our studies no difference in the rate was found.[29] This is the case throughout the whole of the period of chromatolysis lasting from 4 to 90 days. This result may be stated as a principle, namely that fast axoplasmic transport

is an "all-or-none" process in the nerve fiber independent of the level of synthesis of materials in the cell body.

"Routing" Phenomenon

In a study of fast axoplasmic transport in the monkey where lengths of dorsal roots up to 11 centimeters long occur, the crests of outflow of labeled materials seen in the root after L7 dorsal ganglion injection with ^{3}H-leucine showed the same fast rate; however, a threefold to fivefold greater amount of labeled material was found in the crests in the peripheral nerve as compared to the crests in the dorsal root[10] (figure 5). This asymmetry in the amount of transported activity could not be accounted for by a greater diameter of the fiber branches of the T-shaped ganglion cell neurons in the nerve branch as compared to the root branch.[61] Furthermore, the numbers of microtubules (and neurofilaments) counted in electron micrograms of nerve fibers in the two branches were not dissimilar. The disparity in the amount of labeled components carried down the two branches of the L7 neurons depends therefore on a separate routing of components by an internal system in the fibers. On the transport filament mechanism we consider two sets of microtubules passing down individually into the two branches of the same neuron. The cell body exerts a control over the amount and type of materials destined for routing elements to its several terminals. The various materials carried are likely to be related to some degree to the different functions of the branch terminals, one ending as a receptor peripherally, the other as a presynaptic terminal centrally.[61]

In accord with the transport filament hypothesis, the microtubules are seen to be separately channeled down each of the two branches with no branching or other division of the organelles.[62]

Transport in CNS Nerve Fibers

The dorsal root fibers entering the cord branch with a local collateral in the segment and another branch ascending in the dorsal columns of the spinal cord. After ganglion injection with ^{3}H-leucine, a crest of activity was seen in the dorsal columns at a distance from the ganglion depending on the usual rate as is the case in the sciatic nerves.[10] When the fibers in the dorsal column were interrupted by applied pressure or by freezing at temperatures below −20° C, a failure of fast axoplasmic transport occurred with a damming of

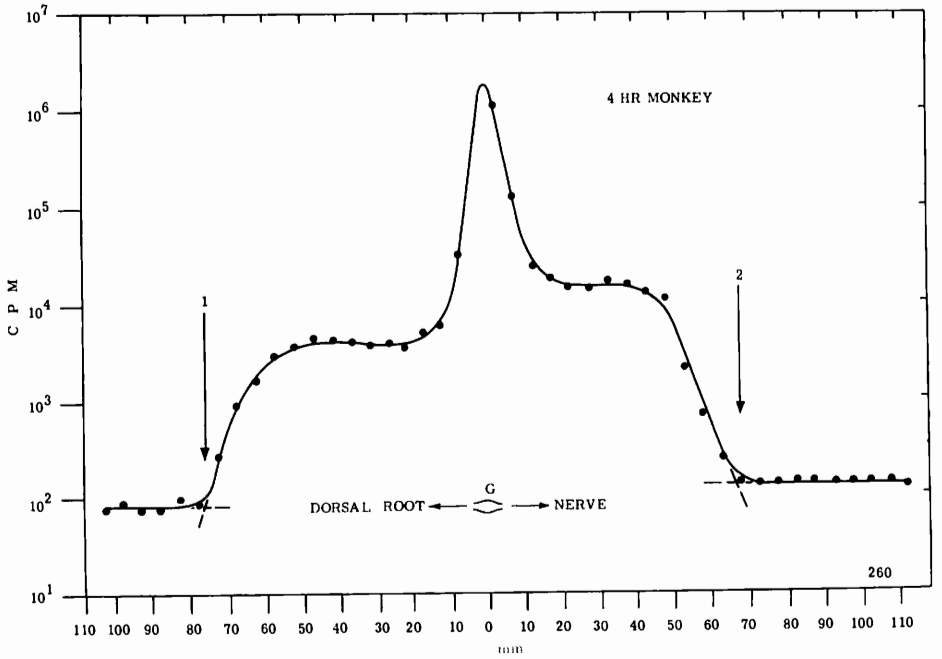


Fig. 5. *Transport of dorsal root and sciatic nerve.* L7 dorsal root ganglion was injected with ^{3}H-leucine; 4 hours later the monkey was sacrificed and the distribution of activity in dorsal root and comparable length of sciatic nerve compared. Arrow 1 shows the furthest extent of the crest of activity of fast axoplasmic transport in the dorsal roots, arrow 2, the crest showing transport into the sciatic nerve. The larger height of the crest in the nerve branches represents some 3–5X more material than in the root branches.

activity seen, a result similar to that earlier found in peripheral nerve as expected from an axoplasmic transport in the fibers.

Studies of dorsal column transport make it appear likely that the same fast axoplasmic transport system is also present in other CNS fibers. Transport in the brain and cord was shown for various amine fiber systems by making transections along their course and finding an increased accumulation of the amines in the fibers proximal to the lesion facing the cell bodies with their depletion in the fibers distal to the lesion.[63, 64] This technique was used to show dopamine containing fibers passing from the substantia nigra to the striatum and noradrenaline and serotonin containing fibers passing from identified brain stem nuclear groups into the hypothalamus, thalamus, and cortex. The mapping of monoamine pathways can be clearly shown in their entirety in the smaller brain of the rat by the use of combined lesions and fluorescent techniques.[65]

Such information has already found clinical application. The nigrostriatal system has been related to neural motor control mechanisms; decreased dopamine levels in parkinsonism led to the introduction of L-DOPA, a precursor of dopamine, as a successful therapeutic agent for the disease.[66] Changes in the amount and types of neurotransmitter-related materials at the terminals of the dopamine, noradrenaline, and possibly serotonin fiber systems or their postsynaptic receptors have been correlated with changes in alertness and emotional state and thus serve as a rationale for the use of psychotrophic agents.[65-69]

The axoplasmic transport of other materials is likely to be important for clinical medicine in a general sense. Alterations of the amount or types of materials synthesized by the nerve cell bodies as a result of disease can lead to long-term alterations. These may come about through a changed level of trophic substances supplied by the nerve fiber to other cells. Some indication that such a trophic supply may be directly invested has been suggested by labeling studies.[70, 71] Just what the trophic materials may be and how they act on the cells receiving them is at present an important field for study.[72] It is most reasonable to expect that further knowledge of this protean system will lead to a better understanding of what are now obscure neuropathological entities and in turn lead to a new and better means of treating diseases of nerve, muscle, cord, and brain.

REFERENCES

1. Lajtha, A. (1964). Protein metabolism of the nervous system. *Int. Rev. Neurobiol.* 6:1–98.
2. Ochs, S. (1974). Systems of material transport in nerve fibers (axoplasmic transport) related to nerve function and trophic control. In: Trophic Function of the Neuron. *Ann. N.Y. Acad. Sci.* 228:202–223.
3. Guth, L. (1969). Degeneration and regeneration of taste buds. In: Handbook of Sensory Physiology, *Vol. 4: Chemical Senses*. Berlin, Springer-Verlag.
4. Weiss, P., and Hiscoe, H. B. (1948). Experiments on the mechanism of nerve growth. *J. Exp. Zool.* 107:315–395.
5. Lubińska, L. (1964). Axoplasmic streaming in regenerating and in normal nerve fibers. In: Mechanisms of Neural Regeneration. (*Progress in Brain Research, Vol. 13*). Singer, M. and Schade, J. P. (Eds.) Amsterdam, Elsevier, pp. 1–66.
6. McEwen, B. S., and Grafstein, B. (1968). Fast and slow components in axonal transport of protein. *J. Cell Biol.* 38:494–508.
7. Barondes, S. H. (1969). Axoplasmic transport. In: Handbook of Neurochemistry, *Vol. 2: Structural Neurochemistry*. Lajtha, A. (Ed.) New York, Plenum Press, pp. 435–446.
8. Ochs, S., Johnson, J., and Ng, M. -H. (1967). Protein incorporation and axoplasmic flow in motoneuron fibers following intracord injection of leucine. *J. Neurochem.* 14:317–331.
9. Karlsson, J. O., and Sjöstrand, J. (1971). Synthesis, migration and turnover of protein in retinal ganglion cells. *J. Neurochem.* 18:749–767.
10. Ochs, S. (1972). Rate of fast axoplasmic transport in mammalian nerve fibers. *J. Physiol.* 227:627–645.
11. Ochs, S. (1972). Fast transport of materials in mammalian nerve fibers. *Science* 176:252–260.
12. Byers, M. R., Fink, B. R., Kennedy, R. D. Middaugh, M. E., and Hendrickson, A. E. (1973). Effects of lidocaine on axonal morphology, microtubules, and rapid transport in rabbit vagus nerve in vitro. *J. Neurobiol.* 4:125–143.
13. Ochs, S., and Jersild, R. A., Jr. (1974). Fast axoplasmic transport in nonmyelinated nerve fibers shown by electron microscopic radioautography. *J. Neurobiol.* 5:373–377.
14. Gross, G. W., and Beidler, L. M. (1973). Fast axonal transport in the C-fibers of the garfish olfactory nerve. *J. Neurobiol.* 4:413–428.
15. Gross, G. W. (1973). The effect of temperature on the rapid axoplasmic transport in C-fibers. *Brain Res.* 56:359–363.
16. Edstrom, A., and Hanson, M. (1973). Temperature effects on fast axonal transport of proteins in vitro in frog sciatic nerves. *Brain Res.* 58:345–354.
17. Ochs, S., Sabri, M. I., and Johnson, J. (1969). Fast transport system of materials in mammalian nerve fibers. *Science* 163:686–687.
18. Sabri, M. I., and Ochs, S. (1973). Characterization of fast and slow transported proteins in dorsal root and sciatic nerve of cat. *J. Neurobiol.* 4:145–165.
19. Grafstein, B. (1969). Axonal transport: communication

between soma and synapse. In: *Advances in Biochemical Psychopharmacology*, Vol. 1:11–25.

20. McEwen, B. S., Forman, D. S., and Grafstein, B. (1971). Components of fast and slow axonal transport in the goldfish optic nerve. *J. Neurobiol.* 2:361–377.
21. Elam, J. S., and Agranoff, B. W. (1971). Transport of proteins and sulfated mucopolysaccharides in the goldfish visual system. *J. Neurobiol.* 2:379–390.
22. Abe, T., Haga, T., and Kukokawa, M. (1973). Rapid transport of phosphatidylcholine occurring simultaneously with protein transport in the frog sciatic nerve. *Biochem. J.* 136:731–740.

23a. Dahlstrom, A. (1971). Axoplasmic transport with particular respect to adrenergic neurons. *Phil. Trans. Roy Soc. B.* 261:325–358.

23b. Geffen, L. B., and Livett, B. G. (1971). Synaptic vesicles in sympathetic neurons. *Physiol. Rev.* 51:98–157.

24. Lubińska, L., and Niemierko, S. (1970). Velocity and intensity of bidirectional migration of acetylcholinesterase in transected nerves. *Brain Res.* 27:329–342.
25. Ranish, N., and Ochs, S. (1972). Fast axoplasmic transport of acetylcholinesterase in mammalian nerve fibers. *J. Neurochem.* 19:2641–2649.
26. Ochs, S. (1971). Characteristics and a model for fast axoplasmic transport in nerve. *J. Neurobiol.* 2:331–345.
27. Ochs, S. (1974). Systems of material transport in nerve fibers (axoplasmic transport) related to nerve function and trophic control. In: Trophic Function of the Neuron, Drachman, D. B. (Ed.), *Ann. N.Y. Acad. Sci.* 228:202–223.
28. Ochs, S., and Burger, E. (1958). Movement of substance proximo-distally in nerve axons as studied with spinal cord injections of radioactive phosphorus. *Amer. J. Physiol.* 194:499–506.
29. Ochs, S., Dalrymple, D., and Richards, G. (1962). Axoplasmic flow in ventral root nerve fibers of the cat. *Exp. Neurol.* 5:349–363.
30. Miani, N. (1963). Analysis of the somato-axonal movement of phospholipids in the vagus and hypoglossal nerves. *J. Neurochem.* 10:859–874.

31. Ochs, S. (1974). Retention and redistribution of fast and slow axoplasmic transported proteins in mammalian nerve. *Third Int. Cong. on Muscle Diseases.* Abstr. Excerpta Medica Int. Cong. Series *334*: 26.

32. Barondes, S. H. (1966). On the site of synthesis of the mitochondrial protein of nerve endings. *J. Neurochem.* 13:721–727.
33. Jeffrey, P. L., James, K. A., Kidman, A. D., Richards, A. M., Austin, L. (1972). The flow of mitochondria in chicken sciatic nerve. *J. Neurobiol.* 3 : 199–208.
34. Khan, M. A., Ranish, N., and Ochs, S. (1971). Axoplasmic transport of AChE, LDH, and MAO in mammalian nerve fibers. *Soc. Neurosci. Abst.* 1 : 144.
35. Dahlström, A. (1968). Effect of colchicine on transport of amine storage granules in sympathetic nerves of rat. *Europ. J. Pharmacol.* 5:111–113.
36. Kreutzberg, G. W. (1969). Neuronal dynamics and axonal flow. IV. Blockage of intraaxonal enzyme transport by colchicine. *Proc. Nat. Acad. Sci.* (U.S.A.) 62:722–728.
37. Fernandez, H. L., Huneeus, F. C., and Davison, P. F. (1970). Studies on the mechanism of axoplasmic transport in the crayfish cord. *J. Neurobiol.* 1:395–409.
38. Samson, F. E. (1971). Mechanism of axoplasmic transport. *J. Neurobiol.* 2:347–360.

39. Ochs, S. (1975). Axoplasmic transport. In: *The Nervous System*, Tower, D.B. (Ed.), Vol. 1: *The Basic Neurosciences*, Raven Press, N.Y., pp. 137–146.

40. Ochs, S., and Hollingsworth, D. (1971). Dependence of fast axoplasmic transport in nerve on oxidative metabolism. *J. Neurochem.* 18:107–114.
41. Ochs, S., and Smith, C. (1971). Fast axoplasmic transport in mammalian nerve in vitro after block of glycolysis with iodoacetic acid. *J. Neurochem.* 18:833–844.
42. Ochs, S. (1974). Energy metabolism and supply of nerve by axoplasmic transport. In: Symposium on Metabolism in Relation to Function in Ganglia and Peripheral Nerves of Vertebrates. Larrabee, M. (Ed.) *Fed. Proc.* 33:1049–1057.
43. Sabri, M. I., and Ochs, S. (1972). Relation of ATP and creatine phosphate to fast axoplasmic transport in mammalian nerve. *J. Neurochem.* 19:2821–2828.
44. Khan, M. A., and Ochs, S. (1974). Magnesium or calcium activated ATPase in mammalian nerve. *Brain Res.* 81: 413–426.
45. Berl, S., and Puszkin, S. (1970). Mg^{2+}—Ca^{2+} -activated adenosine triphosphatase system isolated from mammalian brain. *Biochemistry* 9:2058–2067.

46. Ochs, S., (1971b). Local supply of energy to the fast axoplasmic transport mechanism. *Proc. Nat. Acad. Sci.* (U.S.A.) 68:1279–1282.
47. Leoni, J., and Ochs, S. (1973). Reversibility of fast axoplasmic transport following differing durations of anoxic block in vitro and in vivo. *Abst. Soc. Neurosci.* 3:147.
48. Lundborg, G. (1970). Ischemic nerve injury. *Scand. J. Plastic and Reconstr. Surg. Suppl.* 6:3–113.
49. Fowler, T. J., Danta, G., and Gilliatt, R. W. (1972). Recovery of nerve conduction after a pneumatic tourniquet: Observations on the hind-limb of the baboon. *J. Neurol. Neurosurg. and Psych.* 35:638–647.
50. Cragg, B. G. (1970). What is the signal for chromatolysis? *Brain Res.* 23:1–21.
51. Ochs, S., Booker, H., and DeMyer, W. E. (1961). Note on the signal for chromatolysis after nerve interruption. *Exp. Neurol.* 3:206–208.
52. Brattgard, S.-O., Edstrom, J.-E. and Hyden, H. (1958). The productive capacity of the neuron in retrograde reaction. *Exp. Cell Res. Suppl.* 5:185–200.
53. Watson, W. E. (1965). An autoradiographic study of the incorporation of nucleic acid by neurons and glia during nerve regeneration. *J. Physiol.* 180:741–753.
54. Lubińska, L., and Niemierko, S. (1970). Velocity and intensity of bidirectional migration of acetylcholinesterase in transected nerves. *Brain Res.* 27:329–342.
55. Ranish, N., and Ochs, S. (1972). Fast axoplasmic transport of acetylcholinesterase in mammalian nerve fibers. *J. Neurochem.* 19:2641–2649.
56. Kristensson, K., and Olsson, Y. (1971). Uptake and retrograde axonal transport of peroxidase in hypoglossal neurons. Electron microscopical localization in the neuronal perikaryon. *Acta Neuropathol.* (Berl.) 19:1–9.
57. LaVail, J., and LaVail, M. (1972). Retrograde axonal

transport in the central nervous system. *Science* 176:1416–1417.

58. LaVail, J., and LaVail, M. (1974). Intra-axonal transport of horseradish peroxidase following intravitreal injections in chicks. *Abst. Soc. Neurosci.* 4:299.
59. Kristensson, K. (1970). Morphological studies of the neuronal spread of herpes simplex virus to the central nervous system. *Acta Neuropath (Berl.)* 16:54–63.
60. Kristensson, K., Lycke, E., and Sjöstrand, J. (1971). Spread of herpes simplex virus in peripheral nerves. *Acta Neuropathol.* (Berl.) 17:44–53.
61. Ochs, S., and Erdman, J. (1974). "Routing" of fast transported materials in nerve fibers. *J. Abst. Soc. Neurosci.* 4:359.
62. Ha, H. (1970). Axonal bifurcation in the dorsal root ganglion of the cat—a light and electron microscopic study. *J. Comp. Neurol.* 140:227–240.
63. Heller, A. (1972). Neuronal control of brain serotonin. *Fed. Proc.* 31:81–90.
64. Anden, N. E., Fuxe, K., Hamberger, B., and Hökfelt, T. (1966). A quantitative study on the nigrostriatal dopamine neuron system in rat. *Acta Physiol. Scand.* 67:306–312.
65. Ungerstedt, U. (1971. Stereotaxic mapping of the monamine pathways in the rat brain. *Acta Physiol. Scand.* Suppl. 367:1–48.
66. Hornykiewicz, O. (1972). Dopamine and extrapyramidal motor function and dysfunction. In: Neurotransmitters. Kopin, I. J. (Ed.) *Res. Pub. Assoc. Nerv. Ment. Dis.* 50:390–415.
67. Phillis, J. W. (1970). *The Pharmacology of Synapses.* New York, Pergamon Press.
68. Cooper, J. R., Bloom, F. E., and Roth, R. H. (1970). *The Biochemical Basis of Neuropharmacology.* New York, Oxford University Press.
69. Kopin, I. J. (Ed.) (1972). Neurotransmitters, Vol. 50. *Res. Pub. Assoc. Nerv. Ment. Dis.* Baltimore, Williams & Wilkins Co.
70. Korr, I. M., and Appeltauer, G. S. L. (1974). The time course of axonal transport of neuronal proteins to muscle. *Exp. Neurol.* 43:452–463.
71. Grafstein, B. (1971). Transneuronal transfer of radioactivity in the central nervous system. *Science* 172:177–179.
72. Drachman, D. B. (Ed.) (1974). Trophic function of the neuron. *Ann. N.Y. Acad. Sci.* 228, pp. 1–423.

Nerve Root Compression: Effects on Neural Chemistry and Metabolism

DAVID PLEASURE
Associate Professor, Neurology
University of Pennsylvania, and
Children's Hospital of Philadelphia

INTRODUCTION

Nerve compression results in two distinct pathological processes. Trauma of relatively low intensity (pressures of 150 to 1,000 millimeters of mercury) causes segmental demyelination.[1-3] Electrophysiological studies demonstrate slowing of conduction velocity in the injured segment, or conduction block.[2,4] Distal to the compressed segment, conduction velocity and neuromuscular junction function are preserved. To restore normal function, all that is required is new myelin synthesis in the injured segment; this is ordinarily a rapid, successful process.

When trauma is more severe, Wallerian degeneration occurs. The distal nerve segment becomes electrically inexcitable, and neuromuscular junctions and sensory end-organs degenerate.[5,6,7] To restore function, the neurons must synthesize large quantities of axonal structural proteins, the axons must sprout through the distal nerve segment to reestablish synaptic contact with muscle, and finally these axons must be remyelinated.[7,8] This complex regenerative process is usually incomplete and unsatisfactory, especially in adults.[9]

This paper will outline the biochemical alterations in peripheral nerves and nerve roots during Wallerian degeneration. Unfortunately, traumatic segmental demyelination has not yet been studied biochemically, though it is probably more frequent than Wallerian degeneration as a cause of neurological disability after nerve compression. Morphological and electrophysiological investigations of the pathogenesis of traumatic segmental demyelination will be reviewed, as will recent biochemical studies of nontraumatic segmental demyelination.

WALLERIAN DEGENERATION

A. Axonal changes. Most axonal structural proteins and enzymes are synthesized in the neuronal perikaryon.[10,11] The major axonal structural proteins include tubulin, the monomeric form of microtubules;[12,13] filarin, the subunit of neurofilaments;[14] and the growth-cone microfilamentary subunit, an actin-like protein.[15,16] Chemical properties of these proteins have recently been reviewed.[17] Axons also contain a variety of enzymes, including those associated with transmitter vesicles and mitochondria.

Within 24 hours after nerve transection, disintegration of microtubules and neurofilaments, mitochondrial swelling, and rupture of the axolemma occur distal to the injury.[6,7,18] Prior to axonal fragmentation, there is a transitory accumulation of transmitter vesicles and other axonal organelles in the distal nerve stump, probably a consequence of residual reverse axoplasmic flow.[18,19] Miniature end-plate potentials continue for several hours after the transection, longer when the injury is far from muscle, suggesting continued forward axoplasmic flow.[20] Similarly, forward and reverse axoplasmic flow continue for several hours in vitro in isolated nerve segments, indicating that such flow is not dependent upon continuity with the neuronal perikaryon.[21,22]

Neurofilaments, mitochondria and transmitter vesicles accumulate in the proximal nerve stump during the first days after axonal transection.[23,24] The concentration of cyclic 3′,5′-adenosine-5′-

monophosphate in the proximal stump also increases;[25] this cyclic nucleotide stimulates the polymerization of tubulin into microtubules[26] and accelerates axonal sprouting.[27] Axoplasmic transport of tubulin and the actin-like microfilamentary monomer is accelerated, whereas axoplasmic flow of transmitter vesicles is reduced.[10] Axonal sprouting begins within a few days; each sprout is tipped by a bulbous growth cone, containing contractile microfilaments that are inserted into the plasma membrane.[15] Axonal microtubules extend into the sprouts, serving as rigid structural members, and perhaps functioning in axoplasmic transport.[28-30]

Retrograde axoplasmic flow, from axonal terminals toward the perikaryon, is probably also important during nerve regeneration. In autonomic axons, nerve growth factor is translocated proximally and may serve to enhance axonal sprouting.[31] There is a transitory increase in reverse axoplasmic flow of proteins at the time regenerating axons reach denervated muscle.[32]

B. Anterior horn changes. Neurons actively synthesize proteins under normal circumstances, and this synthesis is further accelerated after axonal transection.[28, 33-35] Uptake of RNA and protein precursors by the perikaryon accelerates within a day after transection, and there is increased production of the axonal structural proteins.[10, 34, 36] Other normal neuronal functions are neglected during the period of axonal sprouting; synthesis of transmitter vesicles declines,[10] and the number of perikaryal boutons and the extent of dendritic arborization are reduced.[35]

Anterior horn glial cells also participate in the response to axonal transection. During the first few days after trauma, perineuronal microglia proliferate and astrocytes increase in cell mass. Oligodendrocytes do not respond early, but enlarge at the time of end-organ reinnervation.[35, 37]

C. Changes in Schwann cells. Schwann cells make up 90 percent of the endoneurial cell population.[38, 39] All Schwann cells have the capacity to form myelin when in contact with an appropriate axon,[40] and myelinating Schwann cells are highly specialized plasma-membrane synthetic cells. The function of those Schwann cells surrounding unmyelinated axons is unclear, though they may synthesize proteins for export into the axons.[41]

Myelin is particularly rich in lipids, especially unesterified cholesterol and the galactolipids.[42, 43] Peripheral nerve myelin contains three principal proteins, a glycoprotein ("P_0," molecular weight about 30,000 daltons) and two basic proteins ("P_1" nearly identical to CNS myelin basic protein in molecular weight, amino acid sequence, and antigenic properties, and "P_2," molecular weight about 12,000 daltons). In addition, a variable number of higher molecular weight proteins are present in trace amounts; it is uncertain whether these are intrinsic to the myelin sheath, are non-myelin Schwann cell plasma membrane proteins, or contaminants.[44-46]

During the first week after axonal transection, the amount of myelin that can be isolated from the distal nerve segment by sucrose density gradient ultracentrifugation falls, and essentially no myelin can be isolated after 2 weeks.[47] There are corresponding reductions in distal nerve segment content of the lipids and proteins characteristic of myelin, and increases in myelin degradation products, including cholesterol ester and lysophosphatidylcholine.[48,49] During this period, the rate of incorporation of radioactive lipid and protein precursors into myelin by the distal nerve segment falls far below normal.[47] Electron microscopy suggests that the Schwann cells themselves are responsible for myelin catabolism during Wallerian degeneration.[50]

During the first days after axonal transection, the rate of mitosis of Schwann cells in the distal nerve segment increases markedly;[51] subsequently, the Schwann cells line up to form bands of Bunger. When axonal sprouts have penetrated these Schwann cell bands and reached a size suitable as a framework for new myelin formation, there is a dramatic increase in myelin protein and lipid synthesis.[47] The amount of myelin in the regenerated nerve, however, never fully returns to normal.[8, 52]

D. Changes in collagen metabolism. Collagen is present in larger quantity than any other protein in peripheral nerves.[53] The relative amount of collagen is greatest in small, superficial sensory nerves, intermediate in large nerve trunks, and lowest in the nerve roots.[54, 55] Two forms of collagen are present: interstitial collagen microfibrils; and collagen covalently bound to glycoproteins in basement membrane. The chemistry of these two forms has recently been reviewed.[56, 57] Endoneurial interstitial collagen microfibrils are synthesized, at least in part, by endoneurial fibroblasts. Recent tissue culture studies indicate that

cloned malignant Schwann cells, like most ectodermal derivatives, are able to synthesize basement membrane collagen.[58] It seems likely, therefore, that the basement membrane that surrounds all Schwann cell processes in the endoneurium is synthesized by the Schwann cells themselves.

After nerve crush or transection, collagen accumulates at the site of trauma, and continues to increase in concentration for a year or more.[52, 53, 59] Collagen deposition is restricted to the traumatic zone, and there is no increase in this protein in the distal nerve segment, remote from injury.[52] Dense collagen scar in the injured zone may be a major impediment to entry of axonal sprouts into appropriate fascicles of the distal nerve segment,[60] and the progressive increase in scar with time after trauma may explain the poor functional return achieved with late secondary repair of transected nerves.[61-63] Specific pharmacologic inhibition of collagen scar formation in experimental animals subjected to sciatic nerve transection results in an increase in the rate of myelination of the distal nerve segment.[52]

E. Changes in metabolism of muscle. Motor axons have a trophic effect on muscle, controlling the mechanical and metabolic properties of the muscle fibers.[64] The nature of this influence of nerve on muscle is controversial; some evidence points to acetylcholine as the trophic substance, while other investigations suggest the existence of an as yet uncharacterized trophic protein.[65-67]

After nerve transection, there are early changes in muscle membrane composition and metabolism, including increases in sialic acid content and in phosphatidyl inositol turnover,[68, 69] and spread of the distribution of the acetylcholine receptor protein from the subneural apparatus to larger areas of the plasma membrane.[70] The denervated muscle becomes atrophic, and later, there are "myopathic" degenerative changes as well. Though the denervated muscle appears fibrotic, whole muscle content of collagen does not increase (Pleasure, unpublished data).

Muscle has a trophic effect on regenerating nerve as well. Reverse axoplasmic flow increases after restoration of contact between axonal sprouts and muscle;[32] and sprouts that reach muscle increase rapidly in diameter, whereas unsuccessful sprouts do not.[71, 72] Axonal enlargement is less pronounced if the muscle tendon is cut.[72]

SEGMENTAL DEMYELINATION

With trauma of relatively low intensity, there is initial distortion of internodal myelin, followed by paranodal and then segmental demyelination.[1-3] Conduction time along demyelinated internodes is prolonged, though saltatory conduction is not lost, and the "safety factor" of demyelinated internodes is reduced, so that small increases in temperature or in intracellular sodium content result in conduction block.[73-75]

With each episode of demyelination, Schwann cells divide and there is deposition of interstitial and basement membrane collagen in the endoneurium.[76] Recurrent segmental demyelination may lead to excessive concentric proliferation of Schwann cells and collagen around central denuded axons.[76, 77]

Under normal circumstances, each of the lipids and proteins in myelin has a discrete half-life, and steady-state levels of myelin are maintained by synthesis of each lipid and protein by Schwann cells at a rate adequate to match losses. Segmental demyelination occurs if Schwann cell synthesis of a myelin constitutent falls below the normal catabolic rate, as occurs in diphtheritic neuropathy,[78] or if destruction of myelin increases to beyond the capacity of Schwann cells to provide replacements, as occurs when the myelin is destroyed by a surface-active agent.[79]

These two possibilities, depressed Schwann cell synthetic capacity or accelerated destruction of preformed myelin, have both been invoked to explain traumatic segmental demyelination. When a segment of nerve is compressed, blood flow is reduced, and Schwann cell ischaemia occurs; if sufficiently severe, this might depress synthesis of myelin lipids and proteins. If ischaemia alone were responsible for pressure-induced demyelination, however, then demyelination should be present throughout the segment of nerve beneath an occluding tourniquet. In fact, demyelination after acute compression occurs only at the upper and lower borders of the tourniquet, with sparing of the central zone.[3,4] This suggests that mechanical deformation induced by a pressure gradient, rather than ischaemia, is responsible for acute compression-induced segmental demyelination. The relative contributions of ischaemia and of mechanical deformation to segmental demyelination induced by chronic compression remain unknown.

CONCLUSIONS

The biochemical changes during Wallerian degeneration have been studied extensively, and knowledge of the functioning of axonal organelles during sprouting, and of changes in collagen metabolism at the site of trauma, permits the design of rational therapies to optimize nerve regeneration.[52,80] No comparable studies of traumatic segmental demyelination are available; such studies might also lead to measures that would decrease the incidence of long-term disability after nerve and nerve-root compression.

REFERENCES

1. Bentley, F. H., and Schlapp, W.: The effects of pressure on conduction in peripheral nerve. *J. Physiol.* 102:72–82, 1943.
2. Ochoa, J., and Marotte, L.: The nature of the nerve lesion caused by chronic entrapment in the guinea pig. *J. Neurol. Sci.* 19:491–495, 1973.
3. Rudge, P., Ochoa, J., and Gilliatt, R. W.: Acute peripheral nerve compression in the baboon. *J. Neurol. Sci.* 23:403–420, 1974.
4. Fowler, T. J., Danta, G., and Gilliatt, R. W.: Recovery of nerve conduction after a pneumatic tourniquet: observations on the hind-limb of the baboon. *J. Neurol. Neurosurg. Psychiatry* 35:638–647, 1972.
5. Korthals, J. K., Wisniewski, H. M., Ghetti, B., and Cook, R. D.: The fate of the axon and its terminal in the Pacinian corpuscle following sciatic nerve section. *J. Neurocytol.* 3:385–403, 1974.
6. Ballin, R. H. M., and Thomas, P. K.: Changes at the nodes of Ranvier during Wallerian degeneration: an electron microscope study. *Acta Neuropath.* (Berl) 14:237–249, 1969.
7. Donat, J. R., and Wisniewski, H. M.: The spatio-temporal pattern of Wallerian degeneration in mammalian peripheral nerves. *Brain Res.* 53:41–53, 1973.
8. Schroder, J. M.: Altered ratio between axon diameter and myelin sheath thickness in regenerated nerve fibers. *Brain Res.* 45:49–65, 1972.
9. Ballantyne, J. P., and Campbell, M. J.: Electrophysiological study after surgical repair of sectioned human peripheral nerves. *J. Neurol. Neurosurg. Psychiatry* 36:797–805, 1973.
10. Frizell, M., and Sjostrand, J.: The axonal transport of slowly migrating [^{3}H] leucine labelled proteins and the regeneration rate in regenerating hypoglossal and vagus nerves of the rabbit. *Brain Res.* 81:267–283, 1974.
11. Dahlstrom, A.: Axoplasmic transport (with particular respect to adrenergic neurons). *Phil. Trans. Roy. Soc.* B 261:325–358, 1971.
12. Davison, P. F., and Huneeus, F. C.: Fibrillar proteins from squid axons. II. Microtubular protein. *J. Molec. Biol.* 52:429–439, 1970.
13. Borisy, G. G., Olmsted, J. B., and Klugman, R. A.: In vitro aggregation of cytoplasmic microtubule subunits. *Proc. Nat. Acad. Sci.* USA 69:2890–2894, 1972.
14. Shelanski, M. L., Albert, S., DeVries, G. H., and Norton, W. T.: Isolation of filaments from brain. *Science* 174: 1242–1245, 1971.
15. Tennyson, V. M.: The fine structure of the axon and growth cone of the dorsal root neuroblast of the rabbit embryo. *J. Cell Biol.* 44:62–79, 1970.
16. Fine, R. E., and Bray, D.: Actin in growing nerve cells. *Nature (New Biol.)* 234:115–118, 1971.
17. Pleasure, D. E.: The structural proteins of peripheral nerve. In Dyck, Thomas and Lambert, *Peripheral Neuropathies*, W. B. Saunders, in press, 1975.
18. Bray, G. M., Peyronnard, J. M., and Aguayo, A. J.: Reactions of unmyelinated nerve fibers to injury. An ultrastructural study. *Brain Res.* 42:297–309, 1972.
19. Partlow, L. M., Ross, C. D., Motwani, R., and McDougal, D. B., Jr.: Transport of axonal enzymes in surviving segments of frog sciatic nerve. *J. Gen. Physiol.* 60:388–405, 1972.
20. Miledi, R., and Slater, C. R.: On the degeneration of rat neuromuscular junctions after nerve section. *J. Physiol.* 207:507–528, 1970.
21. Schlaepfer, W. W.: Effects of nerve constriction on oxygenated excised segments of rat peripheral nerve. *J. Neuropath. Exp. Neurol.* 32:203–217, 1973.
22. Ochs, S.: Fast transport of materials in mammalian nerve fibers. *Science* 176:250–260, 1972.
23. Kapeller, K., and Mayor, D.: An electron microscopic study of the early changes proximal to a constriction in sympathetic nerves. *Proc. Roy. Soc. B.* 172:39–51, 1969.
24. Garcia, A. G., Kirpekar, S. M., Prat, J. C., and Wakade, A. R.: Metabolic and ionic requirements for the axoplasmic transport of dopamine beta-hydroxylase. *J. Physiol.* 241:809–821, 1974.
25. Jeffrey, P. L., and Austin, L.: Axoplasmic transport. *Neurobiology* 2:207–255, 1973.
26. Puck, T. T., Waldren, C. A., and Hsie, A. W.: Membrane dynamics in the action of dibutyryl adenosine 3′,5′-cyclic monophosphate and testosterone on mammalian cells. *Proc. Nat. Acad. Sci.* USA 69:1943–1947, 1972.
27. Roisen, F. J., Murphy, R. A., Pichichero, M. E., and Braden, W. G.: Cyclic adenosine monophosphate stimulation of axonal elongation. *Science* 175:73–74, 1972.
28. Smith, A. D.: Summing up: some implications of the neuron as a secreting cell. *Phil. Trans. Roy Soc. Lond.* B. 261:423–437, 1971.
29. Tilney, L. G.: the assembly of microtubules and their role in the development of cell form. *Develop. Biol. Suppl.* 2:63–102, 1968.
30. Daniels, M.: Colchicine inhibition of nerve fiber formation in vitro. *J. Cell Biol.* 53:164–176, 1972.
31. Hendry, I. A., Stockel, K., Thoenen, H., and Iversen, L. L.: The retrograde axonal transport of nerve growth factor. *Brain Res.* 68:103–121, 1974.
32. Kristensson, K., and Sjostrand, J.: Retrograde transport of protein tracer in the rabbit hypoglossal nerve during regeneration. *Brain Res.* 45:175–181, 1972.
33. Price, D. L., and Porter, K. R.: The response of ventral horn neurons to axonal transection. *J. Cell Biol.* 53:24–37, 1972.
34. Jeffrey, P. L., and Austin, L.: Axoplasmic transport. *Neurobiology* 2:207–255, 1973.

35. Watson, W. E.: Cellular responses to axotomy and to related procedures. *Br. Med. Bull.* 30:112–115, 1974.
36. Watson, W. E.: An autoradiographic study of the incorporation of nucleic-acid precursors by neurones and glia during nerve regeneration. *J. Physiol.* 180:741–753, 1965.
37. Kreutzberg, G. W.: Autoradiographische untersuchung uber die beteiligung von gliazellen an der axonalen reaktion im facialskern der ratte. *Acta Neuropath.* 7:149–161, 1966.
38. Thomas, P. K.: The connective tissue of peripheral nerve: an electron microscope study. *J. Anat.* 97:35–44, 1963.
39. Ochoa, J., and Mair, W. G. P.: The normal sural nerve in man. I. Ultrastructure and numbers of fibers and cells. *Acta Neuropath.* (Berl) 13:197–216, 1969.
40. Peters, A., and Vaugn, J. E.: Morphology and development of the myelin sheath. In Davison and Peters, *Myelination*, Charles C Thomas, 1970, pp. 3–79.
41. Lasek, R. J., Gainer, H., and Przybylski, R. J.: Transfer of newly synthesized proteins from Schwann cells to the squid giant axon. *Proc. Nat. Acad. Sci. USA* 71:1188–1192, 1974.
42. O'Brien, J. S., Sampson, E. L., and Stern, M. B.: Lipid composition of myelin from the peripheral nervous system. Intradural spinal roots. *J. Neurochem.* 14:357–365, 1967.
43. Oulton, M., and Mezei, C.: Developmental changes in lipid composition of chick sciatic nerve myelin. *Lipids* 8: 235–238, 1973.
44. Brostoff, S., Burnett, P., Lampert, P., and Eylar, E. H.: Isolation and characterization of a protein from sciatic nerve myelin responsible for experimental allergic neuritis. *Nature (New Biol.)* 235:210–212, 1972.
45. London, Y.: Preparation of purified myelin from ox intradural spinal roots by rate-isopycnic zonal centrifugation. *Biochem. Biophys. Acta* 282:195–204, 1972.
46. Singh, H., and Spritz, N.: Polypeptide components of myelin from rat peripheral nerve. *Biochem. Biophys.* Acta 351:379–386, 1974.
47. Rawlins, F. A., and Smith, M. E.: Metabolism of sciatic nerve myelin in Wallerian degeneration. *Neurobiology* 1:225–231, 1971.
48. Berry, J. F., Cevallos, W. H., and Wade, R. R., Jr.: Lipid class and fatty acid composition of intact peripheral nerve and during Wallerian degeneration. *J. Amer. Oil Chem. Soc.* 42:492–500, 1965.
49. Mezei, C.: Cholesterol esters and hydrolytic cholesterol esterase during Wallerian degeneration. *J. Neurochem.* 17:1163–1170, 1970.
50. Morris, J. H., Hudson, A. R., and Weddell, G.: A study of degeneration and regeneration in the divided rat sciatic nerve based on electron microscopy. I. The traumatic degeneration of myelin in the proximal stump of the divided nerve. Z. Zellforsch 124:76–102, 1972.
51. Bradley, W. G., and Asbury, A. K.: Duration of synthesis phase in neurilemma cells in mouse sciatic nerve during degeneration. *Exp. Neurol.* 26:275–282, 1970.
52. Pleasure, D. E., Bora, F. W., Jr., Lane, J., and Prockop, D. J.: Regeneration after nerve transection: effect of inhibition of collagen synthesis. *Exper. Neurol.* 45:72–78, 1974.
53. Abercrombie, M., and Johnson, M.: Collagen content of rabbit sciatic nerve during Wallerian degeneration. *J. Neurol. Neurosurg. Psychiatry* 9:113–118, 1946.
54. Gamble, H. J.: Comparative electron-microscopic observations on the connective tissues of a peripheral nerve and a spinal nerve root in the rat. *J. Anat.* 98:17–25, 1964.
55. Gamble, H. J., and Eames, R. A.: An electron microscope study of the connective tissues of human peripheral nerve. *J. Anat.* 98:655–663, 1964.
56. Grant, M., and Prockop, D.: The biosynthesis of collagen. *New Engl. J. Med.* 286:194–199, 242–249, 291–300, 1972.
57. Kefalides, N.: Structure and biosynthesis of basement membranes. *Int. Rev. Connect Tissue Res.* 6:63–104, 1973.
58. Church, R. L., Tanzer, M. L., and Pfeiffer, S. E.: Collagen and procollagen production by a clonal line of Schwann cells. *Proc. Nat. Acad. Sci.* USA 70:1943–1946, 1973.
59. Abercrombie, M., and Johnson, M.: The effect of reinnervation on collagen formation in degenerating sciatic nerves of rabbits. *J. Neurol. Neurosurg. Psychiatry* 10:89–92, 1947.
60. Sunderland, S.: *Nerves and Nerve Injuries.* Williams and Wilkins, 1968.
61. Pollard, J. D., and Fitzpatrick, L.: An ultrastructural comparison of peripheral nerve allografts and autografts. *Acta Neuropath.* (Berl) 23:152–165, 1973.
62. Holmes, W., and Young, J. Z.: Nerve regeneration after immediate and delayed suture. *J. Anat.* 77:63–96, 1942.
63. Sanders, F. K., and Young, J. Z.: The role of the peripheral stump in the control of fibre diameter in regenerating nerves. *J. Physiol.* 103:119–136, 1944.
64. Guth, L.: "Trophic" influences of nerve on muscle. *Physiol. Rev.* 48:645–687, 1968.
65. Drachman, D. B.: Is acetylcholine the trophic neuromuscular transmitter? *Arch. Neurol.* 17:206–218, 1967.
66. Korr, I. M., and Appeltauer, G. S. L.: The time-course of axonal transport of neuronal proteins to muscle. *Exp. Neurol.* 43:452–463, 1974.
67. Giacobini, G., Filogamo, G., Weber, M., Boquet, P., and Changeux, J. P.: Effects of a snake alpha-neurotoxin on the development of innervated skeletal muscles in chick embryo. *Proc. Nat. Acad. Sci.* USA 70:1708–1712, 1973.
68. Andrew, C. G., and Appel, S. H.: Macromolecular characterization of muscle membranes. I. Proteins and sialic acid of normal and denervated muscle. *J. Biol. Chem.* 248:5156–5163, 1973.
69. Appel, S. H., Andrew, C. G., and Almon, R. R.: Phosphatidyl inositol turnover in muscle membranes following denervation. *J. Neurochem.* 23:1077–1080, 1974.
70. Chiu, T. H., Dolly, J. O., and Barnard, E. A.: Solubilization from skeletal muscle of two components that specifically bind alpha-bungarotoxin. *Biochem. Biophys. Res. Comm.* 51:205–213, 1973.
71. Aitken, J. T., Sharman, M., and Young, J. Z.: Maturation of regenerating nerve fibres with various peripheral connexions. *J. Anat.* 81:1–22, 1947.
72. Eisen, A. A., Carpenter, S., Karpati, G., and Bellavance, A.: The effect of muscle hyper- and hypoactivity upon

597-036 O - 76 - 14

fiber diameters of intact and regenerating nerves. *J. Neurol. Sci.* 20:457–469, 1973.

73. Rasminsky, M., and Sears, T. A.: Internodal conduction in undissected demyelinated nerve fibres. *J. Physiol.* 227:323–350, 1972.
74. Koles, Z. J., and Rasminsky, M.: A computer simulation of conduction in demyelinated nerve fibres. *J. Physiol.* 227:351–364, 1972.
75. Rasminsky, M.: The effects of temperature on conduction in demyelinated single nerve fibers. *Arch. Neurol.* 28:287–292, 1973.
76. Dyck, P. J.: Experimental hypertrophic neuropathy. *Arch. Neurol.* 21:73–95, 1969.
77. Pleasure, D. E., and Towfighi, J.: Onion bulb neuropathies. *Arch. Neurol.* 26:289–301, 1972.
78. Pleasure, D. E., Feldmann, B., and Prockop, D. J.: Diphtheria toxin inhibits the synthesis of myelin proteolipid and basic proteins by peripheral nerve in vitro. *J. Neurochem.* 20:81–90, 1973.
79. Hall, S. M., and Gregson, N. A.: The in vivo and ultrastructural effects of injection of lysophosphatidyl choline into myelinated peripheral nerve fibres of the adult mouse. *J. Cell Sci.* 9:769–789, 1971.
80. Pichichero, M., Beer, B., and Clody, D. E.: Effect of dibutyryl cyclic AMP on restoration of damaged sciatic nerves in rats. *Science* 182:724–725, 1973.

Discussion: Papers of Sidney Ochs and David E. Pleasure

IRVIN M. KORR
Professor, Dept. of Biomechanics
College of Osteopathic Medicine
Michigan State University

We have just heard two splendid papers on axonal transport, trophic functions and neural chemistry and metabolism. They are comprehensive, though concise summaries of current knowledge and hypotheses and require no detailed comment from this discussant. As a physiologist who has had the long-time advantage of working in an osteopathic environment, I believe that my best contribution would be to relate this material to the subject of the conference, spinal manipulation. I propose, therefore, to identify and, where possible, characterize the ways in which the functions, processes and mechanisms discussed by Drs. Ochs and Pleasure might be affected by nerve-trunk deformations of musculoskeletal origins which in turn are amenable to manipulative therapy. To avoid duplication, I shall limit the discussion to those circumstances in which axons survive, disabled though they may be. We shall not, therefore, be concerned with degeneration and regeneration, but only with first degree injury, in Sunderland's classification.[1] I hope this may be a useful effort even if, other than a few clues, the only products are more unsolved questions. Certainly, I have few answers.

These are my questions: How may compression, stretching, angulation, or other deformations of nerves and nerve roots by surrounding structures influence neural chemistry and metabolism and the synthesis and axonal transport of macromolecules and subcellular structures? And how do these neural effects in turn influence the tissues, organs and processes under neural control and regulation?

I do not need to remind this group that a nerve or nerve root is far more complex than a mere aggregate of axons and their myelin sheaths. A nerve may even be viewed as an integrated *organ* consisting of many different kinds of cells, tissues and their products. Nevertheless, and for obvious reasons, the prevailing emphasis of studies on neural mechanisms and their impairment has been on the axons.

Axonology, however, does not offer enough of an answer to the questions I have just asked about the neurochemical and neurotrophic consequences of neural deformation – central questions before this conference. What else do we need to know? We need to know how the nonaxonal components of nerve trunks, especially those which serve nutritive functions and which provide or control the environment of the axons, are affected by deformation and how these in turn affect neuronal chemistry, metabolism, synthesis, axonal transport and trophic relations. These include the Schwann cells and myelin; the vascular elements; the various mesenchymal components; the endoneurial fluids and their flow channels.

Unfortunately, little is yet known about how some of these various components contribute to neural economy under *normal* circumstances, much less under those engendered by mechanical insult. I shall try to suggest how these various components of the nerve may be affected by deformation. I shall start with axons, about which one feels a little more secure.

AXONS

Axoplasmic Transport

Proximodistal (Cellulifugal) Transport

Attenuation of the axon by compression or other deformation, as has been described, impedes axoplasmic transport beyond the compression. The axon becomes swollen with dammed-up axoplasm immediately proximal to the compressed segment, while distally the axon may become reduced in diameter. A smaller swelling on the distal side of the compression reflects dammed-up axoplasmic components moving *toward* the cell. As long as sufficient axoplasmic passage through the stenotic segment is permitted, axoplasmic continuity is preserved and the trophic relations between the perikaryon and its long cytoplasmic process continue, though probably with some impairment. The axon distal to the lesion survives and Wallerian degeneration is forestalled.

Moreover, trophic maintenance of the axon ensures continued trophic support of innervated structures. As long as connectedness is maintained between nerve cell and, for example, muscle cells, whether or not impulses are being or even *can* be transmitted, the muscle cells remain intact and functional. Acetylcholine sensitivity remains, as in the completely normal motor unit, limited to the junctional area and no fasciculation is seen.

We do not as yet know, however, the effects either on the axon (other than attenuation) or on the innervated tissues of the slowing of the axoplasmic supplies or whether indeed the change in axoplasmic transport is purely quantitative. The motoneuron, for example, is essential not only for the *maintenance* of muscle, but also because it exerts a variety of trophic influences on the structure, excitability, contractile properties, enzyme activity, metabolism, etc., indeed on its genic expression.[2,3] These influences, as Dr. Ochs has indicated, seem to be based on axoplasmic transport and, I believe, on the actual transsynaptic delivery of neuronal proteins and other substances to the muscle cells.[4-6] How does the slowing, and possible qualitative alteration, of this delivery affect the functions and properties of the muscle (or other innervated structure)? Such questions are of immense clinical as well as theoretical interest.

We do know that under such conditions of axonal continuity, the axon distal to the lesion, though thinner, remains capable of excitation and conduction. Whether or not the neuron as a whole is functional, i.e., can conduct impulses from one end to the other, depends on the state of the injured segment. At that site the axon may transmit impulses normally; or conduction may be blocked; or it may be hyperexcitable; and, in the living, breathing, moving animal or human, it may oscillate back and forth from one state to the other. Conduction block may, however, be persistent, and recovery delayed for hours, days or months, even after a momentary deformation, as in concussion. The affected fibers suddenly reawaken to activity, as Dr. Sunderland describes it. The chemical and structural basis for the functional states—normal, conduction block, hyperexcitability—is unknown. We can only assume that they are somehow related to the degree of polarization of the resting membrane, although Dr. Pleasure has also drawn our attention to the state of para-nodal myelin.

Hence, excitatory function and trophic function are completely dissociated in this sublethal degree of axonal deformation. Similar dissociation may be produced experimentally (or therapeutically) by chemical or physical agents applied to the nerve which block conduction even for long periods of time (e.g. local anesthetics, cold, pressure) without interrupting the axon.[7] It is of historic interest that only recently was it possible to produce, experimentally, the reverse dissociation, in which a trophic influence was blocked, by pharmacological interference with fast axoplasmic transport, while the nerve remained functional as a transmitter of impulses.[8] Of course, when axoplasmic transport is reduced to the point that it is no longer adequate for axonal maintenance, conduction soon fails and Wallerian degeneration ensues. Up to this point, removal of the compressing force permits redistribution of dammed-up axoplasm and the recovery of the neuron.

Retrograde (Cellulipetal) Transport

As reviewed by Dr. Ochs, at this and other symposia,[3] axonal transport *toward* the cell body has also been demonstrated and studied. It has been shown that proteins in muscle may enter the nerve and be conveyed to the cell body. This transport, too, may be expected to be retarded by axonal compression. Insofar as this serves a feedback function which chemically "informs" the neuron about circumstances in the periphery and

thereby regulates perikaryal morphology, metabolism, protein synthesis, synaptogenesis and possibly even gene expression, this retardation may be expected to have deleterious effects. This is another important area for investigation, not only for obvious theoretical reasons, but because it relates to the clinical impact of axonal impingement and the value of spinal manipulation.

SCHWANN CELLS AND MYELIN SHEATHS

Aside from the role of the Schwann cells in the formation of myelin sheaths, which are so important in impulse conduction, there is evidence of a role in axonal nutrition and energy exchange also. Though not yet well understood, it appears that these cells may supply essential substances, possibly through the Schmidt-Lantermann clefts, that are not supplied by the neuron or via blood and tissue fluids.[9] Deformation of a nerve almost invariably involves deformation, and even disruption of Schwann cell and myelin layers. As the work of Denny-Brown and Brenner[10,11] and others cited by Dr. Pleasure has shown, these structures, especially in the vicinity of the nodes of Ranvier, may undergo considerable disruption in injured segments of nerve. The axons, though they remain intact, undergo conspicuous chemical changes, reflected in altered staining properties at the affected nodes. However, little is known about the effects of such changes on axonal chemistry, metabolism, transport or trophic function, in contrast with the more substantial understanding of the role of myelin in excitation and conduction. In view, however, of the demonstration by Ochs and his coworkers that fast axoplasmic transport is energized by metabolic processes within the axon,[12] reduced access of Schwann-supplied metabolites may be expected to impede or even block such transport. Since the substances, such as glycoproteins, which are rapidly transported seem to be especially important to the axonal endings and synaptic and myoneural transmission, even local disorganization of the Schwann cell and myelin layers may have serious consequences.

BLOOD VESSELS

Deformation of a nerve or spinal root almost inevitably produces deformation and even occlusion of the vessels supplying the nerve. Occlusion of the vasa nervorum of a peripheral nerve over even a considerable stretch of nerve does not necessarily result in ischemia, much less anoxia, because of the rich anastamosis and diffusion of oxygen from surrounding tissues and adjacent nerve segments. The effects of experimental ischemia, with the use of blood-pressure cuffs or tourniquets, on neural function in humans and animals, as reflected in sensation and in motor and autonomic function, have been studied by many investigators, with varying, and even conflicting results.[1] Interpretations of the results are even more ambiguous because, with these methods, it is not possible to distinguish between the effects of ischemia and those of neural compression. However, studies of the effects of local deformation or of anoxia on exposed or isolated nerves, clearly indicate that the respective mechanisms leading to conduction block are quite different, as reflected, for example, in different orders of susceptibility among fiber types, and in very different dynamics of induction and recovery from block.

Of course, ischemia of sufficient intensity and duration does cause degeneration of the ischemic and distal portions. Less severe or briefer ischemia produces conduction block without degeneration. Hyperexcitability occurs during the induction and post-ischemic periods. When the perfusion rate is chronically reduced, but not to the point of complete ischemia, different fibers in the same nerve may at the same time be in various functional states ranging from normal conduction, to hyperexcitability (ectopic, supernumerary impulses) to conduction block, to degeneration; and the degree of ischemia and neural dysfunction may fluctuate with motion, blood pressure and other variables. The functional state—normal, nonconducting or hyperexcitable—of the hypoxic (ischemic) nerve fibers is related to the resting membrane potential, which is also energized by oxidations in the axon.[13,14]

Mechanical insults or relatively moderate pressures which selectively occlude the veins draining the nerve introduce disturbances other than, or in addition to, ischemia. As in other tissues, venous obstruction produces hyperemia, increased transudation and edema. Accumulation of interfibrillar fluids almost certainly retards the exchange of substances between the axons and other elements in the nerve, with detriment to axonal metabolism and to the processes that depend on axonal metabolism for energy and for specific

substances. Alterations in spatial relationships within the edematous nerve and in osmotic relations may also have effects on excitation and conduction. Another factor to be considered as a complication of the increased transudation is the accumulation of protein in the extra-axonal fluids, and the resultant tendency toward fibrosis. This occurs also in chronically ischemic nerves.

The consequences of circulatory embarrassment in the spinal roots may be expected to be the same as for peripheral nerves. There are several factors, however, which render the roots much more vulnerable in this regard:

1. Their location within the intervertebral foramen is in itself a great hazard.
2. Spinal roots lack the protection of epineurium and perineurium.
3. Since each root is dependent on a single radicular artery entering via the foramen, the margin of safety provided by collateral pathways is minimal.
4. Venous congestion may be more common in the roots because the radicular veins would probably be immediately compressed by any reduction of foraminal diameter. There is also the possibility of reflux from the segmental veins through pressure-damaged valves; and venous congestion would have additional consequences because the swelling, being within the foramen, would contribute to compression of the other intraforaminal structures.
5. Circulation to the dorsal root ganglion is especially vulnerable for anatomic reasons shown by Bergmann and Alexander.[15]

It is interesting to speculate to what degree ischemia (or venous congestion) of the dorsal root ganglion affects its protein metabolism or the "routing" mechanism discussed by Dr. Ochs which apparently controls the relative rates of exportation of neuronal proteins toward the spinal cord as compared to that toward the periphery. It is important to remember in this connection that sensory neurons are no less potent trophically than motor neurons. One should remember, also, that because the radicular arteries contribute substantially to the blood flow of the spinal cord, reduced radicular flow over several segments could also affect central neurons and intraspinal axons.

INTERSTITIAL FLUIDS IN THE NERVE

The foregoing discussion of edema draws attention to the existence of fluid spaces within the nerve, provided by, or enclosed by, endoneurium (and possibly perineurium) and to the evidence for circulation of the endoneurial fluids in both directions.[16–21]

Little or nothing is known of the origins or fate of the endoneurial fluid, the channels of flow or the propulsive mechanisms. Indeed, if the observations of Weiss et al.[16] are correct that proximo-distal flow continues in the living nerve after arrest of circulation and even in completely excised nerves, then we are presented with still another mystery regarding the source and site of the motor power.

As to connections of endoneurial spaces with other fluid compartments there is good evidence, I think, for connections, direct or indirect or across selectively permeable membranes, with subarachnoid space[17], and subdural space[22] centrally, and with lymphatics distally[23] and in the epineurium. Brierly,[17] for example, proposes that the periradicular cul-de-sac of the subarachnoid space is a subsidiary site for the excretion of cerebrospinal fluid (in which the roots are bathed) which may pass into the spinal nerve as well as into segmental lymph channels.

What is the role of the endoneurial fluid in the nutrition, ionic and osmotic balance and metabolism of the neuron, and in the processes which depend upon them, including excitation, conduction, axonal transport and trophic functions? Unfortunately, we know as little about the function of the endoneurial flow as we do about its mechanisms. We can assume that at the very least it serves the same function as interstitial fluids in other tissues, i.e., as medium of exchange between the tissue cells and the blood. We can also assume, however, that the longitudinal arrangement, the apparently channelized flow and the interchange with fluid compartments of the central nervous system underlie some additional functions specific to nerve. Whatever they are, they may also be expected to be highly vulnerable to mechanical insult to the nerve.

THE CONNECTIVE TISSUES

The structure and relatively obvious functions of the epineurium, perineurium and endoneurium are reasonably well understood, and have been concisely summarized by Sunderland.[1] The fa-

miliar functions include that, especially of the epineurium, of imparting compactness and cohesiveness to the nerve; that of support; that of compartmentalization, the perineurium binding together and encompassing the funiculi, the endoneurium ensheathing the individual fibers with their Schwann and myelin layers; that of guiding regenerating axons back to their appropriate terminals; that of providing and supporting channels for flow of interstitial fluids, blood and lymph; that of diffusion barriers; that of defense against mechanical, chemical and microbial insult.

In connection with neural chemistry, metabolism and axoplasmic transport, I think we need to examine the likelihood that in the nerve, the mesenchymal components may play an unusual, perhaps unique role in the regulation of fluid volume and composition and of osmotic equilibrium. In the normal nerve the dynamic balance between hydrostatic and oncotic pressures seems to be between the radial pressure exerted by the epineurium and water absorption by endoneurial and interstitial collagen, so abundant in nerve, as Dr. Pleasure has emphasized. As Lorente de Nó showed, when the epineurium is mechanically breached or weakened, the nerve swells enormously, with progressive thickening of the endodeurium and enlargement of the interfibrillar spaces.[24] Under these conditions, one may expect disturbances in exchange between blood and axons, in neural chemistry, metabolism, transport, and electrical properties. This is another area for study with modern methods, for its neurophysiological interest, and in relation to the subject of this conference.

THE NEURON

Having called the roll of axonal and nonaxonal components of nerves and roots with a view to their involvement in the effects of deformation, it is now time to return to the neuron. It is important to remember that neural chemistry, metabolism and axonal transport are subject to alteration not only by neural and radicular deformation but by the activity of the corresponding nerve-cell bodies. Almost any factor which more or less enduringly exaggerates the rate of impulse-discharge by neurons also affects the energy requirements, metabolism and, almost inevitably, protein synthesis and turnover.

As Dr. Denslow will discuss tomorrow, intervertebral and other musculoskeletal strains, designated as osteopathic lesions and responsive to manipulative therapy, are associated with facilitated segments of the spinal cord. In those segments, motoneurons and neurons of the sympathetic nervous system are maintained in a hyperirritable state, presumably by disturbed patterns of afferent input from proprioceptors and other endings in the stressed tissues.[25] In this state, and under conditions of daily life, the affected neurons tend to be in constant or relatively high activity when corresponding neurons in neighboring and contralateral segments are quiescent or only mildly active. The effects of such chronic activity, provoked and sustained by musculoskeletal disturbances, on neural chemistry, metabolism, axonal transport and trophic influence and on the retrograde influences from the target tissues have hardly been explored, yet are of great importance to the subject of this conference.

Finally, it is important to re-emphasize the evidence that peripheral nerves not only conduct impulses to or from the cells, tissues and organs that they supply, but also deliver to them substances synthesized in the cell body, most notably proteins, that are essential for their development, maintenance and self-repair, that influence their various characteristics and functional capacities, and that condition their responses to various factors, both physiologic and noxious.

Any factor which for a protracted period alters the quality or quantity of the axonally transported substances, might not only affect impulse transmission, but could cause the trophic influences to become adverse and detrimental, thereby contributing to disease. In considering the neurologic impact on human health of postural and biomechanical defects in the body framework that are amenable to manipulative therapy, we can no longer limit ourselves to disturbances in impulse traffic. Conspicuous and distressing as are the resultant pain and the motor, sensory and autonomic dysfunction, the more subtle and insidious trophic consequences of disturbances in axoplasmic composition and transport are no less important. It seems likely that much of the efficacy of manipulative therapy is related to the amelioration of those trophic factors.[26,27]

REFERENCES

1. Sunderland, S. *Nerves and Nerve Injuries*. Williams and Wilkins, Baltimore, pp. 35–43, pp. 127–137, 1968.

2. Guth, L. "Trophic" influences of nerve on muscle. *Physiol. Rev.* 48(4):645–687, 1968.
3. Drachman, D. B., Editor. Trophic Functions of the Neuron. *N.Y. Acad. Sci.* 228:1–423, 1974.
4. Korr, I. M., Wilkinson, P. N., and Chornock, F. W. Axonal delivery of neuroplasmic components to muscle cells. *Science* 155:342–345, 1967.
5. Korr, I. M., and Appeltauer, G. S. L. The time-course of axonal transport of neuronal proteins to muscle. *Exp. Neurol.* 43(2):452–463, 1974.
6. Appeltauer, G. S. L., and Korr, I. M. Axonal delivery of soluble, insoluble and electrophoretic fractions of neuronal proteins to muscle. *Exp. Neurol.* 46:132–146, 1975.
7. Robert, E. D., and Oester, Y. T., Electrodiagnosis of nerve-impulse deprived skeletal muscle. *J. Appl. Physiol.* 28(4):439–443, 1970.
8. Albuquerque, E. X., Warnick, J. E., Tasse, J. R., and Sansone, F. M. Effects of Vinblastine and colchicine on neural regulation of the fast and slow skeletal muscles of the rat. *Exp. Neurol.* 37:607–634, 1972.
9. Singer, M., and Salpeter, M. M. The transport of ^{3}H-L-Histidine through the Schwann and myelin sheath into the axon, including a reevaluation of myelin function. *J. Morph.* 120(3):281–316, 1966.
10. Denny-Brown, D., and Brenner, C. Paralysis of nerve induced by direct pressure and by tourniquet. *Arch. Neurol. Psychiat.* 51:1–26, 1944.
11. Denny-Brown, D., and Brenner, C. Lesions in peripheral nerve resulting from compression by spring clip. *Arch. Neurol. Psychiat.* 52:1–19, 1944.
12. Ochs, S. Energy metabolism and supply of ~ P to the fast axoplasmic transport mechanism in nerve. *Fed. Proc.* 33(4):1049–1058, 1974.
13. Gerard, R. W. Metabolism and function in the nervous system. *In:* Elliott, K. A. C., Page, I. H., and Quastel, J. H., eds. *Neurochemistry: The Chemical Dynamics of Brain and Nerve.* Charles C Thomas, Springfield, Ill., 458–484, 1955.
14. Brink, F. Metabolism of isolated nerve and ganglion. In Richter, D. *Metabolism of the Nervous System.* Pergamon. New York, 187–207, 1957.
15. Bergmann, L., and Alexander, L. Vascular supply of spinal ganglia. *Arch. Neurol. Psychiat.* 46:761–782, 1941.
16. Weiss, P., Wang, H., Taylor, A. C., and Edds, M. V., Jr. Proximo-distal fluid convection in the endoneurial spaces of peripheral nerves, demonstrated by colored and radio-active (isotope) tracers. *Am. J. Physiol.* 143(4):521–540, 1945.
17. Brierly, J. B. The sensory ganglia: recent anatomical, physiological and pathological contributions. *Acta Psychiat. et Neurol. Scand.* 30(4):553–576, 1955.
18. Appeltauer, G. S. L., and Eduardo, E. A. Saá. Incorporation of C–14 Lysine into spinal roots, spinal ganglia and peripheral nerves of the rat. *Exp. Neurol.* 14:484–495, 1966.
19. Brierly, J. B., and Field, J. The fate of an intraneural injection as demonstrated by the use of radio-active phosphorus. *J. Neurol., Neurosurg. Psychiatry.* 12(2):86–99, 1949.
20. Bodian, D. Some physiologic aspects of poliovirus infections. *Harvey Lect.* 52:23–56, 1958.
21. Bakin, Ye. I., Dolgachev, I. P., and Kiselev, P. N. Possibility of movement of substances along a nerve. *Herald of Roentgenology and Radiology* (Transl.) No. 1, pp. 3–6, 1953.
22. Dorang, L., and Matzke, H. A. The fate of radio-opaque medium injected into the sciatic nerve. *J. Neuropathol. Exp. Neurol.* 19:25–32, 1960.
23. Kurdyumov, N. A. Structure of the perineural spaces and efferent lymphatic routes from cerebrospinal nerves. *Arkhiv Anatomii, Gistologii i Embriologii (USSR)* (*Transl.*) 44(3):121–127, 1963.
24. Lorente de Nó, R. Observations on the properties of the epineurium of frog nerve. *Symp. Quant. Biol.* 17:299–315, 1952.
25. Korr, I. M. Proprioceptors and somatic dysfunction. *J. Amer. Osteop. A.* (In press).
26. Korr, I. M. The nature and basis of the trophic function of nerves: Outline of a research program. *J. Amer. Osteop. A.* 66:74–78, 1967.
27. Korr, I. M. The trophic functions of nerves and their mechanisms. *J. Amer. Osteop. A.* 72:163–171, 1972.

Chairman's Summary: Neuroscience Studies

HORACE W. MAGOUN
Professor Emeritus
Department of Psychiatry
University of California at Los Angeles

Seth Sharpless: Susceptibility of Spinal Roots to Compression Block

In 1797, Felice Fontana observed that pressure on a nerve blocks its capacity for conduction, without causing stimulation. Sharpless has recently confirmed this observation with sophisticated graded-pressure on the sciatic nerve and lumbar dorsal spinal roots of animals, leading to blockage of the A components of evoked afferent potentials. In addition, he has discovered that dorsal spinal roots display a differential sensitivity to such pressure as compared with the sciatic nerve. Moreover, within the dorsal root, fibers conducting fast components of the A potential are considerably more susceptible to pressure than are those conducting slower components.

Hopefully, the preoccupation of spinal manipulative therapy with back pain will lead Sharpless to extend his experiments to study the effect of such pressure upon slowly conducting, intermediate and fine-diametered afferent fibers in dorsal roots; these are more directly concerned with mediating pain signals than are the large, rapidly conducting A component fibers.

Extrapolation of Sharpless' observations that large, fast-conducting fibers are blocked before slower components suggests the possibility that still smaller and slower pain fibers may be exceedingly resistant to pressure blockade, as witness the ubiquitous complaint of pain during compression of spinal nerve roots in man. It is conceivable that, during pressure block of epicritic signals (tactile pressure and position-sense) in dorsal roots, pain signals that continue to be conveyed centrally may gain unusual intensity and unpleasantness. Recent transposition of Sherrington's concepts of reciprocal innervation, from motor to somatic afferent systems, holds that maintained high activity in epicritic afferents reduces or inhibits the central transmission of pain signals, leading to analgesia. In the absence of such inhibition from blockade of epicritic signals, activity in the central pain systems may gain an exaggerated intensity leading, by analogy to motor syndromes of disinhibition, to a sort of spasticity of pain.

Akio Sato: The Somatosympathetic Reflexes: Their Physiological and Clinical Significance

Sato has employed current electro-physiological techniques to analyze reflex discharge in the white rami and lumbar sympathetic trunk evoked by stimulation of the somatic afferent fibers in the sciatic nerve in experimental animals. Reflexes evoked by stimulation of myelinated somatic afferents had central pathways traversing spinal, medullary and supramedullary levels; those evoked by stimulation of unmyelinated somatic afferents involved only spinal and medullary levels. The amplitude of spinal reflex discharge was largest when afferent volleys entered the same or adjacent segments of the cord corresponding to the white ramus under study.

By contrast, the amplitude of reflex discharge involving medullary or higher centers was independent of the segmental level of spinal input. In generalization, somatic afferent stimulation has a twofold action on the sympathetic system: a generalized action via supraspinal centers and a more circumscribed segmental action on preganglionic neurons.

In further experiments, Sato has interrelated electro-physiological data on somatic-visceral reflexes with analysis of recorded changes in the

affected visceral organs: Increase in heart rate, inhibition of gastric peristalsis, and increase in bladder pressure, on nociceptive stimulation of thoracic, abdominal and perineal skin.

These studies are establishing the physiological bases for determination in man of the visceral consequences of spinal syndromes for which manipulative therapy is advocated.

Edward R. Perl: Pain: Spinal and Peripheral Nerve Factors

Perl's elegant survey of the peripheral and spinal substrates of pain points out that every tissue likely to be exposed to unusual stress and noxious circumstances in ordinary life, has been found to contain sense organs with features of nociceptors.

Although pain is ordinarily initiated by activation of these specific receptors, whose major characteristic is their high threshold to all kinds of stimuli, Perl calls attention to the pronounced functional lability both of these receptors and their central projections, which display remarkable sensitization and hyperalgesia in the presence of inflammation after tissue damage, as well as during pain-induced reflex contraction of muscles following musculoskeletal injury.

Given this situation, Perl concludes, one should expect that the sensation of pain can be a variable reaction and that many forms of physical and psychological manipulation may alter it.

Sidney Ochs: A Brief Review of Material Transport in Nerve Fibers

Ochs, who has himself contributed much of the current information in this field, points out that the nerve cell body is presently regarded as the major site of synthesis of neural proteins and other metabolites, which are conveyed centrifugally through the axonal and dendritic branches of neurons to their receptor, presynaptic and postsynaptic, or neuro-effector terminals.

Ochs' studies of this internal transport system have recorded the advancing patterns of labeled activity along the sciatic nerves of animals, after injection of ^{3}H-leucine or ^{3}H-lysine into the dorsal root ganglia or anterior horns of the lumbar spinal cord. The characteristic rate of transport, between 410 and 450 millimeters per day, is independent of molecular weights of materials, diameters of nerve fibers, types or lengths of nerve, or species of animals; indicating a common carrier or conveyor-belt type of mechanism. Microtubules appear to form integral elements and the transport filaments involved require energy, presumably supplied by ATP, and are dependent upon local oxidative metabolism along the length of the fiber.

Reciprocally, a centripetal transport mechanism enables some signal substances to ascend the fibers and provide feedback control regulating the level of protein and other synthesis in the cell body. This retrograde transport proceeds at a rate of 220 millimeters per day, about half that of the outward transport system. In addition, some mechanism exists for the internal routing of transported material among different types of neuronal branches. For example, after injecting tagged material into a dorsal root ganglion, the peripheral branches ending in receptors display a 3–5 fold greater amount of labeled activity than do the branches proceeding into the spinal cord and ending centrally in presynaptic terminals.

Major current attention is directed also to CNS axoplasmic transport mechanisms influencing the amounts and types of neuro-transmitter related materials delivered to the presynaptic terminals of dopamine, noradrenaline, and serotonin fiber systems; as well as to their postsynaptic receptor sites. Advances in this area have already found clinical application in the introduction of L-Dopa as a therapeutic agent for Parkinson's disease. Further knowledge will doubtless lead to understanding of other, now obscure disorders and, in turn, to the development of means of treating additional diseases of nerve, muscle, spinal cord, and brain.

David Pleasure: Nerve Root Compression: Effects on Neural Chemistry and Metabolism

In his introduction, Dr. Pleasure points out that nerve compression of relatively low intensity causes segmental demyelinization, characterized by slowing or blocking of conduction in the injured segment. To restore normalcy, all that is required is new myelin synthesis in the segment involved and this is, ordinarily, a rapid and successful process. Although this situation is probably much more frequent than Wallerian degeneration, as a cause of neural disability after nerve compression, such segmental demyelination has not yet been studied biochemically.

Dr. Pleasure goes on to point out that, with each episode of demyelination, Schwann cells divide and there is deposition of interstitial, basement-membrane collagen in the endoneurium. With

excessive recurrence, this may lead to an exaggerated concentric proliferation of Schwann cells and collagen around central denuded axons.

Under normal circumstances, each of the lipids and proteins in myelin has a discrete half-life and steady states are maintained by the synthesis by Schwann cells at a rate adequate to match natural losses. Segmental demyelination occurs if this synthesis of myelin constituents falls below the normal catabolic rate; or if destruction of myelin exceeds the capacity of Schwann cells to provide rapid replacement, as in traumatic injury or even nerve compression.

When a segment of nerve is compressed, blood flow is reduced and, if sufficiently severe, Schwann cell ischemia may depress synthesis of myelin lipids and proteins. In fact, however, demyelination after acute compression occurs only at the upper and lower borders of the compressive force, with sparing of the central zone. This suggests that some feature of local deformation of pressure gradients, of a mechanical, rather than an ischemic nature may be responsible for acute, compression-induced, segmental demyelination.

Considered more generally, overall viability of Schwann cells and the myelin sheaths they have generated around nerve fibers, both in and outside the central nervous system, may be significantly contributed to, or trophically influenced by, or under the control of, their parent cell bodies. Correspondingly, the sheath cells and their products may be dependent upon axoplasmic transport of substances moving out of the neuronal perikaryon, in much the same way that activities at nerve fiber terminals have been shown to be so influenced by Sidney Ochs. Further studies of segmental demyelination following nerve or nerve-root compression may lead to measures that would reduce the incidence of long-term disability following such trauma. Such studies may even provide more fundamental insights that, hopefully, would contribute to a therapy for such major demyelinating diseases of the nervous system as multiple sclerosis.

Summary of General Discussion: What Do the Basic Sciences Tell Us About Manipulative Therapy?

HARRY D. PATTON
Professor and Chairman
Department of Physiology and Biophysics
School of Medicine, University of Washington

The discussion focused mainly on the effect of mechanical forces on nerve trunks, with some discussion of the mechanisms of mechanoreceptors. It was pointed out that in controlled experiments sustained mechanical forces on axons or nerve trunks block rather than excite; this is true whether the force is directed transversely (compression) or longitudinally (stretch). An exception is that transient rapid mechanical distortions (either longitudinal or transverse) depolarize both nerve trunks and mechanoreceptors and generate bursts of impulses usually of short duration. The blocking action of mechanical forces is inconsistent with the basic theory of spinal manipulation, which is aimed at relieving chronic compression or stretching of nerve roots, believed to be the underlying cause of pain. Several suggested explanations were offered, viz., that vascular pulsations cause repeated transient mechanical stimuli to compressed or stretched roots, that receptors in dura, ligaments or other integuments may be the source of pain impulses, or that inflammation rather than mechanical forces per se may be responsible for sustained neural discharge. It was pointed out that hypocalcemia renders axons sufficiently excitable to respond repetitively to mechanical stimuli, an observation which, though true, is of questionable relevance to the problem of low back pain. There was general agreement that further investigation of the response of nerve roots to chronic stretches or compression is needed and that such studies should attempt to create experimental conditions mimicking those believed to be present in spinal subluxations.

Chapter V.

What Do the Clinical Sciences Tell Us About Manipulative Therapy? (Subluxation: Pathophysiology and Diagnosis)

Henry G. West, *Chairman*

The Pathophysiology of the Spinal Subluxation

SCOTT HALDEMAN
Vancouver, B.C., Canada

The term "subluxation" has been used by members of the healing arts since the time of Hippocrates.[1] It has been referred to extensively by classical[2] and modern[3,4] medical practitioners, chiropractors[5,6] and osteopaths.[7,8] However, despite its common usage, the varying concepts of what constitutes a subluxation and the clinical significance ascribed to it by the different professions has been a major source of controversy which has hindered interprofessional discussions.

The earliest English definition of a subluxation appears to be that of Randle Holme in 1688[9] who described it as "a dislocation or putting out of joynt." In the literature both gross dislocations with separation of joint surfaces and very minor misalignments of adjacent articulations have been referred to by this term.

In this paper the definition of the subluxation proposed by the American Chiropractic Association will be used: "A subluxation is the alteration of the normal dynamics, anatomical or physiological relationships of contiguous articular structures."[10] This definition has the advantage of including most of the concepts used by the various health professions and subspecialities involved in the treatment of skeletal disorders. In addition it makes no reference to specific symptomatology and gives no pathophysiological significance to the subluxation, which may therefore be considered a clinical finding rather than a pathological entity. The significance of this finding is dependent upon the degree and type of joint dysfunction, its etiology and more especially its symptomatology and importance in initiating further pathology.

CLASSIFICATION

The spinal subluxation, as it is currently used, is not considered a single entity. The complex biomechanics of the spine allows for numerous

TABLE 1

The classification of spinal subluxations as determined by x-ray (from Basic Chiropractic Procedural Manual.)[10]

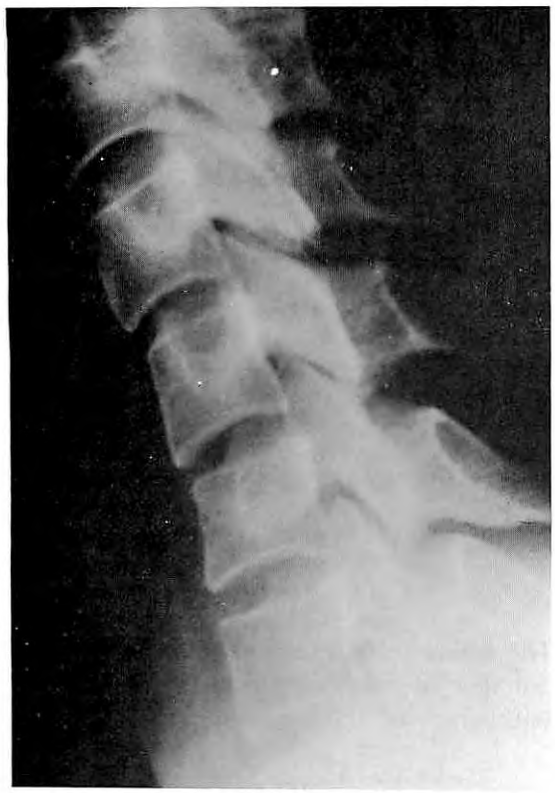
A. STATIC INTERSEGMENTAL SUBLUXATIONS
1. Flexion malposition
2. Extension malposition
3. Lateral flexion malposition
4. Rotational malposition
5. Anterolisthesis (spondylolistheses)
6. Retrolisthesis
7. Laterolisthesis
8. Altered interosseous spacing (decreased or increased)
9. Osseous foraminal encroachments

B. KINETIC INTERSEGMENTAL SUBLUXATIONS
1. Hypomobility (fixation subluxation)
2. Hypermobility (loosened vertebral motor unit)
3. Aberrant motion

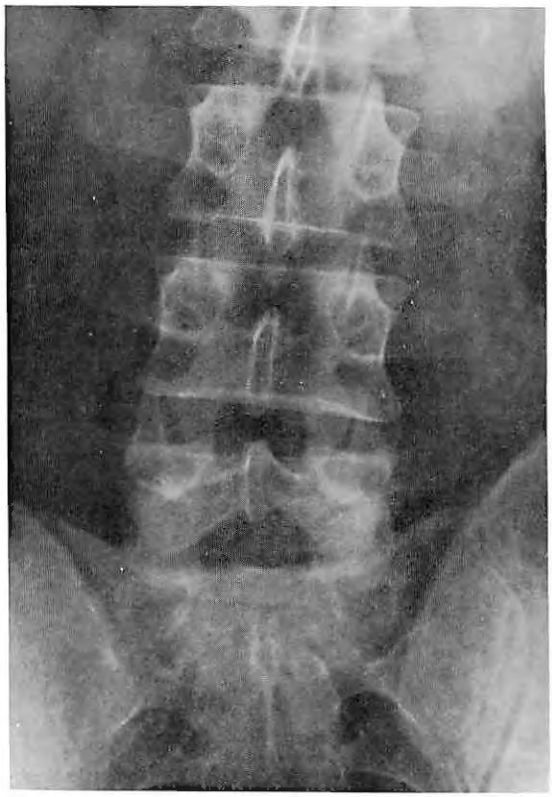
C. SECTIONAL SUBLUXATION
1. Scoliosis and/or alterations of curves secondary to muscular imbalance
2. Scoliosis and/or alterations of curves secondary to structural asymmetries
3. Decompensation of adaptational curvatures
4. Abnormalities of motion

D. PARAVERTEBRAL SUBLUXATIONS
1. Costovertebral and costotransverse disrelationships
2. Sacroiliac subluxations

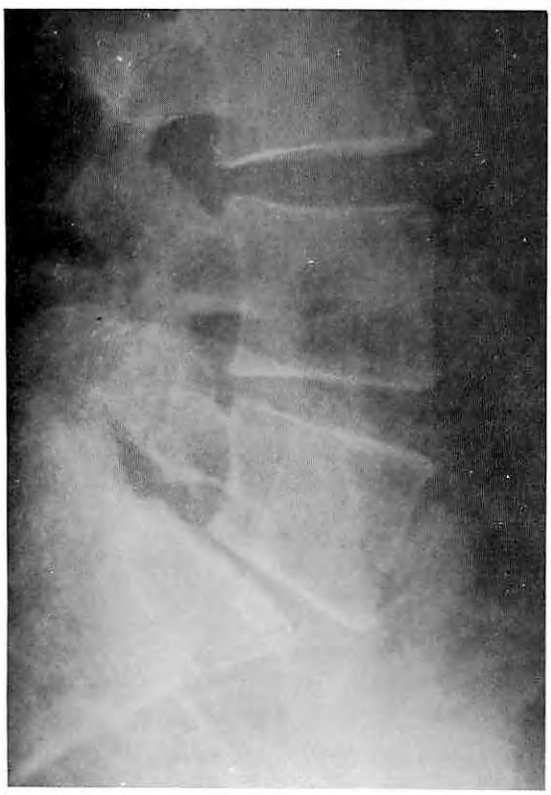
597-036 O - 76 - 15



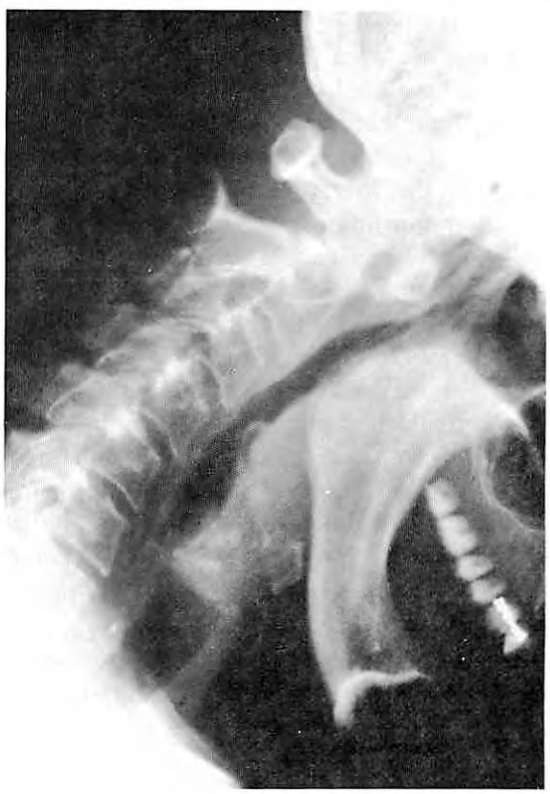
A



B



C



D

Fig. 1. Examples of static spinal subluxation.

subclassifications of intersegmental disrelationships. Table 1 lists one system of classification based on X-ray findings. This system includes descriptive terms for minor and major misalignments, kinetic, sectional, and paravertebral disrelationships.

In addition to the above-mentioned mechanical classifications a number of adjectives have been used to differentiate subluxations on clinical grounds. These descriptive terms may be divided into the following categories:

1. According to Etiology
   - Traumatic
   - Inflammatory
   - Postural
   - Psychosomatic
   - Congenital
   - Reflex
2. Primary, secondary or compensatory
3. According to Symptoms
   - Asymptomatic
   - Compressional
   - Painful
   - Fixated
   - Hypermobile
   - Irritable (suggesting abnormal visceral reflexes)

Figures 1 and 2 present several examples of subluxations which may be demonstrated on X-ray. 1-A shows a flexion misalignment of traumatic origin of C-4 on C-5; 1-B a lateral flexion misalignment associated with a disc lesion of L-4 on L-5. This film also shows an abnormal Hadley's S line on the left; 1-C demonstrates a spondylolisthesis with decreased interosseous spacing and a defect in the pars interarticularis, and 1-D a hypermobile subluxation of the atlanto-axial articulation found commonly in rheumatoid arthritis. Figure 2 shows a normal cervical spine in the upright position (2-A) but with aberrant movement of the atlanto-occipital region on neck flexion (2-B).

DIAGNOSIS AND IMPORTANCE IN SPINAL ADJUSTING AND MANIPULATION

The existence and clinical significance of the spinal subluxation is determined by the procedures listed in table 2. X-ray examinations have been the primary tool for confirmation of the subluxation and the introduction of motion X-ray studies has led to a tremendous expansion in the knowledge

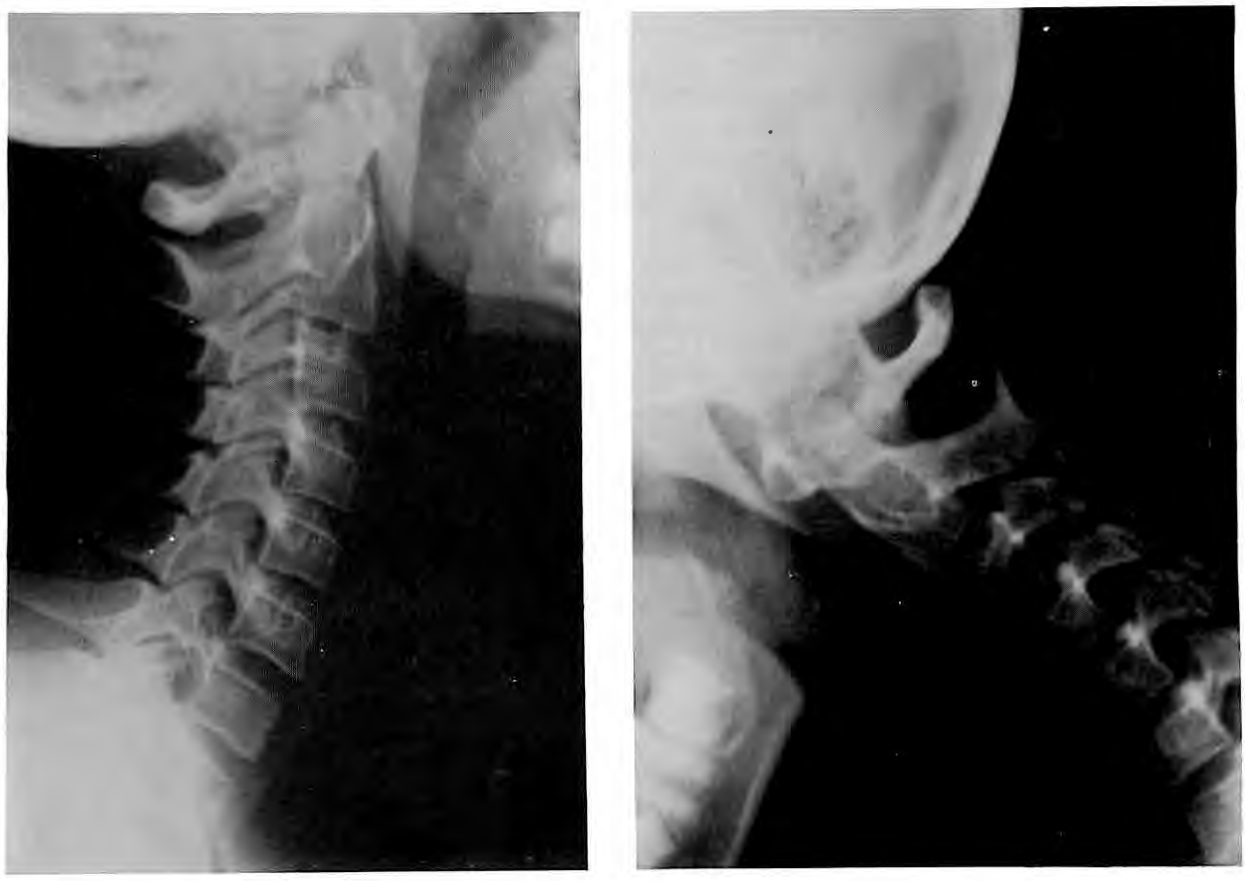
TABLE 2

Procedures Used in the Diagnosis of the Spinal Subluxation

1. HISTORY
   (a) Trauma or inflammatory disease
   (b) Symptoms
2. GENERAL EXAMINATION
3. OBSERVATION
   (a) Deformity
   (b) Posture
   (c) Motion
4. PALPATION
   (a) Static deformity
   (b) Abnormal mobility
   (c) Tenderness and muscle spasm
5. X-RAYS
   (a) Static
   (b) Motion
6. SPECIFIC INSTRUMENTATION
   (a) Postural analysis
   (b) Autonomic nervous system monitoring

of joint disrelationships, and has increased the number of classifications of the subluxation.[4,11] Static and motion palpation in conjunction with the other procedures in table 2 has been and remains, the primary tool for day-to-day assessment of joint relationships and the determination of the clinical significance of the subluxation.[12,13]

This entity has additional importance in the practice of spinal manipulation in that the specific adjustive thrust and the general techniques of spinal manipulation are attempts to reduce positional abnormalities,[5] increase movement at a "locked joint"[14] or the "reduction" of disc lesions.[15,16] Each of these procedures requires movement between vertebral segments to achieve their goal. Therefore, the ease with which an adjustment is given is dependent upon a complete understanding of normal spinal anatomy and mechanics, as well as a thorough examination for anomalies and subluxations in the spine being adjusted. Certain subluxation (e.g. the hypermobile segment in rheumatoid arthritis) are considered contraindications for manipulation. Failure to perform an adequate examination and to take this type of spinal subluxations into account could lead to serious complications.[15,16]



A B

Fig. 2. An example of an aberrant movement subluxation of the atlanto-occipital articulation.

CLINICAL SIGNIFICANCE

The determination of the clinical significance of the spinal subluxation has been clouded by the large number of widely varying, and in many cases, diametrically opposed opinions on this subject which have often been dogmatically adhered to without adequate investigation. The exact clinical significance remains difficult to determine because of the great diversity in its etiology, the complex nature of the subluxation and the comparative lack of research.

To date the only relevant research has been in the field of preemployment examination and workmen's compensation studies on back injuries. A few pertinent statistics from these studies are listed in table 3. From these figures it would appear that:

a. There is a slightly higher incidence of spinal abnormalities and subluxations in patients with back problems than those without problems.
b. There is no one type of subluxation or abnormality which is associated with all forms of back pain.
c. The presence of an abnormality does not mean that the patient is at present suffering from back pain.

To date no conclusive studies have been done to determine the significance of minor misalignments in any symptom of spinal origin.

The incidence and cost of these back problems in the populations of Western Nations is so high that further research is imperative. Nagi et al.[22], in an investigation of the general population, found that 18 percent of people questioned in a survey had persistent symptoms of backache while Hirsch et al.[23] found that 30–50 percent of women in a Swedish study suffered from back pain. It is well known that back injuries are one of the major causes of industrial time loss.[24]

Apart from back pain, a large number of additional symptoms and pathology have been attributed to spinal derangements by individuals in each of the

TABLE 3

The incidence of structural or subluxogenic spinal abnormalities in patients with and without symptoms.

Defect	Asymptomatic patients (percent)	Symptomatic patients (percent)	Reference
1. Any spinal abnormality	59.4	81.0	Barton [17]
2. Congenital anomalies	41.1		Diveley and Oglevie [21]
3. Scoliosis	14.5	27.2	Barton [17]
4. Postural scoliosis	5.0		Diveley and Oglevie [21]
5. Decreased mobility	15.0	43.0	Mensor and Duvall [18]
6. Increased mobility	8.0	13.0	Mensor and Duvall [18]
7. Increased mobility		28.6	Morgan and King [19]
8. Instability		47.9	White [20]
9. Degenerative changes		77.3	White [20]
10. Negative x-ray findings	39.9	13.8	Diveley and Oglevie [21]

health professions.[6,8,12,25] Nerve root and spinal cord compression due to spinal dysfunction has been well documented.[4,15,16] The role of the spinal subluxation in the pathophysiology of visceral disorders, although not well documented, has been widely claimed by members of all professions who practice spinal manipulation.[6,8,25] The implications of these claims in the understanding of health and disease warrants the properly controlled physiologic, clinical, and statistical research necessary to determine their significance.

PATHOPHYSIOLOGY

It is impossible to list and discuss each of the numerous theories which have been proposed to explain the pathophysiology of the spinal subluxation and the results obtained through spinal manipulation. However, most of these theories relate to one or more of the following pathophysiological processes:

1. Intervertebral disc degeneration.
2. Posterior joint dysfunction.
3. Compression or stretch of vital structures such as the nerve roots, spinal cord, perispinal blood vessels and the sympathetic chain.
4. Abnormal somato-visceral reflex activity.

An attempt will be made to discuss the role of the subluxation in each of these processes.

1. DISC DEGENERATION

The intervertebral disc has been shown to function optimally, have the greatest resistance to stress and optimum mobility when the nucleus pulposis is located in the center of the disc, and the height of the disc is of normal proportions and equal in all dimensions.[26,27] The integrity of the disc appears to be dependent on the degree of hydration, the collagen and mucopolysaccharide content, and the amount of stress to which the disc is subjected.[27,28]

The etiology of disc disease is not yet fully understood. A number of factors appear to be involved. Postural stress, structural abnormalities, and autoimmune processes have been considered as possible predisposing factors.[28,31] Normal aging has been shown to reduce the water and mucopolysaccharide content while increasing the amount of collagen in the disc.[29,30] In the presence of these changes, and perhaps even in their absence, the end plates and annular fibers can rupture when sufficient compressional or rotational force is applied to the disc.[26,27] The rupture of these fibers then leads to fissures in the annulus, the protrusion of the nucleus and the production of symptoms. The symptoms of local and referred pain, muscle spasm and neurological deficit are due to irritation of pain sensitive soft tissue structures and the compression of the spinal cord or nerve roots by the bulging nucleus.[4,15]

The investigation of spinal subluxations has been used to determine the degree and stage of disc degeneration.[4] Although there is a great deal of doubt as to the value of static X-rays in the prediction of symptoms attributable to disc lesions,[15] an analysis of spinal position and movement is often included in examinations of spinal disc disease to determine the following four types of subluxations.

a. The Disc Height

The height of the disc is a reflection of the degree of hydration and since the water content decreases with age and degeneration,[29,30] it can be used as an indication of these two factors. It is, however, a late sign of degeneration and normal disc spaces may be present in cases of severe disc degeneration.[27] Macnab's joint body line[4] (figure 3), has been used as a diagnostic criterion for disc thinning. He

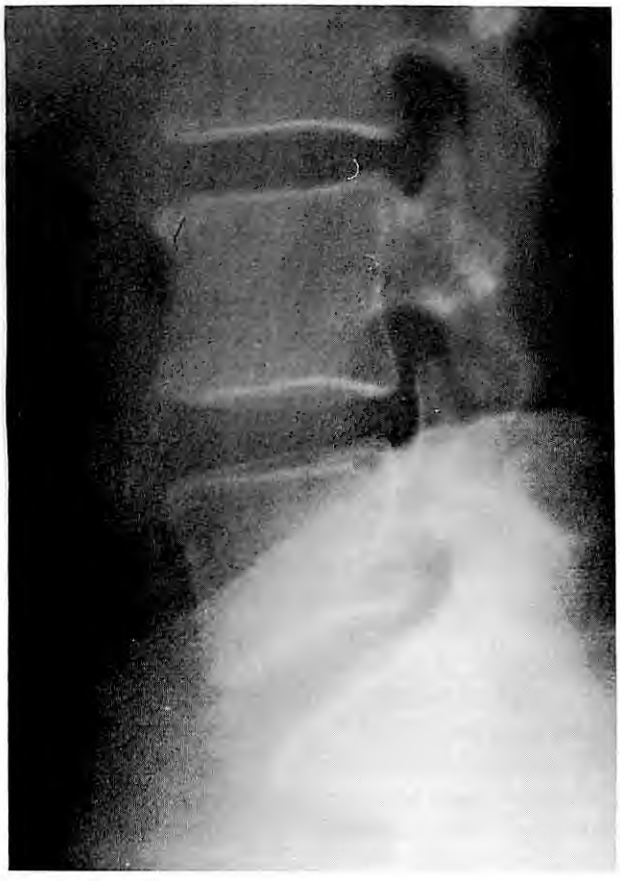


Fig. 3. An example of an abnormal Macnab's joint body line, of the L–4 : L–5 articulation.

feels that in the absence of disc degeneration the tip of the superior articular facet should not reach a line extending backward from the undersurface of the vertebral body above. It is possible, however, to have almost total obliteration of the disc space with no symptoms whatsoever.[31]

b. The Disc Wedge (flexion, extension and lateral flexion)

Nachemson[32] showed that a compressional force placed on a normal intervertebral disc is transmitted via the nucleus evenly throughout the annulus. It has been suggested[16,28] that a wedged disc or maldistribution of forces can cause a shearing effect, thus increasing the wear and tear forces on the disc. Ritchie and Fahrni[33] found that the number of lamellae in the annulus is the same at all points around the disc, but that in the lumbar region these fibers were distorted and compressed by the effect of posture on the secondary curve, thus causing a thickening of the anterior portion of the annulus.

It is possible that the displacement of the nucleus found in scolioses[26] is also a result of an abnormal distribution of forces on the disc. Lindblom[34] investigated the effects of this type of stress on the intervertebral discs in rats' tails. He found that when the tail was fixed in flexion, producing a compression on one side of the disc, degenerative changes and annular ruptures could be produced.

Clinically there is some correlation between disc wedging and disc disease. Most disc lesions occur in the cervical and lumbar regions of the spine, where the greatest degree of physiological disc wedging occurs. Drum[16] has stated that certain spinal configurations such as hyperlordosis are particularly prone to disc disease, while Wiltse[31] feels that there is a high incidence of disc disease in lumbosacral tilt subluxations.

c. Rotational Subluxation

In an extensive study, Farfan[26] found that normal rotation in the lumbar spine, at least in the cadaver, is associated with a forward tilt, which increases the distance between the posterior margins of the vertebral body, thereby stretching the posterior lateral annulus. Excessive rotation was found to result in a bulging in one part of the disc with a depression of another part. The maximum degree of distortion occurred most commonly at the posterolateral angles of the disc, which are the most common sites of disc degeneration. There was also separation of the annular filaments which resulted in a disc which had lost its stiffness and which yielded to touch. On removal of the torsion stress there was an incomplete return of the vertebral segment to the neutral position. If similar events occur in vivo, a chronic rotational unstable subluxation could be expected to remain following such an injury.

d. The Hypermobile Subluxation or Unstable Segment

Knutsson[35] has stated that the first radiological sign of disc disease is abnormal motion on flexion from the upright position. A number of other abnormal movements have been described by Macnab[4] who attributes them to degenerative changes such as inspissation of the nucleus pulposis, tear of the annulus or rupture of the hyaline cartilage plate. Drum[16] in a review of the literature illustrated a number of the major signs of instability (figure 4) which have been described by Hadley[36]

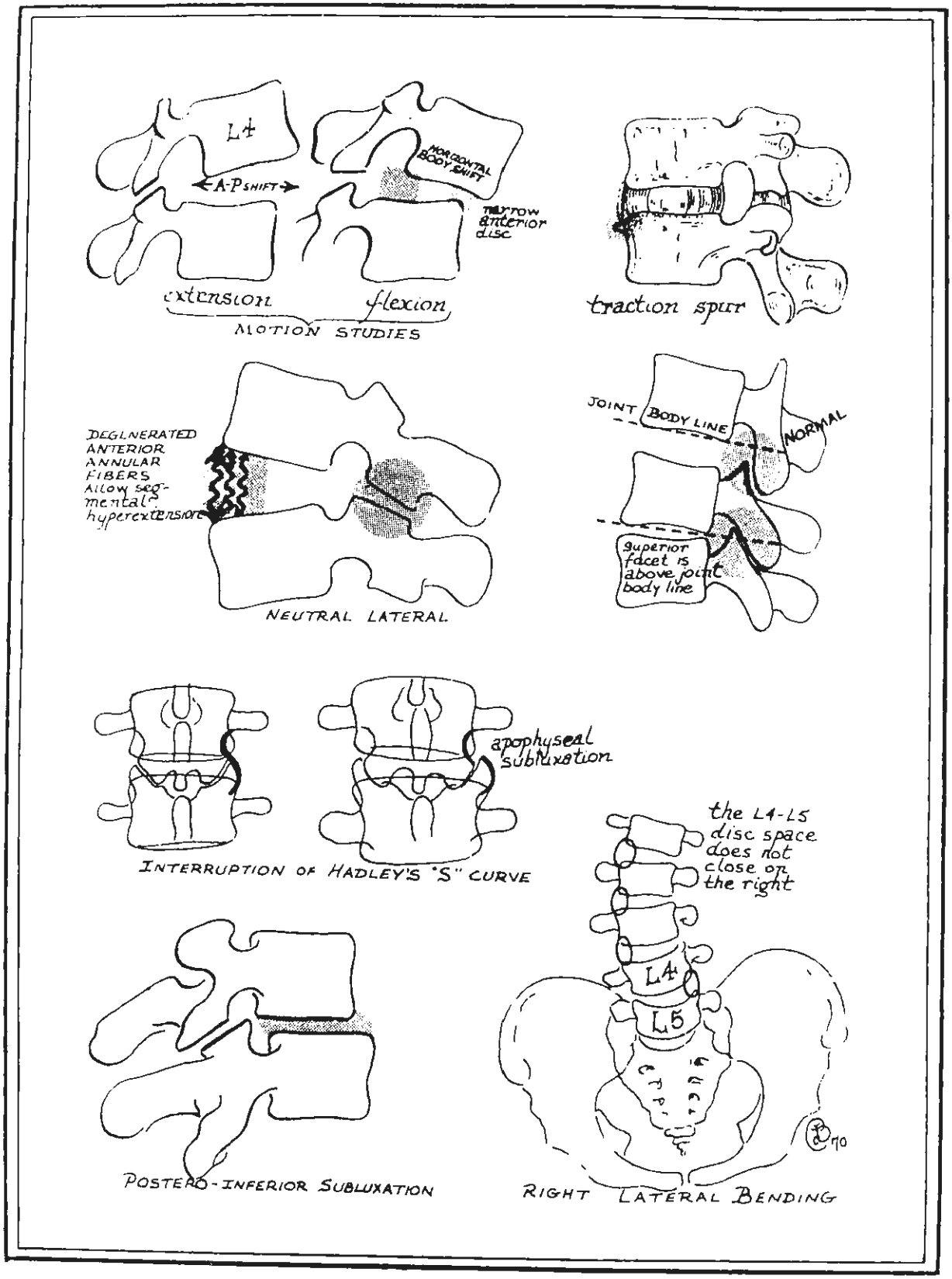


Fig. 4. Diagrammatic illustrations of some of the signs of segmental instability (by permission from Drum [16].

Macnab[4] and others. Macnab[4] attributed most of the pain of disc lesions to repetitive ligamentous strain due to chronic hyperextension of the posterior joints and the resulting posterior joint arthritis. Farfan[26] feels that the more advanced changes which include the formation of marginal osteophytes, degenerative facet changes and pseudospondylolisthesis found in disc disease are also due to abnormal mechanical stresses. Both of these authors consider these changes to be due to instability of the vertebral segment.

2. POSTERIOR FACET DYSFUNCTION

It has become well accepted that disc disease is not the only cause of back pain. In the search for additional sources of back pain, the posterior facets and their associated ligaments have been repeatedly implicated,[14,37] since both of these structures have been found to have a rich sensory innervation.

Mennell[14] has stated that these joints can be injured by active inflammatory disease, or trauma in the same manner as other diarthrodical joints. Abnormal joint relationships may be the result of such injuries or disc disease, and at the same time may decrease the resistance of the joint to injury. Vertebral tilting as seen in disc wedge subluxations changes the relationship of the two articular surfaces resulting in a change in the direction of the compressional forces on these joints. Rotation, on the other hand, causes a jamming or compression of the facets on one side, with opening of the joint space on the other.[26] Experiments by Gritzka et al.[38] have shown that continuous compression which one would expect in these situations, when applied to a peripheral joint which is still mobile and active, can cause erosion of joint cartilage with total destruction of the chondrocytes followed by arthritic changes.

The hypomobile subluxation, fixation or joint locking is considered an important cause of spinal dysfunction.[12,14,37] Mensor and Duvall[18] found that 43 percent of persons with back pain have decreased movement in the lumbar spine, compared with 15 percent of asymptomatic persons. Goldthwaite[39] and Steindler[40] were both of the opinion that this type of restricted motion in a section of the spine made it more susceptible to injury. This might be due to the fact that fixation of any diarthrodial joint for a prolonged period of time results in shortening of the ligaments and eventually degeneration of the joint cartilage.[41] On the other hand, restricted movement at one segmental level may result in an increased strain on joints of adjacent segments.

3. THE COMPRESSION OF VITAL STRUCTURES

The significance of the spinal subluxation as a cause of compression or interference with vital organs such as the spinal cord, nerve roots, blood vessels and the sympathetic chain has been the subject of a great deal of debate.

Nerve root compression has been the major focus of the debate. However, since the anatomy, physiology, and pathology of the intervertebral foramen and nerve root compression have been discussed in the papers presented by Drs. Drum, Sunderland, Schaumberg, and Sharpless, further comment in this paper would be repetitious.

Because of its anatomical position, the spinal cord is vulnerable to compression by vertebral misalignments. However, it would appear that only gross traumatic,[42] rheumatoid subluxations[3] or fracture-dislocations[42] which are not amenable to manipulation can cause sufficient compromise of the vertebral canal to cause compression of the cord. In rare instances posterior disc protrusions may also be so large as to cause cord compression.[15]

The vertebral artery which passes through the foramina transversaria, over the atlas and through the foramen magnum may be compressed by mechanical distortion of the cervical spine.[43] In the presence of anomalous arteries or severe arteriosclerosis this compression has been known to cause cerebral ischemia with severe neurological deficit.[44] This fact is well recognized and precautions are taken against aggravation of these symptoms by persons actively engaged in manipulation.[12,44]

Both Palmer[5] and Kunert[25] have suggested that the sympathetic chain ganglia which lie immediately adjacent to the costovertebral articulations may be irritated mechanically by lesions in the region of the thoracic spine and the heads of the ribs. Kunert bases this theory on the observation that arm movements and body rotation can cause tension on the sympathetic chain. If this is true, then the costovertebral or rotational subluxations may be capable of interfering with the sympathetic chain and cause either blockage of impulses or irritation of the sympathetic nerves. Unfortu-

nately, very little research has been done on this subject. The fact that severe chronic scolioses do not appear to be associated with known sympathetic dysfunction suggests that at least the chronic subluxation is not associated with this type of interference.

4. ABNORMAL SOMATO-SYMPATHETIC REFLEXES

The physiology and significance of somatosympathetic reflexes has been discussed by Dr. Sato. The possibility that abnormal somatosympathetic reflexes may be initiated by musculoskeletal lesions has been actively researched by Korr and his colleagues [45] who feel that there is significant correlation between cutaneous sympathetic activity and these lesions.[46, 47] He has postulated that somatic lesions create a "central excitatory state" in the spinal cord by stimulating local receptors and neurones thus causing bombardment of central neurones with afferent impulses.

The clinical evidence supporting this theory concerning spinal lesions is primarily indirect and based on the correlation of physical symptoms with spinal lesions. For example, Ushio et al.[48] showed that vertigo following a spinal injury could be relieved by applying a corset to the lumbar spine and suggested that overactivity of lumbar proprioceptors may be the mechanism involved, while Maigne [12] described cases of Barre-Lieou syndrome (headache, tinnitis, vertigo, hoarseness, facial flushing, lacrimation etc.) which responded to spinal manipulation. A large number of similar observations have been made by other practitioners of spinal manipulation,[5, 8] but unfortunately no controlled studies have been carried out to eliminate placebo effects and to determine the frequency with which these results occur.

The role of the subluxation in the production of abnormal somato-sympathetic reflexes has yet to be established. However, by definition, the subluxation is an abnormality in position or movement, and in clinically significant situations may be associated with ligamentous and discal injury, joint inflammation and muscle spasm. Since the tissues affected by these processes have a rich sensory innervation [49] it seems possible that any acute subluxation would result in an increased sensory input to the spinal cord, which in turn could result in the "central excitatory state" referred to by Korr.

SUMMARY

As it is currently used, the term spinal subluxation describes a valid clinical finding, the significance of which is dependent on symptoms and signs of local pathology. Since it is used to describe any mechanical or functional derangement between adjacent vertebrae there are a large number of subclassifications of the subluxation. It has been intimately associated with the pathophysiology of intervertebral disc disease, posterior joint dysfunction, the compression of vital perispinal structures and abnormal somato-visceral reflexes. For these reasons an understanding of and the ability to diagnose the subluxation is important in the practice of spinal manipulation.

REFERENCES

1. Adams, F.: *The Genuine Works of Hippocrates.* Translated from the Greek. Vol. 2. Printed for the Sydenham Society (London) 1849.
2. Hieronymus, J. H.: *De Luxationibus et subluxationibus* Ritter. (Jena). 1746.
3. Bland, J. H.: Rheumatoid arthritis of the cervical spine. *J. Rheumatology* 1:319–342, 1974.
4. Macnab, I.: Pathogenesis of symptoms in discogenic low back pain. Chapter 6 in *American Academy of Orthopaedic Surgeons Symposium on the Spine.* C. V. Mosby Co. (St. Louis) 97–110, 1969.
5. Palmer, D. D.: *The Science, Art and Philosophy of Chiropractic.* Portland Printing House Co. (Oregon). 1910.
6. Janse, J.: The vertebral subluxation. *J. National Chiropractic College.* 20, 1:3–7, 1948.
7. Laskei, D. L.: *Principles of Osteopathy:* Third Edition, 1913.
8. Hoag, M., Cole, W. V., and Bradford, S. G.: *Osteopathic Medicine.* McGraw-Hill Book Co. (New York), 1969.
9. Holme, R.: *Academy of Armory.* Printed in Chester by the author. 1688. Reprinted by The Scolar Press Limited in 1972. (Menston, England) 448.
10. Inman, O. B.: Editor; *Basic Chiropractic Procedural Manual.* Printed by the American Chiropractic Association. 1973.
11. Howe, J. W.: The chiropractic concept of subluxations and its roentgenological manifestations. *J. Clinical Chiropractic.* 64–70, September 1973.
12. Maigne, R.: *Orthopedic Medicine—A new approach to vertebral manipulations.* Translated and edited by W. T. Liberson. Charles C Thomas (Springfield, Ill.), 1972.
13. Johnston, W. L.: Segmental behavior during motion. *J.A.O.A.* 72:352–373, 1972.
14. Mennell, J.: *The Science and Art of Joint Manipulation.* Vol. II: The Spinal Column. J. and A. Churchill Ltd. (London) 1952.
15. Cyriax, J.: *Textbook of Orthopaedic Medicine.* Vol. I: Diagnosis of soft tissue lesions. Bailliere, Tindall and Cassell (London) 1969.

16. Drum, D. C.: *An introduction to the study of posture and spinal mechanics.* Published by the author. (Toronto), 1970.
17. Barton, P. N.: *The significance of anatomical defects of lower spine.* Ind. Med. 17:37–40, 1948.
18. Mensor, M. C., and Duvall, G.: Absence of motion at the fourth and fifth lumbar interspaces in patients with and without low back pain. *J. Bone Jt. Surg.* 41–A:1047–1054, 1959.
19. Morgan, F. P., and King, T.: Primary instability of lumbar vertebrae as a common cause of low back pain. *J. Bone Jt. Surg.* 39–B:6–22, 1957.
20. White, A. W. M.: Low back pain in men receiving workmen's compensation. *Can. Med. Assn. J.* 95:50–56, 1966.
21. Dively, R. L., and Oglevie, R. R.: Preemployment examinations of the low back. *J.A.M.A.* 160:856–858, 1956.
22. Nagi, S. Z., Riley, L. E., and Newby, L. G.: A social epidemiology of back pain in a general population. *J. Chron. Dis.* 26:769–779, 1973.
23. Hirsch, C., Jonsson, B., and Lewin, T.: Low back symptoms in a Swedish female population. *Clin. Orthopaedics and Related Research.* 63:171–176, 1969.
24. Kosiak, M., Aurelius, J. R., and Hartfiel, W. F.: Backache in industry. *J. Occup. Med.* 8:51–58, 1966.
25. Kunert, W.: Functional disorders of internal organs due to vertebral lesions. *Ciba Symposium:* 13–3, 1965.
26. Farfan, H. F.: *Mechanical Disorders of the Low Back.* Lea and Febiger (Philadelphia), 1973.
27. Armstrong, J. R.: *Lumbar Disc Lesions.* 3rd Edition. E. and S. Livingston Ltd., (Edinburgh), 1965.
28. DePalma, A. F.: and Rothman, R. H.: *The Intervertebral Disc.* W. B. Saunders Co. (Philadelphia), 1970.
29. Naylor, A.: The biophysical and biochemical aspects of intervertebral disc herniation and degeneration. *Ann. Roy. Coll. Surg. Eng.* 31:91–114, 1962.
30. Puschel, J.: Der Wasserergehalt normaler und degenerierter zwischenwirbelscheiben. *Beitr. Path. Anat.* 84:123–130, 1930.
31. Wiltse, L. L.: Lumbosacral strain and instability. Chapter 4, in *The American Academy of Orthopaedic Surgeons* Symposium on the Spine. 54–83, 1967.
32. Nachemson, A.: Lumbar Intradiscal Pressure. *Acta. Orthop. Scand. Suppl.* 43:1–104, 1960.
33. Ritchie, J. H., and Fahrni, W. H.: Age changes in the lumbar intervertebral discs. *Can. J. Surg.* 13:65–71, 1970.
34. Lindblom, K.: Experimental ruptures of intervertebral discs in rats tails. *J. Bone Jt. Surg.* 34–A:123–128, 1952.
35. Knutsson, F.: The instability associated with disc degeneration in the lumbar spine. *Acta Radiol.* 25:593–609, 1944.
36. Hadley, L. A.: Apophyseal subluxation; disturbances in and about intervertebral foramen causing back pain. *J. Bone Jt. Surg.* 18:428–433, 1936.
37. Wright, J.: Mechanics in relation to derangement of the facet joints of the spine. *Arch. Phys. Ther.* 25:201–206, 1944.
38. Gritzka, T. L., Fry, L. R., Cheesman, R. L., and LaVigne, A.: Deterioration of articular cartilage caused by continuous compression in a moving rabbit joint. *J. Bone. Jt. Surg.* 55–A:1698–1720, 1973.
39. Goldthwaite, J. E.: The variations in anatomical structure of the lumbar spine. *Amer. J. Orthop. Surg.* 2:416–434, 1920.
40. Steindler, A.: *Mechanics of Normal and Pathological Locomotion in Man.* Charles C Thomas (Springfield, Ill.), 1935.
41. Hall, M. C.: Cartilage changes after experimental immobilization of the knee joint in a young rat. *J. Bone Jt. Surg.* 45–A:36–44, 1963.
42. Garber, J. N.: Fracture and fracture-dislocation of the cervical spine. Chapter 3 in *The American Academy of Orthopaedic Surgeons Symposium on the Spine.* 18–53, 1967.
43. Jones, R. T.: Vascular changes occurring in the cervical musculoskeletal system. *South African Med. J.* 40:388–391, 1966.
44. Kleynhans, A. M.: Vascular changes occurring in the cervical musculoskeletal system. *Bull. European Chiropractic Union.* 19–1:24–30, 1970.
45. Korr, I. M., Buzzell, K. A., Hix, E. L., Northup, G. W., and Kugelmass, I. N.: *The physiological basis of osteopathic medicine.* Published by The Postgraduate Institute of Osteopathic Medicine and Surgery (New York), 1970.
46. Korr, I. M., Wright, H. M., and Thomas, P. E.: Effects of experimental myofascial insults on cutaneous patterns of sympathetic activity in man. *Acta Neuroveg.* 23:329–355, 1962.
47. Korr, I. M., Wright, H. M., and Chace, J. A.: Cutaneous patterns of sympathetic activity in clinical abnormalities of the musculoskeletal system. *Acta Neuroveg.* 25:589–606, 1964.
48. Ushio, N., Hinoki, M., Hine, S., Okada, S., Ishida, Y., Koike, S., and Shizuku, S.: Studies on ataxia of lumbar origin in cases of vertigo due to whiplash injury. *Agressologie* 14–D:73–82, 1973.
49. Haldeman, S.: Spinal and paraspinal receptors. *J. Am. Chiropractic Assn.* 9:5:25–31, 1972.

Pathophysiologic Evidence for the Osteopathic Lesion. Data on What is Known, What is Not Known, and What is Controversial

J. S. DENSLOW
Vice President
Kirksville College of Osteopathic Medicine
Kirksville, Missouri

The term "Osteopathic Lesion" is generic and perhaps needs clarification. This term has been used for many years; its origin is obscure. Apparently it came into usage to represent a palpatory experience shared by those who worked with it. Manipulative Therapy directed to this disturbance has often proven beneficial to the patient.

"Somatic Dysfunction" has recently been offered as a substitute for Osteopathic Lesion for use in the Hospital-International Classification of Disease, Adapted (H-ICDA). This term is defined as "impaired or altered function of related components of the somatic (body framework) system: musculoskeletal, arthrodial, and myofascial structures, and related vascular, lymphatic, and neural elements."

There is persuasive evidence that the Osteopathic Lesion is similar to, and possibly identical with, such disturbances as trigger points in the body wall and upper extremities in myocardial disease, splinting and tension of the abdominal wall, flank and paravertebral tissues in intra-abdominal disease, the muscular aches and pains accompanying systemic infections such as influenza, etc.

Despite the fact that Osteopathic Lesions have been diagnosed and treated by many thousands of health professionals and that millions of patients have benefited from such diagnosis and treatment, the proof that such disturbances exist, and can be managed effectively, does continue to be on a subjective basis with regard to both the health professional and the patient. However, it is clear and is generally acknowledged that much of contemporary therapeutics is judged efficacious on subjective evidence alone.

In view of the brief time available, it is necessary to limit these comments to a few examples of "data on what is known, what is not known, and what is controversial." The bibliographies of the papers cited, however, will provide opportunity for a much broader review in this area than can be presented at this time.

WHAT IS KNOWN

The clinical evidence for the Osteopathic Lesion, which leads into a discussion of the Pathophysiologic Evidence is usually obtained by the physical examination methods of inspection and palpation, supplemented at times with such procedures as X-ray, electromyography, and thermography.

This clinical evidence includes:

a. Hyperalgesia (with or without pain)
b. An abnormality in the texture or tone of soft tissues
c. Anatomical Asymmetry*
d. Range and ease of joint motion

Hyperalgesia is identified clinically by the presence of tenderness to digital palpation in the area of the Osteopathic Lesion in contrast to the

* It is recognized, of course, and as Maigne [1] has pointed out, that misshapen bony prominences and the remote location of such prominences, must raise questions as to the validity of palpation in determining the presence or absence of segmental asymmetry.

absence of tenderness in response to similar palpation in a normal area.

Abnormality in the texture or tone of soft tissues is identified by light to moderate digital palpation. The areas of the Osteopathic Lesion may be described as being boggy or thickened non-muscle tissue and rigid muscle. It can be identified by the palpation methods similar to those described by Maigne[1] to identify disturbances in tissue texture and in the ease and range of joint motion.

Hyperalgesia and abnormal tissue texture will be discussed together. Hyperalgesia and abnormal tissue texture probably involve all nonosseous tissues, including the skin and the periosteum, and various tissues between the skin and the periosteum; namely, muscle, fat, various collagenous tissues (e.g. cartilage, fascia, ligaments and tendons) and the vascular, neural and lymphatic networks that are embedded in all of these tissues. The heterogeneity of these tissues is probably the most important single factor accounting for the current lack of objective diagnostic methods for differentiating normal from abnormal somatic tissues (in the way that ECG does for cardiac muscle, the X-ray for bone, and the EEG for the brain). Hence, while normal and abnormal somatic tissues can be differentiated by physical examinations, the precise nature of the abnormality still is not yet known.

The two characteristics of Osteopathic Lesion, hyperalgesia and abnormality of tissue texture, are of overriding importance. They represent a disturbance in local homeostasis which might be due (a) to pathophysiology within these tissues, that might have resulted from trauma, or (b) to changes induced by visceral, emotional, and other disturbances via the controlling and communicating systems (e.g. neural, vascular, lymphatic, hormonal) which govern, sustain, and determine the state of the somatic tissues involved.

Thus, these two characteristics provide direct information concerning the location and severity of the Osteopathic Lesion. The cause of this disturbance must be determined from the history and other diagnostic procedures.

Anatomical asymmetry and range and ease of joint motion will also be considered together. Kendall, Kendall and Boynton[2] have pointed out that, ideally, in the human erect stance, the center of gravity in the sagittal plane bisects the trunk including the pelvis and the vertebrae. Thus, there would be a symmetrical distribution of the body weight, one-half on one side and one-half on the other side of the center line of gravity. In the coronal plane, the anatomical curves would not permit a true bisection but would provide for symmetry with half the body weight anterior to and half posterior to the center line of gravity. Thus, balance in the upright position would depend heavily on weight distribution with a minimum of stress, and subsequent wear and tear, on articulating processes and on nonosseous supporting tissues.

Similarly, where there is symmetry in the sagittal plane, and normal anatomical curves in the coronal plane, normality in both the ease and range of joint motion would be expected. Conversely, asymmetry in the sagittal plane, and/or a diminution or exaggeration of the anatomical curves in the coronal plane would produce (i) abnormal stresses and strains on the cushioning and supporting tissues, and (ii) problems as regards the ease and range of joint motion.

Asymmetry in alignment and joint motion ranges are detected by inspection and palpation supplemented at times with radiography, particularly when done in the weight-bearing position. Methods for taking and interpreting such films have been described.[3]

The discussion of Pathophysiologic Evidence will start with our own work. It had been postulated that the abnormal tissue texture in Osteopathic Lesion was due, at least in part, to muscle contraction. Following the early work of Adrian and Bronk[4] which used electromyography to identify the single motor unit, and the work of Jacobson[5] who showed that resting skeletal muscle is electrically silent it was reasoned that, in normal areas, resting muscle would be relaxed and no muscle action potentials would be seen. Conversely, in areas of Osteopathic Lesions the finding of action potentials would show the presence of muscle contraction. The early work did bear out this hypothesis and the first publication concerning this work reported no muscle contraction in normal areas (as identified by palpation) and the presence of muscle contraction in many areas of Osteopathic Lesion (also identified by palpation).[6]

However, as more experience was gained with these methods and after additional studies reported by other workers, it was recognized that if sufficient care was used in bolstering the subject with pillows

and sandbags the EMG evidence of muscle contraction in some of the abnormal areas disappeared.[7] In other words, under those conditions, palpable abnormal tissue texture could not be due to muscle contraction.

However, another differentiating characteristic between normal areas and areas of Osteopathic Lesion was found. In normal areas the irritation produced by placing needle electrodes through fascia and into muscle frequently results in a burst of action potentials called "insertion potentials." This burst lasts for a brief period and subsides spontaneously; minor movements of the electrodes do not reinitiate it, and the muscle remains electrically silent. In contrast, in areas of Osteopathic Lesion, the insertion potentials persisted for longer periods of time; and, when they did subside, they could be reinitiated by minor electrode movement. Further studies revealed that a wide variety of stimuli, whether applied to the area of Osteopathic Lesion or remotely, would initiate action potentials in that area, unlike the normal area.

This observation suggested that the motoneurons supplying the areas of Osteopathic Lesion were in a state of enduring subliminal excitation, as was confirmed by the low reflex thresholds demonstrated in such areas.[8]

Later, methods were developed by Denslow, Korr, and Krems[9] to (a) quantitate levels of reflex activity and (b) to correlate reflex thresholds with various other segmental phenomena. Briefly, the following was found:

Normal areas (by palpation)	*Areas of osteopathic lesion (by palpation)*
High reflex threshold	Low reflex threshold (facilitation)
Paravertebral tissues	Paravertebral tissues
(a) Normal texture	(a) Abnormal texture
(b) No hyperalgesia	(b) Hyperalgesia
(c) No lasting soreness following minor trauma	(c) Lasting soreness following minor trauma

Probably the most important points to be made here are (a) that there is a direct correlation between the palpable tissue texture abnormality in Osteopathic Lesion and a state of chronic facilitation in the motoneuron pools in the spinal cord that are segmentally related to the Osteopathic Lesion, and (b) since the abnormal tissue texture may not be due to muscle contraction, some other phenomena must be sought to account for it.

The correlation between palpable tissue texture abnormality and other disturbances is comparable to a somewhat similar correlation made by Brendstrup, et al.[10] These investigators examined patients prior to surgery for herniated intervertebral discs. Normal areas and areas of "fibrositis" were identified by palpation. At the time of surgery, tissues from normal and fibrositic areas were taken for microscopic examination and chemical analyses. Although the series of cases was small (12) the findings from the control areas were normal; in contrast, there were positive findings in 10 of the 12 specimens from fibrositic areas. The positive findings included a decrease in potassium and an increase in chloride and hexosamine content (the latter indicated by an excess of acid mucopolysaccharides). Microscopic examination of the fibrositic tissues showed a widening of the interstices, indicating edema, an increased number of mast cells in the connective tissue and an increased number of nuclei in muscle fibers.

As regards fibrositis, in discussing pain thresholds, Procacci et al.[11] commented "In the pathological field: in subjects with fibrositis of the upper limb the cutaneous pain threshold is significantly lower in the limb itself. . . ."

Another possibility that might account for abnormal tissue texture, which is not due to muscle contraction, involves the observation that patients suffering from migraine (and so-called "tension") headaches show evidence of Osteopathic Lesion at the atlanto-occipital and upper cervical areas; the Osteopathic Lesion is severe during acute episodes and present, but less severe, during interim periods.

Dalessio[11] in discussing "Vascular Permeability and Vasoactive Substances: Their Relationship to Migraine" suggested ". . . that migraine is a clinical syndrome of self-limited neurogenic inflammation." He points out that:

> ". . . present evidence implicates at least five groups of vasoactive substances associated with inflammation: (a) catecholamines, (b) other bioactive amines (histamine and serotonin), (c) the peptide kinins, (d) the prostaglandins, which are fatty acids, and (e) SRS-A, an acidic lipid. These vasoactive substances all have potent biologic properties which differ with their structure and include, among others, contraction or relaxation of smooth muscle, constriction or dilation of arteries and veins, induction of water and sodium diuresis, fever, wheal and flare

reactions, and induction of pain, including headaches."

It seems possible, even probable, that this inflammatory process might account for the abnormal tissue texture that is seen in the absence of muscle contraction. The ". . . self-limited neurogenic inflammation" could very well be involved in clinical syndromes, other than migraine headache, including Osteopathic Lesion.

Still another possibility that might account for abnormal tissue texture in the absence of muscle contraction involves the trophic functions of the neuron,[12] discussed elsewhere in this Workshop. It is known, as regards muscle, that maintenance of axonal and junctional integrity between neuron and muscle cell is essential for "normal" trophicity. Axoplasmic flow, rather than impulse traffic, is clearly implicated in a variety of neurotrophic influences, such as those operating in morphogenesis, regeneration, control of genic expression and the maintenance of structural, functional and biochemical integrity of the innervated tissue.[13]

Chamberlain, Rothschild, and Gerard[14] have ". . . shown that a postural asymmetry in the hind limbs, induced by a unilateral cerebellar or vestibular lesion, will persist after mid-thoracic spinal cord transection, providing sufficient time is allowed for this asymmetry to 'fixate' in the cord before transection." Patterson[15] has discussed mechanisms involved in conditioning and in the fixation of functional patterns in spinal mammals. Surely such patterns of functional activity have some bearing on integrity, or lack of it, of the axonal transport mechanisms.

Related to the demonstration of facilitation in motoneuron pools of the voluntary side of the nervous system, Korr and his colleagues[16] have shown that similar phenomena occur in the sympathetic system. They studied regional and segmental variations of sympathetic activity by determining sudomotor and vasomotor activity. They found that in certain spinal cord

> ". . . segments, at least some of the neurons mediating sensory, motor, and autonomic function are maintained in a state of hyperexcitability, which they manifest in their easier, augmented, and prolonged responses to impulses reaching them from many sources, and are therefore susceptible to sustained and exaggerated activity under conditions of daily life.
>
> These segmental disturbances appear to be physiologic lesions related, by nature and location, to the clinical phenomena designated as osteopathic lesions."

This will be discussed further in connection with experimentally produced neuromusculoskeletal disturbances.

Over the years there have been a substantial number of reports of disturbances, or syndromes, in which somatic and visceral problems concur. The somatic problems often show strong resemblance to Osteopathic Lesion. Hence these disturbances should be discussed as part of the "Pathophysiologic Evidence." A few of these reports will be cited as examples.

Ruch et al.[17] comment:

> "Sustained contractions of skeletal muscle likely to cause pain may arise from higher centers or from reflexes of somatic or visceral afferents. Such reflexes are important (i) as diagnostic signs (Kernig's sign, stiff neck of meningeal irritation, abdominal rigidity of appendicitis), and (ii) as secondary sources of pain and discomfort.

They further comment:

> "The muscle contraction may be due to a vicious circle: deep pain—sustained reflex contraction—deep pain—reflex contraction—etc. The success of such single procedures as osteopathic treatments, ethyl chloride sprays and procaine hydrochloride injection of trigger zones may depend on the breaking of the circle."

It is well known by those who have studied the neuromusculoskeletal system carefully in patients with acute infectious diseases, for example in various types of pneumonia, that Osteopathic Lesions are present in the spinal area that is segmentally related to the affected organ or viscus; e.g. there is hyperalgesia, abnormality in tissue texture, and limitations in the ease of joint motion. In addition to relieving the hyperalgesia, abnormality in tissue texture and restrictions in the ease of joint movement, spinal manipulation often has been followed by concurrent improvement in the remote infection.

Speransky et al.[18] carried out well-controlled experiments, in patients with pneumonia, by reducing the related neuromusculoskeletal system

abnormality with a different form of therapy (somatic blockade by local anesthesia). Following a long series of studies on experimental animals, they suggested that treatment of pneumonia in man be directed at the cord segments involved. In patients suffering from lobar pneumonia, they injected the rhomboid area with procaine. They observed that this treatment, when given early, is usually followed by a drop in body temperature, resolution of the pneumonic consolidation, and improvement in the patient's general condition. One might speculate (a) that the muscular disturbance was the result of spinal cord hyperirritability initiated and sustained by afferents from the affected viscera, and (b) that when the feedback from the rigid musculature was moderated, the patient's ability to combat the original infection was improved, presumably by a breaking of the positive feedback circle suggested by Ruch et al.[17] Speransky suggests that "The rationale of this treatment is based on the presumed changes in the lung following restoration of normal nerve function."[18]

Many years ago, MacKenzie[19] called attention to a relationship between cardiac disease and somatic tissues via what he termed an "irritable focus" in the spinal cord. Since then numerous reports concerning this relationship have been published. Of these, two will be cited. Lindgren[20] studied patients with angina pectoris and other cardiac problems by inducing precordial pain with hypoxemia or exercise. She mapped the areas of referred pain and infiltrated them with local anesthetic. It was observed that this procedure lessened both the pain and the accompanying electrocardiographic abnormalities.

Rinzler and Travell[21] used somatic blockade in the management of angina pectoris, related cardiac problems and hyperalgesia in the chest wall; the latter may simulate cardiac problems, but may be due to somatic factors. They studied cardiac and somatic chest pain through the presence of what they called a trigger mechanism in the somatic structures. The trigger area is an abnormal zone of hypersensitivity which, when stimulated by digital pressure or needling, gives rise to a brief reference pattern of pain. They suggested that the essential part of the examination for trigger areas was the discovery, by careful palpation, of topographically discrete areas of exquiste somatic tenderness. At times the spot of hyperalgesia was so acute that when it was palpated a motor response followed, such as wincing or withdrawal. The trigger area was treated by infiltration of procaine or by spraying the overlying skin with ethyl chloride, with the result that in a large majority of cases the pain and disability occasioned by the disease, were eliminated or greatly lessened.

Judovich and Bates[22] in the monograph, "Pain Syndromes" direct attention to the large number of patients who suffer pain and disability due to unrecognized disturbances in the somatic system. They comment, ". . . segmental pain and tenderness may simulate the pain of visceral disease, and many patients who have submitted to medical treatment for visceral disease are not relieved of pain until treatment is directed to the somatic origin of pain and its cause."

The diagnostic methods they describe have much in common with the diagnostic methods used in the search for, and evaluation of, Osteopathic Lesion. They place considerable stress on structural asymmetry and imbalance (and related soft tissue stresses and strains) that are due to an inequality of leg length and that are responsive to the appropriate use of heel lift. They state, for example,

> "A slight shortening of one lower extremity will produce a lateral tilt of the pelvis. The lumbar spine swings to the short side and develops a compensatory scoliosis with strain at the dorsolumbar spine. This may be mild and is many times ignored, yet we have observed and corrected these mild postural changes by using a heel lift, and have often seen chronic pain disappear without using any other form of therapy."

Siehl[23] studied a series of patients who had sciatic pain. All of these patients had lumbar and lumbosacral Osteopathic Lesion and were suspected of having a herniated intervertebral disc. All were studied with electromyography for evidence of nerve root pressure. All received spinal manipulation. Subsequently, it was found that a high percentage of patients with normal electromyograms responded favorably to manipulation, while those with positive EMG evidence of nerve root pressure did not respond favorably, and required surgical intervention.

Another aspect of Pathophysiologic Evidence of Osteopathic Lesion is seen in disturbances that

have been produced experimentally. Lewis and Kellgren[24] point out that (a) stimulation of an interspinous ligament produces segmentally related pain, superficial and deep hyperalgesia and muscle contraction, (b) stimulating appropriate somatic structures produces pain similar to that of angina of effort or intestinal colic, (c) stimulating either spinal muscles or a viscus, such as the pancreas, produces contraction of the muscles of the abdominal wall and (d) visceral disease, and the stimulation of deep somatic structures, both produce segmentally related cutaneous hyperalgesia.

Likewise, Korr, Wright, and Thomas[25] also clearly demonstrated that certain characteristics of Osteopathic Lesion can be produced experimentally. These investigators irritated musculoskeletal tissues by hypertonic saline injections and produced postural stresses by the use of heel lifts and tilt chairs. They found that (a) when saline injection produced referred pain, there was the appearance of segmentally related areas of lowered electrical skin resistance, and (b) experimentally induced postural changes produced both an exaggeration of existing patterns and additional areas of lowered electrical skin resistance. They concluded that their observations suggest a relationship between patterned differences in sympathetic activity, as shown by lowered electrical skin resistance, and painful myofascial and visceral conditions.

WHAT IS NOT KNOWN

The identification of what is not known regarding Pathophysiologic Evidence of the Osteopathic Lesion represents a very extensive work in itself. However, three of the most critical areas will be identified.

1. Objective evidence concerning such things as the etiology, type (acute or chronic), location, severity, etc. of Osteopathic Lesion is almost totally lacking. It is appropriate to recognize at this point that the heterogeneity of the tissues involved, skin, muscle, fat, many kinds of collagens, and the vascular, neural, lymphatic, hormonal systems that are embedded in the tissues, make objective determinations extraordinarily difficult.
2. It is known that there are such phenomena as somatic and visceral reflexes and reflexes initiated by impulses from the higher centers. It is also generally accepted that there are combinations of these, which are involved in psychosomatic, viscerosomatic, and somaticovisceral problems. But how these reflexes interrelate in matters of health and disease is not known. For example, a patient might be ill from a disturbance involving a psychosomatic reflex.

   Currently it is not known how to evaluate either the higher center or the somatic element; hence, it is impossible to determine on the basis of objective evidence which, in any given case, should receive major attention, or if both should be addressed simultaneously. Related to this, it is not known whether one disturbance precedes the other, or, if two or more occur as primary pathophysiologic disturbances, what, if any, influence each exerts on the other.

   This is not to say that the experienced clinician, with competence in the evaluation of, for example, psychiatric and somatic problems, cannot successfully treat patients who present such a combination of problems; he certainly can do so, on an empirical basis. However, objective evidence, to support his empirical observations, is not available.
3. Finally, and again related to 2 above, it is not known how to objectively assess the usefulness and effectiveness of therapy, particularly manipulative therapy, directed at Osteopathic Lesion.

THE MOST CONTROVERSIAL

There are two major and one minor controversial issues.

First, there is the question as to whether or not Osteopathic Lesion contributes to pathophysiologic disturbances in organs and systems outside of the somatic system itself. Strong evidence has been presented above that a pathophysiologic disturbance in the neuromusculoskeletal system (Osteopathic Lesion) at least contributes to the total clinical picture that the patient presents. Since emotional, visceral, and other disturbances are generally acknowledged to cause certain somatic disturbances, it is reasonable to assume that the reverse might also occur (e.g. that somatic disturbances might have comparable deleterious influences on higher center, visceral, and other functions). The fact that the latter possibility is almost totally ignored in texts and treatises in medicine and surgery has given rise to major controversy. It might be said here that it is the lack of appropriate research, particularly involving

experimental animal research, rather than negative results of research, that has given rise to this controversy.

Second, controversy exists as to whether or not the manipulative and other management of the Osteopathic Lesion contributes to the amelioration or elimination of emotional, visceral, and other disturbances. Here again, this controversy is not due to negative observations from carefully controlled studies, but rather to a lack of appropriate research.

A third controversy of lesser significance, particularly among those health professionals who use, and are skilled in manipulation, involves the breaking of the synovial seal in arthrodial joints, which sometimes occurs in the course of manipulation. This procedure is frequently accomaccompanied by a noise often referred to as a "pop" or a "snap." Roston and Haines,[26] and later Unsworth, Dowson, and Wright,[27] have conclusively shown that the breaking of the joint seal permits an increase in motion (particularly motion not under voluntary control) for a period of time reported as being 15 to 20 minutes. There are those that hold that this is an unimportant phenomenon and is not involved in the beneficial effects of properly applied joint manipulation. Livingston[28] disagrees. On the basis of personal experiences with attacks of low back pain he comments:

> "An unsuccessful manipulation of the back seems to aggravate the pain, but a successful one causes a snap that may be audible. When this occurs, there is an immediate sense of relief, even though much of the pain and the muscle spasm are still present. The residual muscle stiffness and soreness gradually disappear once the underlying source of irritation has been corrected, and within three or four days I am back playing badminton without the slightest back disability.

This speaker, like many of his colleagues, agrees with Livingston since in many situations, breaking the joint seal in the course of manipulation of Osteopathic Lesion is followed by an immediate sense of relief on the part of the patient, an immediate reduction in the palpable abnormality in the texture of the tissue, and an improvement in joint motion that is evident to the individual administering the manipulation.

Here again, this controversy can only be resolved through appropriate and well-controlled research.

REFERENCES

1. Maigne, R.: *Orthopedic Medicine: a New Approach to Vertebral Manipulations*. Springfield. Charles C Thomas. 1972.
2. Kendall, H. O., Kendall, F. P., and Boynton, D. A.: *Posture and Pain*. Baltimore. Williams and Wilkins Co. 1952.
3. Denslow, J. S., Chace, J. A., Gutensohn, O. R., and Kumm, M. G.: Methods of taking and interpreting weight-bearing X-ray films. *J. Am. Osteop. A.* 54:663–670, July, 1955.
4. Adrian, E. D., and Bronk, D. W.: The discharge of impulses in motor nerve fibres, part II. *J. Physiol.* 6:81–101, 1928.
5. Jacobson, E.: *Electrical Measurement of Activities in Nerve and Muscle*. New York. McGraw-Hill Book Co. 1934.
6. Denslow, J. S., and Clough, G. H.: Reflex activity in the spinal extensors. *J. Nurophysiol.* 4:430–437, 1941.
7. Buchthal, F., and Clemmeson, S.: On the differentiation of palpable muscle affections by electromyography. *Acta Med. Scand.* 105:48–66, 1940.
8. Denslow, J. S., and Hassett, C. C.: The central excitatory state associated with postural abnormalities. *J. Neurophysiol.* 5:393–402, 1942.
9. Denslow, J. S., Korr, I. M., and Krems, A. D.: Quantitative studies of chronic facilitation in human motoneuron pools. *Am. J. Physiol.* 105:229–238, August, 1947.
10. Brendstrup, P., Jespersen, K., and Asboe-Hansen, G.: Morphological and chemical connective tissue changes in fibrositic muscles. *Ann. Rheum. Dis.* 16:438, 1957.
11. Bonica, J. J., Editor: *Advances in Neurology (Vol. 4): International Symposium on Pain*. New York. Raven Press. 1974.
12. Drachman, D. B., Editor: Symposium: trophic functions of the neuron. *Ann. N.Y. Academy Science.* 228:1–423, 1974.
13. Korr, I. M.: The trophic functions of nerves and their mechanisms. Louisa Burns Memorial Symposium. *J. Am. Osteop. A.* 72:163–171, October 1972.
14. Chamberlain, T. J., Rothschild, G. H., and Gerard, R. W.: Drugs affecting RNA and learning. *Proc. N.A.S.* 49: 918–925, 1963.
15. Patterson, M. M.: Mechanisms of classical conditioning and fixation in spinal mammals. (In Press).
16. Korr, I. M.: Clinical significance of the facilitated state. Symposium on the functional implications of segmental facilitation. *J. Am. Osteop. A.* 54:265–282, January 1955.
17. Ruch, T. C., Patton, H. D., Woodbury, J. W., and Towe, A. L.: *Neurophysiology*. 2nd Ed. Philadelphia. W. B. Saunders Co. 1966.
18. Speransky, A. D.: *Experimental and Clinical Lobar Pneumonia*. Dolevni Pneuvmonii. 3–17, 1942.
19. MacKenzie, J.: *Symptoms and Their Interpretation*. London. Shaw and Sons. 1912.
20. Lindgren, I.: Cutaneous precordial anaesthesia in angina pectoris and coronary occlusion (an experimental study). *Cardiologia.* 11:207–218, 1946–47.
21. Rinzler, S. H., and Travell, J.: Therapy directed at the somatic component of cardiac pain. *A. Heart J.* 35:248–268, February 1948.

597-036 O - 76 - 16

22. Judovich, B., and Bates, W.: *Pain syndromes*. 4th ed. Philadelphia. F. A. Davis Co. 1953.

23. Siehl, D., Olson, D. R., Ross, H. E., Rockwood, E. E.: Manipulation of the lumbar spine with the patient under general anesthesia: evaluation by electromyography and clinical-neurologic examination of its use for lumbar nerve root compression syndrome. *J. Am. Osteop. A.* 70:433–440, January 1971.

24. Lewis, T., and Kellgren, J. H.: Observations relating to referred pain, visceromotor reflexes and other associated phenomena. *Clinical Science*. 4:47–71, 1939.

25. Korr, I. M., Wright, H. M., and Thomas, P. E.: Effects of experimental myofascial insults on cutaneous patterns of sympathetic activity in man. *Acta Neurovegetativa, Bd. XXIII, Heft 3.*

26. Roston, J. B., and Haines, R. W.: Cracking in the metacarpophalangeal joint. *J. Anat.* 81:165–173, 1947.

27. Unsworth, A., Dowson, D., and Wright, V.: "Cracking joints" a bioengineering study of cavitation in the metacarpophalangeal joint. *Ann. Rheum. Dis.* 30:348–358, July 1971.

28. Livingston, W. K.: *Pain Mechanisms*. New York. Macmillan Co. 1947.

Discussion: Papers of J. S. Denslow and Scott Haldeman

LYLE A. FRENCH
Vice President for Health Sciences Affairs
University of Minnesota

Mr. Chairman, ladies, and gentlemen: I appreciate very much the privilege of the invitation to this workshop and the privilege of discussing the reports of Dr. Denslow and Dr. Haldeman. In some ways the reports are complementary and in other ways the opposite is true.

The term "osteopathic lesion" as described by Dr. Denslow, based on terminology commonly used by osteopathic physicians, implies an abnormal structural change in a tissue. He describes the lesion as tenderness to digital palpation with abnormal texture and tone of the soft tissues which "may be described as being boggy or thickened nonmuscle tissue and rigid muscle." Part of the evidence presented for the existence of a structural change is one report by Brendstrupp et. al.,[1] published in 1957, in which, in 10 or 12 areas of fibrositis identified by palpation, there were positive findings as opposed to negative findings in tissue removed from control sites. The positive findings were very minor and are of doubtful significance unless confirmed by independent observers. There is no additional bibliography to support this claim of positive findings.

There is nothing really novel about the presence of localized or focal tenderness associated with a variety of disorders and these are well known to all physicians who treat patients with painful conditions. A classical example is a patient with a cervical intervertebral disc protrusion. Many of these people have typical paravertebral foci of pain or point tenderness which are often associated with muscle spasm and on palpation an increase in bulk or firmness can readily be detected. This I believe would qualify as an "osteopathic lesion" by Dr. Denslow. Traction, however, if correctly applied, relieves the pain and the spasm promptly in some instances and the "osteopathic lesion" disappears since there is neither pain nor palpable residual evidence for it. Thus the so-called "osteopathic lesion" may not be a true structural abnormality but rather, simply local muscle contraction brought on by nociceptive input. Furthermore it has been well established experimentally that Group III afferents (nociceptive) activate motor neurons via interneurons in the cord so that lowered firing thresholds of the motor neurons visualized in the electromyogram is an expected consequence. In fact, failure to find increased firing would be more surprising than its presence.

The "osteopathic lesion" is also said to be similar to and possibly identical with "the trigger points in the body wall and upper extremities in myocardial disease, with splinting and tension of the abdominal wall, flank and paravertebral tissue in intraabdominal disease, and the muscular aches and pains accompanying systemic infection such as influenza, etc." I doubt if there is really any acceptable evidence in the literature for any of these symptoms of disease being associated with a lesion, as defined earlier. When the pain is diffuse as in myocardial ischemia, in which the whole of the medial aspect of the upper extremity is hyperalgesic and hyperesthetic, with extension into the chest wall, and when there is also muscle spasm, using the criteria for "osteopathic lesion", one would be led to the conclusion that most of the limb now had this lesion. The same would be true of the entire abdominal wall in cases of peritonitis. Furthermore, as in the case of the

protruded intervertebral disc, when the cause or primary disease is removed, the "osteopathic lesion" disappears either immediately or leaves only a mild residual tenderness. I question if we are really talking about a true structural abnormality.

In fact, what has been described is simply the observation that nociceptive input leads to motor neuron excitation and thus to muscular contraction. It does not lead to the development of a structural lesion, but rather to palpably increased muscle tension. The so-called nodules of myositis and fibrositis are elusive in most instances and, when palpated, are most probably only localized areas of muscle contraction. The microscopic findings in the one report referred to in which pathological examinations were done (Brendstrupp et. al., 1957) are stated to have been "widening of the interstices, indicating edema, and increased number of mast cells in the connective tissue, and an increased number of nuclei in the muscle fibers." It seems highly improbable that such histologically observed findings could be palpated with any degree of assurance.

A report that pneumonia could be rationally treated by local blockade of the input to the appropriate segments of the spinal cord comes from the Russian literature of 1940 (Speransky et. al.).[4] The investigator, Speransky, suggested that "the rationale of this treatment is based on the presumed changes in the lung following restoration of normal nerve functioning." But there is no evidence given that there was anything but normal nerve functioning. Rather than bolstering the case for an "osteopathic lesion", inclusion of such a bizarre and obsolete and unverified claim tends to make the reader question the judgment of accepting it. The literature can be used uncritically to support virtually any thesis. It seems to me that the words of Karl Popper[2] exemplify this when he stated that "it is easy to obtain confirmation, or verification, for nearly every theory if we look for confirmation." It is important that we do not see only what we do not want to see. Observations must be selective.

The relief of pain which results from local anesthetic block of the so-called trigger points is well known to most observers and, when successful, may well be due to interruption of a vicious cycle as proposed by Ruch[3] and others. Again this is an old observation dating from the beginning of the century. It does not need confirmation. The blocking of the so-called trigger point, however, even though it may partially relieve symptomatology, is seldom if ever an adequate method of relieving the basic problem.

To sum up, it seems to me that Dr. Denslow has gathered data in support of the existence of an "osteopathic lesion" by recourse to work by others, some of whom are well-known scientists, and correlated these data with clinical observations made by himself and many others over the past century. I believe the attempted correlation is still inconclusive since the data submitted are inadequate to establish the existence of a true tissue abnormality. The sum total of the paper, in fact, repeats a well established principle that input of stimuli from a disease process can be followed by the development of secondary hyperalgesic, painful areas associated with muscle spasm of more or less discrete extent. The clinical and neurophysiological literature is in accord with this concept. If good evidence that a structural tissue abnormality is present could be provided by biopsy, or other means, this would be most interesting. It would also be necessary to establish in precisely what condition the lesion appeared. Furthermore, it would be appropriate to verify that the tissue had returned to normalcy by osteopathic therapy. In essence the report appears to me to be a good example of taking those pieces of information which support one's hypothesis and then using them to give scientific stature to a theory that is lacking in substance, that is, that there actually is such a thing as an "osteopathic lesion." Yes, there is muscle spasm and you can feel it, and I can feel it, we all know this, but so far as I'm concerned, the case for a structural lesion has not been fully made. Whether one calls point tenderness with muscle spasm just that, or uses another term such as somatic dysfunction or "osteopathic lesion," is a matter of semantics as I see it. The semantics become tendentious when they are used to give validity to a thesis.

The report by Dr. Haldeman is a straightforward presentation of some of the ways of investigating spinal abnormalities. It does begin with a definition of subluxation that is all inclusive so that most any variance from the normalcy of a newborn could be considered pathological. The result is that many of the consequences of the subluxation, as so defined, are vague, ill defined and most difficult to relate scientifically to specific clinical entities. With such a broad definition it is really difficult to differentiate cause and

effect. The statement is made that subluxation is "important in initiating further pathology." The concept promoted is that there is a potential in subluxation for positive feedback, that spinal joint dysfunction, produced by minor mechanical stresses, may not be self-correcting because the reflex and tissue abnormalities that develop may exacerbate the joint dysfunction, tending to maintain it. Thus it is presumed, even in the absence of gross pathology, that in susceptible individuals nerve compression does occur as a result of forces to which all of us are exposed every day. It is concluded that this feedback then will lead to further development of subluxation and eventually to various visceromotor alterations. I think everyone will accept the notion that nerve roots are always being subjected to mechanical forces of some degree throughout their course and that they are especially vulnerable at the intervertebral foramina, especially in the presence of abnormal conditions. There is reason to believe that nerve roots are more susceptible to compression damage than peripheral nerves, in part because of their surrounding tissues and in part because of their anatomical structure, i.e., lacking a perineurium, etcetera. This was shown by Sunderland and Bradley[5] in 1961. There is no unchallengeable evidence, however, that symptoms supposedly alleviated by chiropractic manipulation are, in fact, due to spinal nerve compression. In fact it is believed that such evidence will be hard, if not impossible, to obtain. This is because of the extremely minor variations from normal, or even abnormal situations, in which manipulation is applied. This is in contradiction to the nerve root compressions that are observed by neurosurgeons and orthopedists in which specific neurological evidence of such compression is usually present preoperatively. I think one of the points that Dr. Haldeman is attempting to make is that a very minimal compression over a period of time may well cause somatovisceral symptoms which can be alleviated by manipulation. I would conclude from his discussion that symptoms which may be alleviated by chiropractic manipulation are due to pathological changes so minor that neurological and roentgenological diagnostic procedures will not offer help in obtaining definitive evidence of spinal nerve compression. Certainly many of the X-rays presented today would be considered by the medical profession as being within the range of normal.

In the section on "Compression of Vital Structures," an attempt is made to implicate problems with the sympathetic nervous system, on the premise that there is a lack of understanding of the function and anatomy of this system. It is my impression that a great deal is known about the sympathetic nervous system. It is unclear to me what Dr. Haldeman really means by costovertebral rotational subluxations as a source of sympathetic irritation, particularly in view of the rather significant rotatory deformities one sees in scoliosis as well as the fact that compression fractures and fracture dislocations of the thoracic spine are rarely associated with visceral symptoms, even when the sympathetic chain itself has been severely damaged. I do believe that the quoted work of Ushio[6] makes good sense and confirms the observations of many others that applying a lumbosacral corset will decrease vertigo following spinal injury. However it is believed that this is not accomplished by decreasing activity of the lumbar proprioceptors, as suggested by Dr. Haldeman, but rather that a lumbosacral support will decrease visceral pooling of blood by increasing intra-abdominal pressure. This diminishes the recurrent hypotensive episodes observed in these patients. I believe that the conclusions obtained in regard to the review of the literature concerning somatosympathetic reflexes leads to a conclusion totally speculative and really detracts rather than adds to the report.

In summary, I have enjoyed reading and hearing the report of Dr. Haldeman. The paper certainly has virtue if listed simply as a summary of spinal alignment abnormalities, but it really has limited significance in terms of advocating manipulation of the spine.

REFERENCES

1. Brendstrupp P., Jespersen K., and Asboe-Hansen, G. Morphological and Chemical Connective Tissue Changes in Fibrositic Muscles. *Am. Rheum. Dis.* 16:438, 1957.
2. Popper, K. R. *Conjectures and Refutations: The Growth of Scientific Knowledge*. Routledge and Kegan Paul, London 1969.
3. Ruch, T. C., Patton, H. D., Woodbury, J. W., and Towe, A. L. *Neurophysiology*. Second edition, W. B. Saunders Co., Philadelphia 1966.
4. Speransky, A. D. Experimental and Clinical Lobar Pneumonia. Dolevni Pneuvmonii, 1942. (Cited by Denslow, J. S.)
5. Sunderland, S., and Bradley, K. C. Stress-Strain Phenomena in Human Spinal Nerve Roots. *Brain* 84:120–124, 1961.
6. Ushio, N., Hinoki, M., Hine, S., Okada, S., Ishida, Y., Koike, S., and Shizuku, S. Studies on Ataxia of Lumbar Origin in Cases of Vertigo Due to Whiplash Injury. *Agressologie* 14-D:73–82, 1973. (Cited by Haldeman, S.)

The Role of X-Ray Findings in Structural Diagnosis

JOSEPH W. HOWE
Professor and Chairman
Department of Roentgenology
The National College of Chiropractic
Lombard, Illinois

Roentgenology plays an important role in diagnosis in all phases of the healing arts. It has special importance in structural diagnosis as related to a manipulative approach to therapy due to its unique ability to depict anatomy, pathology, and certain aspects of articular mechanics or function that are otherwise not obtainable.

Although there is considerable overlap and interrelationship among the following arbitrary categories, this paper will discuss the role of X-ray in structural diagnosis relative to: (1) Anatomical depiction, (2) functional assessment, (3) pathological considerations, with comments regarding the present levels of knowledge in each of these areas and some ideas for further investigation. The important consideration of the ionizing radiation incidental to X-ray examination will also be discussed.

ANATOMICAL DEPICTION

The ability to depict certain aspects of anatomy, especially to allow visualization of anomaly and anatomical variation, has made the use of diagnostic X-ray an invaluable tool in structural diagnosis. This anatomical information must be placed in clinical perspective if it is to be used beneficially. Differences of opinion regarding the importance of anomaly and anatomical variation are widespread.

Janse,[1] Calliet,[2] Ferguson,[3] and others [4, 5, 6] have attached clinical significance to spinal anomaly and anatomical variation, while Higley,[7] Splithoff,[8] Fullenlove,[9] and DePalma [10] claim that they find no correlation between such variants and low back pain. A study presently under way at the National College of Chiropractic has found approximately equal incidence of several lumbosacral anomalies among 72 patients with no history of low back pain compared to 284 patients with low back symptoms. Cox [11] has observed that facet tropism in the presence of posteromedial disc protrusion is a poor prognostic sign relative to response to the manipulative treatment he propounds. Farfan [12, 13] notes that not every person with tropism develops backache, but that there is a higher incidence of tropism in people with backache. He also notes a high correlation between tropism and disc pathology. Books by Ferguson,[3] Schmorl, and Junghanns,[14] Hadley,[15] and Epstein [16] have become classic references relating to the radiological appearances of the myriad variations of spinal morphology and other aspects of spinal roentgenology, but their ideas regarding the significance of some of these findings vary. The presence of anatomical variation as related to present or previous symptoms has not been documented. Further, knowledge about the presence of variants is important to manipulative efforts since such procedures must be planned with knowledge about the structure and function affected by these variants.

The American College of Radiology recently published the proceedings of a conference on low back X-rays as part of preemployment physical examinations.[17] In addition to papers by Epstein,[18] Moreton,[19] and others [20, 21] dealing specifically with X-ray findings, and Trout [22] who spoke on radiation safety, the publication contained an 11-page bibliography of papers dealing with low back X-ray

studies. Conference participants included radiologists, orthopedists, epidemiologists, and other interested people. A 583-page annotated bibliography on the spine, much of which is radiological in nature, was published by the Ontario Labour Ministry in 1972.[23] Both publications concluded that spinal X-ray findings must be evaluated in light of the clinical situation, not as screening procedures, because of the ambiguity of the significance of many X-ray findings.

Additional studies with reference to specific spinal variants as well as to the general category of spinal anomaly and anatomical variation are necessary in light of the varying opinions of experts. Such investigations might well consider response to treatment in people with anomaly and incidence of degeneration in such people as well as the relationship of symptoms to the abnormality.

FUNCTIONAL ASSESSMENT

The most widely accepted definition of spinal subluxation in chiropractic is: "A subluxation is the alteration of the normal dynamics, anatomical or physiological relationships of contiguous articular structures."[24] This definition along with manifestations, significance, and a classification of radiological manifestations is presented in the following table. These concepts are currently taught in the chiropractic colleges.

The chiropractic concept of subluxation is largely one of functional derangement. The import of subluxation, though anatomical disrelationship may be the most recognizable aspect radiographically, depends upon the articular dysfunction and the resultant pathophysiological alterations. Subluxation frequently accompanies articular pathology.

X-ray manifestations, classified as "static intersegmental subluxations," have functional import since they may affect spinal dynamics and either cause or be caused by pathophysiological alteration. If a radiographically demonstrable subluxation causes no presently demonstrable clinical problems, its significance may be questionable

TABLE

Definition

A subluxation is the alteration of the normal dynamics, anatomical or physiological relationships of contiguous articular structures.

Manifestations

In evaluation of this complex phenomenon, we find that it has—or may have—biomechanical, pathophysiological, clinical, radiologic, and other manifestations.

Significance

Subluxations are of clinical significance as they are affected by—or evoke—abnormal physiological responses in neuromusculoskeletal structures and/or other body systems.

Radiological Manifestations

In considering the possible radiological manifestations of subluxations, it is important to emphasize that clinical judgement is necessary to determine the advisability of exposing a patient to the potential hazards of ionizing radiation. An important purpose of exposure, besides the evaluation of subluxations, is the determination of the evidence of other pathologies.

The radiographic procedures necessary to determine possible fractures, malignancies, etc., may not be the specific views needed to evaluate the possible radiological manifestations of subluxation. When subluxation can be evaluated by other clinical means, it may be prudent to avoid radiation exposure.

Classification of Radiologic Manifestations:

A. Static intersegmental subluxations.
   1. Flexion malposition.
   2. Extension malposition.
   3. Lateral flexion malposition (right or left).
   4. Rotational malposition (right or left).
   5. Anterolisthesis (Spondylolisthesis).
   6. Retrolisthesis.
   7. Lateralisthesis.
   8. Altered interosseous spacing (decreased or increased).
   9. Osseous foraminal encroachments.

B. Kinetic intersegmental subluxations.
   1. Hypomobility (fixation subluxation).
   2. Hypermobility (loosened vertebral—motor—unit).
   3. Aberrant motion.

C. Sectional subluxations.
   1. Scoliosis and/or alteration of curves secondary to musculature imbalance.
   2. Scoliosis and/or alteration of curves secondary to structural asymmetries.
   3. Decompensation of adaptational curvatures.
   4. Abnormalities of motion.

D. Paravertebral subluxations.
   1. Costovertebral and costotransverse disrelationships.
   2. Sacroiliac subluxations.

but, as with anomaly, the possibility of later degenerative or other problems resultant from such disrelationship has not been sufficiently investigated.

Hadley,[15,25] McNab,[26] Shapiro,[27] Williams,[28] and many others refer to radiologically demonstrable subluxations, ascribing to them the ability to cause pain both locally and peripherally. Their descriptions of subluxations correspond with some of the chiropractic classifications.

Attempts to demonstrate spinal disrelationship by chiropractors and adjustive procedures designed to correct or improve such disrelationships have led to some special approaches to spinal roentgenology. Spinography, the term traditionally applied to describe upright spinal radiography, done particularly for mensuration, comprises by far the most frequent X-ray procedure done by or at the direction of doctors of chiropractic. The methodology used to produce such radiographs and the interpretation thereof differs widely among those from different schools of thought in chiropractic.

One approach to spinography is the use of "full spine" radiography (14 x 36-inch single exposure radiographs of the entire spinal column). Though this procedure was developed by a chiropractor[29] and is used mainly in chiropractic, its acceptance by at least two[30,31] noted medical scientists is noteworthy. Winterstein[32] and Hildebrandt[33] have indicated that this procedure allows a conceptual analysis of postural and intersegmental relationships that cannot be gained from any other radiographic method. Logan,[34] Gonstead,[35] Winterstein,[36] Hildebrandt[33] and others have devised systems of measurement from such full spine radiographs to assess intra-pelvic, vertebro-pelvic, and intervertebral relationships.

Vladeff,[37,38] Gregory,[39] Messer,[40] Pettibon,[41] and Blair[42] among others have advocated methods of precise patient alignment and stressed exacting alignment of X-ray tube to film plane as part of their systems of spinographic analysis. Mears,[43,44,45] in addition to his spinographic system, has reported a high incidence of certain abnormal cervical spine configurations in patients with mental illness and certain other syndromes. Vladeff's turntable is applicable to either full spine radiography or to radiography of smaller spinal areas. The others cited advocate the use of smaller films of specific spinal areas to decrease distortion.

Radiographic distortion is a constant factor in any geometric evaluation of spinal relationships. This is especially true with full spine radiography. The use of long target-film-distances (72-inch or 84-inch) for upright spinographs has therefore been almost universally employed to reduce such distortion as much as possible. Figures 1, 2, 3, and 4 show mathematical representations of distortion incidental to radiography. One result of such distortion is that significant deviations from horizontal can be made to appear on the film through slight rotations of the patient's body relative to film plane and central ray. A major difficulty in attempting to solve the complicated problem of defining intervertebral relationships via radiography is that

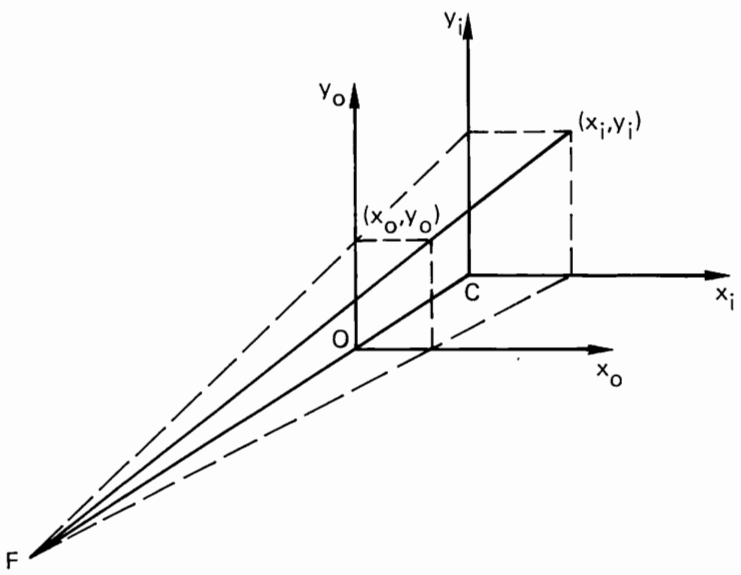


Fig. 1. *Distortion due to distance between object and film.* The problem is defined on Figure 1 where (x_0, y_0) is a point of the object, (x_i, y_i) is its image.

Let:

$$a = CF, b = OF, d = CO$$

Then:

$$\frac{x_i}{x_0} = \frac{y_i}{y_0} = \frac{a}{b}$$

and if all points were in the x_0y_0 plane, the image would be simply a magnification of the object, in the ratio a/b. However, the image is three dimensional and there will be distortions due to the varying of the perpendicular distance between points of the object and film.

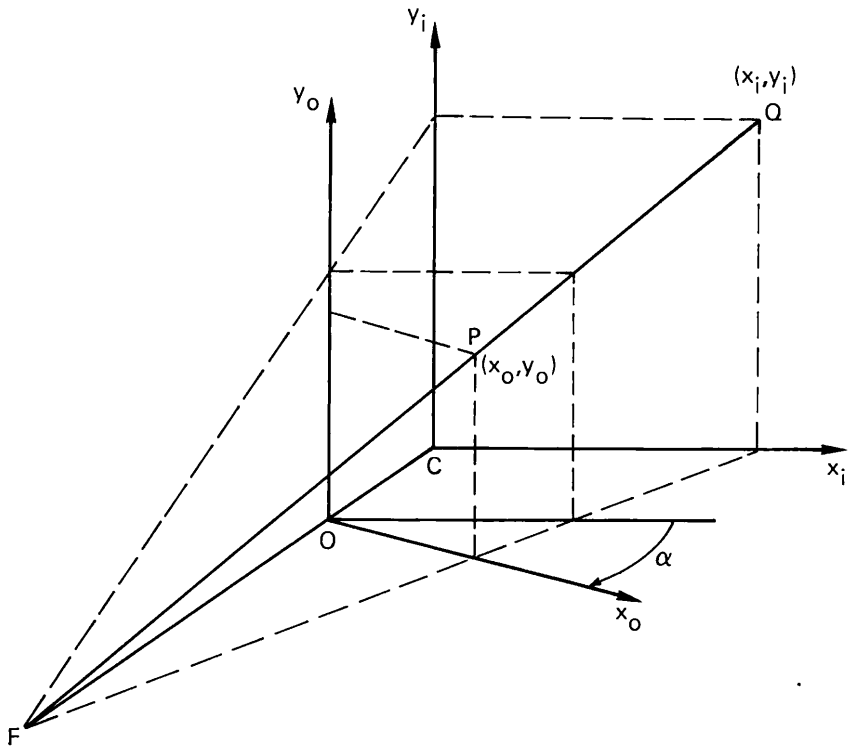


Fig. 2. *Distortion due to rotation of plane of object.* Even though certain structures may lie approximately in a plane, the object has been so positioned that the plane of interest is not exactly parallel to the film, but rotated at an angle α, as shown in Figure 2. The image Q of a point $P(x_0, y_0$. will be at:

$$x_i = \frac{a x_0 \cos \alpha}{b - x_0 \sin \alpha}$$

$$y_i = \frac{a y_0}{b - x_0 \sin \alpha}$$

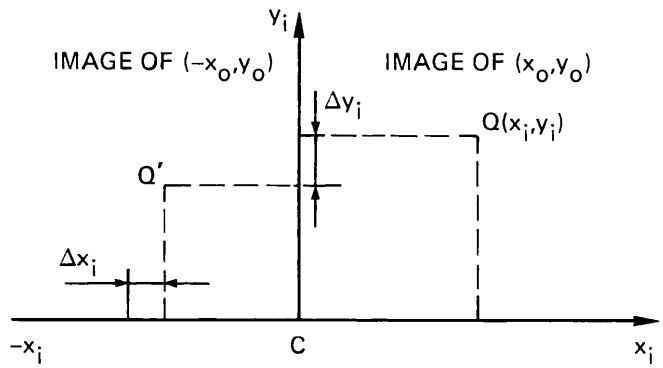


Fig. 3. Consider now an object point P' $(-x_0, y_0)$ symmetrical to P with respect to the y_0-axis. Its image Q' will be at $(-x_i+\Delta x_i, y_i-\Delta y_i)$ as shown on Figure 3. Δx_i and Δy_i are most conveniently expressed in terms of x_i, y_i instead of x_0. y_0 since coordinates of film points are the more readily available ones.

$$x_i = \frac{2x_i \sin \alpha}{b} \frac{(a + x_i \tan \alpha)}{(a + 2x_i \tan \alpha)}$$

$$y_i = \frac{2x_i y_i \tan \alpha}{a + 2x_i \tan \alpha}$$

If $2x_i \tan \alpha << a$, and α is small, these can be approximated by:

$$\Delta x_i \approx 2x_i \alpha / b$$

$$y_i = 2x_i y_i \alpha / a$$

with better than 10 percent accuracy for all standard film sizes and distances a, and angles α less than 15°.

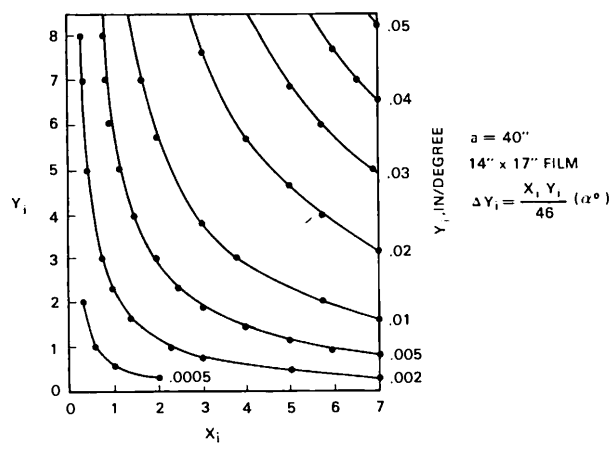


Fig. 4. The approximatons shown in Figure 3 are most useful in that, for a given distance, a family of hyperbolas drawn in the $x_i y_i$ plane will give a quick estimate of the amount of vertical deviation to be expected, per degree of rotation α, between the two image points Q and Q'.

The straight horizontal line PP' will project on the film as the sloping line QQ'; actually QQ' is slightly curved, concave up, but for practical purposes this curvature can be ignored.

the body does not present either parallel planes or symmetry of its structures. Offcentering of patient to central-ray and film center is nearly unavoidable to at least some degree in either the vertical or horizontal planes. This makes it nearly impossible to duplicate patient positioning from one radiograph to another.

The use of full spine radiography has drawn criticism from some sources, both as to the diagnostic film quality obtainable and the irradiation of the entire trunk. Use of varying screen speeds in the 14 x 36-inch cassette has been one method used to improve radiographic quality. This method, however, exposes the thinner body areas to as much radiation as the heavier areas. Use of compensating filters reduces radiation to thin body parts and also results in a balance of radiographic densities which produces radiographs of acceptable quality.[32]

Levine, Howe, and Rolofson[46] used thermoluminescent dosimetry and an Alderson phantom while simulating chiropractic radiographic techniques to measure the surface exposure and organ doses incidental to such radiographic procedures. Their data showed that good quality radiographs using full spine techniques did not cause higher organ doses than were produced by X-rays of the several spinal regions. Data collected by the Nationwide Evaluation of X-ray Trends Survey System[47] also shows that exposure from full spine radiographs is not excessive as compared to radiography of smaller spinal areas.

The spinographic systems previously cited and others in use, while giving adequate ability to evaluate spinal structure and relationships for clinical purposes, do not allow exacting determinations. Suh[48] has devised a computer graphics method to allow very exacting measurements needed to better assess manipulative effectiveness. Frigerio, Stowe, and Howe[49] have taken a somewhat different approach and by orthogonal radiography and photogrammetry have demonstrated movement in the sacroiliac joints, a subject long disputed by clinicians and anatomists. Further investigations in these areas should yield helpful information relating to spinal function and the effects of spinal manipulation.

Functional spinal radiography has been used via static stress studies and cineradiography to enhance knowledge of spinal integrity and functional ability. Hviid,[50] Davis,[51] Jackson,[52] Cobb,[53] Krausova and Lewit,[54] Mehta,[55] Jirout[56] and others have discussed the efficacy of flexion-extension lateral films of the spine of side-bending frontal spinal films to demonstrate intersegmental mobility and integrity of spinal retaining structures. These methods are frequently used in chiropractic, osteopathy, and orthopedics for such purposes. Fielding,[57] Jones,[58,59,60,61,62,63] Bard and Jones,[64,65] Woesner,[66] Rich,[67,68] and Howe[69,70] have shown the efficacy of cineradiography in detailing normal and abnormal intervertebral movements. These studies have allowed somewhat better understanding of spinal mechanics which should have practical application. Some of these observations have cast doubt upon a few traditional concepts of intervertebral movement.

Penning[71] illustrated flexion and extension in the mid-cervical region as producing a rocking-gliding movement of a vertebral body over the disc below with the fulcrum of motion approximately in the center of the vertebral body below. Rich[68] and Jones[59] both noted altered intervertebral mobility associated with spinal degenerative disease. Howe[70] in confirming this has postulated that the earliest sign of disc degeneration would be alteration of the usual rocking-gliding movement noted by Penning.[71] With degeneration the motion becomes more identifiable with some anterior and posterior slippage replacing some of the rocking. This also results in changes in the usual gliding facetal movements. This abnormal motion has been demonstrated in the cervical spine with degeneration; some observations in the lumbar spine also suggest such applicability.

Rich[68] reported that on lateral flexion of the low back the L-5/S-1 functional unit showed little mobility and that L-4/L-5 was the lowest truly mobile unit. Sandoz[72] aptly likened this to the semirigid portion of an electric or telephone cord which distributes stress over a distance so that early breakdown will not ensue where a highly flexible area functions in close proximity to a more rigid unit. Recent observations in the cineradiological laboratory of the National College of Chiropractic have shown, however, that when the spine is held relatively immobile and the pelvis moved freely thereunder (as in a hula dance) that the L-5/S-1 junction is more freely movable than the functional units above it.

Janse and Illi[73] observed that side-bending and rotation of the spine are concomitant motions. Janse[74] then proposed the hypothesis that in scoliosis or subluxation the spine will deviate *more*

easily into distortion (increase the distortion) than it will deviate out of distortion (correct or reverse the distortion). Yochum[75] and Taylor[76] by somewhat different methods verified this on a few patients. Further studies under direction of this author have also shown it to be true as related to scoliosis or lateral distortion, but in hyper- or hypo-lordoses of the lumbar or cervical spine, the principle has not held true except in those cases where the distortion was due to antalgic muscle contraction.

Cox[11,77,78] has suggested a method of assessing disc protrusion or prolapse from plain-film radiography, along with a manipulative technique for treatment of this common problem. His diagnostic procedures were derived from the work of Herlin[79] with radiographic concepts adapted from cineradiographic observations by Rich[68] relating to abnormal intervertebral movements in patients with disc hernia, and from Winterstein's[36] method of diagnosing disc protrusion from upright radiographs. This work is being continued.

PATHOLOGICAL CONSIDERATIONS

Depiction of spinal pathology is one of the very important aspects of spinal roentgenology. Pathological changes may be present that are not readily discernible except by radiography. Some of these could be contraindications for manipulative therapy. Other pathologies, while not being contraindications to adjustive procedures may have profound effects upon spinal function and therefore must be carefully considered in manipulative efforts. Still other pathological changes may point toward dysfunctions which are not readily apparent in static radiographs and therefore lead to better understanding of subluxation of dysfunction patterns that can be found clinically but are poorly appreciated radiographically. Some types of pathologies may be entirely incidental to the biomechanical approaches of manipulative treatment, but the approach of any doctor must encompass the totality of the patient's being so that no information is unimportant. X-ray detection of "silent" gall stones, abnormal intestinal patterns, changes in kidney contours, and other abnormalities frequently found may be incidental relative to the primary reason for the spinal X-ray examination, but differential diagnosis and consideration of all aspects of health are not incidental to any physician.

The emergence of diagnostic roentgenology as a specialty in chiropractic has given this aspect of roentgenology and the development of more inclusive approaches to X-ray diagnosis significant impetus in the last 15 years. The radiological approach to spinal diagnosis must include all aspects—anatomical, biomechanical, and pathological.

EXPOSURE TO IONIZING RADIATION FROM SPINAL ROENTGENOLOGY

One of the great concerns among all who deal with X-rays is the exposure to ionizing radiation incidental to radiological examination. Although patient doses from diagnostic X-ray are low and unlikely to cause demonstrable damage if proper radiological safety measures are used, it must be recognized that, except for background radiation, diagnostic procedures comprise the greatest amount of radiation exposure to the general public. In spinal radiography this is of special concern since all critical organs: Gonads, bone marrow, thyroid, eye lens, and much of the body's epithelium are either within or very close to the primary beam during at least some of the studies. This concern has led to a 7-year major effort within the chiropractic profession to promulgate proper measures of patient and operator radiation safety through publications[80,81,82,83] and continuing education courses.[84,85]

The chiropractic profession has accepted the recommendation of the BEIR Report[86] that "No exposure to ionizing radiation should be permitted without the expectation of a commensurate benefit." Through its colleges and radiological organizations, the chiropractic profession has promulgated the notion that spinal X-ray examination should be done only to fulfill clinical needs as shown from other examination procedures and history, and that repeat radiographic examinations should be avoided unless they are clinically necessary. The profession has also gone on record with the Bureau of Radiological Health, U.S. Department of Health, Education, and Welfare, as advocating the use of gonad shielding where such use will not interfere with the clinical objective of the X-ray examination.

In light of the seriousness of the problem of exposure to ionizing radiation incidental to spinal X-ray examination, the action of the U.S. Congress which requires X-ray demonstration of spinal subluxation in order to qualify a patient for reimbursement of chiropractic services under Medicare and the Federal Workmen's Compensation Act seems inequitable, capricious, and detrimental to the pub-

lic interest. This is especially true since radiological depiction is only one method of detection of subluxation and not as reliable as other clinical methods in some cases because of the functional nature of subluxation and the static nature of most X-ray examinations. The use of X-ray in spinal diagnosis is significant, as this paper has tried to demonstrate, but the use of X-ray in any diagnosis should be at the discretion of the clinician and dictated by other clinical findings.

SUMMARY

Spinal X-ray examination gives significant information in the diagnosis of spinal normalcy, abnormality, and disease. Its benefits also extend to giving guidance for therapeutic procedures, especially in manipulative or adjustive approaches where intraspinal disrelationship, distortion, deformity, or disease may necessitate special consideration or even contraindicate such ministrations. Of special importance to chiropractors and others whose therapeutic thrust is biomechanical is the fact that some radiological procedures give information that is helpful in understanding spinal function. Such use must be dictated by clinical needs in light of possible patient benefit. X-ray examinations should be designed to give the information of greatest benefit to the clinician in his diagnostic and therapeutic efforts and therefore routine use of or stereotyped methods of X-ray examination may not serve the best interests of patient or doctor.

In order that the common complaints of man associated with musculoskeletal function may be better understood and more effectively managed additional investigations in the following areas are needed:

1. the significance of anatomical variation, anomaly, spinal distortion, and subluxation or dysfunction;
2. the effects of various disease processes on the biomechanics of the body; and
3. the ranges of normal functioning of the spinal column and other supporting body structures.

Radiological investigation is indispensable in such studies.

REFERENCES

1. Janse, J.: The mechanical pathologies involved in the low back and sciatic neuralgia syndrome. *J. National College of Chiropractic.* 25: No. 1:3–7, 16–22, 1953.
2. Calliet, R.: Miscellaneous low back conditions and their relationship to low back discomfort and disability. Chap. 6 in *Low Back Pain Syndrome.* (Philadelphia) Davis, 1966.
3. Ferguson, A. B.: The spine. Chap. 6 in *Roentgen Diagnosis of the Extremities and Spine.* 2nd edition. (New York) Harper and Brothers, 1949.
4. Chiak, R.: Variations of lumbosacral joints and their morphogenesis. *Acta Univ. Carol. Med.* 16: No. 12:145–165, 1970.
5. Ryan, M. J.: Low back pain—the role of radiology. *Ir. J. Med. Sci.* 351–361, August 1964.
6. Stinchfield, F. E.: Sinton, W. A.: Clinical significance of the transitional lumbosacral vertebra. *J.A.M.A.* 157:1107–9, 1955.
7. Higley, H. G.: *Lumbosacral Spine Facet Facings.* Published by the Foundation for Chiropractic Education and Research, (Glendale, Calif.), 1967.
8. Splithoff, C. A.: Lumbosacral junction—roentgenolographic comparison of patients with and without backache. *J.A.M.A* 152:1610, 1953.
9. Fullenlove, T. M.: Williams, A. J.: Comparative roentgen findings in symptomatic and asymptomatic backs. *Radiology* 68:572, 1957.
10. DePalma, A. F.: Rothman, R. H.: *The Intervertebral Disc.* Saunders (Philadelphia) 262, 265–7, 1970.
11. Cox, J. M.: Lumbar disc herniation: statistics on an innovative diagnostic and therapeutic approach. *J. Clin. Chiropractic,* Radiographic Edition 20–44, 1973.
12. Farfan, H. F.: Sullivan, J. D.: The relation of facet orientation to intervertebral disc failure. *Can. J. Surg.* 10:179, 1967.
13. Farfan, H. F.: *Mechanical Disorders of the Low Back.* Lea and Febiger (Philadelphia) 1973.
14. Schmorl, G. and Junghanns, H.: *The Human Spine in Health and Disease.* 2nd American Edition, translated by E. F. Beseman; Grune and Stratton (New York) 1971.
15. Hadley, L. A.: *Anatomico-roentgenographic Studies of the Spine.* (Springfield, Ill.) Thomas, 1973.
16. Epstein, B. S.: *The Spine: Radiographic Text and Atlas.* 3rd Edition, Lea and Febiger (Philadelphia) 1969.
17. *Conference on Low Back X-rays in Preemployment Physical Examinations.* Published by the American College of Radiology (Tuscon, Ariz.) 1973.
18. Epstein, B. S.: Radiological anatomy of the lower spine. *Conference on Low Back X-rays in Preemployment Physical Examinations,* 21–31. Published by the American College of Radiology (Tucson, Ariz.). 1973.
19. Moreton, R. D.: A radiologists's viewpoint. *Conference on Low Back X-Rays in Preemployment Physical Examinations,* 141–7. Published by the American College of Radiology (Tucson, Ariz.). 1973.
20. Erickson, G. E.: Morphological perspectives of the back. *Conference on Low Back X-rays in Preemployment Physical Examinations,* 2–7. Published by the American College of Radiology (Tucson, Ariz.). 1973.
21. Ford, L. T.: Orthopedic considerations. *Conference on Low Back X-rays in Preemployment Physical Examinations,* 33–45. Published by the American College of Radiology (Tucson, Ariz.). 1973.
22. Trout, E. D.: Radiation Safety. *Conference on Low Back X-rays in Preemployment Physical Examinations,* 65–72. Published by the American College of Radiology (Tucson, Ariz.). 1973.
23. Brown, J. R.: *Manual Lifting and Related Fields, An Annotated Bibliography.* Published by the Ontario Ministry of Labour. (Ontario, Canada) 1972.

24. Inman, O. B.: Basic chiropractic procedural manual. published by the *American Chiropractic Assn.* (Des Moines) 1973.
25. Hadley, L. A.: Intervertebral joint subluxation, bony impingement and foraminal encroachment with nerve root changes. *Am. J. Roentgenol. Radium Ther. Nuc. Med.* 65:377–402, 1951.
26. McNab, I.: Pathogenesis of symptoms in discogenic low back pain. American Academy of Orthopedic Surgeons, Symposium on the Spine. Mosby (St. Louis) 1969.
27. Shapiro, R.; Youngberg, A. S.; and Rothman, S. L. G.: The differential diagnosis of traumatic lesions of the occipito-atlanto-axial segment. *Radiol. Clin. North Am.* 11: No. 3:1973.
28. Williams, P. C.: *The Lumbosacral Spine.* McGraw-Hill (New York) 1965.
29. Sausser, W. L.: Achievment! Entire body X-ray technique is perfected. *The Chiropractic Journal* 4: No. 2:17, 1935.
30. Hoppenfeld, S.: *Scoliosis, A Manual of Concept and Treatment.* Lippincott (Philadelphia) 1967.
31. Keim, H. A.: *Scoliosis. Clinical Symposium.* 24: No. 1, Ciba-Geigy, 1972.
32. Winterstein, J. F.: Leverone, R. A.: Full spine radiography, its methods and value. *Dig Chiropractic Econ.* 17: No. 1:26–30, 1974.
33. Hildebrandt, R. W.: *Synopsis of Chiropractic Postural Roentgenology.* 2nd Ed. Published by the National-Lincoln School of Post-Graduate Chiropractic Education (Lombard, Ill.) 1974.
34. Logan, H. B.: *Textbook of Logan Basic Methods.* Edited by V. F Logan. Published by Logan Chiropractic College (St. Louis) 1950.
35. Herbst, R. W.: *Gonstead Chiropractic Science and Art.* Sci-Chi Publications (Mount Horeb, Wis.) 1971.
36. Winterstein, J. F.: *Chiropractic Spinographology.* Published by the National College of Chiropractic (Lombard, Ill.) 1970.
37. Vladeff, T., Hardy, M.: The theory of the fixation points determined by the double full spine turntable technique. *National Chiropractic J.* 15: No. 6:20–3, 56–7, 1945.
38. Vladeff, T., Ulrich, S., Hardy, M.: The value of postural spinography in the practice of scientific chiropractic. *National Chiropractic J.* 18: No. 3:13–6, 1948.
39. Gregory, R. R.: *Manual for Upper Cervical X-ray Analysis.* published by the National Upper Cervical Chiropractic Assn. (Monroe, Mich.) 1971.
40. Messer, A. R.: The cervical subluxation, its correction and effect upon the body. *J. Clin. Chiropractic Archives.* Ed. 2:122–34, 1972.
41. Pettibon, B. R.: *Pettibon Method Cervical X-ray Analysis and Instrument Adjusting.* Published by B. R. Pettibon and Assoc. (Tacoma, Wash.) 1968.
42. Blair, W. G.: Blair Clinic of Lubbock, Texas. *Digest of Chiropractic Economics* 14: No. 1:10–2, 1971.
43. Mears, D. B.: Analysis of lateral–cervical X-ray. *Digest of Chiropractic Economics* 14: No. 3:32–3, 1971.
44. Mears, D. B.: Analysis of AP–cervical X-ray. *Digest of Chiropractic Economics.* 14: No. 1:44–5, 1971.
45. Mears, D. B.: Analysis of lateral–cervical X-ray. *Digest of Chiropractic Economics* 14: No. 4:36–7, 1972.
46. Levine, J. I., Howe, J. W., Rolofson, J. W.: Radiation exposure to a phantom patient during simulated chiropractic spinal radiography. *Rad. Health Data and Reports* 12: No. 5:245–51, 1971.
47. *Nationwide Evaluation of X-ray Trends Tabulation, Representative Sample Data.* Published by the Bureau of Radiological Health, FDA, USDHEW, 1974.
48. Suh, C. H.: The fundamentals of computer aided X-ray analysis of the spine. *J. Biomech.* 7:161–9, 1974.
49. Frigerio, N. A.: Stowe, R. S.: Howe, J. W.: Movement of the sacroiliac joint. *Clin. Orthop.* 100:370–7, 1974.
50. Hviid, H.: Functional radiography of the cervical spine. *Scientific Yearly of the Lincoln Chiropractic College* 1:60–88, 1966.
51. Davis, A. G.: Injuries of the cervical spine. *J.A.M.A.* 127: No. 3:149–56, 1945.
52. Jackson, R.: *The Cervical Syndrome.* 3rd Ed. Thomas *(Springfield, Ill.) 1966.*
53. Cobb, J. R.: *Outline for the Study of Scoliosis.* American Academy of Orthopedic Surgeons, Instructional Courses. Edwards (Ann Arbor) 5:261–75, 1948.
54. Krausova, L., and Lewit, K.: The mechanism and the measuring of the mobility in the craniovertebral joints during lateral inclination. *Acta Univ. Carol Med.* 21:123–6, 1965.
55. Mehta, M. H.: Radiographic estimation of vertebral body rotation in scoliosis. *J. Bone Joint Surg.* 55B: No. 3:513–20, 1973.
56. Jirout, J.: The normal mobility of the lumbosacral spine. *Acta Radiol.* 47:345–8, 1957.
57. Fielding, J. W.: Cineroentgenography of normal cervical spine. *J. Bone Joint Surg.* 39A:1280–8, 1957.
58. Jones, M. D.: Cineradiographic studies of the normal cervical spine. *Calif. Med.* 93: No. 5:293–6, 1960.
59. Jones, M. D.: Cineradiographic studies of degenerative disease of the cervical spine. *J. Can. Assoc. Radiol.* 12:52–5, 1961.
60. Jones, M. D.: Cineradiographic studies of the collar-immobilized cervical spine. *J. Neurosurg.* 17: No. 4:633–7, 1960.
61. Jones, M. D.: Cervical spine cineradiography after traffic accidents. *Arch. Surg.* 85:974–81, 1962.
62. Jones, M. D.: Cineroentgenographic studies of patients with cervical spine fusion. *Am. J. Roentgenol. Radium Ther. Nuc. Med.* 87:1054–7, 1962.
63. Jones, M. D.: Cineradiographic studies of the high cervical spine. *Arch. Surg.* 94:206–13, 1967.
64. Bard, G.; and Jones, M. D.: Cineradiographic recording of traction of cervical spine. *Arch. Phys. Med. Rehab.* 45:403–6, 1964.
65. Bard, G.; and Jones, M. D.: Cineradiographic analysis of laminectomy in cervical spine. *Arch. Surg.* 97:672–7, 1968.
66. Woesner, M. E.; Mitts, M. G.: The evaluation of cervical spine motion below C–2: a comparison of cineroentgenographic and conventional roentgenographic methods. *Am. J. Roentgenol. Radium Ther. Nuc. Med.* 115:148–54, 1972.
67. Rich, E. A.: Observations noted in 11,000 feet of experimental cineroentgenographic film. *J. Am. Chiropractic Assn.* 1: No. 3:11–12, 1964.
68. Rich, E. A.: Cineroentgenographic observations of the human systems. *Scientific Yearly of the Lincoln Chiropractic College* 1:1–9, 1966.

69. Howe, J. W.: Observations from cineroentgenographic studies of the spinal column. *J. Am. Chiropractic Assn.* 7: No. 10:65–70, 1970.
70. Howe, J. W.: Cineradiographic evaluation of normal and abnormal cervical spine function. *J. Clin. Chiropractic Archives.* Ed. 2:76–88, 1972.
71. Penning, L: Nonpathological and pathological relationships between the lower cervical vertebrae. *Am. J. Roentgenol. Radium Ther. Nuc. Med.* 91:1036–50, 1964.
72. Sandoz, R.: Newer trends in the pathogenesis of spinal disorders. *Annals Swiss Chiropractic Assn.* 5:93–179, 1971.
73. Janse, J.: Dr. Illi does research at National College. *J. National College of Chiropractic* 22: No. 1:9, 1950.
74. Janse, J.: *Basic Clinical Phenomena as they Relate to the Art of Specific Reduction of Spinal Segmental Dysarthrias.* published by the National College of Chiropractic (Lombard, Ill.) 1973.
75. Yochum, T. R.: Stress studies of thoracolumbar scoliosis. *J. Clin. Chiropractic Radiographic* Ed. 9–19, 1973.
76. Taylor, D. B.: Biomechanics of the spine under stress. *J. Clin. Chiropractic Education* Ed. 61–73, 1973.
77. Cox, J. M.: Diagnosis, mechanism, and treatment of lumbar disc protrusion and prolapse, part 1. *J. Am. Chiropractic Assn.*
78. Cox, J. M.: Diagnosis, mechanism, and treatment of lumbar disc protrusion and prolapse, part 2. *J. Am. Chiropractic Assn.*
79. Herlin, L.: *Sciatic and Pelvic Pain Due to Lumbosacral Nerve Root Compression.* Thomas (Springfield, Ill.) 1966.
80. Howe, J. W.: Reduction of patient radiation exposure through higher kilovoltage-lower milliamperage techniques. *J. Am. Chiropractic Assn.* 5: No. 9, 1968.
81. Howe, J. W.: Buehler, M. T.: Diagnostic roentgenology with reduced exposure factors. *J. Am. Chiropractic Assn.* 4:S17–22, 1970.
82. Howe, J. W.: Roentgenology in chiropractic, state of the art – 1974. *J. Am. Chiropractic Assn.* 8:S66–72, 1974.
83. Hildebrandt, R. W.: *Synopsis of Chiropractic Roentgenologic Technology.* 2nd Ed. published by the National-Lincoln School of Post-Graduate Chiropractic Education (Lombard, Ill.) 1974.
84. *Continuing Education Bulletin* 1974–75. published by the National-Lincoln School of Post-Graduate Chiropractic Education (Lombard, Ill.) 1974.
85. College Seminar Calendar. A monthly listing of postgraduate courses. *J. Am. Chiropractic Assn.* 1969–74.
86. *The Effects on Population of Exposures to Low Levels of Ionizing Radiation* (the BEIR report). Published by the National Academy of Sciences and the National Research Council (Washington, D.C.) 1972.

The Role of Static and Motion Palpation in Structural Diagnosis

WILLIAM L. JOHNSTON
Professor, Department of Biomechanics
College of Osteopathic Medicine
Michigan State University

I have felt definitely challenged by the fact that there is only one topic in this conference dealing expressly with the term "palpation." Since "hands-on" is the setting of our subject matter, I will try and report in this short period what stand out as simple overriding principles, with the broadest application, in the palpatory diagnosis of somatic dysfunction.

Briefly, palpation offers the clinician the opportunity for a personal and directed, on-the-spot evaluation of the neuromusculoskeletal system. It is the source of those fingertip cues which provide him significant diagnostic information about the patient. These, in turn, guide him during his therapeutic approach.

First of all, what will I not be considering?

1. I am not going to be analyzing any single kind of anatomical structure—not muscle alone, not just bony structure, not ligament or fascia per se.
2. I will not be commenting on intervertebral discs, which are not palpable.
3. I will not be dealing with slackening of ligaments or pinched nerves.
4. I will not be handling terms like "minor intervertebral derangements of discal herniations,"[1] "posterior intervertebral articulations of interspinal ligaments," nor reviewing the axes of intervertebral movements.[2]
5. I will not be talking about facet-joint processes nor joint surfaces.

I will be talking about somatic tissues in toto, tissues in movement, tissues in action, the perception of what it is the tissues reflect when they are apparently static, the perception of tissues in their response to a demand for movement and what they reflect during that movement—specifically what they reflect to the palpating hand.

In communicating about palpatory findings, the clinician's first obligation is to accurately locate where his palpating hand is sensing.[3,4] Is it overlying a particular spinal segmental level? Is it located centrally, laterally, bilaterally, specifically where? In this manner, the site of contact is accurately localized, and the size. That is, does it involve the contact of just the fingertips, fingerpads, or larger to include the whole hand?

Such designations may seem simple, but they are primary in recording the facts; my initial premise is that careful observation is scientific. It is scientific whether you are in a laboratory looking through a microscope, whether you are at the bedside auscultating on the surface of a chest or whether you are in the office palpating on the surface of the back. Palpation has not yet been widely recognized as a careful form of observation that can have a scientific validity. What it demands to be scientific is the factual reporting of what is actually observed, and how it is observed.

This brings me to my second premise, which is closely allied to the palpating and the recording. The observing—the situation of the observer and the observed—bears heavily on the implications of what is to be recorded as fact. I can't emphasize this too strongly because we have all observed in the history of our professions that physicians have observed the same neuromusculoskeletal phenomenon with their fingertips, have climbed onto

597-036 O - 76 - 17

an interpretive framework and then ridden off in all directions. There is a need to carefully delineate what is perceived at the hand of the observer and what is conceived in the mind of the observer. Often the relation between the two becomes a tenuous one.

It will be helpful at this point if you will presume that I am standing in front of a huge blackboard (see page 252), picture my left side of the blackboard as listing items of theory and my right side as listing items of fact. What can the palpating physician (observer) report on the right side of the blackboard as a first level record of careful observation? First, the rigors of static palpation. The subject is seated and the operator runs two fingers lightly down the paraspinal areas overlying the thoracic spine. He can report encountering differences at different levels. He can report that this area differs from that area. (Experience with braille can verify this fact.) So at the top of our board on my right we have differences, areas of tissue texture changed [3,4] from their immediate surroundings as reportable palpatory fact that should be able to be confirmed by a second observer. If one observer describes that difference as congestive or tense or ischemic or fibrotic, the statement emerges as a level of interpretation that may, or may not, be confirmable.

Second, he can factually report the location of this finding. An example would be T2–T4 on the right or even bilaterally at the level of T6. The size and extent of the difference can be carefully observed and measured. Example: a unilateral patch on the right extending vertically 4 centimeters and horizontally 1.5 centimeters or a bilateral strip extending vertically 1 centimeter and horizontally 7 centimeters. Even with approximate numerics, the basic picture emerges reasonably factually.

By an appreciation of the amount of pressure in palpation, we may gauge the approximate depth of our finding and begin to interpret the tissue we're palpating. The gradation of pressure can be recorded on the right, the interpretation on the left.

Pain and sensitivity [1,5,6] represent significant criteria and are recordable as fact. However, they rely specifically on the subjective nature of the patient's response. As such, they are of a lesser reliability than the objective palpatory cues of a skilled observer.

One of the tissues we are beginning to palpate here is bony. We can sense irregularities of the underlying soft and bony tissue, prominences, depressions and asymmetries. The perceptions are recordable on my right, as to location, size, distribution, (facts); the interpretation would be here on my left, that is, bony malposition,[7] subluxation, sprain (concepts).

Where deep pressure palpation ends and joint probing begins, there may be a point for controversy. The situation is still one of static palpation with the subject resting. The technique of pressure probing may involve one thumb or one reinforced by the other.[8] The actual scene of this kind of palpation might involve the subject in a prone position with pressure probing by the operator downward over the paraspinal musculature at a given segmental level, comparing one side with the opposite, right with left. Encountering a sense of resistance at one side (as compared to a sense of yield at the other side) could be recorded here on my right, locating the point of resistance to pressure probe. On my left side you might have the interpretation of a zygapophyseal lock,[9] or of the vertebra positioned with the transverse process posterior on the side expressing resistance.

One variation of this kind of joint probing involves pressure with one thumb over a bony prominence at a joint area (spinous or transverse process), often with the second thumb creating a counter pressure on an adjacent bone.[10] Each still senses resistance to pressure probe recorded here on my right. If a resistance is elicited by pressure against the side of the vertebral spinous process, it is often interpreted as restricted rotation. If it is elicited by pressure in a medial direction against the articular process, it is often interpreted as restricted sidebending; if bilaterally in an anterior direction (patient still prone) it may be interpreted as restricted extension. These interpretations are all recordable as theory. Other terms like "hypermobility," "joint lock," "stretched ligaments," "roughened articular cartilages," "irregular joint surfaces," and "adhesions," [11] are also recordable under theoretical data.

On my left side of the backboard, a number of concepts are accumulating that have given rise in the past to much heated discussion. On the right side, items of skilled perception that need more careful recording are noted.

So far we have been talking about local asymmetry and tissue change. I am proposing that these represent two cues of a triad that is equally as reflective of problems in the neuromusculoskeletal

system as the triad of rate rhythm and murmur in the cardiovascular system. The third cue is motion change. In dealing with the issue of motion palpation, let me begin by suggesting that this is not necessarily a familiar methodology.

Most joint motion testing procedures have separated the joint as an isolate from the rest of the body.[2,6,12] With the body static and resting, a joint structure has been evaluated with some type of pressure probe or testing procedure for the joint itself and its motion range. Such methods have led to a concept and a terminology of motion that relates the position of one bone to another, and implies a restriction of motion as a locking of a joint in a given position. The facts have emerged as a snapshot photograph.

Consider for a moment the commonly used movement patterns of the subject's musculoskeletal system. These are not movements of one bone alone, nor just at a single joint. They are movement patterns involving many segments, with each segmental part organized and contributing to the total patterned performance, whether the pattern involves primarily segments of a spine or segments of a limb.

With a subject seated, the operator can introduce a gross movement passively, palpating first of all for the sense of total range, the extent of the total performance, and record limitation and asymmetry. Rotational, lateral, forward and backward bending elements are all appropriate for gross motion testing.

We are talking about the dynamics of total body movement, or at least the movement of a region of the body, involving a pattern that is segmented, the summation of many parts. Does each part easily go along with and contribute to the gross movement pattern—or does it express resistance to the particular demand for movement?

Motion palpation deals with the behavior of these segmental parts *during* motion.[13] Once identifying a local segmental area of tissue texture change, the operator palpates for the motion of that segmental part by monitoring it individually while introducing a variety of gross movement patterns (gross rotation of the shoulders, gross forward bending of the head and neck, and so on).

It is a light palpation, during motion, with the fingerpads overlying the defined segmental level as it participates in a particular gross movement being introduced. The procedure utilizes the same sensations for cues at the fingertips with the operator registering the same sense of either compliance or resistance, but this time during movement. These are picked up by a light palpatory technique[14] that has been described as "nonperturbational"; it does not intrude in the action and therefore offers an increased validity in a scientific setting.

Findings during motion palpation add further specific descriptors to our phenomenon here on my right under perceived facts. Up until now, during static palpation, we've localized an area of tissue texture, say overlying T2 bilaterally (changed from its surrounding areas above and below), and noted its subjective sensitivity and the asymmetry of its relationships and the resistances to pressure probe. Now in motion palpation we can record at T2 an immediate increasing resistance (active binding) during the initiation of gross rotation of the shoulders to the left as compared to its increasing compliance, (active easing) when the shoulders are rotated to the right. Range is not measured here. This is not a resistance to all movements. Rather, it is a constantly changing response of local segmental tissues during movement—bind accelerating during some movements, bind decelerating during others.

The conceptual framework is behavioral. The relationship is of a segmental part, not to the one below but to the whole, that is to the whole movement of which its behavior is being evaluated as it contributes its individual segmental part. Here the phenomenon is viewed as one of lesioned segmental behavior within the dynamics of movement; this time the picture is a motion picture not a photograph. We are not asking the question: "A lesion of what?" with a conceptual answer of a bone, joint, ligament, a muscle, and so on. Rather, we are asking the question: "Lesioned in response to what?" with a factual answer: "In response to the particular gross demand for movement that initiates the response." This is a definable, reportable fact. It is a careful observation that is scientific, one that can be confirmed by a second observer.

Let's consider for a moment what Sherrington had to say about motion and position. His statement was that position follows motion like a shadow.[15] The corollary to this statement is that motion is position on the run. The fact to be considered is that motion and position are integral stages of the same process. Realizing that we had already identified a lesioned phenomenon of t.t.a. (tissue texture abnormality) during a so-called static palpation in the resting

position, we might now consider asking our same question in this static situation: "Lesioned in response to what?" and suggest as a plausible answer that it may be lesioned in response to the demand to assume a position. Actually this is a particular *postural* positioning, the one that the subject has been asked to take during the examination; each segment is being asked to contribute to that positioning so that each segment is in the right place in the sequence. If this is so, we can easily probe this assumption by monitoring the already tense tissue texture at T2, as we alter the subject's position. When we do, the tissue texture immediately changes,[7] gets more resistant or more compliant depending on the new position being demanded. But from one position to another position is movement, bearing out the direct relationship of our phenomenon to position and/or motion. Binding has existence only as a response to the demand being currently placed on it—be it motion or position.

There is an impact to this kind of observation, attentive to the methodology of palpation and the facts perceived in palpation. It allows us to integrate all of the diagnostic findings, in both static and motion palpation on the right side, put them together and organize a single common conceptual framework on the left side that will deal with all descriptors of the phenomenon of somatic dysfunction—deal with palpatory findings in direct relation to the dynamics of demand and response—and deal with the commonality of demands for both position and movement and their existence together as a single continuum of demand. With proper attention to the methodology of palpation (*how* the facts are observed) and the clinical diagnostic findings of palpation (*what* is factually observed), we have procedures that can be reproduced and findings that can serve as specific descriptors within a laboratory setting.

Palpation is a careful form of observation that can be scientific. I have tried to address a verbal *logic*, and suggest a kind of physio*logic*, as the basis for a theoretical model which will be an appropriate guide for clinical research.

Somatic Dysfunction*

Fact	Theory (caused by)
Static Palpation	
Subject seated:	
Tissue texture abnormality-paraspinal	
Level T2 bilateral	
1 cm. vertical, 7 cm. horizontal	Sprain
(subjective pain)	Subluxation
Irregularity, asymmetry bony prominences	Bony malposition, zygapophyseal lock
Subject prone:	
Resistance to pressure probe at rt.	T2 on T3, transverse process posterior on right
Resistance to pressure in a medial direction from rt.	T2 on T3, sidebent to left
Resistance to pressure in an anterior direction, bilaterally	T2 on T3, restricted in extension, positioned in flexion
	T2 joint lock on T3 (roughened articular cartilages, irregular joint surfaces, adhesions)
T.T.A. at T6-increased compliance to pressure probe from posterior, bilaterally	T6-hypermobile joint stretched ligaments
Motion Palpation**	
Subject seated: ↔ t.t.a. at T2	Binding in relation to postural demands (of the seated position)
Head rotation to rt. ↔ t.t.a. increases	
Head rotation to left ↔ t.t.a. decreases	
Head side bending rt. ↔ t.t.a. increases	Active bind, accelerating or decelerating in relation to the particular demand for movement.
Head side bending lft. ↔ t.t.a. decreases	
Head forward bending ↔ t.t.a. increases	
Head backward bending ↔ t.t.a. decreases	

*Three kinds of palpatory facts about somatic dysfunction: asymmetry, tissue change, motion change.

**The tissues about a moving part constantly reflect its compliance or resistance (behavior) in response to specific movement patterns (position and changing position).

REFERENCES

1. Maigne, R., *Orthopedic Medicine*, translated and edited by Liberson, W. T., Springfield, 1972, Charles C Thomas.
2. Ho, R., Testing Intervertebral Joint Movement, *J.A.O.A.* 61:635-639, April 1962.
3. Denslow, J. S., Pearson, J., and Gutensohn, O., An Approach to Skeletal Components in Health and Disease, *J.A.O.A.*, 50:399-403, April 1951.
4. Johnston, W. L., Manipulative Specifics, *J.A.O.A.* 61:535-539 March 1962.
5. Denslow, J. S. Palpation of the Musculoskeletal System, *J.A.O.A.* 63:1107-1115, August 1964.
6. Mennell, J. McM. *Joint Pain*, Boston, Little Brown & Co., 1964.
7. Downing, C. H., *Principles and Practice of Osteopathy*, Kansas City, Mo., Williams, 1923.
8. Beal, M. C., Motion Sense, *J.A.O.A.* Vol. 53, No. 3, 151-153, 1953.
9. Northup, G. W., Osteopathic Lesions, *J.A.O.A.* 71:854-865, June 1972.
10. Wilson, P. T., Principles and Techniques of Individual Lesion Diagnosis, Yearbook *AAO*, pp. 193-194. 1955.
11. Stoddard. A., *Manual of Osteopathic Practice*, New York. Harper & Row, 1969.
12. Strachan, W. F., Joint Motion Testing and Forces Involved In Passive Motion. *J.A.O.A.* 66:271-277, November 1966.
13. Johnston, W. L., Segmental Behavior During Motion, Part I: A Palpatory Study of Somatic Relations, *J.A.O.A.* 72:352-361, Dec. 1972.
14. Bowles, C. H., Functional Orientation for Technic, Part III: Yearbook *AAO*. 53-57, 1957.
15. Swinyard, C.A., ed.: *Role of the Gamma System in Movement and Posture*. Association for the Aid of Crippled Children, New York, 1964, p. 45.

The Role of Thermography and Postural Measurement in Structural Diagnosis

MARTIN E. JENNESS
Director of Research
Palmer College of Chiropractic
Davenport, Iowa

INTRODUCTION

The purpose of this paper is twofold: (1) To determine how thermography and posture have been used in structural diagnosis and (2) to determine in what way the analytical tools employed in thermographic and postural assessment may be applied or modified to extend our knowledge in the analysis and/or diagnosis of human structure. As these tools are quite disparate with respect to materials, methods, and application in evaluating or diagnosing structure, they will be considered separately. The use of thermography will be considered first.

THERMOGRAPHY

Hippocrates (400 B.C.) was the first to recognize the significance of body temperature regulation relative to pathological changes in man. Over 2,000 years later, Claude Bernard (1879) discovered that the maintenance of body temperature was under the control of the nervous system.

Since the introduction of the neurocalometer in 1924 by Evins,[1] skin temperature measurements have played a significant role in the clinical practice of chiropractic. Correlation of roentgen and other clinical findings with skin temperature patterns have assisted doctors of chiropractic in determining the presence of vertebral subluxations.

Heat loss through the skin surface may occur by four different physical mechanisms: Radiation, convection, conduction, and evaporation. In the basal state, most body heat is lost through radiation.[2, 3, 4, 5]

Thermography is a technique that records infrared radiations and produces a pictorial recording or "map" of the temperature pattern over the area scanned. Typical thermographic patterns of the back follow (figures 1–4).

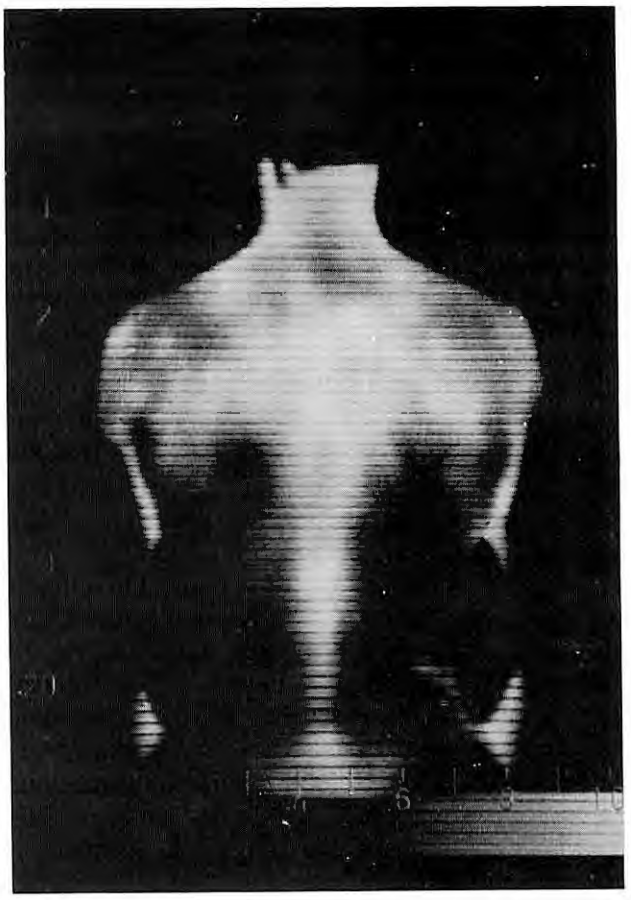


Fig. 1. On this male subject, warm areas are represented as light areas and cold areas appear dark. Note the warm streak along the vertical median line of the back, the warm areas between the shoulders, and the cold areas on the lateral margins of the torso.

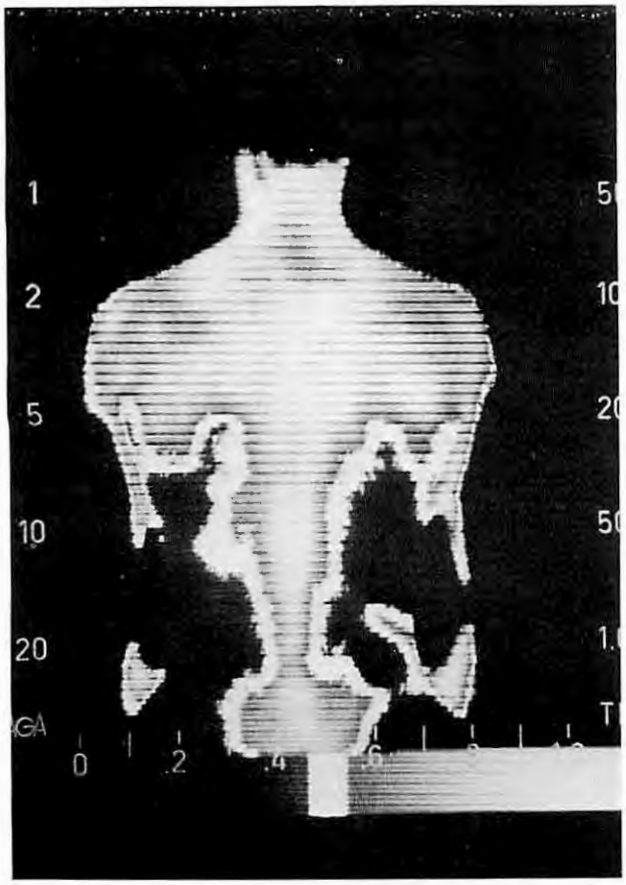


Fig. 2. This is the same subject as in Figure 1. Cold areas are outlined in brilliant white. Note that these are found on the lateral margins of the lower thoracic and lumbar regions of the torso.

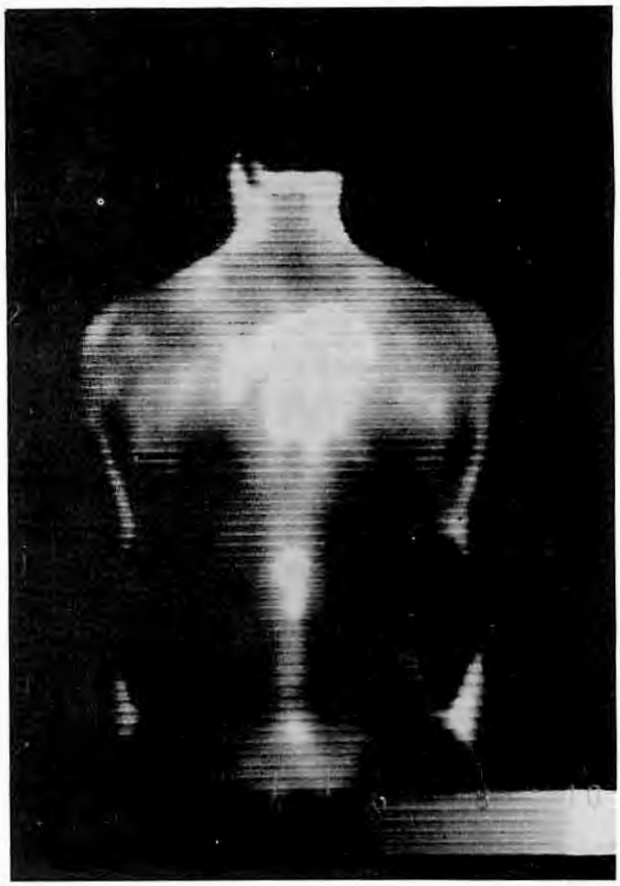


Fig. 3. Thermograph of the same subject with the warm areas highlighted in white. Note the warm area between the shoulders and the lower thoracic-upper lumbar area.

The clinical application of infrared scanning systems to map skin temperature distributions stems from the observation by Lawson[6] that the skin over a malignant tumor of the breast is frequently between 1° to 3° hotter than surrounding skin.

The role of thermography in clinical practice is still to be established. It is a young diagnostic tool in which experience is rapidly accumulating. In cancer, vascular disease, neurology, and orthopedics, enough data are available to merit attention.

In orthopedics, thermographic techniques have been found useful in evaluating musculoligamentous injuries to the spine.[7] In most instances, the thermogram localizes the hyperemia incident to the local insult.[8, 9]

In evaluating herniated discs and nerve root compression in the lumbar region of the spine, investigators have reached conflicting conclusions regarding the diagnostic merits of thermography.[10, 11, 12, 13, 14] The dichotomies are apparently due to a disparity of opinion as to what constitutes a normal thermogram. Also, because of anatomically reflected radiation, the normal patient often exhibits increased heat in the lumbosacral area, the most common site for clinically significant disc disease.[11, 14, 15] Some investigators are avoiding this problem by basing their diagnosis on thermograms of the buttocks, thigh, and calf.[16, 17]

A number of physiologic mechanisms are involved in forming skin temperature patterns. Muscular contraction plays a significant role in determining the nature of thermographic patterns.[18] In muscle, heat appears as a byproduct of muscular contraction and recovery.[7, 19]

The role of the nervous system in skin temperature regulation is of paramount importance, particularly in the practice of chiropractic. There are two systems which regulate the effector systems of temperature control: the skin receptor mechanism and the central regulating mechanism.[20, 21, 22]

The amount of blood passing to the skin is governed by local vascularities, which in turn are

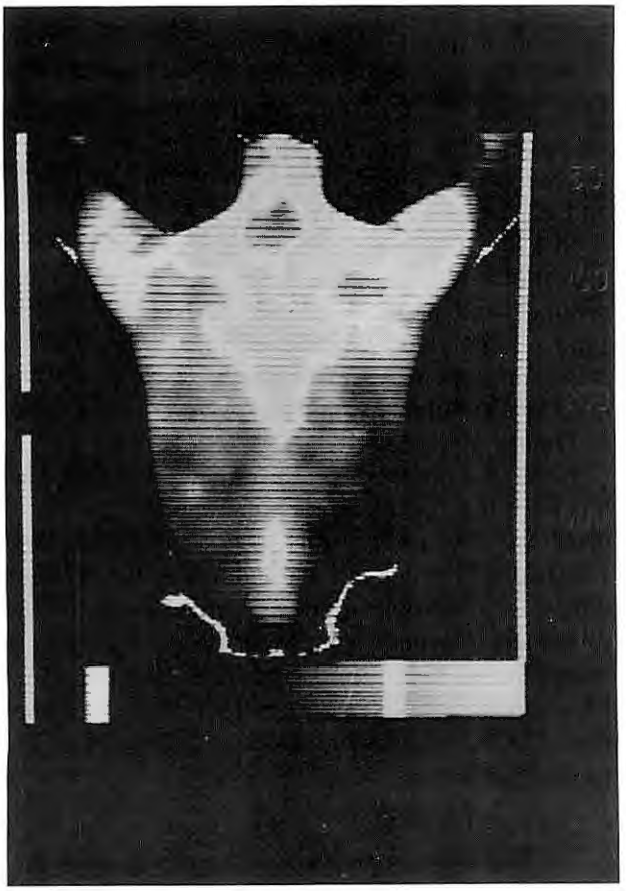


Fig. 4. Both warm and cool areas have been highlighted on this different subject. Note warm areas in the upper back and cool areas at the lateral inferior margins of the torso.

controlled by the sympathetic nervous system.[23, 24, 25, 26, 27]

Central temperature regulation is mediated by the hypothalamus in response to changes in blood temperature.[20, 22, 25, 26] In addition, sensory impulses from the skin on cooling can act via the hypothalamic mechanism to increase body heat. This is accomplished through shivering, piloerection, and vasoconstriction.

Stimulation of sympathetic fibers supplying the skin causes constriction of the cutaneous vessels in the area supplied by the stimulated fibers. Vasoconstriction causes the skin to blanch, and eventually cool. Conversely, if sympathetic pathways are interrupted through surgery, small arteries and arterioles dilate, causing an increase in blood flow and a rise in skin temperature. Efferent sympathetic fibers carry persistent electrical discharges, which are chiefly concerned with vasoconstriction.

Nerve irritation, such as partial nerve injury, produces cooling; whereas a complete interruption of nerve impulse transmission produces warming.[28, 29, 30, 31] The thermogram, therefore, may be a valuable tool in the differential diagnosis of neuropathies. Besides determining whether nerve injury is partial or complete, thermography may help determine whether a neuropathy is toxic, traumatic, or compressive in nature.[14] Injury to the spinal cord also results in marked changes in skin temperature patterns.

A number of orthopedic conditions which may or may not involve neuronal dysfunction have exhibited thermographic manifestations. In cases of spondylolisthesis, consistently warm areas were demonstrated over the area of subluxation.[11] In addition, inflammation from within, such as osteoarthritis arising from inflammation of synovial membranes or rheumatoid arthritis with inflammation around joints, may be displayed by thermography.[32, 33, 34, 35]

It is clear that the role of the nervous system in peripheral vascular control is of primary importance. The thermograph is a valuable indicator of the blood vascular system's response to neural control systems. In chiropractic, the greatest potential of thermography would appear to be its use as an adjunct in determining the relationship between structural disrelationships and neuropathic processes.

POSTURE

The term posture generally implies a characteristic stance or position of the body. In the literature, most definitions are coupled with the adjective "good"; however, there is little agreement among authorities as to what constitutes good posture or how it should be defined. This was made clear in a review by Massey[36] in which he stated:

> In the literature related to standards of "good" posture, there were many definitions describing the correct upright position. Authorities emphasized segmental alignment, pelvic inclination, carriage of the head and neck, the distribution of weight on the feet, the curves of the spine, abdominal protuberance, the position of the chest, and the center of gravity in the trunk. When considered collectively, there seemed to be general agreement in the choice of criteria used to describe the conditions for "good" posture.

Thus, "good" posture should perhaps be treated

as a general abstraction, implying an idealized relationship between structural form and function, with specific attributes tied operationally to the particular touchstone used in its assessment.

In a limited paper of this nature, it is not possible to consider all aspects of posture. In keeping with the title of this review and the purpose of this workshop, the following topical delimitation expressed in question form seemed appropriate: (1) How can posture be assessed? (2) Is there a relationship between posture and health? and (3) How can poor posture be prevented and/or corrected?

Assessment of Posture

Man's unique erect posture has attracted study by physical educators and various members of the healing arts. These investigations have ranged from the highly subjective visual grading of posture to more objective methods utilizing photographs, X-rays, silhouettes, center of foot pressure apparatus, electromyography, and moiré topography. This review is limited to a brief description of the more objective methods.

In 1909, Reynolds and Lovett [37] determined the line of gravity of the body and related it to the A–P spinal profile as determined by a spinal "conformateur." This approach to postural measurement was later modified and employed by Cureton and Wickens [38] and Jenness, Speijers, and Silverstein.[39] However, many of the early postural investigations utilized objective refinements of previous subjective work. The silhouettograph described by Fradd [40] was used in the thirties to describe various body contours, landmarks, and angles of intersection of body parts. In 1943, Massey [36] in addition to his own criteria, took measurements utilizing landmarks previously described by Goldthwait and Kellogg and correlated them with subjective postural ratings. Similarly, MacEwan and Howe,[41] in 1932, correlated subjective ratings with various body measurements taken from photographs.

In 1938, Hellebrandt and Braun [42] measured shifts of the line of gravity, or more correctly, the center of foot pressure in the A–P and lateral planes. Thomas and Whitney [43] in 1959, utilized a force analysis platform and accelerometer to relate center of gravity deviations with center of foot pressure shifts. In 1971, Stevens and Tomlinson [44] described a method using displacement transducers to record movements of the center of gravity, an improvement over earlier drawing pens attached to the head or shoulders of postural subjects.[45, 46]

The dynamic and static aspects of erect posture have been demonstrated by electromyographic measurements of postural muscles by Joseph and Nightingale,[47] Floyd and Silver,[48] Nachemson,[49] and Klausen and Rasmussen.[50]

In 1906, Fitz [51] devised a celluloid grid to measure spinal curves, scolioses, body contour, and scapular and pelvic levels. A modification of this type of apparatus capable of measuring rotation of body parts was developed by Johnston [52] and used by Vilholm [53] in an extensive postural study of Danish children.

Finally, a method of describing irregular surfaces by the use of moiré topography was developed by Takasaki [54] and used by Terada and Kanazawa [55] to depict the human skull in three dimensions. Application of moiré topography to the erect human posture, as described by Free (paper presented at the Symposium on Biomedical Photogrammetry, September 13, 1974, Washington, D.C.), shows promise as a new tool for postural analysis.

Posture and Health

The literature provides a rich and varied assortment of specific symptoms, conditions, and diseases that have been associated with poor posture. In addition, a number of general statements relating posture and health have been forwarded by various investigators. Hansson [56] stated:

> The medical profession was slow in accepting poor posture and poor health as one of cause and effect. In 1740, Nicholas Audry (Andry) taught that many illnesses in children had their origin in imperfect body mechanics. A generation ago the foundation for our present conception of body mechanics and health was brought out by Goldthwait and continued by Osgood. All surveys of posture in our primary schools show less illness, as proved by absences among children taught good body mechanics. Similar surveys in our colleges indicate a definite correlation between good functional health and good body mechanics.

This position was not shared by Keeve [57] who contended:

> Unfortunately, much of physical education and training has (unknowingly)

incorporated the tenets of cult medicine with its emphasis on "body mechanics" and posture.

Other generalized statements were made by Hallock[58] who asserted that poor posture has an adverse effect on the general health and, more recently, by Kuhns[59] who stated that poor posture was associated with many pathological conditions and that good posture may prevent serious disease processes. Thompson[60] lists a large number of conditions involving every major organ system of the body that may be "cured" through postural correction. Finally, Garner[61] averred that proper posture aids in "minimizing fatigue" and "building up a resistance to infections."

As indicated earlier, poor posture has been linked with many specific symptoms, conditions, and pathologies. Dickson[62] tabulated the following conditions that he considered to be tied to faulty posture: Leg, knee, and foot ache; fatigue; nervousness and irritability; failure to gain weight; restlessness at night; constipation; and periodic gastrointestional attacks. Cochrane[63] said that chronic conditions such as gastrointestinal disturbances and arthritis yielded to postural correction. Canter[64,65] emphasized the prophylactic function of good posture in preventing "footstrain, backache, neuritis, and arthritis." Forrester-Brown[66] stated that rheumatism and chronic digestive trouble are commonly related to faulty body mechanics. In an article discussing the importance of the quality of sleep, Mattison[67] said that posture "is generally admitted to affect circulation, respiration, nutrition, and the tonus of the muscles."

A number of studies have been made to try to determine the validity of some of the foregoing claims. In 1917, at the instigation of Brown,[68] medical and postural data were collected on Harvard freshmen. He found that persons having poor posture suffered seven times as many backaches and had one and a half times as many appendectomies as those having good posture. More recently, Fox[69] determined that the incidence of dysmenorrhea amongst college women was significantly greater in those having excessive lumbar curvature than in the controls. Moriarty and Irwin[70] found that "there is a significant association between poor posture and certain physical and emotional factors, namely: Disease, fatigue, self-consciousness, fidgeting, hearing defects, restlessness, timidity, underweight, heart defects, and asthma."

A review on posture would be remiss without reference to structure and low back pain (LBP). That LBP is a common malady deserving continued research attention has been recently demonstrated by a number of investigators. Magora[71] found that 12.9 percent of a large sample of men and women (n = 3316) suffered from LBP. With respect to incidence of LBP in industry, Rowe[72] determined that over a 10-year period, "35 percent of the sedentary workers and 47 percent of the heavy handlers made visits to the medical department for low back pain." In a radiographic comparison of a primitive "squatting tribe in West Central India" with "American and Swedish radiographic studies," Fahrni and Trueman[73] suggested that lumbar lordosis may be implicated in intervertebral disc degeneration. Similar findings had been reported earlier by Jones,[74] who stated that LBP resulted from an increased sacral angle, and by Shannon and Terhune[75] who said that increased lumbar curve "is the most common cause of low back pain." Thus, it would appear that LBP is related to excessive lumbar curvature. This would seem to be particularly significant in that Hagen[76] has shown that there is a strong tendency toward progressive accentuation of the lumbosacral angle during maturation and it is known that the incidence of LBP increases with age.

It would appear that the foregoing mélange of associations between posture and health or disease could quite properly be subsumed and explained by the basic chiropractic hypothesis, which links structural disrelationships to aberrant function of the nervous system, with concomitant tissue dysfunction and/or pathology.

Postural Modification

There is considerable evidence to support the hypothesis that many disease processes are related to faulty posture. This leads to the third question that was posed at the beginning of this review: "How can poor posture be prevented and/or corrected?" Time allows but brief mention of techniques that have been suggested or employed for effecting postural changes.

Some of the most common methods that have been suggested for the prevention or correction of poor posture are: (1) general and specific postural exercises; (2) education (inculcation of an awareness of good postural habits); (3) proper structural balance of the spine, legs, and feet; (4) proper support of the body when sitting or reclining; (5)

good nutrition; and (6) adequate rest. Many authorities consider most or all of the aforementioned factors to be important in the postural equation.[77, 78, 79, 80, 81] Others have emphasized the importance of (1) correct support while sitting;[82] (2) foot or leg imbalance;[83, 84] and (3) postural awareness.[85, 86, 87, 88] However, the greatest attention has been devoted to the use of exercise as a modifier of posture.

Stewart[89] described dynamic postural exercises designed to appeal to the playful nature of children. Haller and Gurewitsch[90] have developed postural exercises based on primitive motion patterns. Individualized exercise programs with frequent testing have been described by Kraus and Weber.[91] Other prescriptive postural exercises have been elaborated by Kendall and Kendall[92] and Pheasant.[93] An exercise to reduce lumbar lordosis for eliminating backache was described by Lankford.[94] Kiernander[95] and Beneš, Fiala and Krtička[96] reported studies which seemed to indicate that exercise is effective in improving posture and/or correcting faulty posture in schoolchildren.

Finally, because of the nature of this conference, one other postural factor deserves special attention; that is, the effectiveness of chiropractic adjustments on changing spinal conformation and balance. That chiropractic adjustments were capable of effecting structural changes of the spine has been demonstrated by comparing spinal X-rays taken before and following chiropractic care.[1, 97, 98, 99] Refer to figures 5 and 6.

In addition, from X-ray measurements taken on "normalized" (corrected) spines, Toftness[99] established normal ranges for spinal shape and balance. Thus, there is objective evidence to support the value of chiropractic adjustments as an effective modifier of posture.

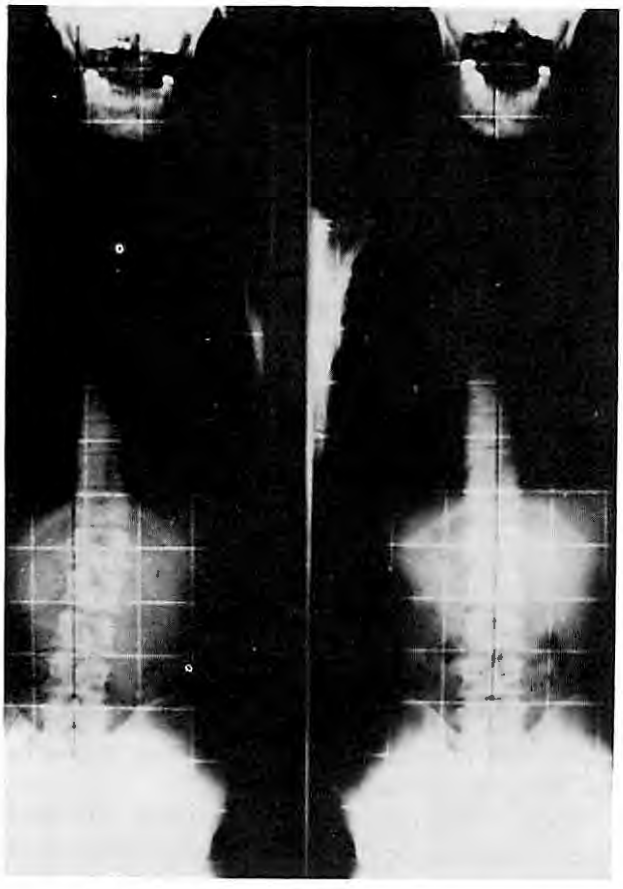


Fig. 5. Male patient, age 41. Suffered low back pain and sciatica. Left: A–P spinal X-ray before chiropractic adjustments. Right: Similar X-ray view taken after 2 months chiropractic care. Patient returned to work 1 month after starting adjustments. (Courtesy Dr. I. N. Toftness, Cumberland, Wisc.)

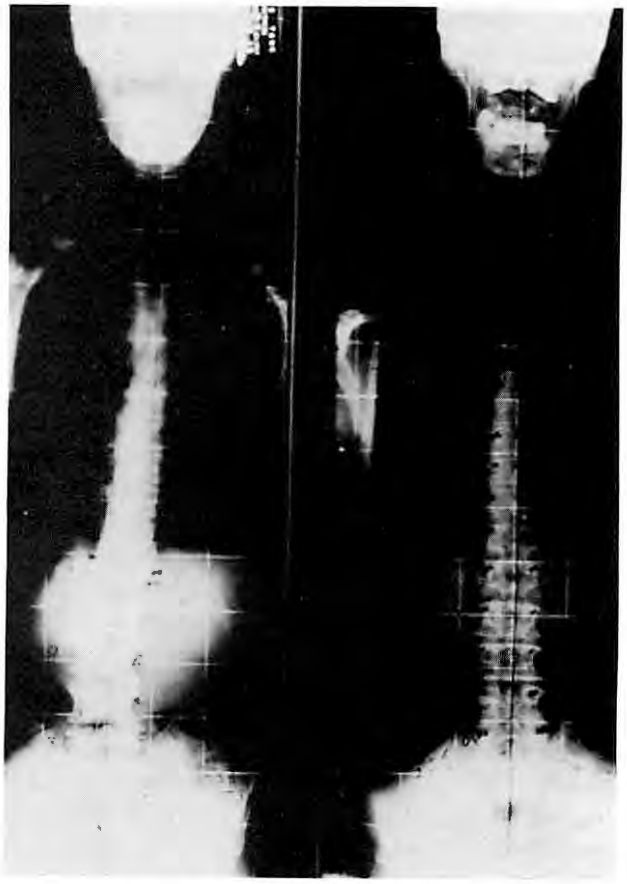


Fig. 6. Male patient, age 51. Medically diagnosed herniated disc between 5L and sacrum. Left: A–P spinal X-ray before chiropractic adjustments. Right: Similar X-ray view taken after 2½ months of chiropractic care. Patient returned to farming job. (Courtesy Dr. I. N. Toftness, Cumberland, Wis.)

SUMMARY AND CONCLUSIONS

It was the purpose of this paper to determine how thermography and postural analysis have been and may be used in structural diagnosis. Although currently being used for diagnosing a variety of conditions and diseases, thermographic techniques may prove to be of great value for investigating the relationship between structural malposition and neuronal dysfunction.

Posture was discussed with respect to definition, methods of assessment, relationship to health and disease, and modification. Considerable evidence was found to suggest that many symptoms, conditions, and diseases are associated with faulty posture. Factors thought to be involved in postural modification included: Exercise; education; proper structural balance of the spine, legs, and feet; proper support of the body while sitting or reclining; good nutrition; and adequate rest. Studies dealing with these modifiers of posture were discussed, including the value of chiropractic adjustments in normalizing the spine. However, the need for further research was made evident by the paucity of controlled longitudinal studies involving all of the aforementioned factors that may affect posture.

REFERENCES

1. Palmer, B. J. *Precise posture-constant spinograph comparative graphs* (Vol. XX), Davenport, Palmer School of Chiropractic Press, 1938.
2. DuBois, E. F. *The mechanisms of heat loss and temperature regulation.* Lane Medical Lectures, Stanford, California, Stanford University Press, 1937.
3. Hardy, J. D., Soderstrom, G. F. "Heat loss from nude body and peripheral blood flow at temperatures of 22C to 35C." *J. Nutr.*, 16, 493–510, November 1938.
4. Hardy, D. "Radiation of heat from human body." *J. Clin. Invest.*, 13, 593–604, July 1934.
5. Hardy, J. D.; Muschenheim, C. "Radiation of heat from human body; emission reflection and transmission of infrared radiation by human skin." *J. Clin. Invest.*, 13, 817–31, 1934.
6. Lawson, R. N. "A new tool in the investigation of breast lesions." *Canadian Services Medical Journal*, 13, 517, 1957.
7. Karpman, H. L., Knebel, A., Semel, C. J., and Cooper, J. "Clinical studies in thermography, II. Application of thermography in evaluating musculoligamentous injuries of the spine—a preliminary report." *Arch. Environ. Health*, 20, 412–417, 1970.
8. Connell, J. F., Jr., Morgan, E., Rousselot, L. M. "Thermography in trauma." *Ann. N.Y. Acad. Sci.*, 121, 171–176, Oct. 9, 1964.
9. Tai, P. L. "An investigation of clinical applications of mesomorphic cholesteric liquid crystals in podiatric medicine. A preliminary study." *J. Am. Podiatry Assoc.*, 63(4), 119–28, April 1973.
10. Edeiken, J., Wallace, J. D., Curley, R. F., and Lee, S. "Thermography and the herniated disc." *Am. J. Roentgenol.*, 102, 790–796, 1968.
11. Heinz, R. E., Goldberg, H. I., Taveras, J. M. "Experiences with thermography in neurologic patients." *Ann. N.Y. Acad. Sci.*, 121, 177–189, Oct. 9, 1964.
12. Albert, S. M., Glickman, M., and Kallish, M. Thermography in orthopedics. *Ann. N.Y. Acad. Sci.*, 121, 157–170, Oct. 9, 1964.
13. Goldberg, H. I., Heinz, E. R., and Taveras, J. M. Thermography in neurological patients. *Acta. Radiol. Diagn.*, 5, 786–795, 1966.
14. Marinacci, A. A. Thermography in the detection of non-neurological imitators of peripheral nerve complexes. *Bull. Los Angeles Neurol. Soc.*, 30(1), 1–11, March 1965.
15. Abernathy, M. R., Ronan, J. A., and Winsor, D. T. Diagnosis of coarctation of the aorta by infrared thermography. *Am. Heart J.*, 82(6), 731–41, December 1971.
16. Duensing, F., Becker, P., and Rittmeyer, K. Thermographic findings in lumbar disk protrusions. *Arch. Psychiatr. Nervenkr.*, 217(1), 53–70, 1973.
17. Lebkowski, J., Polocki, B., Borucki, Z., Dudek, H., Szpakovicz, P., and Tomczyk, H. Determination of the level of prolapsed intervertebral disk in ischalgia by means of an electric thermometer. *Pol. Tyg. Lek.*, 28(24), 907–908, 1973.
18. Hill, A. V. Thermodynamics of muscle. *Nature*, 167(4245). 377–380, 1951.
19. Cooper, T., Randall, W. C., and Hertzman, A. B. Vascular convection of heat from active muscle to overlying skin. *J. Appl. Physiol.*, 14, 207–211, 1959.
20. Cranston, W. I. Temperature regulation. *Br. Med. J.*, 5505, 69–75, 1966.
21. Roberts, T. D. M. Temperature control in fever. *Lancet*, 2, 1015–1018, 1970.
22. Benzinger, T. H. Heat regulation: homeostasis of central temperature in man. *Physiol. Rev.*, 49(4), 671–759, October 1969.
23. Pickering, G. W. The vasomotor regulation of heat loss from the human skin in relation to external temperature. *Heart*, 16, 115–135, July 1932.
24. Lewis, T., and Pickering, G. W. Vasodilatation in the limbs in response to warming: with evidence for sympathetic vasodilator nerves in man. *Heart*, 16, 33–51, October 1931.
25. Hemingway, A., and Price, W. M. The autonomic nervous system and regulation of body temperature. *Anesthesiology*, 29(4), 693–701, 1968.
26. Barbour, H. G. Heat-regulating mechanism of body. *Physiol. Rev.*, 1, 295–326, 1921.
27. Uprus, V., Gaylor, J. B., Williams, D. J., and Carmichael, E. A. Vasodilation and vasoconstriction in response to warming and cooling the body; study in patients with hemiplegia. *Brain*, 58, 448–455, 1935.
28. Winsor, T., and Bendezu, J. Thermography and the peripheral circulation. *Ann. N.Y. Acad. Sci.*, 121, 135–156, Oct. 9, 1964.
29. Lippmann, H. I., Winsor, T., and Hume, M. Medical instrumentation in peripheral vascular disease; peripheral vascular disease study group sub-committee on peripheral

vascular disease instrumentation. *Circulation*, XLV, A-285-A-291, January 1972.
30. Normell, L. A., and Melander, O. Thermographic method for application in clinical neurology. *Acta Neurol. Scand.* (Suppl), 51, 471-472, 1972.
31. Winsor, T. Vascular aspects of thermography. *J. Cardiovasc. Surg.*, 12(5), 379-88, 1971.
32. Boas, N. F. Thermography in rheumatoid arthritis. *Ann. N.Y. Acad. Sci.*, 121, 223-234, Oct. 9, 1964.
33. Agarwal, A. , Lloyd, K. N., Dovey, P. Thermography of the spine and sacro-iliac joints in spondylitis. *Rheumatology and Physical Medicine*, X(7), 349-355, 1967.
34. Huskisson, E. C., Berry, H., Browett, J. P., and Balme, H. W. Measurement of inflammation. II. Comparison of technetium clearance and thermography with standard methods in a clinical trial. *Ann. Rheum. Dis.*, 32(2), 99-102, 1973.
35. Viitanen, S. M., and Laaksonen, A. L. Thermography in juvenile rheumatoid arthritis. *Acta. Rheum. Scand.* 16, 91-98, 1970.
36. Massey, W. W. A critical study of objective methods for measuring anterior posterior posture with a simplified technique. *Research Quarterly*, 14(1), 3-21, 1943.
37. Reynolds, E., and Lovett, R. W. A method of determining the position of the center of gravity in its relation to certain bony landmarks in the erect position. *Am. J. Physiol.*, 24, 286-293, 1909.
38. Cureton, T. K., Jr., and Wickens, J. S. The center of gravity of the human body in the antero-posterior plane and its relation to posture, physical fitness, and athletic ability. *Research Quarterly*, 6, 93-105, 1935.
39. Jenness, M. E., Speijers, F. H., and Silverstein, H. T. Use of the conformateur and line of gravity apparatus in a new technique for assessing posture. *The Digest of Chiropractic Economics*, 17(3), 14-18, November-December, 1974.
40. Fradd, N. W. A new method of recording posture. *J. Bone Joint Surg.*, 5, 757-758, October 1923.
41. MacEwan, C. G., Powell, E. and Howe, E. C. An objective method of grading posture. *The Physiotherapy Review*, 15(5), 167-173, September-October, 1935.
42. Hellebrandt, F. A., and Braun, G. L. The influence of sex and age on the postural sway of man. *Am. J. Phys. Anthropol.*, 24, 347-360, January-March, 1939.
43. Thomas, D. P., and Whitney, R. J. Postural movements during normal standing in man. *J. Anat.*, 93, 524-39, October 1959.
44. Stevens, D. L., and Tomlinson, G. E. Measurement of human postural sway. *Proc. R. Soc. Med.*, 64, 653-5, February 1971.
45. Eichkern, G. C., and Skaggs, E. B. Some studies in body sway. *Papers of Michigan Academy of Science Arts and Letters*, 10, 369-379, 1929.
46. Sheldon, J. H. The effect of age on the control of sway. *Geront. Clin.*, 5, 129-138, 1963.
47. Joseph, J., and Nightingale, A. Electromyography of muscles of posture: leg muscles in males. *J. Physiol.*, 117, 484-491, 1952.
48. Floyd, W. F., and Silver, P. H. S. The function of the erectores spinae muscles in certain movements and postures in man. *J. Physiol.*, 129, 184-203, July 1955.
49. Nachemson, A. Electromyographic studies on the vertebral portion of the psoas muscle. *Acta Orthop. Scand.*, 37, 177-190, 1966.
50. Klausen, K., and Rasmussen, B. On the location of the line of gravity in relation to L5 in standing. *Acta Physiol. Scand.*, 72, 45-52, January-February, 1968.
51. Fitz, G. W. A Simple method of measuring and graphically plotting spinal curvature and other assymmetries by means of a new direct-reading scoliometer. *American Physical Education Review*, 11, 18-23, 1906.
52. Johnston, L. C. *Biogravitational Adaptation, Theory Measurement and Interpretation.* Toronto, Canada: Posture Research Foundation, 1900 Bayview Ave., 1972.
53. Vilholm, F. Statistical evaluation of posture in 1000 schoolchildren. *Annals of the Swiss Chiropractors' Association*, 5, 55-79, 1971.
54. Takasaki, H. Moiré topography. *Applied Optics*, 9(6), 1467-1472, June 1970.
55. Terada, H., and Kanazawa, E. The position of euryon on the human skull analyzed three-dimensionally by moiré contourography. *J. of Anthropological Society of Nippon*, 82(1), 10-19, March 1974.
56. Hansson, K. G. Body mechanics and posture. *J.A.M.A.*, 128, 947-953, July 28, 1945.
57. Keeve, J. P. Fitness, posture and other selected school health myths. *J. of School Health*, 37, 8-15, January 1967.
58. Hallock, H. The school child's posture. *Public Health Nursing*, 32, 533-538, September 1940.
59. Kuhns, J. G. Diseases of posture. *Clin. Orthop.*, 25, 64-71, 1962.
60. Thompson, J. Knox. The erect posture. *Lancet*, 1, 107-109, Jan. 14, 1922.
61. Garner, J. R. Posture and fatigue. *International Journal of Medicine and Surgery*, 45, 27-28, January 1932.
62. Dickson, F. D. The effect of posture on the health of the child. *J.A.M.A.*, 77, 760-764, September 1921.
63. Cochrane, W. A. The importance of physique and correct posture in relation to the art of medicine. *Brit. Med. Jour.*, 1, 310-313, February 1924.
64. Canter, B. M. The value of proper body mechanics. *International Journal of Medicine and Surgery*, 45, 134-135, March 1932.
65. Canter, B. M. Importance of posture in health. *M. J. & Rec.*, 137, 483-484, June 1933.
66. Forrester-Brown, M. Posture and its relation to health. *J. Royal San. Inst.*, 55, 429-435, February 1935.
67. Mattison, N. D. A new estimate of sleep quality. *M. Rec.*, 148, 133-134, August 1938.
68. Brown, L. T. A combined medical and postural examination of 746 young adults. *Am. J. Orth. Surg.*, 15, 774-787, November 1917.
69. Fox, M. G. The relationship of abdominal strength to selected posture faults. *Research Quarterly*, 22, 141-144, May 1951.
70. Moriarty, M. J., and Irwin, L. W. A study of the relationship of certain physical and emotional factors to habitual poor posture among school children. *Research Quarterly*, 23, 221-225, May 1952.
71. Magora, A. Investigation of the relation between low back pain and occupation. *Industr. Med. Surg.*, 39, 465-471, 1970.
72. Rowe, M. L. Low back pain in industry. *J. Occup. Med.*, 11, 161-169, 1969.

73. Fahrni, W. H., and Trueman, G. E. Comparative radiological study of the spines of a primitive population with North Americans and Northern Europeans. *Bone Joint Surg.*, 47, 552–555, August 1965.
74. Jones, L. Nerve tension and inflammation. *Postgrad. Medicine (Minneapolis)*, 7, 441–449, June 1950.
75. Shannon, P. W., and Terhune, S. R. The low back problem. *American Surgeon*, 17, 1106–1112, December 1951.
76. Hagen, D. P. A continuing roentgenographic study of rural school children over a 15-year period: the lumbosacral angle. *J. Am. Osteopath. Assoc.*, 64, 1163–70, July 1965.
77. Wiggins, R. H. Posture in children. *Canad. Med. Assoc. J.*, 24, 820–825, June 1931.
78. Brown, W. D. Some unattained objectives in pediatrics. *Texas State J. of Medicine*, 38, 335–38, September 1942.
79. MacNamara, J. Posture fundamentals. *Med. J. Aust.*, 2, 592–595, October 1949.
80. Howorth, M. B. Extremities in young children. *N. Y. J. Med.*, 51, 2868–2874, December 1951.
81. Henrichon, M. Posture and the school age child. *Canad. Nurse*, 55, 826–828, September 1959.
82. Garner, J. R. Posture and woman. *International J. Med. & Surgery*, 45, 195–199, April 1932.
83. Krynicki, F. X. The chronic low backache. *Indust. Med.*, 21, 111–114, March 1952.
84. Pepin, W. A., and Kasch, F. W. Induced postural changes resulting from orthopedic programming. *American Podiatry Assoc. Journal*, 50(10), 805–9, October 1960.
85. Ewerhardt, F. H. Bodily mechanics. *Arch. Physical Therapy*, 10, 216–221, May 1929.
86. Denniston, H. D. The value of exercise in the control of posture. *J. Health Phys. Educ.*, 9, 556–557, 581, November 1938.
87. Metheney, E. The balanced posture. *Amer. J. Occup. Therapy*, 2, 133–144, June 1948.
88. Barlow, W. Discussion on postural re-education—a critical examination of methods (postural deformity). *Proceedings Royal Soc. of Med.*, 49, 667–674, September 1956.
89. Stewart, H. E. Principles underlying modernized corrective exercises. *Arch. Phys. Therapy*, 19, 103–106, February 1938.
90. Haller, J. S., and Gurewitsch, A. D. An approach to dynamic posture based on primitive motion patterns. *Arch. Phys. Med.*, 31, 632–640, October 1950.
91. Kraus, H., and Eisenmenger-Weber, S. Fundamental considerations of posture exercises: guided by qualitative and quantitative measurements and tests. *Physiotherapy Review*, 27, 361–368, November–December 1947.
92. Kendall, H. O., and Kendall, F. P. Developing and maintaining good posture. *Phys. Ther.*, 48, 319–36, April 1968.
93. Pheasant, H. C. Practical posture building. *Clin. Orth.*, 25, 83–91, 1962.
94. Lankford, L. L. Postural backache. *Amer. J. Orth.*, 6(7), 150–153, June–July 1964.
95. Kiernander, B. Discussion on postural re-education—a critical examination of methods. *Proceedings Royal Soc. of Med.*, 49, 667–674, September 1956.
96. Beneš, V., Fiala, L., and Krtička, Z. Prevention of spinal abnormalities in school and preschool children. *Cesk. Pediat.*, 16, 932–4, October 1961.
97. Logan, H. B. *Textbook of Logan Basic Methods*. Chesterfield, Mo.: Logan Basic Chiropractic College, 1950.
98. LBM, Inc. *Chiropractic Corrections*. Normandy, Mo.: LBM, Inc., 1961.
99. Toftness, I. N. *"Re-Searching" the Chiropractic Adjustment*. Cumberland, Wis.: I. N. Toftness, D.C., 1961.

Discussion: Comments on Subluxation — Pathophysiology and Diagnosis

ROBERT SHAPIRO
Chairman, Department of Radiology
Hospital of St. Raphael
New Haven, Connecticut

Although spinography furnishes an overview of various static spinal conditions, e.g. curvatures, it has serious inherent deficiencies. Moreover, the limited information it provides can be obtained in other ways. The limitations of spinography may be categorized as follows: (1) technical, (2) interpretive, and (3) excessive radiation.

1. *Technical.*—Good bone detail requires a small focal spot and sharp coning. The substantial scatter produced by radiation of a large volume of tissue is incompatible with optimal resolution. Because of poor film quality, focal as well as generalized bone diseases may be overlooked on spinography.

The inability to visualize important anatomical structures on the anteroposterior spinogram is also a distinct disadvantage. Structures such as the intervertebral foramina, apophyseal joints, pars interarticularis and the sagittal diameter of the bony spinal canal can only be seen on oblique or lateral projections.

2. *Interpretive.*—The great gamut of normal variations facilitates misinterpretation and erroneous diagnosis in the anteroposterior projection—e.g. asymmetry of the articular facets at the same or different levels, asymmetry of the dens and lateral masses of the atlas, variations in the shape of the pedicles, spurious unilateral compression of a cervical articular process,[7] spurious vertebral malalignment (subluxation).

Subluxation.—Subluxation should be a definite objective finding that any trained observer can see and measure. Hence, I prefer to define subluxation as a partial disruption of the normal articular alignment instead of the all-inclusive chiropractic definition of "an alteration of the normal dynamics, anatomical or physiological relationships of contiguous articular structures." Subluxation is diagnosed erroneously all too freely because of the inability to make and reproduce accurate measurements from the spinograms and the misinterpretation of the normal wide range of motion.

Atlanto-axial rotary subluxation is an excellent example of the existing confusion.[6] This diagnosis is often incorrectly made because the spinous process of C2 is seen off the midline. This finding per se has no significance in the absence of an increased distance of the dens from the anterior atlantal arch in the lateral projection. The atlanto-axial joint has convex opposing lateral articular surfaces which are not exactly reciprocal. Hence, there is a telescoping effect during rotation as each lateral atlantal mass slides forward or backward on the corresponding articular surface of C2. As a consequence of the telescoping, the anteroposterior radiographs show relative widening between the articular surfaces on the side of anterior gliding and relative narrowing on the side of posterior gliding. This is demonstrable on anteroposterior tomograms which are often misinterpreted because the two lateral masses are not in the same plane.

Pseudosubluxation.—Because the ligaments in infants and children (up to age 10–12 years) are laxer than in adults, the range of motion in the former is greater. Thus, the body of C2 may project several millimeters anterior to C3 in forward flexion of the neck in children. This should not be misinterpreted as a dislocation since it disappears in the neutral position or in extension. In

597-036 O - 76 - 18

adults, the apex of maximal cervical motion shifts caudad to C5–C6 (sometimes C4–C5).

In my experience, vertebral subluxation in the normal spine is rare. The popular chiropractic practice of diagnosing subluxation on the basis of spinography is inexcusable and should be abandoned.

There is the additional difficulty of correlating various pathologic findings on the roentgenogram with the patient's clinical problem. McCrae[4] has shown that disc protrusions occur in all areas of the spine in almost everyone over 40 years of age. Similarly, Pallis, Jones and Spillane[5] reported the presence of cervical spondylosis in 50 percent of non-neurological hospitalized patients over the age of 50 years. It is important, therefore, not to attribute etiologic significance to such findings unless there is good neurological segmental correlation.

3. *Excessive radiation.*—I cannot agree with Howe's statement that "good quality radiographs using full spine technique do not cause higher organ doses than those produced by well-collimated radiographs of the several spine regions." In the first place, the quality of the radiographs is not comparable. Films produced by the regional technique with sharp collimation are far superior in quality to spinographic films for reasons previously mentioned. If roentgenograms of the spine are indicated, they should be of the best quality. Secondly, although Howe states that the radiation delivered to the gonads in both techniques is comparable, the data he presents are misleading.[3] In his measurements with the full spine technique he used high speed screens for the caudal three-fifths of the cassette. On the other hand, he used par speed screens in the regional technique. Had high speed screens also been used for the regional technique, the radiation dosage to the gonads would have been significantly less than in the full spine technique.[2]

In practice, we are all guilty of a somewhat cavalier attitude toward radiographic examination of the spine in females in the child-bearing age. Perhaps, we should be asking ourselves whether we would be ordering the examination if the patient were known to be pregnant.

REFERENCES

1. Howe, J. W. *The Role of X-Ray Findings in Structural Diagnosis.* Presented at NINDS Conf. on Spinal Manipulation, February 4, 1975.
2. Kennelly, E.: Unpublished data.
3. Levine, J. I., Howe, J. W., and Rolofson, J. W. Radiation Exposure to a Phantom Patient During Simulated Chiropractic Spinal Radiography. *Radiol. Health Data and Reports* 12:245–251, May 1971.
4. McCrae, D. L. Asymptomatic Intervertebral Disc Protrusions. *Acta Radiol.* 46:9–27, 1956.
5. Pallis, C., Jones, A. M. and Spillane, J. D. Cervical Spondylosis: Incidence and Implications. *Brain* 77:274–289, 1954.
6. Shapiro, R., Youngberg, A. S. and Rothman, S. L. G.: The Differential Diagnosis of Traumatic Lesions of the Occipito-Atlanto-Axial Segment. *Rad. Clin. N. Am.* 11:505–526, 1974.
7. Vines, F. S. The Significance of "Occult" Fractures of the Cervical Spine. *Am. J. Roentgenol., Rad. Ther. and Nucl. Med.* 107:493–504, 1969.

Chairman's Summary: What Do the Clinical Sciences Tell Us About Manipulative Therapy?

HENRY G. WEST, JR.
Pocatello, Idaho

It is acknowledged that manipulative therapy has been utilized universally since antiquity for the treatment and relief of back pain. Back pain is one of the most common complaints met in practice. It has been stated that back pain affects probably 80 percent of the members of the human race at some time in their lives. Some have ascribed their symptoms to mechanical malfunction, while others may blame the symptoms on psychosomatic disorders. The symptoms, therefore, lend themselves to evaluation and treatment if the normal mechanisms are known and deviations from normal are recognized.

Dr. Augustus A. White stated "that many symptoms described by patients suffering from back pain are also found in well established psychosomatic types of disease—that the natural course of pain will go away with no assistance in about 99 percent of the cases. Differentiation between natural pain and psychosomatic disease calls for careful clinical trials."

Dr. Alf Nachemson stated "that epidemiological studies show that many people with subluxations do not have pain. He added that additional studies have demonstrated that patients with 2, 3, and even 5 cm. of leg length discrepancies that can not be corrected have not suffered any more back pain than people with equal leg length."

Dr. Peter Tilley suggested that further studies on sacral base unleveling on the erect lumbar spine would be productive in resolving the statements about comparative leg length.

Dr. Scott Haldeman presented a table that demonstrated a higher incidence of spinal abnormalities and subluxations in patients with back problems than those without problems. Also, the presence of abnormality does not mean that the patient is suffering from back pain. "To date," he added, "no conclusive studies have been done to determine whether those asymptomatic persons with subluxations are more likely to suffer from back pain in the future. Also, no research has been done to determine the relationship of spinal subluxation to symptoms other than back pain."

Dr. Joseph Howe stated that "x-ray examinations are widely used by physicians engaged in manipulative therapy for the detection of underlying pathology in order to have some understanding of the structure which he is going to manipulate knowing that structure governs function." According to Dr. Robert Shapiro, one cannot make a diagnosis of a vertebral subluxation from a single radiograph. He also stated that he was "absolutely convinced that a diagnosis of disc disease cannot be made from a plain radiograph in the presence of the normal intervertebral disc space, regardless of the orientation of the pedicles and articular pilars, because of great individual variability." He also pleaded for appreciation of the wide gamut of normalcy. Both Dr. Howe and Dr. Shapiro agreed that multiple films in various postures are necessary before one ventures a diagnosis of an abnormal relationship between two vertebra.

Dr. Peter Tilley added that the question of subluxation or identification of the osteopathic lesion is not one which has been productive in terms of radiographic criteria. Radiographic examination has not adequately defined the osteopathic lesion nor given sufficient corrobroation with postural factors or anomalies, which is an indication

that the compensatory mechanisms which the human body possesses are extremely effective. Therefore, our attention is drawn to the soft tissue.

Dr. Martin E. Jenness said "the lack of quantification of a thermographic picture at the present time has limited the use of thermography as a universally employed diagnostic tool for physicians utilizing manipulative therapy.

Much discussion centered around palpation as a diagnostic tool and whether it could be sufficiently quantified with reliable interpretation by more than one examiner. Dr. Andries Kleynhans quoted references that pathological changes do take place in soft tissue as a result of disturbances of local homeostasis such as edema, fibrotic infiltration, waxy degeneration, increase in cell nuclei of muscle fibers, all contributing to a palpable change – that a trained physician can palpate and interpret this tissue change compared to normal tissue. Discussion also centered around the ambiguities of the role of poor posture and its effect upon health problems. This would appear to be another area of research.

Concepts, theory, philosophy and terminology came into discussion. Terminology has been a major source of controversy which has hindered interprofessional discussions. This workshop will probably contribute to alleviating this major problem.

Dr. Irvin M. Korr summarized the apparent differences in philosophy; that chiropractic appears to be concerned with structural and anatomical approach, whereas osteopathy is more concerned with the interplay of the inputs and outputs via the central nervous system.

Dr. George Northup adroitly stated that "one of the causes of confusion is that we are not talking about a single type of lesion, but about a variety of lesions with influence on a variety of mechanisms. Perhaps there is too much emphasis on static mechanisms rather than on dynamic aberrations of function. He added that there are many changing concepts in medicine and in the theorization of manipulation and the various musculoskeletal lesions that are treated. Manipulative practitioners are faulted for their theories on manipulation on the premise that we do not know the exact mechanism which is observed clinically in the musculoskeletal system. Yet there were six or seven different theories of pain for which there is little understanding about how that pain is caused or transmitted. He added that, in the field of manipulative therapy, we cannot cast aside clinical experience, clinical reporting and clinical observation as being merely anecdotal.

Dr. John Mennell repeatedly emphasized the importance of manipulation for the extremities as well as the spine. The best summarization for the discussion was given by Dr. Robert Maigne, "if a patient is in pain and is treated with manipulative therapy and he has no more pain, what has happened? Why?"

The purpose of this workshop was not to prove or disprove chiropractic, osteopathy or spinal manipulative therapy (SMT), but rather to establish what is known and what is not known about manipulation, and to suggest areas for further research. We can summarize by saying that the exact nature of the lesion which responds to manipulation has never been fully established and that exactly what is accomplished by manipulation no one has fully explained. The fact that manipulation relieves patients is self evident. The following areas of research were suggested during the discussion period:

1. What is the nature of the lesion that responds to manipulative therapy?
2. What is actually accomplished by manipulation in the terms of anatomical, pathological, and physiological changes?
3. Epidemiological studies of biomechanical back pain.
4. Sacral base unleveling and its symptomatic effect on an erect lumbar spine.
5. Epidemiological studies of patients with symptoms (e.g. pain) and "normal spines" as compared to patients with symptoms and "abnormal spines."
6. The relationship of spinal subluxations to symptoms other than back pain and the role of spinal subluxation in the pathophysiology of visceral disorders.
7. Biomechanical dynamics of a normal spine.
8. Quantification of thermography for structural diagnosis.
9. The effects of abnormal posture on problems of health.
10. Quantification of disturbances of local homeostasis.
11. Quantification of palpation for diagnosis.

Chapter VI.

What Do The Clinical Sciences Tell Us About Manipulative Therapy? (Therapeutic Studies)

Andrew B. Wymore, *Chairman*

Treatment of Pain by Manipulation

JAMES CYRIAX
St. Thomas's Hospital
London

So many concepts have been put forward during the last 2 days that I feel I must start by clearing the air before putting forward a unifying concept.

I do not believe that any minor subluxation of a lumbar vertebra causes any symptoms at all. This view has been confirmed by many radiographic studies in thousands of people with and without backache. They show no difference.

I do not believe that manipulation has any effect (other than momentary) on the position of one vertebra on another. Any lasting alteration results from shift of a displaced intra-articular fragment of disc. Normal movement returns when its correct position is restored, whether by the passage of time, rest in bed, manipulation, traction, or laminectomy.

I do not believe that disc degeneration causes symptoms. We all know that lumbar symptoms come and go, yet it is not conceivable that disc narrowing can cease, nor that an osteophyte gets smaller. Statistics from everywhere inform us that backache is commonest at the age group 40–50, declines slightly by the age of 60, but has fallen by two-thirds by 70. Were backache caused by degeneration of the disc or the facet joint, the frequency of backache would increase as age advanced. Again, we have all met with the old man who has lost several inches in height and has no disc spaces at all at any lumbar level, with consequent gross incongruence of the facets and their fixation in the position of full extension; yet he has no backache.

If a patient's pain is elicited by a lumbar movement, it must arise from a joint. Since most lumbar pain starts centrally, this must be the central intervertebral joint. Lumbar pain, in my view, results from a minor displacement of a fragment of disc; it does not matter if the fragment comes from a thick or a thin disc, supported or not by osteophytes.

Here then lies the point where physicians and nonmedical manipulators meet. The former maintain that any theory involving bony subluxations is faulty – correct. The latter maintain that manipulation stops the pain by correcting a displacement – correct again. But the subluxation is of a small fragment of radio-translucent tissue – cartilage. I hope that this concept will prove acceptable to both parties, and enable them to come together at last.

Vertebral manipulation has lain under a cloud all this century. Understandably so; for most medical men do not manipulate the spinal joints at all. By contrast, nonmedical manipulators manipulate nearly all comers. Both these policies are mistaken and smack of bigotry. The proper attitude lies midway between the two extremes.

Manipulation of the spine is important to physicians for three reasons:

1. It is the only method of treatment required in his daily work that he was taught nothing of as a student.
2. The lesions that respond, though restricted in number, occur very commonly and provide the most frequent reason for a fit man being off work.
3. Every time a patient, relievable by spinal manipulation, visits his doctor and this is not done, a gratuitous advertisement is afforded to nonmedical manipulators. We all know that some of these men claim to cure disorders that no manipulation could possibly affect, but this justified scepticism should not blind us to the fact that some of their patients are greatly benefited. It behooves medical doctors to study not their failures, but their successes. We must avoid such patients having to look beyond the medical profession for

relief either by manipulating them ourselves, or (as has been my policy for the last 30 years) handing them over to physiotherapists trained in spinal manipulation.

PURPOSES OF MANIPULATION

Manipulation of joints has three purposes. (No mention will be made of reduction of fractures, dislocations, hernias, etc., since no controversy exists there.)

1. *To break adhesions.* Minor adherent scars may form when a sprained ligament unites, restricting its mobility. They can be ruptured by a sharp jerk in the direction of the limitation. Major adhesions severely restrict movement at the joint after, say, immobilization in plaster after fracture. These require rupture by a strong stretch under anesthesia.

2. *To stretch out a contracture.* Both congenital and acquired contractures need elongation by gradual increasing sustained pressure. Congenital torticollis and talipes equinovarus are obvious examples; arthritis at shoulder and hip represent acquired capsular contracture.

3. *To reduce an intra-articular displacement.* Here lies the main object but, curiously enough, also the most controversial aspect of manipulation. In general, a physician's first thought when a displacement is found present is the feasibility of reduction. In fracture, dislocation, hernia, or breech presentation or indeed a subluxated meniscus at the knee or jaw joint, the advisability is considered at once. But manipulative reduction appears scarcely to figure in medical thought when a fragment of disc is found out of position at an intervertebral joint. Before 1929,[1] when Dandy first ascribed sciatica to a disc-protrusion, the disorder was regarded as "sciatic neuritis" for which manipulation would have been absurd. Until 1945,[2] when I put forward the concept of a postero-central displacement of a fragment of disc as the cause of lumbago, this had been regarded as the result of spontaneous inflammation of muscle, which manipulation could only aggravate. Now, however, when the pathological concepts that we advanced have been accepted everywhere, reason surely demands logical treatment based on this mechanical etiology.

By no means will all disc lesions respond to manipulation. Suitability is based on the size, duration, position, and consistency of the displacement. Moreover, the patient's age, occupation, and sensitivity to pain must all be taken into account. In the lumbar region, my experience is that two-thirds of all cases of backache, but only one-third of all sciaticas, prove reducible.

Reduction of a fracture or of a dislocation is ascertainable objectively. By contrast, at a spinal joint, it is a subjective event. It is only with the patient's cooperation that the operator can tell when all the spinal movements have become free, or straight-leg raising has become painless at full range. The patient is examined immediately before the session starts and after each maneuver. The immediate result is ascertained; also, which measure has the best effect. All this knowledge is denied to the manipulator under anesthesia, who cannot even tell if he is making the patient better or worse, let alone when to go on and when to stop. Since so much spinal manipulation is carried out in Britain either under anesthesia or by untrained persons, it occasions little surprise that many medical men regard it as dangerous or useless.

CERVICAL DISC LESIONS

These present themselves in five different ways and, to add to the confusion, have been given names that distract attention from the actual lesion.

Clinical Examination

This has five purposes. The function is assessed of:

1. *The joints.* The partial articular pattern indicates internal derangement. Of the six movements, two, three, or four hurt; four, three, or two do not. Moreover, the pain is usually unilateral.

2. *The cervical muscles.* Movement, attempted against such resistance that none of the joints move, discloses the state of each muscle group in turn.

3. *The cervical nerve roots.* Monoradicular palsy indicates a disc protrusion. Neuralgic amyotrophy, neuroma, secondary neoplasm, neuritis, and pulmonary sulcus tumor each set up weaknesses in wholly different patterns.

4. *The spinal cord.* Whether the pyramidal deficit is caused by a disc protrusion or not, objective signs of spinal cord involvement wholly contraindicate manipulation.

5. *The upper limb.* This may well contain a separate lesion causing pain in the arm.

Radiography

None of this vital information is obtainable by inspection of radiographs. A displaced fragment of disc within an osteoarthrotic joint is often just as

reducible by manipulation as one in a radiographically normal joint. Every time a medical physician pays excessive attention to a few harmless osteophytes, he is creating one more opportunity for nonmedical manipulators to score. The only reliable basis for a decision on whether to manipulate or not rests on careful and informed evaluation of clinical data. By contrast, normal radiographic appearances must not be allowed to lull the manipulator into a false sense of security; since chordoma, myeloma, neuroma, and early secondary neoplasm do not show up at first.

Clinical Types of Disc Lesions

1. *Acute torticollis.* This is the analog at a cervical joint of lumbago. The young patient wakes with his neck fixed in a posture of gross deformity. Marked limitation of one rotation and one side-flexion movement is present.

Reduction is secured in patients under 30 by manipulating during strong traction only in the direction of full range. When this measure has secured as much improvement as possible, the patient lies down and his head is pushed over more and more in the direction of limited range. It may well be 1 or 2 hours before full range is restored by this means. In patients over 30, manipulation during traction, first in painless direction, then in the painful, suffices.

2. *"Scapular fibrositis."* This is the unfortunate name that has been given to cervical disc lesions causing, as they usually do, pain felt in the muscles about the scapula. The lesion is neither scapular nor is it caused by inflammation of fibrous tissue. Clinical examination shows that the passive, but not the resisted movements of the cervical spine bring on the pain, thus showing its cervical articular origin, and that the resisted movements of the scapula are neither weak nor painful, thus exculpating the structures about the scapula. In other words, positive signs at a joint of the neck are corroborated by negative signs from the circumscapular tissues.

Medical physicians are accustomed to cervical lesions causing scapular pain and accept this extrasegmental reference. But reference to the pectoral area is rare. When it does occur diagnoses like pseudoangina may be reached. When now a nonmedical manipulator manipulates the neck and relieves this symptom, both he and the patient may well imagine that the manipulation has cured some obscure form of heart disease. Medical physicians must be on the lookout for such cases, for they strengthen the assiduously fostered idea that manipulation by nonmedical men cures visceral disease.

Manipulation during traction is simple and usually completely successful in one or two sessions. The distraction relieves pain, thus enabling the patient to relax; it doubles the width of the joint (1954[3]), thus giving the fragment room to move. It also exerts centripetal force on the displacement both by suction and by tautening the posterior longitudinal ligament. Manual traction can be used early in cases with basilar ischaemia (but not during anticoagulant therapy).

Nonmedical manipulators inexplicably avoid adequate traction; in fact they squeeze the vertebrae together. Naturally, such compression militates against a successful result, and this type of "adjustment" (locking the facets) may require many sessions or may fail altogether. Also, judging by the literature it tends to be dangerous.

3. *"Brachial neuritis."* There are many reasons for pain and paraesthesia in the upper limb, but the common cause is a disc protrusion compressing a cervical nerve root; if so, the lesion is neither brachial nor a neuritis.

If no root palsy is present when the upper limb is examined and the spinal cord conducts normally, reduction is often still possible provided that unilateral radiation to the arm has lasted less than 2 months. If a root palsy has supervened and muscle weakness is apparent, manipulation always fails and spontaneous recovery from pain (3 to 4 months since the brachial pain, not the scapular pain, started), and from the muscle paresis (6 to 8 months) must be awaited. Manipulation is also apt to fail when one or more of the neck movements provoke the pain down the upper limb, and when the symptoms appear in the reverse of the usual order, i.e. paraesthesia in the hand, then aching in the limb, then scapular pain.

In Britain a very annoying situation exists. In cervical root compression, the pain in the scapula and arm goes on getting worse for 2 to 3 weeks. During this time, the patient's physician prescribes him ever stronger analgesics. By the third or fourth week the pain is at its worst, and lack of progress leads to reference to hospital. There examination reveals the root palsy, confirmed by electromyography. Physiotherapy, traction or a collar are employed, all in vain. At the end of 2 months, just when the symptoms are about to

wane, the despairing patient takes himself off to a nonmedical manipulator. Since his treatment starts at the same moment as spontaneous subsidence of the pain, manipulation twice a week for, say, 6 weeks coincides in time with the advent of spontaneous recovery. Again, both the nonmedical manipulator and the patient mistakenly ascribe recovery to the manipulations.

4. *Acroparaesthesia.* Bilateral root pressure may set up pins and needles in both hands together with a vague aching in the upper limbs. (Differentiation between the thoracic outlet syndrome and a bilateral carpal tunnel syndrome may present difficulty.)

Manipulation may help. Often the disorder proves intractable, but the symptoms are never severe.

5. *Posterolateral sclerosis.* Evidence of pressure on the spinal cord contraindicates manipulation. Pins and needles in the hands and feet (or postural vertigo indicating basilar ischaemia) are not an absolute bar, provided the methods of nonmedical manipulators are avoided; these are dangerous and death has resulted. Strong traction without rotation may succeed and no lasting harm has resulted from such measures. If the apex of the spur compressing the spinal cord consists of a fragment of cartilage, manipulation during strong traction can still shift it. If the point is osseous, manipulation must fail and the prevention of paraplegia due to compression of the anterior spinal artery is now laminectomy.

Prevention of cord pressure is feasible. The osteophyte arises in the first place by traction on the posterior longitudinal ligament from a postero-central bulging of the disc. The periosteum at the edge of the vertebral body is elevated and bone grows to reach its limiting membrane. The propylaxis of an osteophyte increasingly menacing the spinal cord is to have carried out manipulative reduction years ago.

HEADACHE

There is one type of headache that physicians often fail to recognize—that arising from the ligaments about the occipito-atlantoid and atlanto-axial joints. These joints are developed within the first and second cervical segments and therefore refer pain along the relevant dermatomes in the usual way, i.e., to the back of the head (C1) and the forehead (C2). The patient is an elderly man (women are almost immune) who describes occipito-frontal headache every day on waking. At first it has eased by midday, later by the afternoon: it never lasts all day. At his age, some elevation of blood pressure may be found present. The headache attributed to that, the more so since the radiographs of the upper neck show no more osteophytosis that anyone that age often has. One session of manipulation of the neck during traction nearly always affords full relief lasting at least a couple of years. The nonmedical manipulator may cure this type of headache. If so, again both he and the patient understandably, but mistakenly, take for granted that high blood pressure has been relieved. This not uncommon misdiagnosis provides nonmedical manipulators with renewed "evidence" that they can cure visceral disease.

THORACIC DISC LESIONS

These also present under misleading names e.g. fibrositis of chest wall, muscle strain, pleurodynia (because a deep breath hurts), intercostal neuritis. Diagnosis is not difficult if thoracic disc lesions are kept in mind. The influence of posture and exertion on the pain is elicited in the history and the spinal movements therefore tested.

The difficult cases are those with a primary postero-lateral onset, the root pain felt in the anterior thorax or abdomen coming on without previous backache. Exhaustive examination of visceral junction naturally reveals no abnormality, and such patients are often dismissed as neurotic, or alternatively, some vague label such as gastritis or chronic cholecystitis is applied. A. T. Still, the founder of osteopathy, describes how he had pain in the region of his own heart, which ceased with a click during pressure at his mid-thoracic vertebrae. In this type of case pain exists that the nonmedical manipulator can easily abolish; however it is wrongly ascribed to some vague visceral disorder. Obviously, vertebral manipulation relieves not visceral disease, but those pains actually of spinal origin that have been mistakenly ascribed to a viscus. Neither patient nor nonmedical manipulator realizes that, nor would it suit the latter's book if he did have doubts.

Examination

This comprises eliciting:

1. *Articular signs.* The partial articular pattern indicates internal derangement. Some, but not all, of the six movements prove painful.

2. *Dural signs.* Neck flexion and scapular ap-

proximation draw the dura upwards and increase the thoracic pain.

3. *Root signs.* Though root pain felt as a rule along the lower costal margin is common, neurological deficit is rare and suggests a neuroma rather than a disc lesion.

4. *Cord signs.* If evidence of pyramidal pressure exists, manipulation is wholly barred; laminectomy should be considered.

Articular signs accompanied by dural signs clearly indicate a posterior disc displacement, since the dura mater lies behind the joint. Manipulative reduction during traction is usually very easy.

LUMBAR DISC LESIONS

Here, too, the situation is obscured by many different names for the same disorder—pulled muscle, lumbago, sciatica, sacroiliac strain, sprung back, lumbar or gluteal fibrositis, spinal arthritis, or spondylosis. The same phenomenon as is so conspicuous at the neck—extra segmental reference from the dura mater with a secondary localized tender spot within the painful area—occurs also in lumbar disc lesions. Since a postero-central disc protrusion bulges the posterior ligament out far enough to compress the dura mater, remarkable areas of reference are reported by sufferers from acute lumbago, e.g., to one or both groins, to the lower abdomen, up to the lower posterior thorax. When the referred pain overshadows local pain, it is not unknown for a low lumbar disc lesion to be mistaken for chronic appendicitis, since the way the dura mater refers pain misleadingly is not recognized by most doctors. Clearly, spinal manipulation may well relieve such a pain in the iliac fossa, and the mistaken notion of nonmedical manipulation curing visceral disease is once more strengthened.

Detailed diagnosis is most important for it is by no means enough to know that a lumbar disc lesion is present. The lesion's duration, size, position, consistency, and stability have all to be correlated with the patient's occupation, age, and sensitiveness. A small cartilaginous displacement should be reduced by manipulation; a small nuclear protrusion should be reduced by daily traction. If the protrusion is large neither method is applicable and the desensitization of the nerve root at the point of impact by the induction of epidural local anasthesia is the treatment of choice.

Examination

Four data are sought:

Articular signs. These comprise: 1. visible deviation; 2. limitation of movement in some directions but not in others. In early disc lesions a painful arc, usually on trunk flexion, is often present. The partial articular pattern indicates internal derangement.

Dural signs. Lumbar pain produced by neck flexion and bilateral limitation of straight-leg raising indicate that the mobility of the dura mater is impaired on stretching from above or below. When, in sciatica, the straight leg is raised as far as possible, neck flexion causes added root pain, again as the result of pulling on the tense nerve root via the dura mater.

Nerve root mobility. At the third root, this is tested by prone-lying knee flexion.

At the fourth and fifth lumbar levels, the mobility of the dural sheath of the roots L4 to S2 is assessed by straight-leg raising.

Nerve root conduction. Muscle weakness, impaired reflex, cutaneous analgesia indicate a degree of protrusion too great for manipulation or traction to help.

Radiography

Choice of treatment in disc lesions rests on what is found when these four essential elements in clinical evaluation are correlated. None of these findings emerges from inspection of a straight radiograph nor is appreciable help afforded by positive or negative myelographic appearances.

Manipulative reduction should form the physician's first approach, and be performed forthwith unless clinical examination has disclosed a contraindication.

CONTRAINDICATIONS TO MANIPULATION

The contraindications are:

1. *Danger to the fourth sacral root.* Any complaint of weakness of bladder or rectum or of perineal, testicular or saddle paraesthesia suggests severe stretching of the posterior longitudinal ligament. If this should rupture during manipulation, massive protrusion of the whole disc may result, leading to severe bilateral sciatica and damage, possibly permanent, to the innervation of the bladder. In such cases laminectomy is urgently required.

2. *Hyperacute lumbago.* Most cases of lumbago respond very well to manipulation. However, in a few cases the patient is so fixed that the slightest movement provokes such sharp stabs of pain that the attempt becomes unthinkable. If so, epidural local anesthesia is induced, whereupon the displacement impinges against the now-insensitive dura mater and all pain ceases for the time being. Spontaneous reduction is aided during this period of painless mobility if the patient lies prone for as long as the anesthesia lasts.

3. *Pregnancy.* During the last month manipulation is impracticable. During the first 4 months, prone pressures as well as the rotation manipulations are quite safe.

4. *Neurosis.* Very nervous patients, or those who, owing to a legal suit pending, have to maintain disablement are not suited to manipulation.

MANIPULATION USELESS BUT NOT HARMFUL

1. *Too large.* Reduction is impossible when the protrusion is larger than the aperture whereby it emerged. Sciatica with a marked lumbar lateral deviation, or signs in the lower limb of impaired conduction at the nerve root (muscle paresis, loss of reflex, cutaneous analgesia) show that this is the situation.

2. *Too long.* When root pain has lasted 6 months or more in a patient under 60 years of age, the attempt is almost sure to fail.

3. *Too soft.* Nuclear protrusions require 1 to 3 weeks' daily traction for 30 to 45 minutes, at a distracting force of 80 pounds (minimum for a frail woman) to 200 pounds (for a large strong man). The treatment is entirely painless (1950[4]). It should never be used for acute lumbago with twinges, which is made much worse.

LUMBAR MANIPULATION

Manipulative technique is not difficult to master. The patient lies on a low couch (15 in. high) and pressure on his lumbar spine, accompanied by a jerk, is applied as he lies prone, or rotation of the pelvis on the thorax is secured while he lies on his side or supine. Patients who present with much lumbar deviation do best on the rotational maneuvers.

The family doctor should be prepared to carry out these maneuvers as soon as the patient attends: for he sees the case early and thus offers treatment at the most favorable moment. Alternatively, he should instruct the physiotherapist of his choice to carry out these measures at once. This insures that the patient remains under medical supervision throughout, and is treated by trained personnel. I found this policy welcomed in England, and wherever my graduates have gone, including the U.S.A. and Canada, they have been esteemed by physicians and patients for their skill (Cyriax, 1974).[5]

SUMMARY

In my experience, the only good reason for spinal manipulation between the third cervical and fifth lumbar vertebrae is an endeavor to reduce a displacement of a small fragment of disc. This is what nonmedical manipulators, without realizing it, have been doing for the past 100 years and have gained many kudos thereby. Their successes have led to untenable hypotheses. An attempt is made to substitute a valid anatomical explanation.

REFERENCES

1. Dandy, W. E. Loose cartilage from intervertebral disc simulating tumor of spinal cord. *Arch. Surg.* 19 ii, 660, 1929.
2. Cyriax, J. Lumbago: The Mechanism of Dural Pain. *Lancet* ii, 427, 1945.
3. Cyriax, J. *Textbook of Orthopaedic Medicine, Vol. I,* plates 5 and 6. London: Bailliere, Tindall, 1954.
4. Cyriax, J. Treatment of Lumbar Disc-Lesions, *Brit. Med. J.* ii, 1434, 1950.
5. Cyriax, J. *Textbook of Orthopaedic Medicine, Vol. II,* Eighth edition. London: Bailliere, Tindall, 1974.

The Treatment of Pain by Spinal Manipulation

RONALD GITELMAN
Canadian Memorial Chiropractic College
Toronto, Ontario, Canada

*Chiropractic is a discipline of the scientific healing arts concerned with the pathogenesis, diagnostics, therapeutics and prophylaxis of functional disturbances, pathomechanical states, pain syndromes and neurophysiological effects related to the statics and dynamics of the locomotor system, especially of the spine and pelvis.**

Man has used manipulation as a means of relieving pain and disability since his beginnings. Hippocrates (1) described manipulation and "how to press down hard on a gibbus of the spine" in order to relieve pain.

Sir James Pagett [1866] and Dr. Walton P. Hood [1871] wrote "Cases That Bone Setters Cure", and "On Bone Setting," respectively.

At that time the medical profession looked upon manipulation with disdain, primarily because of the theoretical explanation of its practice. In spite of being scorned or even ostracized by their colleagues, many individuals utilized manipulation for the benefit of their patients. The advent of osteopathy and chiropractic helped fill the need for this form of treatment. The fact that both of these disciplines developed outside the medical mainstream did little to soften the attitude of traditior.. ' medicine. Even so, many dedicated medical scientists[2-16] urged that manipulation was a valid procedure and should no longer be set aside.

Travell[17] in 1946 applied manipulation in over 400 selected low back patients. No deleterious effects were observed and many cases secured immediate relief of pain.

Henderson[18] in 1954 reported on 500 unselected patients with low back pain. Of the group treated by manipulation, more than half were relieved or improved. Another group of 20 patients did not improve with physiotherapy or exercise until after manipulation was instituted.

Fisk[13] (1971) treated 327 unselected patients with manipulation and claimed a 90 percent success rate. Wilson[19] prepared 18 patients with herniated intervertebral discs with interspinal pantopaque and studied the results of manipulation radiographically. Crude rotational manipulations to both the right and left side of the spine were employed. Surprisingly, three patients experienced relief of the back and leg pain for 48–72 hours and only one case had a slight increase in the discal defect.

Mensor[2] (1955) using manipulation under general anesthesia reported good to excellent results with 64 percent of 72 private patients and 45 percent of 133 industrial accident cases with lower back pain. Many clinicians feel that manipulation under anaesthetic is unsafe, due to the patient's protective reflexes being abolished.[6-8, 10, 11, 14]

The first controlled series was conducted by Coyer and Curwen[21] (1955). One hundred and thirty-six patients were divided into two groups. The first group of 76 patients was treated by manipulation and the second group of 60 with bed rest and analgesics. In the first group 50 percent were free of signs and symptoms within 1 week as compared to 27 percent in the second group. At the end of 6 weeks, 12 percent of the first group and 28 percent of the second group were still afflicted. A number of additional studies have been conducted by individuals who have reported excellent results with manipulation, often after more traditional methods have

*Definition of Chiropractic as taught at The Canadian Memorial Chiropractic College, Toronto, Ontario, Canada.

failed.[11, 12, 14, 22–26] These studies must be viewed slightly more critically, as a man is reporting the results of his own work.

A more objective report was made by Poul Bechgaard[27] in a series of 807 patients. Sixty-four percent received a primary dramatic improvement with one treatment. The subjects were referred for manipulation by a second individual and then he evaluated the results.

An independent research group did an objective review[28] of the Workmen's Compensation Board reports of all cases of spinal sprains and strains in the State of Florida for the year 1956. Cases numbering 19,666 were reviewed by comparing costs and days lost. Under chiropractic care, the average total cost was $60 and days lost were 3. Under medical treatment (nonspecialist, nonhospitalization), the average cost was $102 and days lost were 9.

The *Lancet*[29] in 1974 published a similar study on 300 compensation cases which showed that the physician was somewhat less effective than the chiropractor in the treatment of the acute low back. The Palmer College investigation (1950)[30] of 1,000 low back syndromes, and the National College investigation (1970)[31] of 100 cases with osteogenic changes and vertebrogenic pain reported impressive results. The effectiveness of the treatment of headache by manipulation is well established in the high percentage which are vertebrogenic.[10, 11, 27, 32–53, 136] Research material from the scientific communities of Czechoslovakia and Germany shows that they are investigating a wide variety of applications for manipulation.[54–58] For example, 72 cases of Meniere's syndrome were studied with excellent results in 53 patients; also 52 cases of cervicogenic vertigo (Barre and Lieou syndrome) were studied with excellent results in 35 of the 52 cases treated.[59] The growth and development of osteopathy and chiropractic with its tens of thousands of relieved patients, the growing interest of medicine in manipulation with the formation of the International Federation and the American Academy of Manipulative Medicine and the fact that this Conference is being held, are indications that manipulation is effective. I am confident that any fairminded thinker does not doubt the fact that manipulation works, however, I feel that a meaningful research study could be conducted if for no other reason than to convince the remaining skeptics. This study, however, must be conducted in such a way as to not only test one technique or the ability of one practioner to deliver it.

We must also bear in mind that while manipulation is the mainstay of chiropractic treatment it is usually preceded by soft tissue massage, passive stretching and mobilization and/or triggerpoint therapy. Manipulation is not the end of treatment since the practioner must not only reeducate the joint's range of movement, restore normal motor tone, and power, but give the patients advice about their lifestyle including work habits and leisure time activities, in order to restore full function and attempt to prevent recurrence. The major problem for the scientific community is still an explanation as to the mode of action of manipulation and the scope of its application.

The symptom of pain usually indicates tissue threat. It is the end result of a long process (except in the case of acute trauma). It is not merely the stimulation of receptors that marks the beginning of the painful process; that stimulus enters a nervous system which is already a total of past experience, trauma, anxiety, cultural factors, et cetera. These higher processes, these past experiences, and the state of the nervous system at the time of stimulus participate in the selection, abstraction and synthesis of the information from the total sensory input.[60]

To understand the mode of action of manipulation we must have a greater appreciation of the combinations of functional reflexes which are disturbed, and which, under normal circumstances, enable the organism to adapt itself to its internal and external environment. While the removal of pain represents the ultimate test of any therapy, the pathophysiological process and its interruption at any given stage is also a method of understanding and establishing its worth.

A great deal of attention has been paid to nerve root compression syndromes at the intervertebral foramina, but we must bear in mind that these are usually the result of a prolonged process and represent a small percentage of the subluxations which are manipulated in the everyday experience of the average practioner. These are primarily the conditions which we hope to prevent. We do not know the precise nature of the cause of subluxations or the neurophysiological mechanisms by which they exert their influence. We do know that trauma is a major cause, whether sudden or slight or the result of false or poorly judged movements. Prolonged or repeated postural or occupational

stresses result in muscle tensions, postural imbalance and contractions of muscles,[61-63] which produce a low-grade type of traumatic inflammation and have an effect upon the articular and periarticular structures and functions of the involved areas.[64]

Activity of the muscle itself causes some degree of ischemia,[65,66] which can result in pain,[67,68] probably through the transfer of "P" substance across the muscle membrane into the tissue fluid,[69] which gains access to pain endings. The pain itself brings about a tonic reflex muscle contraction which intensifies the ischemia leading to a vicious cycle. These contractions and their effects upon capsules, tendons, fascia, ligaments and joints produce postural asymmeteries[70] and limitation of movement,[71-77] and the resultant proprioceptive bombardment may be the initiating factor of anterior horn facilitation.[78,79]

This in turn leads to the further perpetuation of the muscular contraction by any additional excitation into the same or related neural segments.[80-82] The resultant stress on the ligamentous capsules and articular surfaces can lead to local inflammation, adhesions and early breakdown of the facet articulations.[83,84] These articulations and ligaments have a rich nerve supply and local and radiating pain results.[4,8,9,85-89]

Limitation of mobility decreases the efficiency of the hydraulic system of the vertebral motor unit which is so necessary for the integrity of the intervertebral discs and contributes to its breakdown,[9,10,86,90-93,84] hence to the production of hypermotoricity in the same or adjacent segments. Subluxations of the apophyseal joints may follow.[86,93,95] We believe that a hypermotoricity for example, of the fifth lumbar could be due to fixation and aberrant movement of the pelvis and/or hip joints with the resultant disturbances of the dynamics of the postural muscles; for example, the psoas[96] could produce asymmetrical stresses at the lumbosacral articulations. This is a matter for future research.

We begin to see that subluxation is a process and not a static condition; a state of living tissue undergoing constant change. These pathologic changes are as follows: hyperemia, congestion, edema, minute hemorrhage, fibrosis, local ischemia, atrophy, and eventually rigidity and adhesions which form not only within the joint capsule but also within the ligaments and tendons and muscles themselves. The end result of this is osteogenic change within the facet,[97] premature aging of nucleus pulposis,[86] breakdown of the annulus of the disc,[9,10,86,90-94] facet capsular swelling,[4,8,9,85-89] approximation of pedicules,[13,86] spinal stenosis[86] edema within the foramina, perineural adhesions, radiculitis,[98] subluxation of the articular processes,[86,98] and exostosis.[98,193] As a result, a compression of the nerve trunk may occur within the intervertebral foramina even to the point of affecting neural conduction.[7,86,93,99] How can we explain the relief of root compression and pain by manipulation when we know that pressure on a nerve causes paresthesiae, not sustained pain.[88,100,101]

The x-ray picture of exostosis and disc thinning remains the same, but the pain is relieved. How do we explain Dr. McNab's observation[101] of some cases of instant relief of pain with injection of chymopapain, when we know the enzymic effect of the drug will not immediately reduce a pulpy protrusion and that myelograms show no such immediate change in the defect. Could it be that the enzyme had its dramatic effect by changing the chemistry in such a way as to neutralize pain-producing polypeptides which stimulate hitherto unknown nociceptors in the disc or perhaps destroy them?

Studies of asymptomatic and symtomatic spines[102] at autopsy showed there was no significant difference between the two groups.[103] Manipulation can break adhesions and free fixations[10,16,109] of the facets and even in some cases affect the discal contents,[7,93,104] but surely we cannot explain on this mechanical basis alone the more far-reaching neurological effects.[10,34,55,56,87,99,105-109]

If there were no other signs and symptoms, the placebo effects of the immediate environment, the white coat, and the laying on of hands could account for much of the relief our patients receive. However, there are other parameters which we can measure, not only in determining the stage of the involvement but in monitoring the results of our manipulation objectively. Facilitated anterior horn cells produce changes which can be detected by the educated palpating finger[10,180,110-113] and by electromyography[58,114-118] in the innervated muscles of that segment. These muscles are in a state of increased tone[114,119-121] even under anesthesia.[122]

The appropriate manipulation produces palpable improvement in the muscular tenderness and tension.[88,59] An increase in mobility of the spinal segments[10,113,123,124] can be demonstrated by x-ray;[64,72,110,125-129] measurable electromyographic changes locally and in the extremities have been

reported.[122, 130, 131] Improvements in the motor supply to the eye have been recorded by the nystagmograph.[59, 125, 132] Nystagmographic changes—whether accomplished by modifying the proprioceptive input into the facilitated segment[133-135] or by decompression of the vertebral artery to relieve a temporary relative ischemia of the vestibular centers, or by other reflex effects[122]—is a question that is open for further investigation.

There are other symptoms associated with subluxation which show evidence that the facilitation takes place segmentally in the lateral horn cells as demonstrated by the changes in the sudomotor,[137] visceromotor[138-141] and vasomotor[142-147, 181] activities. Pre- and post measurements of manipulative procedures have demonstrated the effectiveness of manipulation in restoring more normal tone in these sympathetic activities.[148-150] Audiograms,[59, 125, 132, 151] blood pressures,[152-156] and serologic profiles[156-160] also show an improvement after manipulation. The question of how pain is relieved by manipulation still remains unanswered. In the case of myalgia, adhesion and facet subluxation, manipulation could decrease the stimulation of nociceptors by passive stretching to relieve the muscle spasm,[161] by breaking the adhesions and/or by mobilizing the fixated facet. This then decreases the nociceptor stimulus just as Dr. McNab's chymopapain did when he injected it into the discs of patients with root compression.

Kibler and Nathan[162] showed that pain could be abolished in cases of cord and root damage by injecting a local anaesthetic distal to the region of damage. This indicates that the lesion was not the point which generated the pain but impulses from undamaged tissues were necessary to trigger the pain. The questions then arise: Does the lesion block inhibitory impulses or is there a decrease of inhibitory impulses due to a lack of normal motorisity? Is the manipulation successful because it relieves the nerve root pressure or is the manipulation successful because it increases the amount of inhibitory stimuli by restoring movement to the level that would provide a beneficial proprioceptive bombardment? It is interesting to note that acupuncturists are routinely relieving pain[163] and even producing surgical anaesthesias[164-166] by needling of points distal to the site of pain. Kakizaki and Manaba[167] have performed caesarean section after applying static electricity to the hands and feet. This demonstrates that sensory bombardment does affect pain.

There are other questions, does the lesion irritate the recurrent meningeal nerve to cause vasoconstriction to the innervated structures? Does the lesion give rise to proprioceptive impulses which stimulated anterior horn cells to cause somatic vasoconstriction, ischemia and pain? Could this vasoconstriction take place within the cord and lower the threshold of excitation to contribute to a "central excitory state"?

There are several questions which must be answered with regard to the "central excitory state" and the spinal mechanism as they relate to pain, such as specificity or convergence of impulses transmitted to higher structures and the effects of descending controls upon them.

SUMMARY

In this paper I have attempted to review the nature of the painful vertebral lesions. I have presented data which would indicate that manipulation is effective in relieving pain and other symptom complexes which result from vertebral dysfunctions. The exact mode of action of manipulation is not known. In this regard I have presented questions, not answers. It would seem to me that one thing is clear; if we direct all of our efforts into the investigation of nerve compression at the intervertebral foramina, we will obtain only partial answers to our questions. Foraminal compression is the end result of a pathological process. We must investigate the process itself. Investigation must be aimed at the understanding of the normal and abnormal mechanics of the spine and the effects of joint function on the afferent side of the nervous system. Somato-somatic and somatovisceral reflexes must be clearly understood if we are ever to appreciate the mechanism of vertebrogenic pain and other symptomatologies and their relief by manipulation. This quote from the latest edition of Gray's Anatomy[55, 168] is instructive: "The autonomic nervous system is intimately responsive to changes in the somatic activities of the body and while its connections with the somatic elements are not always clear in anatomical terms, the physiological evidence of visceral reflex activities stimulated by somatic events is abundant."[13, 69, 140, 169-175, 178, 179]

We recognize other etiological factors over and above the vertebrogenic.[134, 141, 176, 177] We are also aware of the fact that visceromotor reflexes frequently cause characteristic spinal distortions.[140, 141]

It is these phenomena that we must clearly comprehend when we discuss scope of practice.

One cannot study chiropractic only by investigating manipulation as if it were one modality in a long list of modalities of physical medicine, for it is a whole system of analysis, diagnosis, therapeutics and prophylaxis. Chiropractic emphasis is on the unity of the human body—the individual patient. This is perhaps the major contribution chiropractic can make.

The main thrust of research should be directed to the functional disturbances, pathomechanical states, pain syndromes and neurophysiological effects related to the statics and dynamics of the locomotor system, especially of the spine and pelvis.

This Conference constitutes a threshold which, when crossed with an unbiased scientific attitude, will result in the development of the full potentialities of the manipulative sciences. The result, I believe, will be a quantum step in the evolution of the healing arts for the benefit of all mankind.

REFERENCES

1. Adams, F., *The Genuine Works of Hippocrates*—translated from the Greek. 2:545 Printed for the Sydenham Society, London, 1849.
2. Jones, Sr R., Manipulation as a Therapeutic Measure. *The Royal Society of Medicine* 25:1405–1413, 1932.
3. Fredett, J. W., Manipulative Surgery. *Proceedings of the 21st Annual Session of the American Congress of Physical Therapy*, Pittsburgh, Pa., 1942.
4. Wright, J., Mechanics in Relation to Derangement of the Facet Joints of the Spine. Proceedings of 22nd Annual Session of the *American Congress of Physical Therapy*, Chicago, Ill., 1943.
5. Marlin, T., *Manipulative Treatment for the Medical Practitioner*. Edward Arnold, London, 1934.
6. Jostes, F. A., Place of Manipulative Procedures in the Overall Treatment Rationale for Painful Back Conditions. *Arch. of Phys. Therapy*, 1944.
7. Cyriax, J., *Textbook of Orthopaedic Medicine*, Vol. 1. Bailliere Tindall and Cassell, London, 1969.
8. Mennell, J., *The Science and Art of Joint Manipulation*. Churchill, 1952.
9. Mennell, J., *Back Pain*. Little, Brown, Boston, 1960.
10. Maigne, R., *Orthopedic Medicine*. Charles C Thomas, Springfield, Ill., 1972.
11. Bourdillon, J. F., *Spinal Manipulation*. William Heinemann, London, 1970.
12. Parsons, W. B. and Cumming, J. D. A., Manipulation in Back Pain. *Canadian M.A.J.*, July 15, 1958, Vol. 79. Vertebral Manipulation, *Coll. Gen. Pract. J.*, July 1965.
13. Fisk, J. W., Manipulation in General Practice. *N. S. Med. J.*, 74, pp. 172–175, 1971.
14. Ewer, E. G., Manipulation of the Spine. *J. Bone and Joint Surg.*, Vol. 35A, No 2, pp. 347–354, April 1963.
15. Smart, M., Manipulation. *Arch. Phys. Med.* pp. 730–734, December 1946.
16. Foreman, E. W., Face It We Can Learn From Chiropractors. *Medical Economics*, March 5, 1973, pp. 187–205.
17. Travell, J. and Travell, W., Therapy of Low Back Pain By Manipulation and of Referred Pain in the Lower Extremity By Procaine Infiltration. *Arch. Phys. Med.*, September 1946, pp. 537–547.
18. Henderson, R. S., The Treatment of Lumbar Intervertebral Disk Protrusion. *Brit. Med. J.*, September 1952, pp. 597–598.
19. Wilson, J. N., Manipulation of The Herniated Intervertebral Disc. *Am. J. Surg.*, February 1952.
20. Mensor, M. C., The Nonoperative Treatment of Lumbar Disc Syndrome. *J. Bone and Joint Surg.*, Vol. 37A, No. 5, October 1958.
21. Coyer, M. B. and Curwen, I. H. M., Low Back Pain Treated by Manipulation. *Brit. Med. J.*, March 1955.
22. Leemann, R. A., Pain Syndromes Originating In The Vertebral Column and Their Amenability To Manipulative Treatment. *Schweiz med. Wchnschr*, 87: pp. 1289 and 1321, 1957.
23. Fischer, A. G. T., Manipulation in Lumbar Intervertebral Disc Lesions. *The Practitioner*, September 1961, Vol. 187, pp. 319–327.
24. Valentini, E., The Rate of Success of Chiropractic Manipulation in Lumbar Herniated Discs. *Annals of the Swiss Chiropractor's Association*, Vol. iv, pp. 79–85, 1969.
25. Beyele, W., The Treatment of the Chronic Recurrent Lumbalgias. *Annals of the Swiss Chiropractor's Assoc.*, Vol. 2, 1960.
26. Bosshard, R., The Treatment of Acute Lumbago and Sciatica. *Annals of the Swiss Chiropractor's Assoc.*, Vol. 2, 1964.
27. Bechgaard, P., Late Post Traumatic Headache and Manipulation. Corres. *Brit. Med. J.*, June 4, 1966, p. 1419.
28. *A Survey and Analysis of the Treatment of Sprain and Strain Injuries in Industrial Cases*. Published by Florida Chiropractic Association, 1216 Colonial Drive, Orlando, Fla., April 1960.
29. Kane, R. L., Leymaster, C., Olsen, D., Woolley, F. R., Fisher, F. D., Manipulating the Patient. A Comparison of the Effectiveness of Physician and Chiropractor Care. *Lancet*, June 29, 1974, pp. 1333–1336.
30. Research Department International Chiropractors. *A Study of 1,000 Herniated and "Slipped Discs"*. Davenport, Iowa, 1950.
31. Shelby, R. L., Chiropractic Management of Degenerative Joint Disease of the Spine—A Study of 100 Patients. *National College of Chiropractic*, June 1970.
32. Baoke, H. K., Cervical Headache, *Canadian Family Physician*, May 1972.
33. Kovacs, A., Subluxation and Deformation of the Cervical Apophyseal Joints—A Contribution to Aetiology of Headache. *Act. Radiocogica*, Vol. 43, January 1955, Fasc. 1.
34. Travell, J., Mechanical Headache. *Headache*, April 1967, pp. 27–29.
35. Lewit, K., Results of Manipulative Treatment on Childhood Migraine. *Hippocrates*, 34, pp. 308–316, 1963.

597-036 O - 76 - 19

36. Wachenheim, A., Functional Atlanto-Occipital Block. *Neuroradiology*, 3, pp. 80–81, 1971.
37. Blumenthal, L., Injury to the Cervical Spine as a Cause of Headache. *Postgraduate Medicine*, Vol. 56, No. 3, pp. 147–152, September 1974.
38. Parsons, W. B., Vertebral Manipulations. *J. of the College of General Practice of Canada*, pp. 22–24, 1965.
39. Lewit, K., Ligament Pain and Anteflexion Headache. *Europ. Neurol. 5*, pp. 365–378, 1971.
40. Robertson, A. M., Manipulation in Cervical Syndromes. *Practitioner* 200: pp. 396–402, March 1968.
41. Milne, E., Report on Chiropractic. *Med. J. of Australia*, Vol. 54, pp. 78–79, January 1967.
42. Lewit, K., Functional Disorders of the Spine in Children. *Manuelle Therapie*, Chap. 2.7, pp. 50–54, J. A. Barth, Leipzig, 1973.
43. May, F., Indications for and Techniques of Spinal Manipulation. *Med. J. of Australia*, Vol. 55, pp. 555–559, September 28, 1968.
44. Jones, R. T., Osteo Arthritis of the Paravertebral Joints of the Second and Third Cervical Vertebrae as a Cause of Occipital Headaches. *S.A. Med. J.*, May 1964, pp. 392–394.
45. Gutmann, G., *Halswirbelsäuce und manuelle therapie*, pp. 310–343. Chirotherapie—manuelle therapie. Therapie über das nervensystem, Band 7. *Hippokrates*, 1967, Stuttgart.
46. Strohal, R., *Manuelle Therapie Bei Wirbelsäuce Erkrankungen*, pp. 70–73. Urban & Schwarzenberg München-Berlin-Wien, 1973.
47. Lehapfuhl, H., *Die Behandlung des Subluxations Kopfschmerzes Ther Gegenw*. 1951, Heft 5, 175–179.
48. Robertson, A. H. M., Manipulation in Cervical Syndromes. *The Practitioner*, Vol. 200, March 1968, pp. 396–402.
49. Livingston, M., Spinal Manipulation—A One Year Followup Study. *The Canadian Family Physician*, July 1969, pp. 35–39.
50. Lewit, K., Meniere's Disease and the Cervical Spine. *Review of Czechoslovak Medicine*, 1961, VII–2, pp. 129–139.
51. Miller, H. C., Head Pain. *H.A.O.A.*, Vol. 72, pp. 135–142, October 1972.
52. Janda, V., The Cervicocranial Syndrome in Children. *Cs. Pediatrie*, 14, p. 1018, 1959.
53. Kabatnicova, S. and Kabatniz, Z., The Significance of of the Vertebral Column in Children's Headaches. *Lek Pbzor*, p. 361, 1966.
54. Novotny, A. and Pvorak, V., Functional Disturbances of the Vertebral Column in Gynocological Practice. *Manuelle Medizin* 2: 1972.
55. Stary, O., The Concept of Research of Vertebrogenic Disease in CSSR. *Acta Universities Carolinae Med.*, Suppl. 21: pp. 16–18, 1965.
56. Jirout, J., Studies in the Dynamics of the Spine. *Acta Radiol*, 46:55, 956, pp. 55–60.
57. Rettig, H., Observation of an Acute Basedow's Syndrome After Chiropractic Treatment of the Cervical Spine. *Med. Klin.*, 36, pp. 1528–1530, 1955.
58. Pfeiffer, J., Nauer, L., Berkokva, J., and Sussova, J., Electromyography of the Abdominal and Dorsal Muscles in Initial Disorders of Spinal Dynamics. *Acta Universitatis Carolinae Med.*, Suppl. 21: 33–34, 1965.
59. Wyke, B., Paravertebral Spasm Relieved by Manipulation. *Annals of The Royal College of Surgeons*, 41, 45; 1967.
60. Meczack, R., *The Puzzle of Pain*. Basic Books, Inc., New York, 1973.
61. DeVries, H. A., Muscle Tone in Postural Muscles. *A.J. of Phys. Med.*, Vol. 44, No. 6, pp. 275–291, 1964.
62. DeVries, H. A., Quantitative Electromyographic Investigation of the Spasm Theory of Muscle Pain. *A.J. of Phys. Med.*, Vol. 45, No. 3, 1964, pp. 119–134.
63. DeVries, H. A., Fatigue Curves in Postural Muscles—A Possible Etiology of Idiopathic Low Back Pain. *A.J. of Phys. Med.*, Vol. 27, pp. 175–181, 1968.
64. Sandoz, R., Newer Trends in the Pathogenesis of Spinal Disorders of the Intervertebral Motor Unit. *Annals of the Swiss Chiropractor's Association*, Vol. 5, pp. 93–180, 1971, 1964.
65. Barcroft, H. and Millen, J. L. E., The Blood Flow Through Muscle During Sustained Contraction. *J. Phys.*, 97: pp. 17–31, 1939.
66. Kleynhans, A. M., Vascular Changes Occurring in the Cervical Musculoskeletal System. *Bull. European Chiropractic U.*, 194, No. 1, pp. 24–30, 1970.
67. Dorpat, T. L. and Holmes, T. H. Mechanisms of Skeletal Muscle Pain and Fatigue. *Arch. Neurol & Physchiat.*, 74, pp. 628–640, 1955.
68. Travell, J., Referred Pain From Skeletal Muscle. *New York State Journal of Medicine*, 55: 331, February 1, 1955.
69. Lewis, T., *Pain*. New York, MacMillan & Co., 1942.
70. Lewit, K., Verlag Fur Phyikalische Medizin Heidelberg, 1970. Chemical Roentgenological Examination of Spinal Static and Posture. *Manuelle Mexizin Ihr Wissenschattlichen Grundlagen*, pp. 149–156, 1970.
71. Jirout, J., Correlations of the Functional Disturbances. In: The Cervical Spine as Seen Roentgenographically in Grontal and Sagittal Projection. *Svy. Univ. Carolinae*, Suppl. 21: 128–129, 1965.
72. Jirout, J., The Effect of Mobilization of the Segment. Blockage on the Sagittal Component of the Reaction on Lateroflexion of the Cervical Spine. *Neuroradiology*, 3: pp. 210–215, 1972.
73. Krausova, L. and Lewit, K., The Mechanism and Measuring of the Mobility in the Craniocervical Joints During Lateral Inclination. *Acta Univ. Carolinae (Med.)* Suppl. 21: pp. 123–126, 1965.
74. Goldthwaite, J. E., Variations in Anatomical Structure of the Lumbar Spine. *A. J. Orthop. Surg.* 2:146, 1920.
75. Steindler, G. A., *Kinesiology*, p. 145. Charles C Thomas, Springfield, Ill., 1955.
76. Gutman, G., Opinion on the Change of Cervical Spine. *Man. Med.*, 6, pp. 124–134, 1973.
77. Jirout, J., *Manuelle Medicine und lhrd Wissenschaftlichen. Grundlagen*, pp. 156–166, Verlag Fur Physikausche Medizin, Heidelberg, 1970.
78. Korr, I. M., The Concept of Facilitation and its Origin. *J.A.O.A.*, January 1955.
79. Korr, I. M., Clinical Significance of the Facilitated State. *J.A.O.A.*, January 1955.

80. Sherrington, O. C., *The Selected Writings of Sir Charles Sherrington.* Paul B. Hoeber, New York, 1940, p. 532.
81. Denslow, J. S. and Clough, G. H., Reflex Activity of the Spinal Extensors. *J. of Neurophysiol.*, No. 4, pp. 430–437, 1941.
82. Denslow, J. S. and Hassett, C. C., The Central Excitatory State Associated With Postural Abnormalities. *J. of Neurophysiol.*, No. 5, pp. 393–402, 1942.
83. Lewin, T., Osteoarthritis in Lumbar Synovial Joints. *Acta Orthop. Scand.*, Supp. 73, 1965.
84. Sandoz, R., Radiographic Semiology of Posterior Vertebral Arthrosis. *Annals of the Swiss Chiropractor's Association*, Vol. 4. 1969, pp. 107–154.
85. Lazorthes, G., Despeyrou, L., Juskiewewski, S., *Ann. Med. Phys.* 8, 1:67.
86. MacNab, L., Pathogenesis of Symptoms In Discogenic Low Back Pain. *Symposium on the Spine*, American Academy of Ortho-Surgeons, Cleveland, Ohio, November 1967, pp. 67–110.
87. Gayral, L. and Neuwirth, E., Oto-neuro Ophthalmologic Manifestations of Cervical Origin; Posterior Cervical Sympathetic Syndrome of Barre-Lieou. *New York State Journal of Medicine* 54: 1920, July 26, 1954.
88. Wall, P. D., The Mechanisms of Pain Associated With Cervical Vertebral Disease. *Proceedings of the International Symposium* held in Wenner-Gren Centre, Stockholm, Vol. 19, pp. 201-208, January 1971.
89. Badgley, C. E., The Articular Facets in Relation to Low Back Pain and Sciatica Radiation. *J. Bone and Joint Surg.*, 23:481, 1941.
90. Farfan, H., *Mechanical Disorders of the Low Back.* Lea and Febiger (Philadelphia, Pa.), 1973.
91. Armstrong, J. R., *Lumbar Disc Lesions.* Livingston, Ltd., Edinburgh, 1965.
92. DePalma, A. F. and Rothman, R. H., *The Intervertebral Disc.* W. B. Saunders Co., Philadelphia, Pa., 1970.
93. Drum, D. C., *An Introduction to the Study of Posture and Spinal Mechanics.* Published by the author, 1970.
94. Caillet, R., *Low Back Pain Syndrome.* F. A. Davis Co., Philadelphia, Pa., 1965
95. Farfan, H. and Sullivan, J., The Relation of Facet Orientation to Intervertebral Disc Failure. *Canadian J. Surg.*, 10: 179, 1967.
96. Nachemson, A., Electromyographic Studies on the Vertebral portion of the Psoas Muscle. *Acta Orthop Scand.*, 37, pp. 177–190, 1966.
97. Bonney, G., Cervical Spondylarthrosis Orthopaedic Aspects. *Proc. R. Soc. Med.*, 57, pp. 162–163, 1964.
98. Hadley, L. A., Intervertebral Joint Subluxation, Bony Impingement and Foraminal Encroachment With Nerve Root Changes. *Am. J. Roent.*, Vol. 65, 1951.
99. Sharpless, S. K., Neurophysiology of Nerve Compression and Joint Fixation. *5th Annual Biomechanics Conference on the Spine*, December 7–8, 1974, Boulder, Colo.
100. Perl, E. R., *Proceedings of the International Symposium held in Wenner-Gren Centre*, Stockholm, January 1971, pp. 157–164. Mode of Action of Nociceptors.
101. MacNab, I., The Mechanism of Spondylogenic Pain. *Proceedings of the International Symposium held in Wenner-Gren Centre*, Stockholm, January 1971.
102. LaRocca, G., MacNab, I., Value of Preemployment Radiographic Assessment of the Lumbar Spine. *Industrial Medicine*, Vol. 39, No. 6, June 1970, pp. 253–258.
103. McRae, D. L., Asymptomatic Intervertebral Disc Protrustions. *Acta Radiol.*, 46, 1956, pp. 9–27.
104. Mathews, J. A. and Yates, D. A. H., Reduction of Lumbar Disc Prolapse by Manipulation. *Br. Med. J.*, 3 pp. 696–697, 1969.
105. Homewood, A. E., *The Neurodynamics of the Vertebral Subluxation.* Chiropractic Publishers Canada, 1962.
106. Widmann, B. J., Neurofunctional Aspects of Postural Disorders. *Annals of the Swiss Chiropractors' Assoc.*, 1961.
107. Gayral, L. and Neuwirth, E., Clinical Manifestations of the Autonomic Nervous System Sequential to Osteoarthritis of the Cervical Spine. *Lancet*, May 1958, pp. 197–198.
108. Korr, L. M., The Neural Basis of the Osteopathic Lesion presented at the 51st Annual Convention of *The A.O.A.*, July 1947.
109. Janse, J., The Vertebral Subluxation. *National College Journal of Chiropractic*, Vol. 20, No. 1, February 1948.
110. Stoddard, A., *Manual of Osteopathic Practice.* Harper and Row, New York, 1969.
111. Janse, J., Houser, R. H., Wells, B. F., *Chiropractic Principles and Technic.* National College of Chiropractic, Chicago, Ill., 1947.
112. Johnston, W. C., Segmental Behavior During Motion. *J.A.O.A.*, 72: pp. 352–373, 1972.
113. Gillet, H. and Liekens, M., *Belgian Chiropractic Research Notes.* Published by the authors, eighth edition, 1970, 5, rue de la Limite, Brussels, Belgium.
114. Stary, C., Polyelectromyelographic Studies of Proprioceptive Analysis. Analysis Disorders During the Initial Phases of Vertebrogenic Disease in Children. *Acta Universities Carolinae Med.*, Suppl. 21: pp. 21–23, 1965.
115. England, R. W. and Deibert, P. W., Consideration in the Evaluation of Osteopathic Therapy (Electromyelographic Studies). *J.A.O.A.*, 72: pp. 162–164, October 1972.
116. Denslow, J. S. and Clough, G. H., Electromyelographic Studies of Structural Abnormalities. *J.A.O.A.*, Vol. 41, pp. 175–179, December 1941.
117. Denslow, J. S. and Hasset, C. C., Spontaneous and Induced Spasm in Structural Abnormalities. *J.A.O.A.*, Vol. 42: pp. 207–211, January 1942.
118. Mitatsky, Z., Sussova, J., Figar, S., Stary, O., Electroencephalographic Studies of the Origin and Fixation of Conditioned Nociceptive Reflexes in Cases of Lumbar Discopathies. *Acta Universitatis Carolinae Medico*, September 21, 1965.
119. Seno, S., The Motion of the Spine and the Related Electromyogram of the Patient Suffering From Lumbago. *20th Japanese Congress of Electromyography*, 1968.
120. Yamaji, J. and Misu, A., Kinesiologic Study With Electromyography of Low Back Pain. *20th Japanese Congress of Electromyography*, 1968.
121. Miyaski, A. and Sakou, T., Posture and Low Back Pain Electromyographic Evaluation. *20th Japanese Congress of Electromyography*, 1968.
122. Lewit, K., *Manuelle Therapie.* J. A. Bath, Leipzig, 1973.

123. Paris, S. V., Manipulative Decreasing Restriction of Spinal Movement. Gross Spinal Movements and Their Restriction as the Basis of Joint Manipulation. *Prog. Phys. Ther.* 1: pp. 208–213, paper # 709, 1970.
124. Hviid, H., The Influence of the Chiropractic Treatment on the Rotary Mobility of the Cervical Spine. A Kinesiometric and Statistical Study. *Annals of the Swiss Chiropractors' Association*, Vol. 5, p. 31, 1971.
125. Gutmann, G., Possibility of Objectivation of the Chiropractic Effect. *Mediciene et Hygene*, No. 960, pp. 663–667, April 1971.
126. Jirout, J., Changes in the Sagittal Component of the Reaction of the Cervical Spine to Lateroflexion After Manipulation of Movement Restriction. *Cesk. Neurol.*, 68, 1972, C4, pp. 174–180.
127. Jochumsen, O. H., The Curve of the Cervical Spine. *A.C.A. Journal of Chiropractic*, vol. IV, p. 49, August 1970.
128. Gutmann, G., The Roentgenological Diagnosis of Vertebral Fixations. *Chirotherapie Manuelle Therapie.* Therapie uber das Nervensystem (BD7), pp. 212–224, Hippokrates Verlag. Stuttgart, 1967.
129. Illi, F. W., *The Vertebral Column.* Published by The National College of Chiropractic, 1951.
130. Gutmann, G., The *Cervical Spine and Manual Therapy.* Chyrotherapie Manuelle Therapie. Therapie Uber das Nervensystem, Band 7, Hippokrates Verlag, Stuttgart.
131. Grice, A., A Preliminary Electromyographic Study of Muscle Tonus Changes Pre- and Post Manipulation. *J. of the Canadian Chiropractic Association*, 4th Quarter, Vol. XVIII, No. 4, 1974.
132. Krausova, L., Otoneurological Symptomatology in Cervicocranial Syndrome Before and After Manipulative Therapy. *Manuelle Medizin und ilhre wissenschaftlichen Grundlagen, Verlag fur physikalische medizine*, Heidelberg, 1970.
133. Hikosaka, O. and Maeda, M., Cervical Effects on Abducens Motorneurons and Their Interaction With Vestibulo Ocula Reflex. *Exp. Brain Res.*, 18, pp. 512–530, 1973.
134. Igarashi, Miyata, Alford, Wright, Nystagmus after Experimental Cervical Lesions, 1972. *Laryngoscope*, 82: September 1972.
135. Simon, H. and Moser, M., *Manual Medicine and Otorhinolaryngology*, #11. January 1973, Heft 3, p. 49. Abstracted and translated from the German original by T. Rudolf.
136. Hargrave-Wilson, W., The Cervical Syndrome. *Australian J. of Physiol.*, XVIII 4, pp. 144–147, December 1972.
137. Thomas, P. E., Sympathetic Activity in Facilitated Segments. *Sudomotor Studies*, January 1955.
138. Burns, L., Vertebral Lesions and Gastric Mobility. *J. Am. Osteop. A.*, 33: pp. 294–297, March 1934.
139. Burns, L., *J. Am. Osteop. A.*, 34: pp. 133–135, November 1934.
140. Ussher, N. T., Spinal Curvatures – Visceral Disturbances in Relation Thereto. *California and Western Medical J.*, Vol. 38, No. 16, 1933.
141. Kamieth, H., Pathogenetic Importance of the Thoracic Portion of the Vertebral Column. *Arch. Orthop. Unfall Chir.*, 49: pp. 585–606, No. 6, 1958.
142. Wright, H. and Robbins, R. L., Preliminary Studies of the Influence of Acute Postural Stress in the Skin Patterns of the Back. *J. A. Osteo. A.*, vol. 63, No. 873, pp. 984–998, 1966.
143. Figar, S., Stary, O., Hloddka, V., Changes in Vasomotor Reflexes in Painful Vertebrogenic Syndromes. *Review of Czechoslavak Medicine*, X–4, 1964, pp. 238–246.
144. Korr, I. M., Buzelli, K. A., Hix, E. L., Northup, G. W. Kugelmass, I. N., *The Physiological Basis of Osteopathic Medicine.* Published by the Postgraduate Institute of Osteopathic Medicine and Surgery, New York, 1970.
145. Wright, H., Local Vasomotor Disturbances. *J. A. O. A.*, Vol. 56, pp. 217–224, 1956
146. Korr, I. M., Wright, H. M., and Thomas, P. E., Effects of Experimental Myofascial Insults on Cutaneous Patterns of Sympathetic Activity in Man. *Acta Neuroveg.*, 23: pp. 323–355, 1962.
147. Korr, I. M., Wright, H. M., and Chace, J. A., Cutaneous Patterns of Sympathetic Activity in Clinical Abnormalities of the Muscoskeletal System. *Acta Neuroveg.*, 25: pp. 598–606, 1964.
148. Figar, S. K. and Lewit, K., Plethysomagraphic Studies of the Effects of Chiropractic Treatment Intervertebrogenic Syndromes. *Acta Neurology*, 29, 1967, 4, pp. 618–623.
149. Stary, O., The Reflex Vasomotor Disorders Due to Nociceptioc Root Stimulation in Discopostics. *Neurol. and Psychiat.*, cs. 17, 1954, p. 179.
150. Haldeman, S., Observations Made Under Test Conditions With Syncro-therme. *The Journal of the CCA*, October 1970.
151. B. J. Palmer Clinic Report (1945). *Audiometer Changes Under Specific Chiropractic Adjustment.*
152. Hood, R. P., Blood Pressure – 75 Abnormal Cases. *Digest of Chiropractic Economics*, vol. 16, No. 6, May–June 1974, p. 36.
153. Norris, T., A study of the Effect of Manipulation on Blood Pressure. *Academy of Applied Osteopathy 1964 Year Book*, p. 184, 1964.
154. B. J. Palmer Clinic Report (1946). *Electrocardiographic Changes Under Specific Chiropractic Manipulation.*
155. Shapiro, J., Hann, D., Leer, King, G., Effects of Chiropractic Treatment in Blood Pressure, Pulse Pressure, and Pulse Rate. *Journal of the Canadian Chiropractic Assoc.*, 4th quarter, vol. XVIII, No. 4, 1974.
156. Celender, E., Koenig, A. J., Celender, D. R., Effect of Osteopathic Manipulative Therapy on Autonomic Tone as Evidenced by Blood Pressure Changes and Activity of the Fibrinolytic System. *J.A.O.A.*, Vol. 67, May 1968.
157. Fischera, A. P., Celender, D. R., Effect of Osteopathic Manipulation Therapy on Autonomic Tone as Evidenced by Blood Pressure Changes and Activity of the Fibrinolytic System. *J.A.O.A.*, June 1969.
158. B. J. Palmer Clinic Report, *Hematologic Changes Under Specific Chiropractic Adjustment.*
159. Hursch, B. W., et al., Cardiovascular Changes After Chiropractic. *A.C.A. Journal*, vol. 8, March 1974, pp. 33–38.
160. Johnston, R., Vertebrogenic Stress Eosinopenia, Part I. *J. of the Canadian Chiropractic Assoc.*, 4th quarter, vol. XVIII, No. 4, 1974.
161. Granit, R. J., Reflex Self Regulation of Muscle Contrac-

tion and Autogenic Inhibition. *Neurophysiol.*, 13: pp. 351–372, 1950.
162. Kibler, R. F. and Nathan, P. W., Relief of Pain and Paraesthesia by Nerve Block Distal to the Lesion. *J. Neurol. Neurosurg. Psychiatry*, 23, 91–98, 1960.
163. Gunsberger, M., Acupuncture in the Sore Throat Symptomatology. *Am. J. Chinese Med.*, vol. 2, pp. 337–340, 1973.
164. Kac, F. F., et al., Acupuncture Anesthesia in Herniorrhaphy. *Am. J. Chinese Med.*, Vol. 1, No. 2, pp. 327–328, 1973.
165. Roccia, L., Personal Experience With Acupuncture in General Surgery. *Am. J. Chinese Medicine*, Vol. 1 No. 2, pp. 329–335, 1973.
166. Wayne, Y. H. H. and Chan, J. Y. P., Acupuncture Anesthesia: A Report of Two Cases. *Am. J. of Chinese Med., vol. No. 1, pp. 151–154.*
167. Kakizaki, K. and Manaba, Y., Static Potential Analgesic Applied in Caesarean Sections. *Am. J. of Chinese Med.*, vol. 2, No. 4, pp. 429–432, 1974.
168. Grays Anatomy, 35th Edition, Longman, 1973, p. 1065.
169. Pick, J., *The Autonomic Nervous System.* Lippincott, Philadelphia, Pa., 1970.
170. Odovan, D., The Possible Significance of Scoliosis of the Spine in Causation of Asthma. *Annals of Allergy*, March, April 1951, pp. 184–219.
171. Haldeman, S., Interactions Between the Somatic and Visceral Nervous Systems. *J. of Canadian Chiropractic Assoc.*, October 1971.
172. Egli, A. B., Spine and Heart: Vertebrogenic Cardiac Syndrome. *Annals of the Swiss Chiropractors' Assoc.*, vol. IV, pp. 95–106, 1969.
173. Burns, L., Early Pathogenesis Following Vertebral Strain. *J.A.O.A.*, vol. 46, No. 2, October 1946.
174. Hoag, J. M., Muscular Skeletal Involvement in Chronic Lung Disease. *J.A.O.A.*, vol. 71, April 1972, pp. 698–706.
175. Kunert, W., Functional Disorders of Internal Organs Due to Vertebral Lesions. *Ciba Symposium*, 13, #3, 1965.
176. Robuck, S. V., Osteopathic Management of Gastric and Duodenal Ulcers. *J.A.O.A.*, vol. 46, No. 9, May 1947, pp. 465–468.
177. Bondies, I. O. and Stillman, C. J., *Diagnosis and Treatment of Ulcerative Gastritis.* 1935.
178. Verner, R. J., *The Science and Logic of Chiropractic.* Theo. Gaus & Son, New York, 1941.
179. Ebbetts, R., Autonomic Pain in the Upper Limb. *Physiotherapy*, Vol. 57, No. 6, 1971.
180. Denslow, J. S., Palpation of the Musculoskeletal System. *J.A.O.A.*, Vol. 16, August 1964.
181. Wright, H. M., Clinical Significance of the Facilitated State. *J.A.O.A.*, January 1955.

A Critical Look at the Treatment for Low Back Pain

ALF NACHEMSON
Professor and Chairman
Department of Orthopaedic Surgery
University of Göteborg, Sweden

In order to discuss the treatment of back pain by manipulation, an overview of the whole problem along with other types of treatment must be given.

We know this is a field of importance for the individual patient as well as for society. In epidemiological studies performed in Sweden,[1-3] in England,[4] and in Israel,[5] it has been demonstrated that 70–80 percent of the population in modern industrialized societies some time during active years will suffer back pain—the majority between 20 and 55 years of age. The problem seems to be of the same magnitude in the United States.[6]

I will limit my discussion to the treatment of low back pain—by far the most common of the back syndromes and also the most frequently manipulated.[7]

There are two key issues in this conference.

1. What is pain and exactly how is it elicited?
2. What is the cause and origin of low back pain?

It seems clear that some anatomical structure must be diseased or at fault in order to elicit pain.[8]

Having engaged in research in this field for nearly 25 years and having been clinically engaged in back problems for nearly the same period of time, and as a member and scientific adviser to several international back associations, I can only state that for the majority of our patients, *the true cause of low back pain is unknown.*

The origin of pain is most likely looked for in the lower lumbar motion segments. Practically all structures in this region have had their proponents in the etiological discussion, from the skin and subcutaneous tissue down to the disc.

Without discussing each one of them, I would like to mention the lumbosacral fascia in which one investigator[9] has detected an inflammatory reaction and has performed 10,000 lumbar fasciectomies over the years.

The muscles, which some treat for nodulus, fibrositis, and contractures, have never been demonstrated pathoanatomically to show any significant changes, nor have any consistent EMG changes been demonstrated.

The yellow ligament, the ligamentum flavum, was thought to be responsible for sciatica; that structure is still believed by some orthopedic surgeons to cause pain when it is thickened.[10] Such an increase in width is, however, always secondary to severe disc degeneration.[11]

From a theoretical point of view, the intervertebral (facet) joints seem to be a likely site of origin for back pain. It must be stressed, however, that pathoanatomical changes have always been found in these joints secondary to degenerative changes in the intervertebral disc.[12, 13] Medical or paramedical literature does not show facet subluxation or dislocation as being more common in normal individuals than in those with back pain. We do not even have knowledge of what is the normal radiographic picture of these joints.[14-16]

With recently developed techniques of tomographic examination, this seems feasible to obtain.[16, 17] Decreased motion of the facet joints, according to some authors,[14, 15, 17] is always secondary to disc degeneration. In a few older patients with severe osteoarthritic changes in their lumbar facet joints, however, it might be possible that this is responsible for back pain.

For the majority of scientists working in this field, the origin of the pain is most likely in the lumbar disc or its close surrounding, the longitudinal ligaments.

Unfortunately, we lack direct conclusive evidence of the part played by the disc in the etiology, but a number of indirect indications should be mentioned.

1. The disc hernia is usually preceded by one or more attacks of low back pain.[2, 18]

2. Following intradiscal injection either by hypertonic saline or by contrast media, it is often possible in patients with complaints as well as in symptom-free subjects to cause pain of the same type as experienced earlier.[19-21] It is not fully understood if this pain is due to increase in intradiscal pressure or to chemical irritation; either could act on the outermost layer of the annulus where nerve endings are located.[22, 23] Inside the intervertebral disc proper, however, no nerve endings have been found.

3. Investigations have been performed in which during surgery for sciatica, thin nylon threads were fastened to the fascia lumbosacralis, the muscles, ligamentum interspinale, the intervertebral joints, and ligamentum flavum, and also to the posterior part of the annulus fibrosus, to the dorsal longitudinal ligament, and to the nerve root proper.[24] These structures were irritated 3 to 4 weeks after surgery by pulling the threads, but pain resembling that which the patient had experienced previously could be elicited only from the three last mentioned structures.

4. Pathoanatomically, radiating ruptures are known to occur in the posterior part of the annulus reaching toward the areas in which nerve endings are located. The presence of such single ruptures in the lumbar discs is first seen around the age of 25, i.e. the same age at which the low back pain syndromes start to be of clinical importance.[18, 25, 26, 27, 28]

The intervertebral disc has been subjected to a number of morphological and chemical investigations, but they have shed no light on the origin of pain; these investigations have, however, expanded our knowledge of the biological events in that largely avascular structure.[18, 25-35] Eventually all individuals will have degenerative changes in the discs, characterized by increased fibrosis in the nucleus and ruptures in the annulus. Some subjects show such changes earlier than others, but it has been impossible to prove a direct relationship with the low back pain syndrome. Thus, all we can state is it is likely that certain stages in the aging process are significant in the production of the pain syndromes.[36] Evidence in favor of this is that prolapse is known to occur only in discs with degenerative changes. Secondly, in roentgenologically demonstrable disc degeneration, the number of patients who had had low back pain is significantly greater than comparable subjects in whom no roentgenologic changes can be found.[3] On the other hand, it is wrong to state that a roentgenologically degenerated disc definitely means that the patient's troubles are due to that particular interspace.[18, 37-39]

Thus, it seems that the trouble starts during the stages of degeneration which occur before the changes are clearly visible on the roentgenograms. This means that we cannot say to our patients: "It is obvious from the X-rays that you have a bad back." Certainly such a statement will only create further anxiety and will never increase the cure rate. The roentgenologically degenerated disc space probably should be looked upon as "burned out," and chances are greater that it is not the specific cause of the patient's present condition.

Before further discussing different types of treatment for this disease, it must be clearly stated that, since the cause is unknown, there is only symptomatic treatement available.

Dr. Gitelman[40] in his paper quotes a controlled series conducted by Coyer and Curwen[41] where they showed a trend for better results from manipulation in the first week but not after 6 weeks. Their own statement was: "Statistical analysis has not yet been applied because the present preliminary figures are inadequate for this purpose."

There are a few more similar trials on manipulation in the literature, none of which show statistically valid results.[10, 42, 43]

Beechgaard[44] reports that he personally followed 18 patients himself, not 800, and that 64 percent of the 800 patients manipulated—the majority of them in the lumbar area—showed good results.

The only randomized clinical trial in this field, to my knowledge, was recently published by Glover et al.[45] demonstrating no *statistically significant difference between those manipulated and those receiving detuned short wave.*

In a recent article by Kane and associates[46] from Utah, no statistical difference between patients treated by physicians and patients treated by chiropractors was seen except for the number of visits.

The chiropractors saw their patients nearly twice as many times as the physicians.

In Dr. Gitelman's paper[40] reference is given to some papers[47-50] demonstrating the effect of manipulation as measured by electromyography. Careful scrutiny reveals, however, only anecdotal observations with no quantitatively documented results and no comparison with any normal material. There is no statistical evaluation of the results.

Gitelman also said[40] that this conference by its very existence is proof that manipulation is effective and that "any fairminded thinker does not doubt the fact that it works." May I express my doubts. There is no proof that manipulation for acute or subacute low back pain patients has been demonstrably better in large control series than it has with simple bed rest and salicylates.

Several epidemiological studies[1-3,36] have demonstrated that nearly 50 percent of patients improve in a few weeks irrespective of treatment given and some 80 percent, even as high as 86 percent in a recent English study,[4] are well in 2 months. Dr. St. Clair Dixon,[4] in a recent paper given at the annual meeting of the British Association for Rheumatology and Rehabilitation, said that the placebo element of back pain is enormous. Anyone who takes care of the patient will provide help for 70 percent of patients.

In this context it must be emphasized that a careful clinical examination is of benefit. Perhaps this is the reason for the slight trend to be seen in some series[41,42,45] that the first one or two manipulative sessions seem to help a little more than, for example, detuned shortwave or bed rest. I must also mention that radiation therapy,[51] ultrasound,[10] shortwave diathermy,[52] traction,[53] injection of various drugs including hydrocortisone[54] and chymopapain[55,56] have not proven to be more effective. This is also true for lumbar fusion operations for low back pain[57-60] and facet joint denervation.[61,62] Most of us in our present state of ignorance get 70–80 percent good results. There is, however, one important difference. The latter procedures are only performed in patients with more chronic back pain. For these, no evidence exists at all of the percentage helped by manipulations.

The psychological aspects of back pain have recently been emphasized by several authors[63-65] and certainly have to be subjected to further evaluation.

With actually no treatment program being superior to simple measures such as bed rest and salicylates, why do most orthopaedic surgeons[66] refute manipulative chiropractic or osteopathic types of treatment? Perhaps because these latter treatments are based on theories of disease processes that at least for the majority of our patients have never been proven or disproven. Those medical doctors, like Dr. Cyriax,[67] who use manipulative treatment are convinced they are treating a disc lesion. There is evidence that disc morphology is changing from 20 years of age onward; this is also the age at which attacks of low back pain become more common in the population.[1-3,37]

In the medical field, new types of treatment for low back pain are introduced with some frequency, tried for some time, and then proven or disproven by clinical trials in various places around the world. No such attempts have been made by chiropractors or osteopaths for nearly 100 years.

The enormous problem of back pain cannot be solved by "armchair pathology." Yet, there is one single pathological entity in this field where scientific evidence does exist. That is the true disc hernia, which, when found at operation to be pressing on a nerve root, on removal gives a 90–95 percent good result.[10,18,37,64,68-70]

The true herniated disc can be successfully treated conservatively with bed rest,[10,70] with controlled manipulation under anesthesia,[43] or traction,[10,53] with extradural,[10,71] or intradiscal,[54,72] hydrocortisone injections and intradiscal chymopapain injections.[55,72,73] It remains to be demonstrated that the results are equally good as those obtained by surgical removal in patients with a definite disc hernia. These methods are, however, helpful in avoiding operation in some patients.

Unfortunately, this is not much consolation, since few back patients develop symptoms, signs, and pathology of a true prolapsed disc. For the vast majority of our patients we still have to look for the origin of the pain as well as for the "right" treatment.

Why should it be difficult for the lay public and for us who are treating patients to comprehend and explain that we do not know the cause of this disorder? We know and accept that the cause and cure of cancer is still hidden. This is the same with back pain. On the other hand, mankind has a tremendous need for explanation. This demand can be met with information of those few facts that exist. We all agree that patients with low back pain experience an increase of their troubles when

their backs are mechanically stressed. The results obtained by intravital disc pressure measurements [36, 74, 75] constitute a base for patient education. This has led to what we in Sweden call "the low back pain school." [76] The purpose of this is to: (1) create confidence for the patient to be able to cope with his back troubles; (2) avoid excess therapy; (3) decrease the expenses both for the individual and the society.

The back school consists of four group sessions with six patients led by a physiotherapist. The first and second lessons give information on the anatomy and function of the spine and present knowledge on low back pain, its etiology, frequency, and therapeutic efforts. The mechanism of the spine is explained, based on low pressure measurements and movements and positions are analyzed (tables 1 and 2, figure 1). The importance of decreasing the load on the back is stressed at work, at home, and at rest.

TABLE 1

Approximate Load on L3 Disc in 70 Kilogram Individual in Different Positions, Movements, and Maneuvers

Activity	Load
	kg
Supine	30
Standing	70
Upright sitting, no support	100
Walking	85
Twisting	90
Bending sideways	95
Coughing	110
Jumping	110
Straining	120
Laughing	120
Bending forward 20°	120
Lifting of 20 kg, back straight, knees bent	210
Lifting of 20 kg, back bent, knees straight	340

TABLE 2

Approximate Load on L3 Disc in 70 Kilogram Individual in Different Positions and Exercises

Activity	Load
	kg
Standing	70
Bending forward 20° with 10 kg, in each hand	185
Supine	30
Supine in traction (30 kg)	10
Bilateral straight-leg raising, supine	120
Sit-up exercise with knees bent	180
Sit-up exercise with knees extended	175
Isometric abdominal muscle exercise	110
Active back hyperextension, prone	150

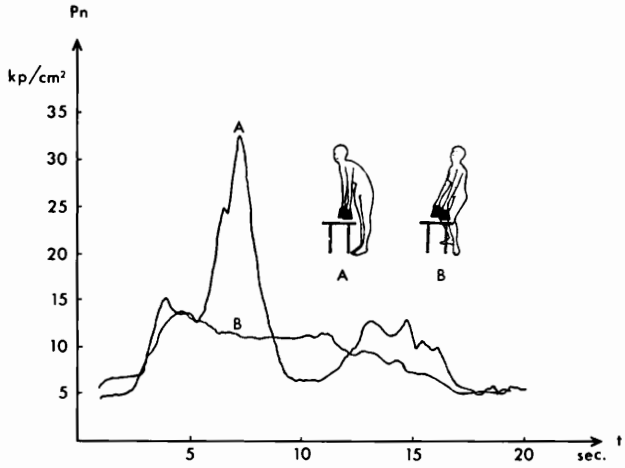


Fig. 1. Pressure recorded from the L3 disc in a 25-year-old male lifting 20 kg with bending of back and knees straight, with back straight and bending of knees.

The third lesson is a practical application of the previous theoretical lessons. Individual advice regarding working and resting positions is given. Isometric abdominal and back muscle exercises and leg exercises are taught, the former because measurements [74] have demonstrated that they load the lumbar spine less than isotonic exercises. The advantage of such an approach has been clinically verified.[76, 77]

The last lesson is mainly a repetition of the course including an examination to avoid misunderstanding and to underline the importance of self-confidence for the future. Physical activities, sports, and plays are encouraged to improve psychological and physical tolerance of pain and stress.

The physiotherapist's primary task is to teach ergonomics and to help patients to cope with their back troubles. The basic therapeutic principle is rest in the semi-Fowler position combined with analgesics, postural, and ergonomic advice. Since most of the patients get well within a couple of weeks irrespective of treatment, this seems to be the most logical approach in the absence of knowledge about the specific cause of the back pain.

In the therapeutic field today the introduction of a new drug is practically impossible without clinical and laboratory tests to prove its effectiveness; we are sensitive to and critical of pharmacological side effects. The same posture should be taken with regard to the different forms of treat-

ment for low back pain. Present methods should be given a critical look.

Thus, while awaiting further knowledge about the back problem, it is in the interest of our patients and ourselves to prescribe simple and inexpensive methods of treatment in which the known clinical, biological, and mechanical factors can serve as guides. A plea is made for all of us to evaluate our belief in a statistically sound manner.

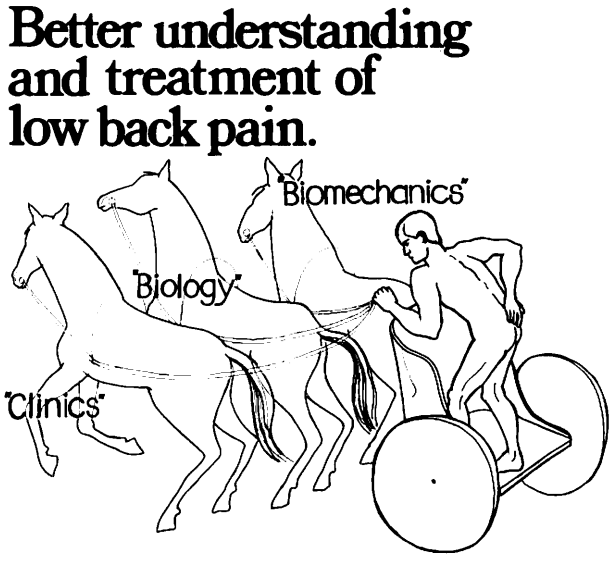


Fig. 2. The troika of low back pain.

As seen from figure 2, only by investigative efforts by clinicians of all kinds, by engineers, by chemists, and by pathologists is it likely that the etiological and therapeutic problems of low back pain may be solved.

REFERENCES

1. Hult, L. The Munkfors investigation. *Acta Orthop. Scand.*, Supp. 16, 1954.
2. Hult, L. Cervical, dorsal, and lumbar spinal syndromes. A field investigation of a nonselected material of 1,200 workers in different occupations with special reference to disc degeneration and so-called muscular rheumatism. *Acta Orthop. Scand.*, Supp. 17, 1954.
3. Horal J. The clinical appearance of low back disorders in the city of Gothenburg, Sweden. *Acta Orthop. Scand.*, Supp. 118, 1969.
4. Dixon, A. St. C. Progress and problems in back pain research. *Rheumatology and Rehabilitation* 12:165, 1973.
5. Magora, A. Investigation of relation between low back pain and occupation. 1. Age, sex, community, education, and other factors. *Ind. Med. Surg.* 39:465, 1970.
6. Chaffin, D. B. Human strength capability and low back pain. *J. Occup. Med.*, 16:248, 1974.
7. Smith, R. L. *At Your Own Risk. The Case Against Chiropractic*. New York. Pocket books. Simon & Schuster, Inc., 1969.
8. Werle, E., and Zickgraf-Ruedel, G. *Intrinsic Pain-Producing Substances of the Body*. Sandorama, Sandoz Ltd., Basle, Switzerland, 1972.
9. Morotomi, T. Affections of spine. Ed. T. Amako. In: *Orthopaedics*, p. 351, Tokyo, Japan, Kanehara, 1960.
10. Hanraets, P. R. M. *The Degenerative Back and its Differential Diagnosis*. Amsterdam, the Netherlands, Elsevier Publishing Co., 1959.
11. Nachemson, A., and Evans, J. H. Some mechanical properties of the third human lumbar interlaminar ligament (ligamentum flavum). *J. Biomechanics* 1:211, 1968.
12. Lewin, T., Moffett B., and Viidik, A. The morphology of the lumbar synovial intervertebral joints. *Acta Morph. Neerl. Scand.* 4:299, 1961.
13. Lewin, T. *Osteoarthritis in Lumbar Synovial Joints*. A morphologic study (thesis). The Department of Anatomy and the Department of Orthopaedics, Göteborg, Sweden, 1964.
14. Lewin, T. Anatomical variations in lumbosacral synovial joints with particular reference to subluxation. *Acta Anat.* 71:229, 1968.
15. Hirsch, C., and Lewin, T. Lumbosacral synovial joints in flexion-extension. *Acta Orthop. Scand.* 39:303, 1968.
16. Reichmann S. *The Lumbar Intervertebral Joints. An Anatomical and Roentgenological Study* (thesis). The Department of Anatomy and the Department of Diagnostic Radiology III, Goteborg, Sweden, 1971.
17. Reichmann, S., Berglund, E. and Lundgren, K. Das Bewegungszentrum in der Lendenwirbelsäule bei Flexion und Extension. *Z. Anat Entwicklungsgesch* 138:283, 1972.
18. Friberg S. and Hirsch C. Anatomical and clinical studies on lumbar disc degeneration. *Acta Orthop. Scand.* 14:222, 1949.
19. Hirsch C. An attempt to diagnose the level of a disc lesion clinically by disc puncture. *Acta Orthop. Scand.* 18:132, 1948.
20. Lindblom K. Technique and results of diagnostic disc puncture and injection (discography) in the lumbar region. *Acta Orthop. Scand.* 20:315, 1950.
21. Holt E. P. The question of lumbar discography. *J. Bone Joint Surg.* 50-A:720, 1968.
22. Hirsch, C., Ingelmark, B.-E., and Miller, M. The anatomical basis for low back pain. *Acta Orthop. Scand.* 33:1, 1963.
23. Jackson, H. C., Winkelmann, R. K., and Bickel, W. H. Nerve endings in the human lumbar spinal column and related structures. *J. Bone Joint Surg.* 48-A:1272, 1967.
24. Smyth, M. J., and Wright, V. Sciatica and the intervertebral disc. An experimental study. *J. Bone Joint Surg.* 40-A: 1401, 1958.
25. Hirsch, C. and Schajowicz, F. Studies on structural changes in the lumbar annulus fibrosus. *Acta Orthop. Scand.* 22:184, 1952.
26. Coventry, M. B., Ghormley, R. K., and Kernohan, J. W. The intervertebral disc: its microscopic anatomy and pathology. Part 1. Anatomy, development, and physiology. *J. Bone Joint Surg.* 27:105, 1945.
27. Coventry, M. B., Ghormley, R. K., and Kernohan, J. W. The intervertebral disc: Part II. Changes in the inter-

vertebral disc concomitant with age. *J. Bone Joint Surg.* 27:233, 1945.
28. Coventry, M. B., Ghormley, R. K., and Kernohan, J. W. The intervertebral disc: Part III. Pathological changes in the intervertebral disc. *J. Bone Joint Surg.* 27:460, 1945.
29. Bijlsma, F., and Peereboom, J. W. C. The aging pattern of human intervertebral disc. *Gerontologia* 18:157, 1972.
30. Jayson, M. I. V. and Barks, J. S. Structural changes in the intervertebral discs. *Ann. Rheum. Dis.* 32:10, 1973.
31. Nachemson, A., Lewin, T., Maroudas, A., and Freeman, M. A. R. In vitro diffusion of dye through the end plates and the annulus fibrosus of human lumbar intervertebral discs. *Acta Orthop. Scand.* 41:589, 1970.
32. Maroudas, A., Nachemson, A., Stockwell, R., and Urban, J. In vitro studies of the diffusion of glucose into the intervertebral disc. To be published in *J. Anat.*
33. Naylor, A. The biophysical and biochemical aspects of intervertebral disc herniation and degeneration. *Ann. Roy. Coll. Surg. Eng.*, 31:91, 1962.
34. Peyron, J.-G. Biologie due disque intervertébral. *Sem. Hop., Paris*, 43:3318, 1967.
35. Feffer, H. L. A physiological approach to lumbar intervertebral disc derangement. Ed., J. P. Adams. In: *Curr. Pract. Orthop. Surg.*, 1, p. 111, St. Louis, C. V. Mosby Co., 1963.
36. Nachemson, A. Low back pain. Its etiology and treatment. *Clin. Med.*, 78:18, 1971.
37. Spangfort, E. V. The lumbar disc herniation. A computer-aided analysis of 2,504 operations. *Acta Orthop. Scand. Supp.*, 142, 1972.
38. Inman, V. T., and Saunders, J. B. Anatomicophysiological aspects of injuries to the intervertebral disc. *J. Bone Joint Surg.*, 29:461, 1947.
39. LaRocca, H. and MacNab, I. Value of preemployment radiographic assessment of the lumbar spine. *Canad. Med. Assn. J.*, 101:383, 1969.
40. Gitelman, R. The treatment of pain by spinal manipulation. Paper given at the NINDS-NIH workshop on the *Research Status of Spinal Manipulative Therapy*, Bethesda, Md., February 2–4, 1975.
41. Coyer, A. B. and Curwen, I. H. M. Low back pain treated by manipulation. A controlled series. *Brit. Med. J.*, March:705, 1955.
42. Edwards, B. C. Low back pain and pain resulting from lumbar spine conditions: A comparison of treatment results. *Aust. J. Physiother.* 15:104, 1969.
43. Chrisman, O. D., Mittnacht, A., and Snook, G. A. A study of the results following rotatory manipulation in the lumbar intervertebral-disc syndrome. *J. Bone Joint Surg.* 46-A:517, 1964.
44. Beehgaard, P. Late post traumatic headache and manipulation. *Corres. Brit. Med. J.*, June 4:1419, 1966.
45. Glover, J. R., Morris, J. G., and Khosla, T. Back pain: a randomized clinical trial of rotational manipulation of the trunk. *Brit. J. Industr. Med.* 31:59, 1974.
46. Kane, R. L., Leymaster, C., Olsen, D., Woolley, F. R., and Fischer, F. D. Manipulating the patient. A comparison of the effectiveness of physician and chiropractor care. *Lancet*, June, p. 1333, 1974.
47. Siehl, D., Olson, D. R., Ross, H. E., and Rockwood, E. E. Manipulation of the lumbar spine with the patient under general anesthesia: Evaluation by electromyography and clinical-neurologic examination of its use for lumbar nerve root compression syndrome. *J. Amer. Osteopath Assn.* 70:433, 1971.
48. Denslow, J. S. and Hassett, C. C. Spontaneous and induced spasm in structural abnormalities. *J. Amer. Osteopath. Assn.* 42:207, 1943.
49. Denslow, J. S. and Clough, D. O. Electromyographic studies of structural abnormalities. *J. Amer. Osteopath Assn.* 41:175, 1941.
50. England, R. W. and Deibert, P. W. Electromyographic studies: Part 1. Consideration in the evaluation of osteopathic therapy. *J. Amer. Osteopath Assn.* 72:162, 1972.
51. Goldie, I., Rosengren, B., Moberg, E., and Hedelin, E. Evaluation of radiation treatment of painful conditions of the locomotor system. A doubleblind study. *Acta Radiol.* 9:311, 1970.
52. Lehmann, J. F., Warren, C. G., and Scham, S. M. Therapeutic heat and cold. *Clin. Orthop.*, 99:207, 1974.
53. Weber, H. Har traksjonsbehandling noen effekt ved ischias forårsaket av skiveprolaps? *Tidsskrift for Den norske laegeforening* 33:2243, 1972.
54. Feffer, H. L. Therapeutic intradiscal hydrocortisone. A long-term study. *Clin. Orthop.* 67:100, 1969.
55. MacNab, I., McCullouch, J. A., Weiner, D. S., Hugo, E. P., Galway, R. D., and Dall, D. Chemonucleolysis. *Canad. J. Surg.*, 14:280, 1971.
56. Ford, L. T. Clinical use of chymopapain in lumbar and dorsal disk lesions. An end-result study. *Clin. Orthop.*, 67:81, 1969.
57. Rolander, S. D. Motion of the lumbar spine with special reference to the stabilizing effect of posterior fusion. *Acta Orthop. Scand.*, Supp. 90, 1966.
58. Stauffer, R. N. and Coventry, M. B. Posterolateral lumbar-spine fusion. Analysis of Mayo Clinic series. *J. Bone Joint Surg.* 54-A:1195, 1972.
59. Stauffer, R. N. and Coventry, M. B. Anterior interbody lumbar spine fusion: Analysis of Mayo Clinic series. *J. Bone Joint Surg.* 54-A:756, 1972.
60. Rothman, R. H. and Marvel, J. P., Jr. Pseudarthrosis of the lumbar spine. In: *Proc. of the First Meeting of the International Society for the Study of the Lumbar Spine* in Montreal, Canada, June 19–22, 1974. Also to be published in the *Orthopaedic Clinics of North America.*
61. Wyke, B. The neurology of joints. *Ann. Roy. Coll. Surg. Eng.* 41:25, 1967.
62. Shealy, C. N. The role of the spinal facets in back and sciatic pain. *Headache* 14:101, 1974.
63. Wolkind, S. N. and Forrest, A. J. Low back pain: a psychiatric investigation. *Postgrad. Med. J.* 48:76, 1972.
64. Roslund, J. *Indications for Lumbar Disk Surgery.* Factors influencing the long-term prognosis (thesis). The Department of Orthopaedic Surgery, Karolinska Hospital, Stockholm, Sweden, 1974.
65. Westrin, C.-G. Low back sick-listing. A nosological and medical insurance investigation. *Scand. J. Soc. Med. Supp.* 7, 1973.
66. Scham, S. M. Manipulation of the lumbosacral spine. *Clin. Orthop.* 101:146, 1974.
67. Cyriax, J. *Textbook of Orthopaedic Medicine, Vol. 1.* Diagnosis of soft tissue lesions. London, England, Baillière Tindall & Casell, 1969.
68. Sparup, K. H. *Late Prognosis in Lumbar Disc Herniation.* Copenhagen, Denmark, Munksgaard, 1960.

69. Söderberg, L. Prognosis in conservatively treated sciatica. *Acta Orthop. Scand.* Supp. 21, 1956.
70. Hakelius, A. Prognosis in sciatica. A clinical followup of surgical and nonsurgical treatment. *Acta Orthop. Scand.* Supp. 129, 1970.
71. Dilke, T. F. W., Burry, H. C., and Grahame, R. Extradural corticosteroid injection in management of lumbar nerve root compression. *Brit. Med. J.*, June:635, 1973.
72. Graham, C. E. Chemonucleolysis: A double-blind study comparing chemonucleolysis and intradiscal hydrocortisone in the treatment of backache and sciatica. Ed. O. D. Chrisman. In: *Proc. of the 26th Annual Meeting in Boston, Massachusetts*, June 15–19, 1974.
73. Smith, L. Chemonucleolysis. *Clin. Orthop.* 67:72, 1969.
74. Nachemson, A. and Elfström, G. Intravital dynamic pressure measurements in lumbar discs. A study of common movements, maneuvers, and exercises. *Scand. J. Rehab. Med. Supp.* 1, 1970.
75. Nachemson, A. Physiotherapy for low back pain patients. A critical look. *Scand. J. Rehab. Med.* 1:85, 1969.
76. Lidström, A. and Zachrisson, M. Physical therapy on low back pain and sciatica. An attempt at evaluation. *Scand. J. Rehab. Med.* 2:37, 1970.
77. Kendall, P. H. and Jenkins, J. M. Exercises for backache: A double-blind controlled trial. *Physiotherapy* 54:154, 1968.

Treatment of Visceral Disorders by Manipulative Therapy

WILLIAM D. MILLER
Acting Dean
The Postgraduate Institute of Osteopathic Medicine and Surgery
New York City, N.Y.

The relationship of the musculoskeletal system, the soma, and the viscera forms an interdependence recognized by all physicians. Disorders in the neuromusculoskeletal system, at times, and in some instances, may be a factor in visceral disorders and disease.[1] The literature is replete with discussions on disturbances in the neuromusculoskeletal system as possible factors in visceral disease.[2] The literature describes various somatovisceral and viscerosomatic reflexes as relating to a specific disease condition. When an average threshold stimulus is applied to a somatic receptor such as the skin, visceral function is influenced by way of the central connection in the gray matter of the spinal cord. If the sympathetic nervous system is involved, the activation occurs in the cells of the mediolateral column in the thoracolumbar area.[3] Since the sympathetic axones terminate in the contractile mechanism of vascular smooth muscle fibers, the end result could be vasoconstriction. Other sympathetic nerves affect heart rate, bronchodilation, sphincter contraction, etc. There is not a direct action and effect relationship between manipulation and a physiologic response. This point has two parts: (1) Malfunction or disturbances in the neuromusculoskeletal system can affect the physiology of tissue or organs of the body if the body does not compensate adequately; (2) Removal or diminution of disturbances in the neuromusculoskeletal system may be an aid in overcoming visceral disorders.[4]

As an example, the so-called shoulder-hand syndrome, periarthritis of the shoulder following acute myocardial infarction, is believed by some to be attributed to reflex spasm of the muscles and ligaments about the shoulder secondary to cardiac pain impulses referred to the same segment of the spinal cord as innervate the affected muscles and ligaments.[5,6]

The Postgraduate Institute of Osteopathic Medicine and Surgery planned as its main objective to determine whether individuals in whom these somatic disturbances are modified have a significant objective or subjective improvement in their disease and pulmonary function compared to a control group. It must be realized from the onset, alterations of visceral function by producing changes in the somatic musculature represent only one modality for treating visceral disease. In no way does this supplant other methods and procedures in treatment.

This study was brought about by the observation of those with wide experience in osteopathic medicine who had the clear clinical impression that certain osteopathic methods were valuable in diagnosis and therapy. The main objective was to determine whether individuals in whom neuromusculoskeletal disturbances were removed or appreciably modified had a significant improvement in their obstructive lung disease and pulmonary function.

This study was entertained with the idea to review uniformity of objective data and determine if patterns of postural stresses and/or faults in the intervertebral and costovertebral mobility, muscular contractions or other changes in physical structures are coexistent with chronic obstructive lung disease and whether they are statistically significant. In addition, efforts were made to establish the

efficacy of manipulative procedures designed to eliminate or modify musculoskeletal disturbances found in a given subject.

Patients admitted to the study were randomly assigned to the treated or control group. Medical and adjunctive treatment were the same for both groups, with the same breathing exercises given to each patient. In contrast to the control group, those in the treated group also received standardized manipulative treatments twice weekly. Thus, the design called for comparable therapy for the two groups except for the additional osteopathic manipulative treatment administered to the subjects in the treated group. This report in no way should be considered complete as other factors such as smoking, symptomatology as to coughing, occupation, etc. were not included so that the thrust of this report is directed toward the musculoskeletal changes and the results observed here.

METHODOLOGY

Patients admitted to the study had to meet the following criteria:

1. Age: 36 years to 65 years.
2. Height: 145–185 centimeters for females and 157–190 centimeters for males.
3. Weight: 41–85 kilos for females and 50–115 kilos for males.
4. A diagnosis of chronic obstructive lung disease.

Patients with concurrent disease were excluded if the disease was considered to have a sufficient potential for altering their response to treatment.

The diagnosis of the patient and the evaluation, the class, and severity of obstructive disease were made by an internist, using the criteria of Gaensler and Wright [7] before the patient was assigned to one of the two groups. A combination of random allocation and matched pairing was used to determine the group to which the patient would be assigned. Patients were paired according to their sex, race, degree of involvement, and age.

Briefly, this was the method used: The first patient in any given group was assigned at random to the treated or control group. The random allocation was carried out using a standard table of random numbers. The next patient admitted to the study with the same four characteristics however, was automatically assigned to the opposite group. These two patients now constituted a pair, matched on the four basic variables—one in the treated group and the other in the control group. It was expected that this system would produce two groups approximately equal with respect to severity of disease, sex, race, and age distribution, while at the same time minimizing the possibility of bias arising in the process of assigning any specific patient to one or the other group.

The initial examination included a detailed, general history and a series of objective measurements to be used as a basis for comparison in subsequent periodic evaluations made during the course of treatment. The history examination was a modified version of the Questionnaire on Respiratory Symptoms (1966) approved by the Medical Research Council's Committee on Research Into Chronic Bronchitis. In addition a complete blood count, serological test for syphilis, sputum culture, modified blood chemistry, urinalysis, 12-lead electrocardiogram, 2-meter chest X-ray, measurement of gas tensions, and pulmonary function studies including lung volumes, diffusion, maximum voluntary ventilation, vital capacity, FEV 1,2 and midexpiratory flow rate were done. In addition, a special standardized examination of the musculoskeletal system was conducted on each patient. Special attention was made to determine if there were evidences of neuromusculoskeletal stress and/or faults in intervertebral and costovertebral as well as costochondral articulations. Methods in use involved recording hypermobility, costovertebral dysfunction, or alterations in side bending, rotation. Skin drag was used to determine sudomotor changes and red reaction to determine vasomotor changes. Spinal examination recorded curvatures whether anterior-posterior or lateral. Deviations at segmental levels also were recorded both at the right and left sides of the body. In addition, muscle tension was graded both right and left. These matters were recorded with number four as arbitrarily accepted as normal. Above four was considered hyperreactive and below four hyporeactive.

The diagnosis and evaluation of the class and severity of the obstructive disease was then determined. The individual's number was assigned by random selection and placed in either the A-treated group or B-control group. Patients in both groups A and B received the same appropriate chemical, medical, and adjunctive therapy including bronchodilators, aerosol, intermittent positive pressure breathing, breathing exercises, postural drainage

graded exercises, and supplemental oxygen inhalation as indicated. Patients in group A received in addition osteopathic manipulative therapy twice weekly, including specific manipulative procedures. There was uniformity in the application of these procedures in all aspects. The procedures involved methods to hyperextend the dorsal spine using several techniques. Other techniques involved increasing any restrictive motion that could be present within the musculoskeletal system. Another method was to increase lymphatic flow by applying pressure to the muscles of the thoracic cage through anterior compression of the chest.[8,9]

The methods for osteopathic manipulative procedures in chronic obstructive lung disease took into consideration the pathophysiology involved. In chronic bronchitis, there is believed to be increased mucus secretion with decreased ventilatory capacity and, as a result, a decrease in diffusion. This increase in mucus secretion alteration could also result in a decrease in compliance. Osteopathic manipulative therapy was designed to (1) modify somatic factors that directly or by abnormal neural reflex activity caused or aggravated the pathophysiology of the disease and (2) to aid the body in reestablishing the normal physiologic balance that has been caused to veer beyond optimal homeostatic limits.[10]

The neuromusculoskeletal examination was assigned to two physicians for consistency. Neither examiner knew if the subject was A or B.

RESULTS

The first figure shows a compilation on the ordinate of the neuromusculoskeletal findings in the various spinal areas which are represented by the abscissa. The findings on the ordinate were the mean of those variations from parameters recorded at each spinal level. In figure 1, 44 cases, showing findings of chronic obstructive lung disease, demonstrated increased neuromusculoskeletal findings in the dorsal area. This is significant when compared with subjects without chronic obstructive lung disease in which no comparable changes in the dorsal area were noted (figure 2). In figure 3, 23 patients with chronic obstructive lung disease, type A subjects, to be treated with standard modalities as well as osteopathic manipulation reveal their musculoskeletal findings to be similar to that of the 44 cases shown in figure 1. Figure 4 represents 21 cases of the B patients or controls with obstructive lung disease who would receive just the standard treatment. Figure 5 shows the curve for subjects and controls compared with the entire group of 44 patients with chronic obstructive lung disease. This demonstrates that the pattern of neuromusculoskeletal activity is comparable. Figure 6 shows the pairing of subjects and controls with the neuromusculoskeletal changes again most evident in the dorsal area. Figure 7 reveals the findings of control patients with the original examination, and figure 8, the final examination, showing an improvement in

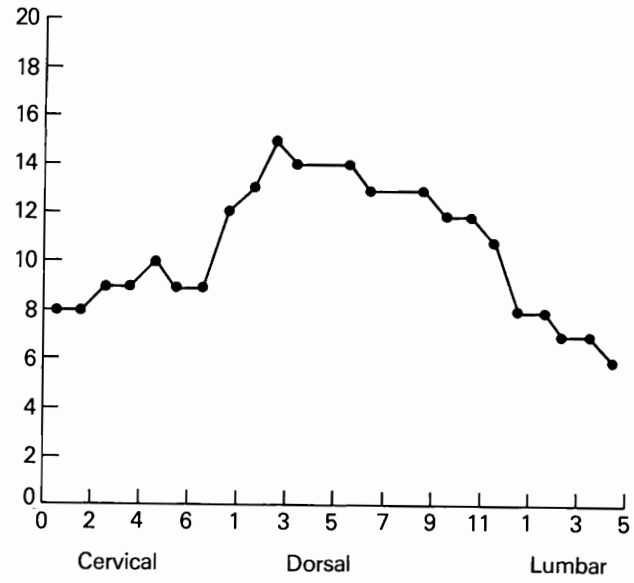


Fig. 1. Subjects and controls with chronic obstructive pulmonary disease (COPD): 44 cases.

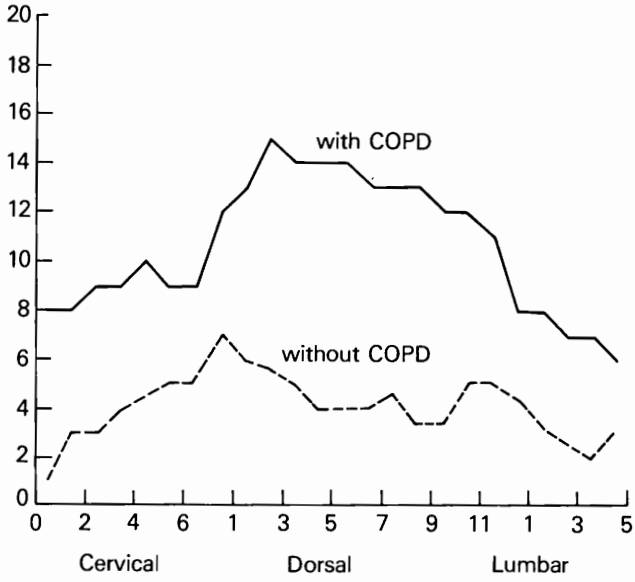


Fig. 2. Comparison of subjects with and without COPD.

597-036 O - 76 - 20

the neuromusculoskeletal findings. This was repeated on several occasions.

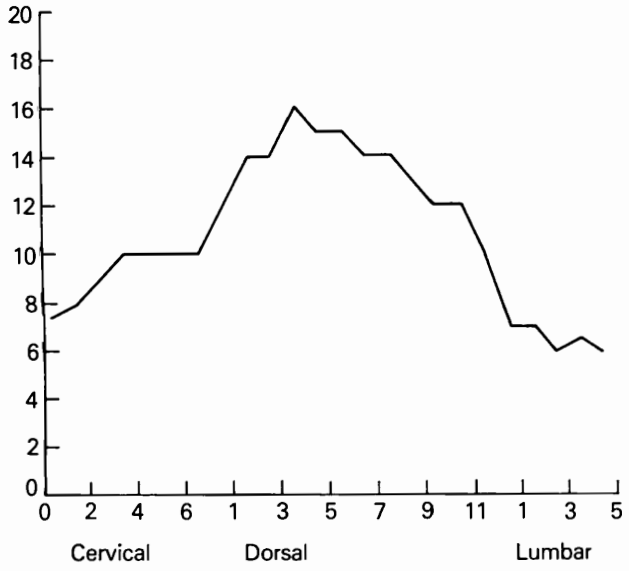


Fig. 3. Twenty-three subjects with COPD: Type A; treated with standard modalities plus osteopathic manipulation.

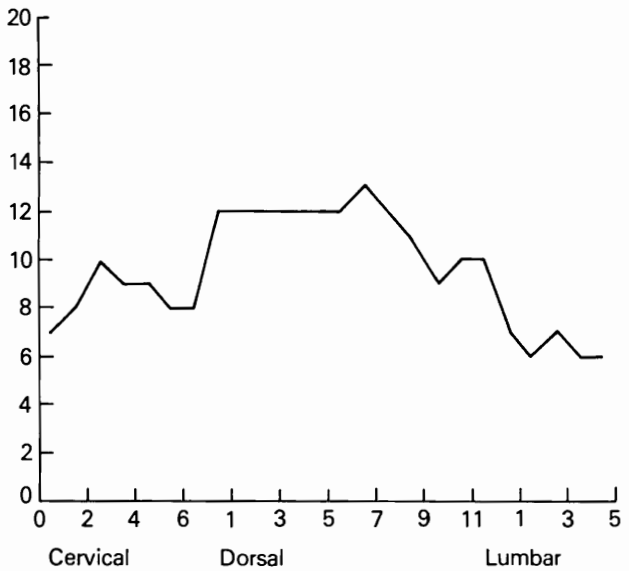


Fig. 4. Controls with COPD: 21 cases. Type B; standard treatment.

The pulmonary function studies revealed the following: (table 1) Blood gas tensions both in the treated and control group remained within relatively the same value. The mean vital capacity was almost identical at the initial examination; the treated group, however, showed an increase in 0.5 liters while the control group increased by 0.1 liter. This was not considered statistically significant ($p > 0.05$). The vital capacity also when expressed as a percent of normal showed similar results. The residual volume increased in the treated group but not in the control group ($p > 0.05$).

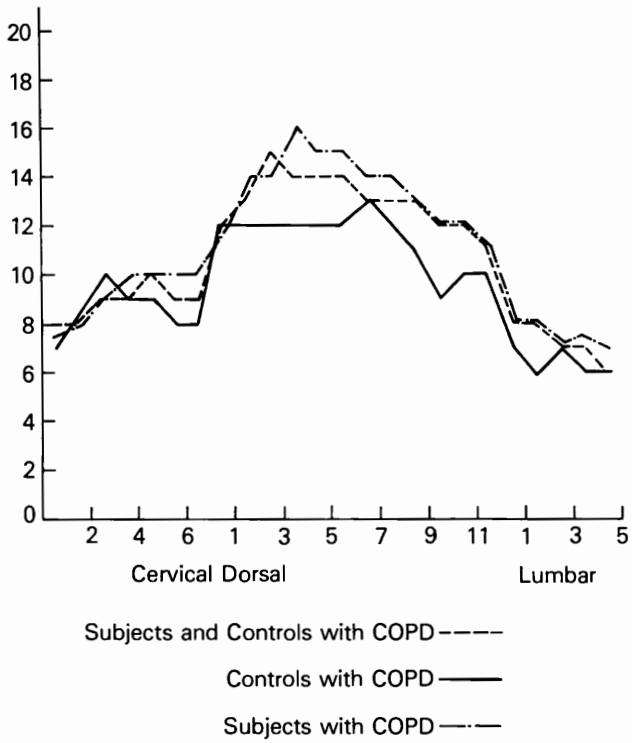


Fig. 5. Subjects and controls with COPD compared with entire group.

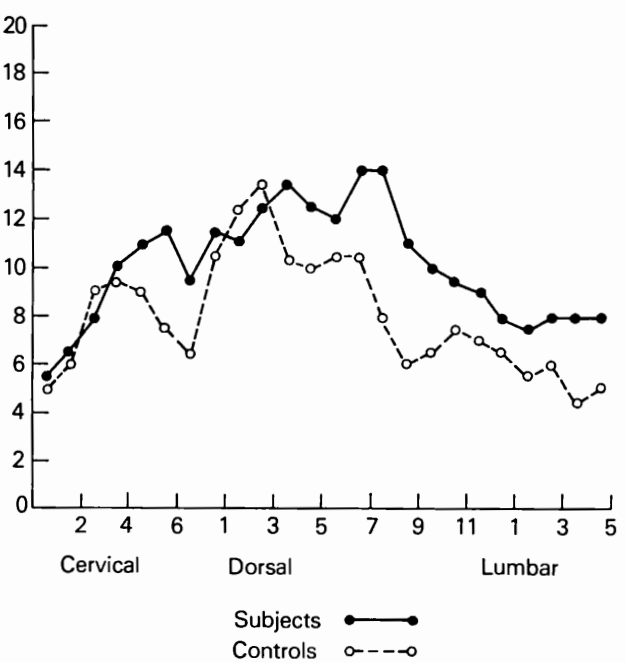


Fig. 6. Pairing of subjects and controls.

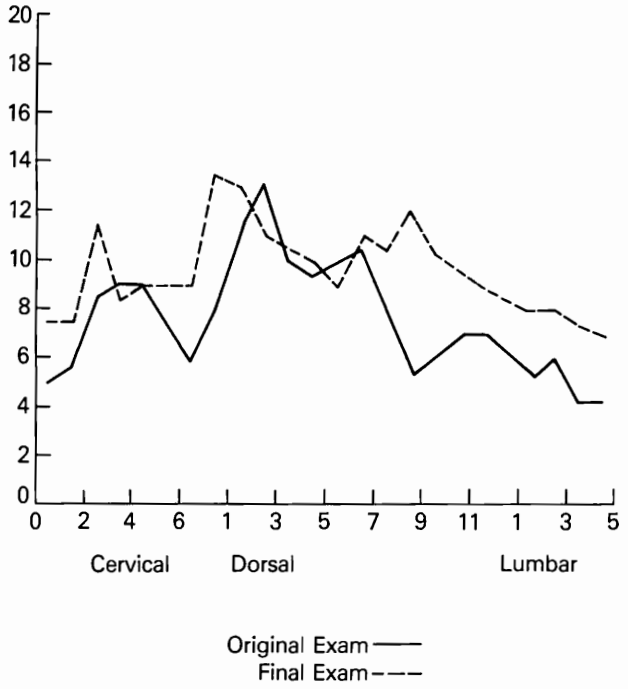


Fig. 7. Controls.

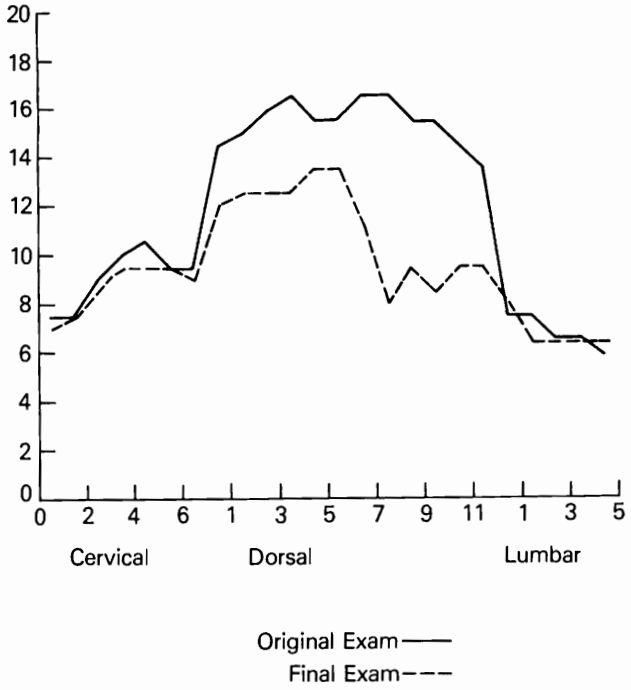


Fig. 8. Comparison of original and final examinations.

The mean total lung capacity increased in both the treated and control groups. (tables 2 and 3.) The increase in the treated group was larger than the increase in the control group. This same applied to the forced expiratory volume both to the control and treated group. (table 4.)

However, there was a tendency for the treated subjects to show a greater improvement in their lung function than the control subjects. It was in the area of subjective improvement that the most marked changes occurred in the treated group. Ninety-two percent of patients in the treated group, when responding to the questionnaire, stated that they were able to walk greater distances, had fewer colds, upper respiratory infections, and less dyspnea then prior to treatment. They were able to function in their normal capacities far better than they had in the past. The cough, in addition, was far less in all aspects.

CONCLUSIONS

Most studies of chronic obstructive pulmonary disease have emphasized significant chronicity and poor response to treatment.[10,11] Yearly changes have been relatively small. Data would suggest that chronic obstructive pulmonary disease is one that begins many years prior to clinical symptoms.[12] Because of its nature this makes early diagnosis rather difficult. The data herein presented is not complete by any means. Despite the relatively small number of individuals studied, the trend noted would indicate a relationship between the neuromusculoskeletal findings of patients with chronic obstructive lung disease compared to a normal population without this condition. The studies of lung capacity suggest that specific osteopathic therapy might increase various lung capacities. Subjectively, a marked change with fewer deviations in the neuromusculoskeletal findings were revealed. This would suggest that there exists a relationship between the neuromusculoskeletal findings and visceral disease involving the lung.

The patients who received osteopathic manipulation in 92 percent of instances, claimed subjective improvement in physical work capacity. This was so consistently observed by the patients that careful observers could not blithely discount its importance. As a consequence, we have been looking for a reconciliation of the physical findings and the patients' increased physical capacity. Perhaps the explanation rests in the area of improvement of patients' oxygen uptake and other similar measurements of physical work capacity.

TABLE 1

Mean Arterial Blood Gases and pH on Initial Exam and After Treatment, for Treated and Control Subjects

Mean arterial blood gases and pH	Initial exam		After treatment	
	Treated (N=13)	Control (N=10)	Treated (N=13)	Control (N=6)
pH	7.4 ± 1.7	7.4 ± 2.4	7.4 ± 2.8	7.4 ± 0.8
PO_2(mm Hg)	86.6 ± 1.6	85.8 ± 1.1	83.0 ± 3.5	82.5 ± 3.2
PCO_2(mm Hg)	39.4 ± 1.5	40.0 ± 1.1	34.4 ± 3.0	36.7 ± 1.1

± Standard error.

TABLE 2

Mean Lung Volume Measurements on Initial Exam and After Treatment, for Treated and Control Subjects

Mean lung volume measurements	Initial exam		After treatment	
	Treated (N=13)	Control (N=10)	Treated (N=13)	Control (N=10)
VC:				
Measured	2.3 ± 0.2	2.4 ± 0.2	2.8 ± 0.2	2.5 ± 0.2
Percent of normal	68 ± 6	67 ± 5	77 ± 6	68 ± 5
Functional residual: capacity				
Measured	2.5 ± 0.3	2.7 ± 0.3	3.2 ± 0.3	2.7 ± 0.3
Percent of normal	80 ± 7	83 ± 7	100 ± 7	85 ± 8
Residual volume:				
Measured	1.9 ± 0.2	2.0 ± 0.2	2.4 ± 0.2	2.0 ± 0.3
Percent of normal	100 ± 8	106 ± 10	129 ± 9	106 ± 11
Total lung capactity:				
Measured	4.1 ± 0.4	4.4 ± 0.4	5.1 ± 0.3	4.5 ± 0.4
Percent of normal	77 ± 5	80 ± 5	94 ± 5	82 ± 5
RV/TLC	.43 ± .03	.43 ± .03	.47 ± .03	.39 ± .05

TABLE 3

Mean of Carbon Monoxide Diffusion Studies and Other Respiratory Function Studies, on Initial Exam and After Treatment, for Treated and Control Subjects

Means of carbon monoxide diffusion studies and MVV	Initial Exam		After Treatment	
	Treated (N=13)	Control (N=10)	Treated (N=13)	Control (N=10)
D_LCO_{SS} – measured	10.26 ± 1.04	11.39 ± 1.87	11.7 + 1.30	11.58 ± 1.90
Percent of normal	72 ± 6	72 ± 7	84 ± 10	71 ± 10
Tidal volume – measured	4.95 ± 0.39	4.32 ± 0.47	5.93 ± 0.43	5.54 ± 0.57
Minute ventilation – measured	8.108 ± 4.95	6.283 ± 7.59	8.745 ± 4.86	9.410 ± 1.221
MVV – measured	51.3 ± 6.0	75.3 ± 14.7	65.7 ± 5.5	75.9 ± 11.4
Percent of normal	60 ± 7	75 ± 9	77 ± 7	68 ± 10

TABLE 4

Mean of Forced Expiratory Volumes and Forced Expiratory Flow Rate on Initial Exam and After Treatment, for Treated and Control Subjects

Flow Studies	Initial Exam		After Treatment	
	Treated	Control	Treated	Control
	($N=13$)	($N=10$)	($N=13$)	($N=10$)
$FEV_{1.0}$	72.4 ± 3.5	77.6 ± 3.3	74.5 ± 2.9	75.2 ± 3.0
$FEV_{2.0}$	88.9 ± 1.4	90.0 ± 3.1	92.3 ± 1.6	91.6 ± 1.8
FEFR	1.5 ± 0.2	1.8 ± 0.2	1.6 ± 0.2	1.8 ± 0.3

REFERENCES

1. MacConail, M. *Muscles and Movements.* Williams & Wilkins, Baltimore, 1969.
2. Campbell, E. *Respiratory Muscles.* Second Ed., W. B. Saunders Co., Philadelphia, 1970.
3. Hoag, J. M. *Osteopathic Medicine.* McGraw-Hill, New York, 1969.
4. Guyton, A. C. *Textbook of Medical Physiology.* W. B. Saunders Co., Philadelphia, 1971.
5. Friedenberg. *Disease of the Heart.* Third Edition, W. B. Saunders Co., Philadelphia, 1968.
6. Hurst, J. W., Ed. *The Heart.* McGraw-Hill, New York, 1970.
7. Gaensler, E. A., and Wright, G. W. Evaluation of respiratory impairment. *Arch. Environ. Health* 12:173, February 1, 1966.
8. Hall, J. Flow of lymph. *New Eng. J. Med.*, 281:720–722, 1969.
9. Allen, T. Use of thoracic pump in treatment of lower respiratory tract disease. *J.A.O.A.*, 67:408–411, December 1967.
10. Allen, T. Management of acute exacerbation of COPD. *J.A.O.A.*, 71:330–333, December 1971.
11. Emirgil, C. Study of the long-term effect of therapy in chronic obstructive pulmonary disease. *Amer. J. Med.*, 47:3–367–377, 1969.
12. Barrow, B. Course and prognosis of COPD. *New Eng. J. Med.*, 280:397–404, 1969.

Grateful acknowledgment is given to the contributions of the faculty of Postgraduate Institute of Osteopathic Medicine and Surgery: Philip E. Fleisher, D.O.; Solomon Gerber, D.O.; Mary E. Hitchcock, D.O.; J. Marshall Hoag, D.O.; William O. Kingsbury, D.O.; Robert G. Thorpe, D.O.; William B. Strong, D.O.

Design of Clinical Trials

JEROME CORNFIELD
Chairman and Professor
Department of Statistics
The George Washington University

It is appropriate that a consideration of the design of clinical trials should come at the end of this informative and interesting set of meetings. This order of discussion recapitulates the course of much modern medical investigation, in which existing concepts and knowledge do not permit the theoretical determination of the effectiveness of suggested therapies. They must, therefore, be subjected to the ultimate pragmatic test—do they work? We have had a recent dramatic demonstration of this process in the treatment of diabetes.[1] Although the condition is characterized by an impaired glucose tolerance, correction of the impairment by lowering of blood glucose does not appear to inhibit the development of the vascular sequelae of diabetes in a large class of patients and this could have been discovered only by trying it and seeing what happened.

It is not as easy as it might seem to answer the question of whether a therapy works. The controlled clinical trial, according to no less a person than the President of Royal College of Surgeons "was as important and valuable as the discovery of penicillin" in its effort to provide a systematic and widely accepted way of evaluating therapy. Its basic value is that it shifts attention from abstract theorizing and the enumeration of special cases to resolvable issues, such as exactly what therapy is under investigation, exactly what is it supposed to do to exactly what class of patients, and exactly what observations are needed to see if it does. I wish I could go on to say that once this is done it is possible to mount a crucial experiment which provides definitive and universally accepted answers. This may sometimes happen, but, as the old story goes, it has never happened to me. The more usual situation is that a number of independent investigations are undertaken; the results are not always in agreement; subclasses of patients are delineated, in some of whom there is reason to think the therapy may work; the literature grows; reviews of articles summarizing the strengths and weaknesses of the various trials get written; and increasing numbers of investigators come to realize that the expectation of quick and easy answers was naive and that the issues are more complex than was originally thought. Lest this picture be thought overdrawn, I point to the present status of anticoagulant therapy in acute myocardial infarction, to cholesterol-lowering therapies, whether by drugs or by diet, in postcoronary patients, and to the oral hypoglycemic agents in diabetes. Furthermore, clinical trials are not exactly most people's idea of fun since they demand unremitting attention to detail, and, in collaborative trials, working with other investigators whose views must be accorded respectful attention, no matter how doubtful they may seem. So, on the assumption that there are pressing questions about manipulative therapy for which answers simply must be found, I shall try to sketch some of the design issues that will arise. But be forewarned that they won't be as easy to implement as might be thought.

The first issue is how to allocate patients among the therapies to be compared. Control and experimental patients must be alike in all relevant factors if differences in the outcome are to be attributed to the therapy under study. But the only safe way now known to achieve this comparability is by random allocation. The fundamental difference between the clinical trial and its second cousin, the epidemiologic investigation, is that in one the patients are randomly assigned to therapy

and in the other they assign themselves, with effects on comparability that cannot always be readily determined. Numerous examples could be given of the noncomparability of patient groups formed by methods other than random allocation, so that the burden of proving comparability of groups not formed at random rests on the investigators. As I understood Dr. Gitelman's interesting summary of published evaluations of manipulation, none of them involved randomization. Dr. Miller does attempt such an allocation and for this he deserves much credit.

Even with such an allocation it has become necessary to convince critics that the randomized groups are in fact comparable with regard to a range of characteristics relevant to the outcome variables being investigated. Dr. Miller has presented us with a comparison, particularly in figure 5 and in his tables, of what I take to be baseline characteristics of the two groups. The ordinates of the graphs are stated to be "neuromuscular findings." I do not know what this parameter is and consequently am unable to comment further on the graph. The blood gas and pulmonary volume and function measurements provide additional information on comparability; but since the number of patients for whom these measurements are available is 23 rather than the 44 originally randomized, these results are difficult to interpret.

Most experts in chronic obstructive lung disease would regard equality in baseline smoking status as mandatory to establish the comparability of the two groups of patients. Since this information is also lacking, I must conclude that the comparability of the two groups still remains to be demonstrated.

Alternation of patients is not the same as random assignment, even when the first patient in each pair is assigned at random. Such an alternation permits anyone to predict the treatment assignment of half the patients before they are actually assigned. Such predictability would have harmful consequences, as in the initial study of anticoagulant therapy in acute myocardial infarction by Wright and his associates,[2] in which patients were allocated to therapy and control groups on alternate days. This was known to admitting physicians and their reaction to this knowledge, as documented in the final Wright report, was to withhold the admission of patients with more severe infarcts until after midnight of the control day, so that they could receive what was regarded as the superior therapy—the anticoagulant. At least a partial explanation of subsequent nonconfirmation of their results is the noncomparability induced by the alternation.

Excessive dropout of patients after randomization can seriously weaken a study, and has, up to now, made it impossible to evaluate the effect of exercise in postcoronary patients. If the drop in Dr. Miller's tables from 44 to 23 patients is due to dropouts, he is in the same boat.

Subjective outcomes, and this could include performance on pulmonary function tests, present a serious difficulty. Many of the best things in life are, of course, subjective and therapeutic evaluations cannot avoid dealing with them. The problem is not in the subjective response per se but in its interpretation. Even in a randomized clinical trial, subjective improvement in the treated group can be interpreted either as the effect of therapy as compared with the control or as autosuggestion. There are many examples that emphasize that the hypothesis of autosuggestion is not a fanciful one. An early operation for the relief of anginal symptoms, the mammary artery implant, was widely considered as successful until it was shown that a sham operation, consisting of a superficial incision on the chest wall, which was then sewed up, was equally successful.[3] Placebo effects in drug trials are almost beyond enumeration. I need mention only a recently reviewed trial in which the nausea and vomiting of pregnancy was reported by participating patients to have been spectacularly relieved by the placebo.

In drug trials the standard procedure for eliminating both patient and physician bias in evaluation of outcome is blinding; neither the patient nor the physician knows the therapy to which the patient has been assigned. In the nausea and vomiting of pregnancy example, an even greater relief was reported by patients on the therapy so that, while a considerable degree of autosuggestion was present, the therapy had an effect over and above that of autosuggestion.

How could blinding be achieved in the evaluation of spinal manipulation? The answer must eventually be provided by those most familiar with the procedure, but the analogy of the sham operation is suggestive. Patients in the control group would be subjected to a manipulation which could not be distinguished from the standard manipulative

therapy, but which would, in the opinion of the osteopathic and chiropractic community, be ineffective. A similar procedure has been proposed for the evaluation of acupuncture, the insertion of the needles in positions which acupuncture specialists would consider without effect. The therapist administering both the sham and the approved manipulation, would, of course, not disclose which was which to the patient; and the subsequent evaluation of the subjective outcomes would be done by someone who did not know the treatment assignment of the patient. Whether such an approach is feasible must be decided by those more expert than I. But if it is not, it will not be possible to interpret the subjective response that is associated with spinal manipulation as caused by it rather than by the autosuggestion it induces.

What about the ethics of a sham manipulation? It is, of course, necessary that patients be informed in advance that they might be assigned to either group and that different forms of manipulation would be involved. Experience in a wide variety of circumstances, including an ongoing study in which patients with unstable angina, the so-called impending infarct syndrome, are randomly assigned to either coronary bypass surgery or to medical therapy, indicates that most patients will agree to participation in such trials. But how can a therapist who is convinced of the efficacy of his therapy ethically justify withholding it from some patients? The use of a randomized control group, of course, already commits him to such a course and a sham manipulation, with the patient's prior consent to a random allocation introduces no new ethical issues. In some recent trials the patients assigned to the control groups have turned out to be the fortunate ones, since the controlled trial disclosed that the therapy had unanticipated and undesired side effects. It can be argued at least as cogently that subjecting patients to a course of therapy about whose effects nothing definitive is known constitutes the really unethical procedure and that ethics require a controlled evaluation.

There is another general issue involved in appraising the specificity of whatever effect may be found which is related to the question of blinding, but raises additional issues, and that is the question of changes in other variables during the course of the investigation. In studies of diet and heart disease, for example, related variables, such as smoking, obesity and blood pressure may change simultaneously. Blinding can control this effect by inducing similar changes in concomitant variables in treated and control groups, but this must be demonstrated by monitoring the concomitant variables. In the National Diet-Heart Study this was done and it was, therefore, possible to demonstrate quite similar changes.[4] Whether this is also true for the study presented by Dr. Miller cannot be decided on the basis of the evidence presented to us. Did the treated patients, for example, reduce the amount of their smoking more than the controls?

I remark parenthetically, less as a criticism and more in the hope of influencing future studies, that it is customary in controlled clinical trials to present much more detailed information on the study variables than we have seen, or indeed than it was possible to present, today. There are many questions that could be raised in addition to issues already discussed. Is the statement that "there was a tendency for the treated subjects to show a greater improvement in their lung function than the control subjects" a clinical impression of the overall results, or is it the result of a statistical analysis; and if so, what was the analysis and what did it show? Ninety-two percent of the patients in the treated groups reported symptomatic relief, but, entirely aside from questions of specificity and interpretation of the difference, what did the controls report? Similarly, was it really 92 percent for each of the subjective improvements enumerated, as the manuscript seems to state, or did it vary, as one might expect, from improvement to improvement? How long was the period of treatment and how long was the followup? How many patients dropped out? Exactly what do the four specific manipulative techniques used consist of? Were they all used on each patient? One could go on, but I think it perhaps sufficient to suggest that those interested in the clinical evaluation of manipulative therapy, could profitably review the reporting practices in ongoing controlled trials, such as that reported in this week's J.A.M.A. by the Coronary Drug Project. In the absence of such detailed reporting, conclusions must be taken on faith, and this is entirely contrary to the spirit and practice of the modern controlled clinical trial.

I turn now to a set of issues that can be conveniently grouped about the question of "How many patients?" It has been by no means unusual for therapeutic trials to find, at the end of a long and difficult investigation, that the sample size used was too small to supply clear-cut answers to the questions asked. It is advantageous before em-

barking on such an investigation to consider in detail one's objectives and their implications for number of patients. There is a well-worked-out body of theory for how one proceeds in this matter, and while it is always fun for a statistician to expound this theory to a medical audience, I do not think we are quite at the point at which such an exposition is required. The issues at this point appear to me to be more elementary – what are the hypotheses that are to be tested? What specific outcomes in what patient populations is spinal manipulative therapy designed to influence and what are the magnitudes of the effects to be expected? Although the required numbers of patients cannot be estimated until answers to these questions are forthcoming, the answers are required for more than statistical reasons; they are a prerequisite to achieving a scientific and generally accepted evaluation of therapy.

It might be thought sufficient to adopt a shotgun approach, i.e. to collect a wide variety of data, in a wide variety of patient types, without prespecifying a hypothesis or the patient subgroups to whom it applies and to search the results for significant effects. There are dangers in such a course that are best summarized by saying that even if there are no effects present one expects to find significance at the $P=.01$ level in 1 percent of the end points and subgroups examined. One need only examine enough end points and subgroups and a "significant" result is virtually guaranteed, even when none exists.

The difficulties experienced by the Coronary Drug Project in pinpointing on a *post hoc* basis the subgroups in whom excess mortality was occurring, even with over a thousand patients on dextrothyroxine therapy and over two thousand on control, is sobering.[5] If the most rigorous form of controlled clinical observation arrives at unequivocal conclusions only with difficulty, it is not surprising that uncontrolled unverified clinical impressions can lead to nothing but continued controversy.

REFERENCES

1. The University Group Diabetes Program: A study of the effects of hypoglycemic agents on vascular complications in patients with adult-onset diabetes. *Diabetes*, 19 (supplement 2): 747–830, 1970.
2. Wright, I.. S., Marple, C. D., Beck, D. F., Report of committee for evaluation of anticoagulants in treatment of coronary thrombosis with myocardial infarction (progress report on statistical analysis of first 800 cases studied by the committee). *American Heart Journal*, 36:801–815, 1948.
3. Spodick, D. H., Revascularization of the heart-numerators in search of denominators. *American Heart Journal*, 81:149, 1971.
4. National Diet-Heart Study Final Report. *Circulation*, 37 (supplement 1): 279–309, 1968.
5. Coronary Drug Research Group: Findings relating to further modification of its protocol with respect to dextrothyroxine. *J.A.M.A.*, vol. 220, 996–1008, May 15, 1972.

Chairman's Summary: Comments on Therapeutic Studies

ANDREW B. WYMORE
Osawatomie, Kansas

The general discussion on therapeutic studies was initiated by the question: I have not heard anything about complications associated with spinal manipulative therapy. Would some of the speakers be willing to briefly describe their complications? The response included:

- The most severe complications reported have been associated with upper cervical manipulation. Fairly simple clinical measures can be used to determine contraindications to the manipulative procedures.
- In 10 years and more than 50,000 manipulations, no complications have been reported in my clinic. The absence of statistics in the treatment of low back pain by manipulation does not prove that this form of treatment is poor. At one time statistics indicated that fusion was a good form of treatment for low back pain, and this is no longer the case. Sixty percent of low back pain did not originate from the low back, but from T 10, T 11, T 12. There is a method for determining which segment was involved.
- While some low back pain may be self-limiting, if means exist of bringing ready relief they should be used.
- Statistics and not anecdotal observation advance science. In the medical field—in the medicoscientific field—the truth takes a long time to carry through. Studies from Mayo Clinic, Philadelphia (from Rothman), and Seattle, Washington have demonstrated that lumbar fusion is not the operation for low back pain and that it is being abandoned in the United States. In Sweden it was given up some 10 years ago.
- Complications from manipulation have been observed; I've certainly had three instances in my practice. Two were self-limiting within a week. There do exist contraindications whereby manipulation should be avoided.
- It is justified to ask for statistics, and the graph by Glover shown by Dr. Nachemson is statistically favorable to manipulative therapy. The study indicated that manipulations were much more successful than the control therapy during the first 3 days, while at the end of 7 days results were comparable. Is it not advisable to attain the results faster than to wait for 7 days? Medical science and surgery have advanced without always applying double blind studies, and manipulative therapy which is not a standardized therapy, but is in the process of evolution, would be a difficult place to apply a double blind study. In the same light, how about applying double blind studies to surgery?

There was a detailed discussion of the problems associated with controlled clinical trials. Comments made during the discussion included:

- Double blind trials for either surgery or manipulation were not being advocated, rather single blind trials.
- It is possible, though difficult and tedious, to design valid clinical trials of surgical intervention. Lack of prospec-

tive randomized controlled trials is not the sole property of any of the disciplines represented here. While we have heard much about drug treatment of hypertension, in the majority of hypertensives we do not have convincing trial evidence of efficacy or safety. We need much research in medical, surgical, osteopathic, and chiropractic areas directed toward therapeutic evaluation. We need to know in the interest of patients being treated every day just what the safest, most efficacious treatment may be. There is need for standardization of diagnostic measurement. We need to study reliability and validity, sensitivity and specificity in the measurement process, since these clinical measurements do relate to the basic treatment given. We need more data in every specialty and better designed, more scientific, more believable results.

Clinical Research Areas Requiring Further Study

FLETCHER H. McDOWELL
Professor of Neurology
Cornell University College of Medicine

It is an unusual opportunity to have nearly the last word at an important conference. There are a few things I would like to mention about research that seem to me to be important.

First of all, I think it is desirable to organize the discussion of needed investigation into three categories:

1. The evaluation of chiropractic therapy;
2. Chiropractic dianostic technique; and
3. The basic science underpinning of chiropractic concepts.

If these are mixed, we will likely take sides on issues which will interfere with a sensible dialogue.

First, I would like to mention what seem to me to be excellent opportunities for firming up basic science correlation with chiropractic concepts. The correlation of basic science data and clinical observation is often difficult and usually relies on animal experimentation. You have heard today of some rather elegant experimental techniques which could be used to investigate chiropractic theory. The studies of Dr. Sharpless and Dr. Sato could be combined to examine the effects of afferent barrage caused by nerve root compression on the central excitatory state, changes in reflex activity and the effects on autonomic function. Using Dr. Sharpless' technique of nerve root compression, one could sample neural activity in the spinal cord and in higher centers in response to varying degrees of pressure on nerve roots. With this technique, Dr. Sato has the opportunity to study somatic visceral reflexes and to see whether chronic irritation of spinal nerve roots in the experimental animal changes visceral function.

It is certainly not a new observation that severe or noxious stimuli from the periphery alter visceral function or that major alterations in visceral function alter somatic function. What is needed is data that chronic less severe peripheral stimulation can change visceral activity.

At the bedside it is difficult to think of basic science studies which could be carried out to support chiropractic theory. It is possible that the use of cortically evoked responses from peripheral stimulation might be useful in the study of nerve root compression. Altered cortical responses from stimuli in the neural segment of the compressed root might be used to investigate the role of compression in altered neural function.

A major area of basic science–clinical investigation that should be undertaken is more thorough clinical pathological correlation. I did not know of the report that Dr. Nachemson mentioned about the study of changes in vertebral joints, but it seems to be appropriate for neuropathologists and pathologists to look at the vertebral column for telltale evidence of fibrosis, joint thickening, nerve compression and other changes that might suggest a connection between a pathological process and a history of trouble with back pain or other disturbances in the past. It will take a long time to get the information but it is a worthy task.

In the area of diagnostic techniques at the clinical level, careful scrutiny of the accuracy and reliability of chiropractic diagnostic methods is needed. As Dr. Remington mentioned, the accuracy of these methods affects every clinical study. It is clear to any of you who have studied the reliability of clinical diagnostic techniques,

that there is enormous variability between individual observers.

Chiropractic physical diagnostic techniques at the bedside need to be carefully correlated with what is seen on X-rays, and what is found with techniques like thermography, and so forth, on a blinded basis. For instance, one could review 100 back X-rays of patients who come to chiropractors, list the abnormalities found without knowing the patient's history, and then see how good the correlation is with the symptoms and how accurately they have been localized. These studies are often tedious and long, but need to be well designed. They need to be done and if done, will enhance the ability to evaluate therapy correctly.

I suspect that I wouldn't get too much argument from most of you if I stated that in the overall totality of human illness, about two-thirds of it is self-resolving. Regardless of what kind of outside intervention we choose to use, the patient often will get well by himself.

If it were more than that figure, that is, if it were over 80 percent, I suspect there'd be no need for our skills. If it were less than 50 percent self-resolving, we'd either all be out of work or in jail. This demands that anyone who suggests a therapy for a particular condition must demonstrate to the satisfaction of the best possible scientific scrutiny that he can better the natural history of illness.

For those of you who have sat here today and heard the controversy about the use of manipulative therapy, I would suggest that this is a polite, quiet session compared to some where neurologists deal with neurosurgeons about the treatment of stroke or cerebral aneurysm. Often needed studies have been impeded by the fact that individuals choose sides about therapy and refuse to recognize the fact that what must be done is to demonstrate that the natural history of an illness or complaint can be improved.

There are two ways to do this. One is by controlled trials which have as their main desirability that they offer short and accurate routes to desired data.

The other way is to gather anecdotal evidence about the efficacy of therapy through careful clinical observation of patients. This has been the standard means of sorting problems out, but it sometimes takes a decade or longer to do it and sometimes it takes centuries. It is hard to be sure how long it took to determine the usefulness of skull trepanization as an effective therapy. We know it was first practiced by the Egyptians; by the 20th century, we found that there are a few rather specific conditions which are treated by this therapy and that most others are made worse, or not helped by this treatment.

We must evaluate therapy as rapidly as possible, because therapies are being suggested with increasing frequency and the public is anxious for effective relief from its complaints and its diseases. All of us are concerned with relieving human suffering, one way or another.

I'm not bothered by a kind of therapy as long as it can be shown to be effective and is safe. As for manipulative therapy, my experience with it certainly is limited at best. It may be effective. I have the impression that there are obviously going to be some conditions for which it is effective. It is mandatory that we take the steps needed to prove this. First of all, we must focus on the elements of chiropractic therapy, if we are going to talk about effective treatment.

Second, we must sharpen diagnostic techniques and be certain that we are talking about the same thing when we make a particular diagnosis, and know exactly what is being studied.

Perhaps the most important problem related to manipulative therapy is that of quantifying treatment. That is, how often is therapy administered, how vigorously, for how long, and at what stage of the disease.

I need not tell *you* who have dealt with patients that the interaction between a sick individual and his physician is complex. All kinds of things are going on, probably the most important being that the patient is relieved of the responsibility of his illness when he hands it to his physician. That simple act may be the important factor, and much of other therapy may have little to do with the relief of symptoms.

One has to be sure that a therapy is effective at better than chance level. This objective is what the statisticians have helped modern evaluators of therapies and drugs to do.

I think that the time is ripe for carefully controlled clinical therapeutic trials of manipulative therapy for a variety of conditions. I also believe that this should be a cooperative venture between physicians and chiropractors.

As someone pointed out, if we do it separately we may arrive at the same conclusion, but it will take a long time. If we do it together, we may get there more quickly.

U.S. GOVERNMENT PRINTING OFFICE : 1976 O—597-036