APPLIED KINESIOLOGY

SYNOPSIS
2ND EDITION

DAVID S. WALTHER

APPLIED KINESIOLOGY

Synopsis
2nd Edition

APPLIED KINESIOLOGY

SYNOPSIS
2ND EDITION

David S. Walther, D.C.
Diplomate ICAK

Photographs:
Daniel R. Maxson

Illustrations:
David McKinley Gavin

SYSTEMS DC, 275 West Abriendo Avenue, Pueblo, Colorado 81004-1870

SYSTEMS DC
275 West Abriendo Avenue
Pueblo, Colorado 81004-1870
USA

Library of Congress Catalog Card Number: 98-61704

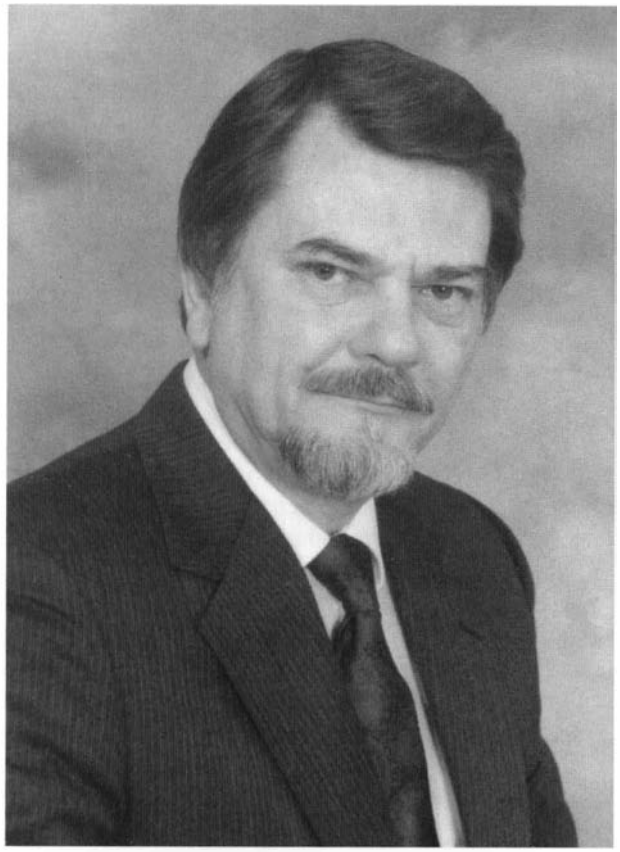
ISBN Number: 0-929721-03-9

Notice: The material in this text is a compendium of clinical and research information developed in the discipline of applied kinesiology (AK). There are references to the standard literature database that help put AK procedures into perspective. AK methods are directed to practitioners who are licensed to be primary health care providers and are to be used in conjunction with their standard methods of diagnosis. The author and publisher have taken care to make certain the information is compatible with information available in the field at the time of publishing. The physician using this material must take into account all of the patient's findings including, but not all-inclusive: clinical history, physical examination, laboratory tests, and other procedures when applicable to arrive at a final conclusion regarding the patient's condition and best approach to treatment. The author and publisher disclaim all responsibility for any liability, loss, injury, or damage incurred as a consequence, directly or indirectly, of the use and application of any of the contents of this volume.

Printed in the United States of America

Table of Contents

Preface



Preface to Second Edition

Applied kinesiology is an augment to standard diagnosis to be used by practitioners licensed to be primary health care providers. It is a system of evaluating how the body reacts to various forms of stimuli applied to the nervous system. It has been described by Schmitt as the evaluation of functional neurology, therefore a person must understand how the body normally reacts to stimulation to the nervous system to be able to put into perspective the results of manual muscle testing.

Applied kinesiology continues a rapid growth among practitioners of the healing arts in the United States and most foreign countries. The most recent Chapter in the International College of Applied Kinesiology is the Russia Chapter.

Unfortunately, "kinesiology" has also grown in use by lay persons who are failing to correlate the results of manual muscle testing with standard methods of diagnosis. Much harm can come from the application of "kinesiology" in this manner. In the first place most of the people I have observed are not using quality muscle testing. Just yesterday I had a patient tell me his experience of attending a meeting the previous night where "kinesiology" was demonstrated. The person held a packet of sugar over what was described as the solar plexus and the subject weakened to the arm-pull-down test. I said, "Let me show you some thing." I tested him with the arm-pull-down test, which demonstrated strength. Next, I did some wild gyrations in front of him with my hand and then retested by the arm-pull-down method and he tested weak. He asked, "What did you do, disrupt my aura?" I then did the same thing and he tested strong. I explained that I simply changed the parameters of the test by changing the test speed. I went on to explain I can make the test appear any way I want by changing different parameters. I told him that I believe the person demonstrating at the meeting he attended is sincere in what he is doing, but it lacks the thoroughness needed to come up with meaningful data and is discouraged. I discussed some of the problems with the arm-pull-down test and the parameters of muscle testing discussed in Chapter eight. There are many factors that must be considered in a complete analysis of a person's health.

The second edition of *Applied Kinesiololgy, Synopsis* contains all of the material of the original text with modifications to bring some of the material up-to-date. In addition there is considerable new material that has developed since the initial volume of this synopsis.

Preface to First Edition

When I began this text, I felt it would be quickly and easily written. Two factors have prevented that assumption from being true: 1) the subject of applied kinesiology is extremely broad, and 2) where does one stop?

Since its inception in 1964, many clinical techniques have been developed in applied kinesiology. The subject deals with the broad scope of body function, providing a superior ability to deal with the integration of function within the body.

The purpose of this text is to provide an overview and working knowledge of applied kinesiology procedures, as well as to present new material not previously written in textbook form. The goal is to provide the applied kinesiology initiate with a thorough and workable introduction to the subject; for the advanced applied kinesiologist, there is reference material and the presentation of some of the newer procedures.

Much of the text provides a true synopsis of applied kinesiology techniques, while in some areas a more in–depth look is presented. Chapter 4 on nutrition is an example of providing more information on how stimulation of the gustatory and olfactory nerve receptors may influence body function, as has been observed clinically in applied kinesiology. Chapter 7 on meridian therapy provides an easy method of understanding and working with this important factor of body function for those not familiar with it. I often think about how much doctors who are not knowledgeable about the meridian system are missing in body language regarding their patients' health problems. For those who have worked with traditional acupuncture, the meridian therapy sec-

tion adds an important dimension in diagnosing meridian imbalance. Acupuncture and the knowledge of the meridian system are thousands of years old; applied kinesiology is one of the very few systems that has added new information to the ancient knowledge.

Practitioners of applied kinesiology sometimes have difficulty diagnosing a condition for insurance purposes. The allopathic health care system is geared toward making a diagnosis upon which treatment is based. But what about the patient with headaches who passes all physical examination and laboratory tests with flying colors? Often he is classified as having a psychosomatic condition. With applied kinesiology examination he may be found to have neurologic disorganization, as discussed in Chapter 5, with many factors contributing to the disturbance. What is the diagnosis? "Headache" is simply a symptom, and the many factors contributing to the neurologic disorganization have not been classified in the International Classification of Diseases.

As in the preceding paragraph, throughout this text patient and physician are referred to most often as "he." This in no way implies disrespect for, or lack of recognition of, the many fine female doctors who are practicing in the healing arts or the women patients treated. The "male" pronoun is used simply as a generic term to allow ease and efficiency in reading, rather than consistently stumbling over the awkward "s/he," "him/her," "she/he" forms of writing.

Applied kinesiology has added the tools of functional evaluation to the health care system, providing the ability to understand functional conditions better than ever before. Because of these tools, primarily developed by George Goodheart, Jr., D.C., the way in which the broad spectrum of functional health problems is treated naturally has been greatly expanded. Using the new–found ability to evaluate function, many methods have been developed by applied kinesiologists to allow the body to return to normal function. A basic applied kinesiology principle is that the body is self–correcting and self–maintaining. The tools in this and other texts on applied kinesiology enable the physician to find the problem(s) impeding the body's normal return to health.

Chapters 11 and 12 on orthopedic and systemic conditions present an overview of applied kinesiology application to specific types of health problems. It is in these areas that I experienced great difficulty in writing a "synopsis" of applied kinesiology. Application of the material presented in the earlier chapters is as beneficial as the physician's ability to put it to practical use. The material in Chapters 11 and 12 is simply putting into application the methods of examination and treatment presented earlier in the text. These chapters give an overview of how applied kinesiology can treat many types of conditions by evaluating function with therapy localization, challenge, and muscle testing. They show how nutrition is important in some conditions but not in others. The application of treatment to the stomatognathic system and its influence on the cranial nerves, as well as the energy patterns of the meridian system and spinal adjusting, are important in many types of conditions. The key is, as Goodheart says, diagnose the need, supply the need, and observe the results.

The material in this text is a result of the contributions of many people and organizations. The innovative, inquiring mind of George Goodheart, Jr., is responsible for the initiation and continuing development of applied kinesiology. It is his original thinking upon which much of this text is based. Without his ideas, the many additional examination and treatment techniques developed by applied kinesiologists would not have been manifested. By applying these methods of examination, new techniques have been developed by applied kinesiologists, many of whom are members of the ICAK. I have learned much from attending the semi–annual meetings, sometimes forgetting from whom I learned a particular bit of information. My apologies to any person I have failed to reference for original ideas. The growth of applied kinesiology has truly been a team effort. My thanks to all who have contributed to the *Collected Papers of the Members of the International College of Applied Kinesiology*, and to the many research scientists cited in this text.

My continuing appreciation goes to my secretary, Carol Ann Hupp, who takes my rough dictation and works with the syntax, improving what you ultimately read.

Daniel R. Maxson has worked with me for many years as Seminar Coordinator and important right–hand person. He is responsible for the photography in this text.

The illustrations in this text, as well as many others used in teaching applied kinesiology, have been done by David M. Gavin. They have also been produced in color and are available as 35 mm slides from Systems DC. They are used by many who teach applied kinesiology.

Finally, but certainly not last, are my appreciation and love for my wife and best friend, Jeanne. Prior to my involvement in teaching and writing about applied kinesiology, she owned and managed her own printing business. Since then she has worked full–time to produce material for applied kinesiology. She is responsible for the typesetting and layout of this text, as well as many other applied kinesiology educational materials.

This text of applied kinesiology techniques can be considered a doctor's toolbox. Use the tools in a scientific manner, and in combination with the usual diagnostic tools available in the healing arts. Applied kinesiology is a valuable addition to the physician's ability to understand health problems, but it is not to be used as a "stand alone" method of diagnosis. In the proper context, the tools of applied kinesiology will provide you and your patients with greater success, enabling health to return to many.

David S. Walther, D.C.
Diplomate, ICAK

Introduction

Introduction to Second Edition

The opportunity to preface a second edition of *Applied Kinesiology – Synopsis* is a repeated pleasure. The introduction to the first edition of the text that follows naturally did not include many new techniques and applications included in this second edition. The reader will be well rewarded by the new material and will be reassured by the careful edition of all new technique material that David Walther does so well. Included are references to new peer-reviewed research that is an ongoing process on applied kinesiology applications. Now, on with the original introduction.

It is again my privilege and pleasure to write this introduction to yet another carefully edited text that my friend and colleague, David Walther, D.C., has produced. By mastering the techniques presented in this volume, the health professional can gain extra dimensions in working with health problems. Walter Schmitt, D.C., also a good friend, former associate, and fellow lecturer in AK, has said, "Muscle testing is functional neurology." Muscle testing provides an opportunity to observe the workings of the nervous system. With the methods presented in this text, the doctor has the ability to diagnose the need, supply the need, and observe the results. This is the standard of an ever–increasing number of health professionals who are interested in the application of the principles of applied kinesiology.

The structural orthopedic application of applied kinesiology is recognition of the interplay of "articular neurology," a term used by neurologist Barry Wycke, M.D. Muscle testing as functional neurology allows diagnosis of an unparalleled accuracy and refinement. Supplying the need established by the diagnosis thus derived allows articular neurology to operate unimpeded by structural, neurological, nutritional, or psychological faults. Observing the result by the same methods carefully applied allows recovery that the healer within can provide when faults are released by treatment without. One does not attempt to heal the body; one does attempt to allow or let the body heal itself. The new knowledge of psychoneuroimmunology conclusively demonstrates the wisdom of the body's physiological homeostasis (innate intelligence). Disease is normal physiology gone awry. To understand it, one must know the normal state of physiology, which may not necessarily be the average. The newer knowledge of the holographic conception of the nervous system, in addition to its connectionism, adds breadth and depth to our understanding of how the body heals itself.

Introduction to First Edition

Applied kinesiology had a simple beginning in 1964, based on the concept that muscle weakness is involved in most muscle spasms and, indeed, is primary. From those early observations, the system has broadened to encompass the many aspects of body function, as indicated by the three–sided triangularity of man; superimposed on it are the five factors of the IVF with biped man in the center, which is the logo of applied kinesiology. Lectures at leading dental schools and multidisciplinary symposia have exposed many dedicated health professionals to the holistic concept that unifies rather than divides the many disciplines. Lectures and demonstrations have been organized by interested members of all disciplines in many countries throughout the world. Norway, England, France, Switzerland, Italy, Germany, Australia, and Japan have ongoing teaching activity provided by many traveling AK diplomates. There are now chapters of the International College of Applied Kinesiology in the United States, Canada, Europe, and Australia. The ICAK is now truly an international organization. To better understand the scientific basis of applied kinesiology, the ICAK is funding research in many of the chiropractic colleges.

Applied kinesiology is based on the fact that body language never lies. Manual muscle testing as an indicator of body language enhances one's ability to observe function and its change. My introduction to the original method of testing muscles and determining their function was through the works of Kendall, Kendall, and

Wadsworth, which remains the primary diagnostic device in applied kinesiology. The testing methods have been refined by many in our group, especially my colleague Alan Beardall, D.C. Once a muscle weakness has been ascertained, a variety of therapeutic options is available, many of which are discussed in this text. The opportunity to use the body as an instrument of laboratory analysis is unparalleled in modern therapeutics, because the response of the body is unerring. With the proper approach, a correct diagnosis can be made, and the body's response is adequate and satisfactory to both the doctor and the patient. The name of the game, to quote a phrase, is to get people better. The body can heal itself in a sure, sensible, practical, reasonable, and observable manner. This is how the healer within can be approached from without. Man possesses an unerring potential for recovery through the innate intelligence or physiological homeostasis of the human structure. This recovery potential with which he is endowed merely waits for the hand, the heart, and the mind of the trained individual to bring it into manifestation, allowing health — which is man's natural heritage — to come forth. This benefit can be performed with knowledge, with physiological facts, and with predictable certainty. It should be done, it can be done, and this book offers a means and measure of how to do it. The references and knowledge contained in this manual, carefully researched and documented, make it a worthy successor to other volumes David has painstakingly written and published. My appreciation to Dr. Walther and his staff for the excellent job done in advancing these principles, and my best wishes are extended to all who read this text.

George J. Goodheart, Jr., D.C. F.I.C.C.
Diplomate, ICAK

Chapter 1

Introduction to Applied Kinesiology

Chapter 1

Introduction to Applied Kinesiology

Applied kinesiology (AK) is a system for evaluating body function that is unique in the healing arts. It has grown rapidly, both in the number of physicians using it and in its concepts and scope. The examination procedures, developed within the chiropractic profession, appear to be such that they can be used in all branches of the healing arts.

Applied kinesiology came into being in 1964 when George J. Goodheart, Jr., D.C., of Detroit, Michigan, began evaluating his patients' muscles with manual tests.[26,29] He observed that sometimes a muscle tested weak, but there was no atrophy or other apparent reason for the weakness. On one occasion he observed nodules at the origin of the patient's serratus anticus muscle; upon deep goading of the nodules, the muscle immediately returned to almost normal strength as compared with the uninvolved side. This led to the original applied kinesiology technique of origin and insertion treatment. Goodheart presented this technique at the charter meeting of the American Chiropractic Association held in Denver, Colorado, in 1964.

The technique of muscle testing Goodheart used was that of Kendall and Kendall.[59] This excellent work on muscle testing is now in its third edition by Kendall and McCreary.[58] Patient positioning and the general methods of testing remain very similar to those originally described by Kendall and Kendall. In applied kinesiology, the timing of the testing procedures has been changed and additional neurologic hypotheses have been developed.[89] Most muscle tests done in applied kinesiology do not evaluate the power a muscle can produce; rather, they evaluate how the nervous system controls muscle function. This has been called "muscle testing as functional neurology."[89] Most practitioners who used muscle testing in the early development of applied kinesiology held the concept of a "strong" or "weak" muscle. In most cases, the results of a test do not depend on whether the muscle is strong or weak, but how the nervous system controls the muscle. The changes in muscle function observed in applied kinesiology manual muscle testing are assumed to be associated with changes in the central integrative state of the anterior horn motoneurons. The central integrative state is defined as the summation of all excitatory inputs (EPSPs) and inhibitory inputs (IPSPs) at the neuron.[93] The terms "conditionally facilitated" and "conditionally inhibited" are more descriptive than strong and weak, respectively. Although the terms "strong" and "weak" have generally been maintained in keeping with their general use in clinical practice, one should think in terms of the nervous system rather than the actual power the muscle is capable of producing. Occasionally in this text the terms "conditionally facilitated" and "conditionally inhibited" are used interchangeably with "strong" and "weak."

The initial development of applied kinesiology was directed toward correcting structural imbalance caused by poorly functioning muscles. The main objective was to support chiropractic adjustments of the spine, pelvis, and other articulations. The improvement, when made, fit well into the structurally oriented chiropractic profession.

In the early development of applied kinesiology there were only a few techniques for changing muscle function. Sometimes the improved muscle function lasted, with no return of dysfunction; on other occasions, the improvement was short-lived. On still other occasions, a dysfunctioning muscle could not be returned to normal. When results were disappointing, Goodheart and others investigated reasons for improper muscle function and tried various therapeutic approaches. This writer developed numerous exercises designed to isolate a particular muscle that tested weak, and trained patients extensively to properly exercise the weak muscle. The results were disappointing; only rarely would the muscle return to normal function as observed by manual muscle testing. This inconsistency of improving function was disconcerting; it caused some physicians to discard the effort to identify individual muscle dysfunction and return it to normal.

Another early problem in applied kinesiology was the apparent inconsistency in manual muscle testing results. One might have identified a psoas muscle that tested weak and stopped to make an entry in his clinical notes, only to re-test the muscle and find it strong. This, too, caused some to discontinue manual muscle testing as an inadequate approach to examination.

Over the years the inconsistency of muscle testing has been largely overcome as the various parameters that change the results of a test have been discovered. For example, it is now known that when a patient places his hand over certain body parts there will be a change in muscle function. This has become known as therapy localization (TL). Another example is seen under some circumstances: when the eyes are turned into a certain direction, muscle function changes as observed by the manual test. This is known as "eyes into distortion." These and other elements that confused early muscle testing have become assets in an applied kinesiology examination. (The methods of applied kinesiology use of manual muscle testing in diagnosis are discussed throughout this text.)

Today it is much easier and less frustrating to learn applied kinesiology. The knowledge has been greatly expanded, and there are usually answers available when there appear to be enigmatic results from an AK examination. Pioneers in the field recognized that a muscle that tested weak, compared to its bilateral counterpart or the muscles of the general body, was not functioning properly. The burning question was **why**.

Those who persevered in their efforts to determine

why muscles tested weak found that many therapeutic approaches were applicable in improving muscle function. Most of the treatment techniques that improved muscle function were not originally developed in applied kinesiology. Some, such as meridian therapy, are used in their classic sense; in addition, they have been modified to be more productive in an applied kinesiology setting.

Applied kinesiology's contribution to many of these techniques is enhanced examination to determine the technique's usefulness for the particular patient being examined. Some new treatment techniques have been developed that are unique to applied kinesiology. The techniques cover a wide range, and one who has mastered all of them has a broad scope of practice in natural health care.

Throughout this text the term "physician" is frequently used to designate the one examining or treating a patient. These procedures should only be done by one thoroughly knowledgeable in physical, orthopedic, and neurologic examination and other examination methodology in the healing arts to properly make a differential diagnosis. Some who have used applied kinesiology muscle testing procedures made an inadequate study of the subject, or they did not have the anatomical, physiological, and clinical expertise necessary for the proper application of the discipline. This includes both doctors and lay people. The findings in an applied kinesiology examination complement information from the usual examination that a physician does. This added information regarding the health status of the examined patient must be put in perspective with the rest of the physician's findings. Applied kinesiology complements the standard examination; it does not replace it!

Techniques used widely in applied kinesiology evaluation and treatment are adjustment of the spinal column and manipulation of extraspinal articulations, nerve receptor treatment, balancing of the meridians and the cranial-sacral primary respiratory system, and nutritional therapy. Again, it is strongly emphasized that an applied kinesiology evaluation of health problems is only part of the total patient work-up. Examination should include standard physical examination, using orthopedic and neurologic tests, laboratory and x-ray when indicated, and the usual complete patient history. All factors of the total examination should correlate; applied kinesiology findings and other factors of differential diagnosis should enhance each other.

The major contribution applied kinesiology makes to standard diagnostic procedures is functional evaluation. Most standard diagnosis without applied kinesiology is directed toward discovering and evaluating pathology. Many individuals clearly pass a physical examination directed toward that end, but they may still complain of headaches, fatigue, and other general health problems. Applied kinesiology helps discover the reason for functional disturbances, and suggests a direction for corrective therapy. When applied kinesiology is used in conjunction with the standard methods of diagnosis developed in medicine, osteopathy, and chiropractic, one has a greater ability to understand a patient's health problem.

Functional Conditions

It is important for the reader to understand what is meant by the term "functional" when applied to a condition. This has been an area of misunderstanding when applied kinesiologists or their patients discuss conditions with other physicians. For example, the allopath considers hypoadrenia as Addison's disease.[2] In applied kinesiology, dysfunction of the adrenal glands is considered in relative states. Absolute Addison's disease may not be present, yet the adrenal glands may not be functioning adequately to maintain an optimal level of health. When hypoadrenia in the functional sense is communicated to an allopath without adequate explanation, he readily recognizes that the patient does not have Addison's disease and discredits the diagnosis, thus widening the gap of misunderstanding between the two philosophies of health care. Functional inadequacy of the adrenal glands is better termed adrenal stress disorder.

Over the years of modern health care, only a few physicians have looked at functional problems as a cause of their patients' symptoms. Harrower[42] wrote in 1922, "We overlook things. Why, the minor form of hypothyroidism is more than commonly ignored entirely! The consideration of the endocrine side of the ordinary troubles that are met every day in general practice has been passed by until very recently. Disorders of the ductless glands had to be 'real diseases' before we recognized them, and hidden functional aberrations were never sought for. Now all is being changed and as our eyes are being opened to the importance of *functional pathology* not only are we recognizing the early influence of endocrine dysfunction, but we are learning to consider a patient as a whole rather than an individual with some obvious disease." Unfortunately, when Harrower said "we" he was referring only to himself and a few followers, for the masses are still looking for "real diseases." Harrower stated in 1922 what is still the prevalent situation today: "Here, then, lies the greatest source of failure in medicine ... we have been treating diseases rather than patients."[42]

In the extensive literature of the health sciences, very little is written about functional disorders. An excellent discussion of the subject is found in Whatmore and Kohli's book, *The Physiopathology and Treatment of Functional Disorders*.[115] They state, "Functional disorders are disorders that have their origin in physiopathology, or functional pathology, instead of the more traditional structural pathology. That is, there is no underlying anatomical lesion such as a tumor or an infection and no biochemical lesions such as an inherited enzyme deficiency, a nutritional deficiency, or a foreign substance in the tissue fluids. Altered function is the primary or basic pathology."

Altered function may extend to the many systems

of the body. The autoimmune system may be unable to fight infection or contain and resolve allergic responses. Poor digestive function may cause malabsorption, with resulting nutritional deficiencies. There may be poor enzyme production and use. The list could go on and on. Improper signaling is a primary cause of these and other dysfunctions within the body. Whatmore and Kohli have coined the word "dysponesis," indicating a form of misdirected effort, to describe improper signaling.

Chiropractic philosophy has given much attention to improper control of body function by the efferent system. Applied kinesiology examination finds that much dysfunction results from the improper stimulation of exteroreceptors and interoreceptors. Homewood, in the conclusion of his book, *The Neurodynamics of the Vertebral Subluxation*,[45] writes, "Perhaps this may serve as a stimulus to increase the interest of all chiropractors in the afferent components of the nervous system, which outnumber the efferents 5 to 1 and are deserving of much more attention in the future than received in the past."

Much of the improper signaling that occurs in functional health problems is a result of improper balance of neurohumeral control. With applied kinesiology techniques, advances have been made in this functional evaluation.[90] Corrections are directed toward nutritional supplementation, improved glandular function, and sometimes the mental aspects of health.

Applied kinesiology recognizes that the body is a self-maintaining, self-correcting mechanism. When health is lost, something is interfering with the body's adaptability and it is unable to cope with the different environmental stresses. Examination effort is directed toward how the body is dysfunctioning, the cause of the dysfunction, and — finally — the therapeutic efforts that will enable it to regain and maintain health.

The difference between this philosophical approach to health and that of medicine produces a substantial difference in the methodology of research between the two.[106] There has been a paucity of research done on the functional health problems with which applied kinesiology and chiropractic deal.[12] Applied kinesiology has opened doors to understanding functional conditions, and it provides countless opportunities for clinical and basic research. Not only has AK provided an opportunity for improved examination of functional conditions, it has also emphasized natural health care and revealed some aspects of drug treatment. Williams[116] states, regarding drug treatment, "The basic fault of these weapons is that they have no known connection with the disease process itself. They tend to mask the difficulty, not eliminate it. They contaminate the internal environment, create dependence on the part of the patient, and often complicate the physician's job by erasing valuable clues as to the real source of the trouble." The last comment is specifically true in applied kinesiology, because drugs often change a muscle's reaction to the manual muscle test so that it does not reveal the patient's true condition.

Cleave,[10] a surgeon captain in the Royal Naval Medical Service who applies the principles of "natural law" to his consideration of body function, states, "...it is often said that to live medically is to live miserably — which is only too true. But to live naturally is to live happily, and there is a world of difference between the two." Exception may be taken to Cleave's statement about misery, but we strongly support his view of the natural state. Improving this condition is the goal of applied kinesiology and this text.

Research

The International College of Applied Kinesiology (ICAK) is an organization of physicians whose main purpose is to improve and expand the scientific use of applied kinesiology in determining the cause of health problems. The organization is open to all who have studied the subject in approved courses and are licensed as primary health care providers. In the USA chapter most members are chiropractors, while in chapters in Europe and other areas there is a more homogenous mixture of medical doctors, osteopaths, dentists, chiropractors, and other disciplines in health care.

A major purpose of the ICAK is to organize and promote education and research in applied kinesiology. The organization provides a forum for practitioners to communicate with each other. Research is being done on clinical and basic science levels.

Research in applied kinesiology follows the sequence of development described by Nordstrom[77]: (1) experience, (2) hypothesis, (3) methodology, and (4) scientific conclusion. Applied kinesiology methods are in various stages of this progression.

Experience. The strongest suit of applied kinesiology is the initial step of experience. This is realistic, because applied kinesiology is a relatively young, although rapidly maturing, part of the healing arts. Experience usually begins as anecdotal, where something is observed for the first time, or it can be a different analysis of a routinely observed pattern. An example is the development of Goodheart's pain control technique. He was tapping a meridian tonification point on a patient who had a recalcitrant clavicular fracture. Although Goodheart's treatment to the meridian system was not being directed to the patient's continuous and severe pain of the fracture, she commented that the pain had been relieved. In an effort to better understand this unusual finding, Goodheart tapped on the meridian's sedation point; the pain returned. Under these circumstances he found one can again tap the tonification point and the pain will be relieved, and again the pain will return when the sedation point is tapped. Goodheart eventually called this pain relief technique Melzack-Wall pain therapy,[33,34] after Melzack and Wall's gate theory of pain,[72] which was the standard explanation of pain control at the time. We will return to this serendipitous experience as we progress through the four steps of development.

Another dimension of experience takes place when one encounters the same occurrence over and over. It has already been mentioned that in the early days of applied kinesiology a muscle would be strengthened, but when the patient retumed for the next examination it would again test weak. Many of the techniques developed in applied kinesiology have resulted from efforts to eliminate this recidivism.

Hypothesis. The next step is the development of a hypothesis, which is necessary for the eventual discussion, testing, and understanding of a procedure. In most cases, these are working hypotheses in the current progression of applied kinesiology. Goodheart's hypothesis for the reduction of pain upon tapping the tonification point of a specific meridian was that it influenced the gate control of pain as described by Melzack and Wall.[72] The hypothesis then must be tested. Ideally this is done with the standard methods of science, including controlled studies with statistical analysis; most AK methodology has not been subjected to this procedure. There has been some effort in the ICAK-USA to get the members to use statistical analysis,[19,20,21] and education is being initiated to teach research methods. These efforts toward the practicing doctor, along with chiropractic schools' increased emphasis on teaching the doctor-scientist role,[57] should increase the testing of these hypotheses. It remains that most ICAK-USA members are clinicians not geared to research methodology. In the last five years the organization has attempted to fill this void by establishing boards of research consultants, scientific review, and standards.

As new examination and treatment techniques are developed in applied kinesiology, there is usually an attempt to correlate the new findings with what is known in the basic sciences and develop a hypothesis. In some instances, observation is made of a dysfunction that is consistently corrected by a therapeutic approach, with no hypothesis for the procedure. At a later time another practitioner may recognize basic neurologic principles that explain the condition and its correction. The lateral cuboid subluxation causing tensor fascia lata muscle weakness, discussed in Chapter 11, is an example of this. The tensor fascia lata muscle weakness is hypothesized to be present as a result of the cuboid subluxation improperly stimulating the "magnet reaction" (also called the "placing reaction").[37,40] This gives a workable neurophysiologic hypothesis to what was previously an empiric method.

Applied kinesiology is a developing science. Few of the hypotheses have developed to the higher level of theory; in fact, most are still in the stage of a working hypothesis. Even when a thought process becomes a theory, it should not be static. Pietsch[83] states, "A scientific theory is a system of thought, a rational explanation of facts or events, and not really an ascertainment of truth. Strictly speaking, true scientific theories do not exist. Does a given theory work? Is it logical? Does it generate new understanding? Can it allow the human mind to fathom what has always seemed beyond the depths of reason? Does it allow the intellect and imagination to place the phrase, 'What if' at the front of new and novel questions about nature? These are the important questions to ask about any scientific theory." This is as it should be. The explanations in applied kinesiology are not written in stone. Many who read this text and other applied kinesiology material are new to the subject, with fresh ideas and intellect developed from different sources. Hildebrandt[43] helps put this in perspective. There are times when a group with a new idea "...fears if it discards a few unworkable ideas of its developers, it will be destroying everything it stands for. Nothing could be further from the truth. Being flexible enough to recognize and discard unworkable ideas is the mark of true science." In addition, one "... should not test or evaluate an idea with any other motive than to uncover the facts; and thus uncovered, readjust his original hypothesis accordingly. Such is also the mark of a science — the ability to readjust original ideas in light of new developments."

Again, the example of Goodheart's application of the Melzack-Wall gate theory puts in perspective the progression of a workable clinical procedure. The hypothesis is expanded to encompass newly developed neurologic knowledge on the interpretation of pain. In this progression, Melzack and Wall have modified their model,[73] and Schmitt[91] has described additional techniques of pain control based on current knowledge of pain interpretation. Schmitt's method, like Goodheart's, accomplishes pain control by tapping on specific meridian points determined by applied kinesiology examination. Without Goodheart's initial observation, the continued development of applied kinesiology pain control methods would probably not have occurred.

Methodology. As we continue consideration of Goodheart's development of the pain control technique, his first question of the initial serendipitous pain relief was whether it would work on others. Would tapping that same acupuncture tonification point give relief to all who have pain? Investigation reveals that it does not. Goodheart then systematically began evaluating the meridian systems of those who had pain of a similar nature but varying from that initial experience. Eventually he found a protocol that consistently identified the proper tonification point to stimulate for pain relief. He also found that the treatment was more effective on pain resulting from trauma, and that it was not an appropriate therapeutic approach for pain from a subluxated sacroiliac or other primary condition.

The development of proper methodology and the subsequent proper application of it are of utmost importance in obtaining consistent results. As noted in the description of the pain control technique in Chapter 7, failure to exactly identify the location of the proper tonification point will result in failure to obtain results when stimulation is applied.

To establish reproducibility in applied kinesiology examination and treatment, the ICAK Board of Standards

has developed a protocol to delineate the methodology of a procedure before it is evaluated by members of the board. Passing this board is a recently established method for accepting new material into the ICAK's recommended procedures.

Scientific conclusion. The final stage, scientific conclusion, rivals the first stage of experience for challenge, creativity, and opportunity. Applied kinesiology must develop its own methodology of research because the principles of health care are different. Applied kinesiology and chiropractic share a philosophy based upon the role an organism plays in response to environmental stressors. This fundamental approach is different from the traditional approach taken by others in the healing arts. Vitelli[106] presents an excellent discussion on the different needs in research between medicine and chiropractic. He states, "Most non-chiropractic disciplines base their views on health and most disease upon the absence or presence of a significant quantity of pathogenic micro-organisms and other agents and toxins considered responsible for the production of disease in the organism. Their approach to health restoration begins with the removal or destruction of such viewed disease-producing agents. Disease is also viewed as a definite morbid process having a characteristic train of symptoms." He goes on to state, "...at the clinical research level due to the different points of view on health and disease, the differences between the professions are great."

Research in applied kinesiology is done on two levels: basic and clinical. Basic research is primarily done in educational institutions. The International College of Applied Kinesiology has an ongoing program to sponsor basic research in these settings. Applications for grants are accepted through the ICAK's Board of Research Consultants.

The ICAK-USA sponsored the development of the Foundation for Allied Conservative Therapies Research (FACTR), which is a separate tax deductible foundation concentrating on applied kinesiology research as it deals with all professions in the conservative health field.

The new discoveries in applied kinesiology have been accomplished on a clinical basis. Goldstein[27] emphasizes the need for clinical studies as well as basic science studies. Clinical studies in areas such as the reliability of clinical diagnosis and the efficacy of therapy are as much a part of research on the scientific basis of any therapy as is an understanding of the fundamental mechanisms underlying it. The scientific basis of manipulative therapy clearly includes both.

Keating et al.[54] describe the time series designs of research that are applicable to clinical use. These studies do not require a large number of subjects and are compatible with the primary purpose of clinical function; that is, to regain optimal health of the patients involved. There are several designs in this system of research. The reversal design is applicable when one can apply and remove the therapy being evaluated. Again, we can use the development of the pain control technique by Goodheart as an example. When one applies stimulation to the appropriate tonification point, pain is relieved. The pain is predictably returned when the sedation point of the same meridian is stimulated. The relief and return of pain can be repeated numerous times and on numerous subjects.

Another time series method is the Multiple Baseline Design. Here the patient's function is plotted over a period of time. A favorable study results when the function predictably improves with treatment application. Function can be measured by range of motion, vital capacity, laboratory methods, or any other parameter applicable to the patient's condition.

Research Opportunity

Many phases of natural health care have stagnated because of lack of properly conducted research. Watkins[111] makes a strong plea for the chiropractic profession to develop chiropractic methodology with properly conducted research programs. Most of Watkins' writings were unpublished and directed to the National Chiropractic Association. His efforts in this direction were made from 1945-1970. Both lack of research and adherence to dogma have limited the growth of chiropractic methods. A main effort of many of the chiropractic colleges has been to "preserve the principle" rather than use investigative efforts to create new and better methods of examination and treatment. Watkins [111] states, "Considerable effort has been expended in the technical advancement of vertebral adjusting, and it is doubtful that there is any possible way of adjusting a vertebra that has not been tried. But after seventy years the adjustment of the vertebra remains the sole contribution of major significance that chiropractic has made to the field of healing — a contribution which was made by Dr. D. D. Palmer before the profession was even organized." Watkins calls preserving the principle "the great mistake in chiropractic."[110] This writer believes that if Watkins had been heeded at the time he first started writing, there would be a greater parity today between medicine and chiropractic, the two largest fields in the healing arts. Watkins stated "...that there is a tremendous need for a second healing science in our society today, competitive with organized medicine on both the scientific and the practice levels."

To prevent this message from being misunderstood, it needs to be stated now that correction of vertebral subluxations and fixations is a major portion of the armamentarium of chiropractic applied kinesiologists. Many of the techniques that have been developed in chiropractic are used. In addition, new methods of examination and adjustment have been developed within applied kinesiology.

The great difference between allopathy and natural health care seems to indicate that the two approaches will not be encompassed into the practice of one individual — at least not in the near future. It is necessary,

however, that the two philosophical viewpoints be understood by practitioners of both approaches. One should recognize the deficiencies and advantages of each approach, and readily refer patients to the other discipline when it is to the patient's advantage.

Douglass[15] says this about his profession of medicine: "To a medical doctor, if you can't see a disease on or in the patient's body, on an x-ray, or in a lab report, then it doesn't exist. Or if it does exist, it isn't very important if it isn't big enough to see." He goes on to comment that the natural health care approach, including chiropractic, recognizes "... subjective complaints for what they are: neurobiological dysfunctions that are not usually measurable by current scientific laboratory methods. How do you measure a headache or a backache? Most chiropractic treatment is not quantifiable because of the subjectivity of the complaints." The subjectivity of complaints has been one of the major problems in dealing with natural health care. Applied kinesiology is adding objectivity to the examination of soft tissue injuries and improper neurologic signaling.

For the allopath and the natural health care provider to communicate, they must speak the same language. Jamison[51] takes the position, "The price of acceptance into orthodox health care is conformity to the institutional structure of medicine. A basic concept of the structure is the 'scientific' nature of modern medicine." She does concede that "It is, however, necessary that the reader be aware that even today all that is accepted as medical science is not necessarily scientific."

Because the mode of therapy in allopathy and natural health care is so divergent, many of the research methodologies cannot be applied to both. Vitelli[106] writes about the medical profession, "Their *methodologies* allowed them to duplicate results supplied by their practitioners and health providers. Their *standards of measurement* allowed them to express these results in a quantitative and qualitative way." Natural health care philosophy demands that approaches to researching health problems be based upon the role that the organism plays in response to environmental stressors, if it is to be kept within its philosophy's guidelines. Natural health care is based upon returning the self-correcting, self-maintaining character to body function. This fundamental approach is almost opposite that of the traditional allopath. Rather than focusing on how the body reacts to environmental and intrinsic stressors, the allopath focuses on the role of the environmental stressors and how they affect the organism. Many answers to improving examination and treatment can be found through research, using proper methodology and standards of measurement unique to applied kinesiology.

Clinical findings in applied kinesiology have opened many doors for basic research and have provided tools for additional clinical understanding of health problems. Research must proceed on both the clinical and basic research levels. The creativity in research seems to come from the clinical level. There are several factors that stifle creativity in basic research.

Selye[96] condemns the process of "grantsmanship" wherein the potential scientist must describe in advance what he expects to discover, how he's going to do it, and what it's going to cost. This writer feels privileged to have heard Selye lecture on the subject. He said that if he had had to follow the scientific method of today, he would never have discovered the general adaptation syndrome of stress. Selye[97] points out the importance of one's ability to recognize what others do not see in his story about Fleming's discovery of penicillin. In the process of culturing pathogens, Fleming observed a mold contamination killing his pure strain of bacteria. Fleming's creative mind recognized this as a possibly useful drug to control and protect against infection. Previous bacteriologists had seen the same thing, but they only considered it as a problem in their attempt to culture a pure strain of bacteria. All they concluded "...was that molds must be kept out of cultures. It took a stroke of genius to see the medicinal promise of the basic observation."[97]

The basic requirement of grantsmanship — to describe what one is going to research — is a talent separate from making new discoveries. Imagine Leonardo da Vinci's creative genius in today's professional and specialization development. Horn[46] discusses Congressional librarian Daniel Bornstein's impression of this aspect of da Vinci. "He would have had to present his credentials before anyone would have looked at a design, but that's not what advances knowledge: it's the people who are willing to have wild imaginings."

Most of us can only recognize what we already know. Scientists, like the rest of us, tend to see what they expect to see. A simple experiment puts in perspective how we see things in our previously established formats. Individuals are asked to identify playing cards. Most of the cards are of the usual design, but some have been changed, e.g., a red six of spades or a black four of hearts. Not only do the subjects identify the normal cards correctly, they also identify the anomalous cards by the usually associated color rather than the design, i.e., the six of spades is identified as the six of hearts, and the four of hearts is identified as the four of spades. The average individual continues to identify them improperly. Some people will eventually see that something is unusual and look closer, finally detecting the anomaly.

Cole[11] emphasizes this with a story about Darwin, who once spent a whole day in a river valley and saw "...nothing but water and rock." Eleven years later he walked in the same valley, this time looking for evidence of glaciers. "I assure you," he wrote a friend, "an extinct volcano could hardly leave more evident traces of its activity and vast powers...the valley about here must have once been covered by at least 800 or 1,000 feet in thickness of solid ice." Once he knew what to look for, it was easy enough to find.

The same factor that causes one to see what he

expects to find can be detrimental to applied kinesiology examination and research. Schwartz[94] warns against having a mental matrix when testing with applied kinesiology methods. One can subconsciously change the parameters of the test to consistently observe what he expects.

When discussing the development of applied kinesiology techniques, it was mentioned that experience is AK's strong suit. This is where the original observations that improve health care are made by doctors confronting everyday health problems their patients present. AK has developed on a need to fix basis. Goodheart's comment is that applied kinesiology is an ongoing research patient-oriented system. His attitude is often, "When I can fix it I don't need to research it any more." This approach performs a special service. We all have different areas of expertise. Some have the creative mind that can look at everyday occurrences and see what others do not, such as Fleming's discovery of penicillin. Others move toward the linear left brain thinking and tie the creative observations into the literature base, developing hypotheses, improving methodology, and — finally — testing the hypotheses to arrive at a scientific conclusion. In any event, without the original experience and creative thought, the other steps in the process do not occur. To some extent, creativity has been stifled by the specialization prevalent in today's health care. So often we see patients who have been shuttled from one specialist to another with no one ever observing the total patient with his integrated systems and functions. Weissman[113] looks at the non-specialized and specialized, stating, "It is often after amateurs (doctors in practice) make distinctive discoveries that the 'ologists' are able to make definitive discoveries."

Viable research can be developed in the physician's office, whether it be natural health care or allopathy. Wiltse[117] states, "...I believe that every spine surgeon's office can be a research center." This is even more applicable in natural health care because of the wide range and integration of the problems encountered. Where one is dealing only with intervertebral disc problems, kidney dysfunction, or some other specialized area, it is easier to design a research study that deals with that single question.

Much has been written in chiropractic literature about clinical research. Keating and Mootz[56] state, "...control-group experimentation is often ill-suited to addressing the questions raised in clinical practice." "Each new clinical case is an opportunity to further explore the validity of chiropractic theory. Science is here considered to be perpetually incomplete, evolving, imperfect." "Research becomes a self-perpetuating activity, for inevitably science raises more questions than it answers." It is the questions that have arisen in applied kinesiology examination that have been the catalyst for rapid development of new examination and treatment techniques.

Many of the techniques developed in applied kinesiology are simple, logical, and fit well with the database of previous knowledge. The uniqueness of the new technique has simply not been observed before. The admonition to pursue simplicity in research is usually the most productive in creative works. Teller[102] characterizes simplicity in a story about 14-year-old Mozart, who listened to a secret mass in Rome that included Allegri's *Miserere*. The composition had been guarded as a mystery. The singers were not allowed to transcribe it, under penalty of excommunication. Mozart heard it only once, but he was able to reproduce the entire score. His feat was not one of amazing memory. As a work of art the piece had threads of simplicity. Mozart did not remember the details of this complicated work, but he could identify the threads, remember them, and re-invent the details, having listened once with complete attention.

As new techniques of examination and treatment are developed in applied kinesiology clinical research, one should be his own devil's advocate. Katch[53] proposes a method of research that he calls the "burden of disproof." This is opposed to the "burden of proof" doctrine that states: "Those who propose a particular hypothesis or theory are responsible for providing proof." Katch goes on to explain, "By the process of disproof, one is able to change, alter, expand, and devise alternative and more appropriate hypotheses which are better explanations of the observed data, and better predictors of new data." Using the "burden of disproof," several alternative hypotheses are developed and crucial experiments devised to exclude non-workable hypotheses. Katch emphasizes the idea that this has been around since the late 1800s, "...that, once we propose a single hypothesis, we necessarily become overly attached to it, to the exclusion of looking for alternative and perhaps contradictory hypotheses." By establishing multiple hypotheses, one does not become attached to a specific one, enabling the researcher to disprove inaccurate approaches and develop new hypotheses that continue building accurate knowledge.

Refusing to relinquish dogma that does not coincide with current knowledge has been a major fault of chiropractic. Occasionally one hears of a research project to prove or substantiate an examination or treatment technique. Projects should be developed to study the mechanism and test the hypothesis. When results do not support the technique, the practitioner, like the scientist, should re-evaluate the initial assumptions and be prepared to set aside previous concepts in light of the new information. The researcher who seeks to prove or substantiate a technique is less likely to do this; indeed, the investigator who seeks to prove or substantiate a technique is misguided and ill-prepared as a researcher.[56]

Wherever a physician's thought process works best — in the experience, hypothesis, methodology, or scientific conclusion scheme — he should apply himself in the role of the scientist-practitioner.[3,12,55] The practitioner is in a daily research setting. The knowledge should be reported to enable more rapid growth. Rose,[88] in the *Journal of the American Dietetic Association*, points out that

"*Research*, simply stated, is a process of identifying a problem, evaluating the alternative solutions, and then selecting the best solution, given the specific constraints at hand." All physicians are doing this on a daily basis, but they generally keep the information to themselves. There is a void in the literature relative to the everyday knowledge the practitioner has developed. Refereed journals intimidate the practitioner, yet most have developed at least a few "jewels" that others are not aware of. Where is this information? The practitioner shakes his head, saying, "Publish? What I have done would interest no one!" Untrue!

The answers to everyday health problems come from confronting them in the clinical setting. As we attempt to solve them, we test the therapeutic approach measured by the results obtained. This may not be a structured research program, with statistics and the other formal methods of research, but it is research nevertheless. Often it fits the time series design[3,54] of research without the formal record keeping. The physician is aware of the efficacy or lack of it in his examination and therapeutic attempts.

The answers to the problems patients present come from our own abilities to apply our education and recognize the uniqueness of each individual situation. As prior knowledge is exercised, it grows in its application. When these experiences are communicated, they stimulate others' intellectual and conceptual capacities and shorten the life cycle of developing improved methods.

The International College of Applied Kinesiology plays several roles in the progression of applied kinesiology. There have been over 2,000 papers published by and for members of the organization. These papers serve as a means of communication, ranging from single-page observations to extensive, well-documented literature reviews and detailed procedures of examination and treatment. This communication consists primarily of observations and efforts to understand phenomena seen in clinical practice. Here are the observations of clinicians dealing with the day-to-day struggles of returning health to the masses. Many of these observations have brought new insights to be refined and researched with formal methods. The organization does not require strict conformity to proper research design for paper presentation. This is done in order to bring out the creativity of the clinicians' observations to stimulate others. There are advantages and disadvantages to this approach. It encourages the dissemination of the clinician's experience, but these people are not researchers and frequently not educated in research design.

The failure of ICAK members' papers to satisfy research criteria is noted by Klinkoski and Leboeuf,[61] who reviewed fifty papers from the ICAK-USA collection of papers. They concluded, "As none of the papers included adequate statistical analyses, no valid conclusions could be drawn concerning their report of findings." They subjected the papers to research methodology, i.e., a clear identification of sample size, inclusion criteria, blind and naïve subjects, and statistical analysis. They state, "Although some papers satisfied several of these criteria, none satisfied all seven of them."

There are other factors besides the seven noted above to be considered in researching alternative therapy. Some of the aspects unique to applied kinesiology are discussed by Motyka and Yanuck.[75]

Some of the new techniques presented in the ICAK papers are submitted to the ICAK Boards of Standards and Scientific Review. This leads to further clinical review, followed by full or provisional acceptance, or unsubstantiated. The unsubstantiated category leaves a procedure open for further investigation, not giving it a totally negative status and thus promoting further observations.[52]

An arm of the ICAK is the International Board of Applied Kinesiology, which confers diplomate status on those who have completed a prescribed course of postgraduate education, performed research in applied kinesiology, and passed written and practical examinations on the subject. Diplomates of the ICAK are eligible to become certified teachers. Those who present classes under the auspices of the ICAK-USA teach only doctors licensed as primary health care providers, or those enrolled in a college leading to a degree to become a primary health care provider. In addition, ICAK-USA certified teachers allow doctors' assistants to attend applied kinesiology classes if the doctor certifies that he is responsible for the diagnosis and treatment of patients with whom the assistant works.

Applied kinesiology will grow or decline on its own merit. There are three basic "courts of accountability." The primary one is the scientific community, which establishes diagnostic and therapeutic efficacy through scientifically controlled clinical trials. Second is the test of time, during which clinical concepts are found to be efficacious (or at least harmless) by empirical application. Finally, the third court of accountability may be a court of law if a diagnostic or therapeutic procedure results in serious consequences for the patient — either directly by the effects of giving improper treatment, or indirectly by the resulting delay in receiving proper treatment, i.e., that generally considered appropriate by peers.[44]

Physicians who qualify for applied kinesiology education by ICAK-USA teachers are under some type of practice license. Boards of examiners usually regulate and administer the license to practice. This is another court of accountability in the third division. There have been doctors who use unusual and sometimes bizarre approaches; when called before a regulatory agency they claim to have been using AK. Unless there are members of the agency who recognize that the questionable activities are not AK, the entire discipline is discredited. The updated ICAK-USA Status Statement that addresses some of these issues is on the organization's Internet web site http://www.icakusa.com.

Another court of accountability is the patient re-

ceiving service. AK patients gain a strong favorable opinion of the system and make many referrals. When relocating applied kinesiology patients seek future treatment from an AK practitioner. They expect the same type of service received in the past. The ICAK-USA office receives numerous requests from people who have received advice from a friend or relative in a distant area. The ICAK-USA web site is accessed frequently for referral information.

Evaluation of applied kinesiology procedures must be done by proper research design. There have been occasions when AK has been criticized that were inappropriate when viewed in light of procedures accepted by the ICAK-USA. Motyka and Yanuck[75] have done a review of the studies in the indexed literature. In their critique of the studies they point out the strengths and weaknesses, and finally address how research projects can be designed to avoid the pitfalls present in studying natural health care, especially applied kinesiology.

A few examples put in perspective studies that place applied kinesiology in a negative light. Often these studies are designed by people who have never practiced applied kinesiology and are studying something that is not in the realm of applied kinesiology as accepted by the ICAK.

A well-designed study of normally functioning subjects attempted to increase a muscle's strength by neuromuscular spindle cell manipulation.[39] Applied kinesiology manipulation of the neuromuscular spindle cell is designed to treat an abnormally functioning muscle that is hypertonic or tests weak by manual muscle testing due to the dysfunctioning spindle cell. One must first identify that the muscle is dysfunctional and then that it is due to the neuromuscular spindle cell. Under those conditions the neuromuscular spindle cell treatment is applicable. This study was of something not a part of applied kinesiology and concluded that the treatment protocol is ineffective. It is foolish to try to make a normal muscle more normal by this technique.

Another study was designed to determine if chewing sugar causes general muscle weakness in a normal population.[23] The study was interpreted as negative toward applied kinesiology because all did not weaken. It has never been stated in AK that this would happen. In fact, it is expected there will be different findings across the subject population, which is what the study found.

The nutrient status of eleven subjects was evaluated with laboratory measures and compared to what was purported to be applied kinesiology.[60] The methods used have never been reported in AK literature, making the study irrelevant to applied kinesiology.

Unfortunately, studies of this nature may be read by people who have no background in applied kinesiology and therefore are unable to recognize the flaws in the research design. The negative impression of applied kinesiology derived from this type of study slows the proper research to better understand and refine the discipline of AK.

Poor quality research has also been applied to chiropractic, with negative results. Vitelli[107] states, "Perhaps impropriety, either in the methods utilized by non-chiropractic researchers or in the lines of approach utilized by these investigators, has been the reason for the criticism when trying chiropractic investigations. In reality everyone has the right, with all statistical validity, to question results if obtained utilizing another profession's criteria or an improper methodology."

Since the initial development of AK in 1964, a large number of physicians have studied the subject and many have contributed to its development. Research is now completed and in the indexed literature that helps understand the mechanisms and supports the AK examination.[22,65,67,68,75,82,92,93] These studies are discussed in this text in the associated locations. Additional studies have been submitted for publication, and others are in the developmental process. A current list of the indexed literature applicable to applied kinesiology is on the ICAK-USA's Internet web site, http://www.icakusa.com.

It appears that the time for applied kinesiology has come. Hildebrandt[43] quotes Beveridge, "When discoveries are made before their time they are almost certain to be ignored or meet opposition which is too strong to overcome, so in most instances they may as well not have been made." Hildebrandt goes on to state that when a new discovery is not placed into proper form for reaction from the scientific community, there is an "attack-escape" reaction. Attack may take the form of ridicule or scientific investigations to disprove the "discovery." Escape reaction is to ignore, which will continue unless there is sufficient aggravation from the proponents of the new "discovery" to cause an attack reaction. We find applied kinesiology as advocated by the ICAK to be experiencing acceptance; still, some who have not thoroughly studied the subject are in the attack-escape reaction.

The examination and treatment techniques presented in this text will be new and revolutionary to some readers. Effort is directed toward understanding dysfunction within the body and finding methods for correcting it. It is assumed that the reader is skilled in the art and science of diagnosis, and will apply these methods along with the applied kinesiology examination. The text is directed toward undergraduates in an educational program leading to a license as a primary health care provider, and to postgraduate practitioners who are currently licensed.

This material will be of interest primarily to those already providing natural health care to their patients. As previously mentioned, the philosophical approach to health differs so greatly between the allopath and the natural health care provider that it is not expected an allopath would be interested in using this material in his practice; however, there is hope that the allopath will read this material and develop an appreciation for natural health care applicable to many patients who have previously been untreatable because no disease was found.

Throughout the development of applied kinesiology,

many new methods have been described. Along the way some have been dropped and others expanded. This is a growth state. We are strongly in agreement with Keating and Mootz's statement[56] that "Research becomes a self-perpetuating activity, for inevitably science raises more questions than it answers." And further, "When initial results do not support the initial clinical hunches, the practitioner, like the scientist, rechecks her/his initial assumptions and preliminary measurements. Like the researcher, the treating doctor is prepared to set aside initial concepts when objective results so require, and, like the scientist, the healer must strike a balance between skepticism and open-mindedness." The approaches presented here should be evaluated by the individual practitioner. Some hypotheses may need to be changed in light of new knowledge; some will continue to expand and grow stronger, to the point of theory. Jamison[51] states, "Theories cannot be proved right, they can only be proved wrong or not wrong. Science is a process which progresses gradually as new problems result from theory breakdown."

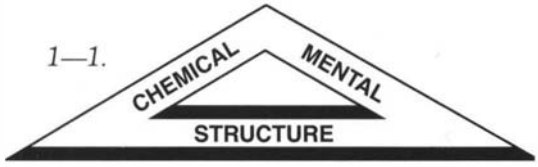
Concluding this chapter are some basic principles adopted by applied kinesiology, such as the triad of health that dates back to the inception of chiropractic, to the "new neurology" of the hologram model of the nervous system and memory. Applied kinesiology will continue to build on the old and established while exploring the new and innovative.

Triad of Health

Many in chiropractic have described the triad of health, beginning with D. D. Palmer.[81] Health is composed of structural, chemical, and mental factors that should be balanced, forming an equilateral triangle. When a person experiences poor health, one of the three factors of the triad of health is always involved. With severe health problems and chronicity, two or all three of the factors may be contributors. Applied kinesiology enables us to evaluate the triad's balance.[31]

Structure

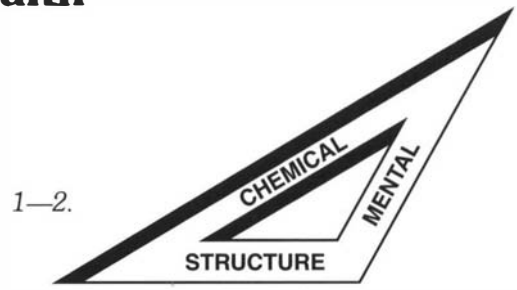
Structure, with which chiropractic has always been involved, is the base of the triangle. Many chiropractors have limited their concentration to the removal of spinal subluxations to improve nerve function. This, of course, has obtained excellent results; it always will. But many health problems with which chiropractors must deal will improve to a higher level when extraspinal structural dysfunction is corrected and examination and treatment is applied to the other two factors of the triad, when involved.



1—1.

Chemical

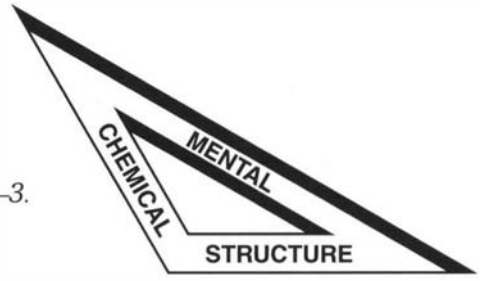
Currently the chemical factor of health is dominated by the medical profession. Nutritionists perform an important role in health care by concentrating on the chemical side of the triad. The difference between the two is that the medical profession often use chemicals to control body function, while the use of nutrition is directed toward building tissue and providing the basic raw materials for normal body function. The allopath attempts to control the other two sides of the triangle with substances such as tranquilizers and antidepressants for the mental aspect, and muscle relaxants and analgesics for structural disturbances.



1—2.

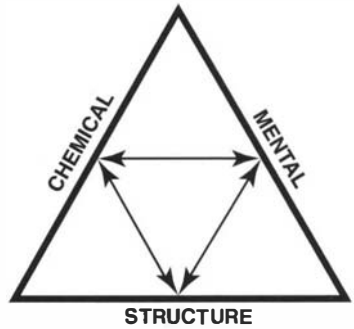
Mental

Mental health care has been dominated by psychiatrists, psychologists, and various types of counselors. Chiropractors and other doctors with strong "personality-dominated" practices often influence this aspect of the triangle.



1—3.

In applied kinesiology there is strong emphasis on examining all three sides of the triad of health and directing therapeutic efforts toward the basic underlying



1—4.

cause of a problem. Often a health problem starts on one side of the triad, and eventually involves all three aspects. It is important to recognize that any one side of the triad can affect the other sides, both as causative factors of health problems and as therapeutic approaches.[41] The key is to determine the primary factor(s).

Applied kinesiology examination helps determine the basic underlying cause of health problems. Structural balance is readily evaluated in the static and dynamic body. The neuromuscular reaction of the body to chemicals helps evaluate the effect of nutrition and adverse chemicals. Insight into the patient's emotional status can be obtained by evaluating the nervous system, using applied kinesiology manual muscle testing. Mental health problems can sometimes be improved by nutritional and structural corrections, determined by manual muscle testing.

Structural Balance

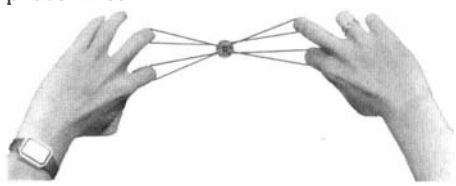
Under normal conditions, structural balance is maintained by the muscles of the body controlled by the nervous system. When the muscles are out of balance, postural distortion is present. Strain develops within the muscles and joints. Often the muscular imbalance is related to hypertonicity, shortness, or frank muscle spasm.

The imbalance of muscular function is an obvious fact that has been observed by physicians of all kinds for centuries. The therapeutic endeavor has frequently been applied to the short, hypertonic muscle. Early in applied kinesiology it was recognized that the short or hypertonic muscle is frequently secondary to poor function of its antagonist muscle. On manual muscle testing the antagonist muscle tests weak, indicating that the nervous system is incapable of adequately controlling it. Therapeutic approaches to the weak antagonist muscle often rapidly relieve the hypertonicity. The usual therapeutic efforts of heat, ultrasound, electrical stimulation, and massage, among others, to the hypertonic muscle may provide only temporary results because the basic cause of the problem is in the poorly functioning antagonist.

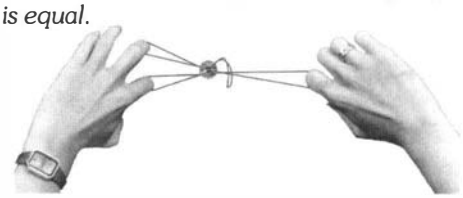
When the antagonist muscle offers poor opposition, the situation is very similar to the "balling up" of the biceps brachii when its tendon ruptures. It may be fortunate that most therapeutic efforts to relax a hypertonic muscle secondary to a weak muscle are unsuccessful; otherwise the patient would end up with two "weak" muscles.

Muscle imbalance is primary to most structural deviations, from vertebral subluxations to major postural imbalances. When muscle imbalance is present, structure pulled toward the stronger muscle cannot be made to stay in place until the muscle imbalance is corrected. A good way to explain this to a patient is to use a button with four rubber bands attached. In each hand hold the ends of two rubber bands, suspending the button in the center. Explain: "The rubber bands represent your muscles pulling on the button, which is your vertebra. The button remains in the center because the pull on it is equal from both sides." At this point drop one of the rubber bands, making a weaker pull from one side. Continue: "This represents the position of a bone when there is muscle dysfunction on one side. The vertebra is pulled out of place, and no matter how many times we center it, it will return to abnormal until we correct the muscle imbalance." This illustration can be used whether dealing with a vertebral subluxation, pelvic, knee, foot, or other imbalance.

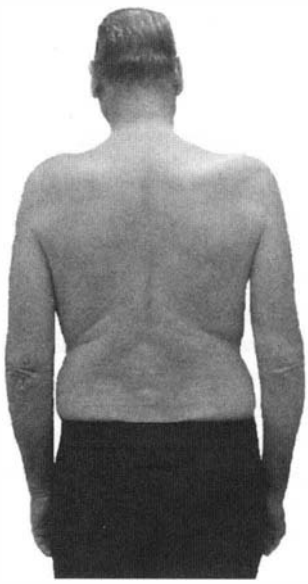
Pain is usually present when a muscle is chronically hypertonic or shortened. Correction of the dysfunctioning antagonist muscle often relieves pain almost immediately in the hypertonic muscle. This can be demonstrated to a patient by having him rate the pain of digital pressure in the belly of the hypertonic muscle before and after correction of the weak antagonist muscle with applied kinesiology procedures.



1—5. Structural balance is present when pull from both sides is equal.



1—6. An imbalance of pull causes the structure to deviate to the stronger side.



1—7. Note the head tilt toward left side and the distance across the shoulder to the neck on the left. In this instance there is imbalance from an upper trapezius weakness on the right and secondary contraction of the upper trapezius on the left. There may be weakness of the cervical flexors and/or extensors on the right. Note the imbalance of the skin folds at the lower portion of the thoracic cage.

Five Factors of the IVF

Most of the examination and treatment procedures in applied kinesiology relate to the nervous, lymphatic, and vascular systems, along with the relationship of cerebrospinal fluid with the cranial-sacral primary respiratory motion, and with the meridian system. Goodheart has related these five factors to the intervertebral foramen (spinal column) and coined the term "five factors of the IVF" to describe the examination and therapeutic approaches used in applied kinesiology. When the term "five factors of the IVF" is used in an examination or therapeutic sense, it refers to evaluating for or treating all factors involved in the applied kinesiology examination or therapeutic armamentarium. These five factors are included with a structural man and the triad of health to form the logo of applied kinesiology.

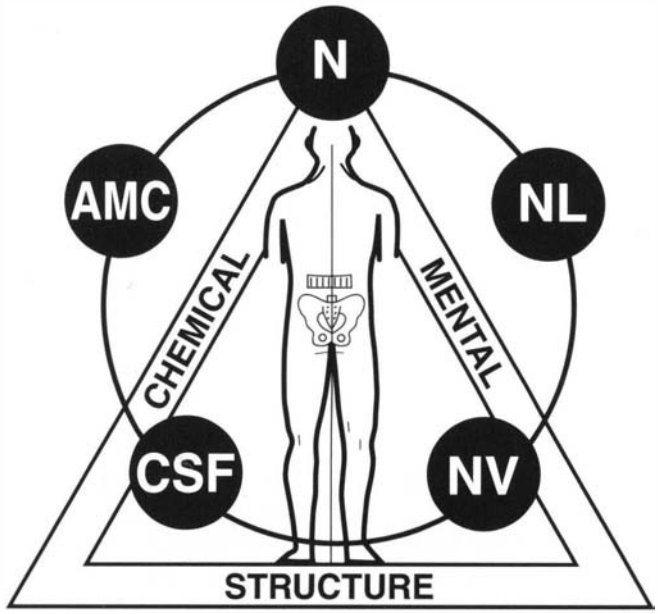
The "N" at the top of the triad refers to the nervous system. It includes disturbance of the nervous system by spinal subluxations, peripheral nerve entrapment, disturbance in neurotransmitters, improper stimulation of the various types of nerve receptors, and nutrition. Nutrition is included in the nervous system because the unique effects of nutritional stimulation on the gustatory receptors have been shown to influence muscle function, as demonstrated by manual muscle testing in applied kinesiology.

The "NL" stands for neurolymphatic reflexes, which are Chapman reflexes[80] that have been incorporated into applied kinesiology. Other examination and therapeutic approaches are also used to influence the lymphatic system to help eliminate major blockages and improve lymphatic flow.

The "NV" stands for neurovascular reflexes, which are the Bennett reflexes[6] that have been incorporated into applied kinesiology. Although Bennett reflexes are located throughout the body, applied kinesiology primarily uses those located about the head. Other applied kinesiology therapeutic factors relating with the blood vascular system are represented by this aspect of the five factors of the IVF.

The "CSF" of the five factors of the IVF represents the cerebrospinal fluid associated with the cranial-sacral primary respiratory mechanism described by Sutherland.[101] It relates to the autonomous movement of the bones of the skull, sacrum, and pelvis, and has become an important part of applied kinesiology examination and treatment. The influence of jaw function on the cranium (the total mechanism is known as the stomatognathic system) is included in both the "N" for nerve and the "CSF" for cerebrospinal fluid.

The final factor of the five is the "AMC," standing for acupuncture meridian connectors that are points along the bladder meridian found in applied kinesiology to relate closely with spinal subluxations. The meridian system has become both an important examination and a therapeutic aspect of applied kinesiology.



1—8.

"Man possesses a potential for recovery through the innate intelligence of the human structure. This recovery potential with which he is endowed merely waits for your hand, your heart, and your mind to bring it to potential being and allow the recovery to take place, which is man's natural heritage. This benefits man, it benefits you, and it benefits our profession. Do it, do it with knowledge, do it with physiologic facts, do it with predictable certainty, do it because it has to be done, and we as a profession are the only ones who can do it effectively."

George J. Goodheart, Jr., D.C.

Muscle-Organ/Gland Association

Early in applied kinesiology Goodheart detected some consistency of specific muscle dysfunction with specific organ or gland dysfunction. For example, when the pectoralis major (clavicular division) muscle tested weak, there was often stomach dysfunction. When the quadriceps muscles tested weak, there was often small intestine dysfunction. When the tensor fascia lata tested weak, there was often colon dysfunction. (The complete muscle-organ/gland association is included in the muscle testing section of this text.) The observation of muscle-organ/gland association was strengthened as new examination and therapeutic approaches were introduced into applied kinesiology.

Treatment to Chapman's reflex for the stomach, now called a neurolymphatic reflex, strengthened the pectoralis major (clavicular division) muscle. Treating the neurolymphatic reflex for the small intestine improved quadriceps muscle function, and the colon neurolymphatic reflex stimulation strengthened the tensor fascia lata muscle.

The Bennett reflexes, now called the neurovascular reflexes, had a similar correlation. Stimulating the small intestine reflex improved the weak quadriceps muscles. Treatment to the colon reflex improved the tensor fascia lata muscle when it had previously tested weak. A similar correlation was found in the neurovascular reflex treatment that improved function of the pectoralis major (clavicular division) muscle. It was discovered that stimulation of Bennett's emotional reflex affected the pectoralis major (clavicular division) muscle. Reflecting that emotions are often considered the cause of stomach problems, especially ulcers, one can readily see a tie-in.

The next factor introduced into applied kinesiology with specific organ/gland association was the meridian system. When treatment was applied, the stomach meridian improved the pectoralis major (clavicular division) muscle, the small intestine meridian improved the quadriceps muscles, and the large intestine meridian improved the tensor fascia lata muscle.

The muscle-organ/gland association of applied kinesiology is clinically valuable in helping find the primary areas of dysfunction when combined with other diagnostic disciplines. The muscle-organ/gland association should not be considered absolute. For example, there may be local muscle dysfunction causing the quadriceps muscles to test weak, with no dysfunction in the small intestine. On the other hand, an individual may have a gastric ulcer confirmed by radiology but the pectoralis major (clavicular division) may not test weak. As one becomes more advanced in applied kinesiology, it is seen that nearly always there is dysfunction of the pectoralis major (clavicular division) muscle, but the body has many ways of adapting to dysfunction. With advanced applied kinesiology techniques, it may be found that the pectoralis major (clavicular division) muscle tests strong in the presence of an ulcer because there is overactivity of the stomach meridian as an adaptive healing effort by the body. Research on the muscle-organ/gland association is ongoing,[7,8,13,50,66,118] with much yet to be learned.

Body Language

The body has a language providing information that can lead to the discovery of the cause of health problems; the key is an ability to understand the language. Failure to read body language regarding health is very similar to an inability to interpret a foreign language. The information written in the language may be very valuable, but unless one can read the language, the information is useless.

A major aptitude that Goodheart, the father of applied kinesiology, possesses is an ability to decipher the language the body presents. It may be a postural imbalance revealing muscle dysfunction related with an organ or gland that provides the primary clue. Add to this an examination of the five factors of the IVF, how the muscles respond to various applied kinesiology tests, and — finally — known body language, such as skin texture, and laboratory, orthopedic and neurologic tests, and a full body language reveals the disturbance causing the health problem.

Applied kinesiology is a discipline that incorporates other diagnostic methods with various applied kinesiology tests evaluated by manual muscle testing. This analysis improves the physician's ability to evaluate functional aspects of health problems. In Goodheart's words, "The body never lies; however, we must ask the right question in the right way." As one delves deeper into applied kinesiology, it is marvelous how body language reveals the functional disturbances causing health problems.

Temporal Sphenoidal Line

The temporal sphenoidal line (TS line) was described by M. L. Rees, D.C. of Sedan, Kansas. A complete treatment procedure using his methods is described in sacro occipital technique (SOT).[9] Points along the temporal sphenoidal line relate with specific organ dysfunction. Goodheart[30] found that the points described in SOT for organ dysfunction correlated with his muscle-organ/gland associations. For example, if the point described in SOT for the stomach was active, the pectoralis major (clavicular division) muscle, associated with the stomach in AK, was quite often weak. This association was made relatively early in applied kinesiology. When therapy localization (page 37) was discovered, it enhanced the use of the TS line as a primary investigating tool to find muscle dysfunction.[32]

The correlation of the temporal sphenoidal line with muscle-organ/gland dysfunction is described here because it has been instrumental in developing greater understanding of functional disturbances and applied kinesiology's evaluation of them. Although there was a fairly consistent correlation of the muscle-organ/gland association in early applied kinesiology, there were many enigmatic findings. For example, an individual might have obvious peptic ulcers and an active stomach TS line point, yet the pectoralis major (clavicular division) muscle associated with the stomach might test normal.

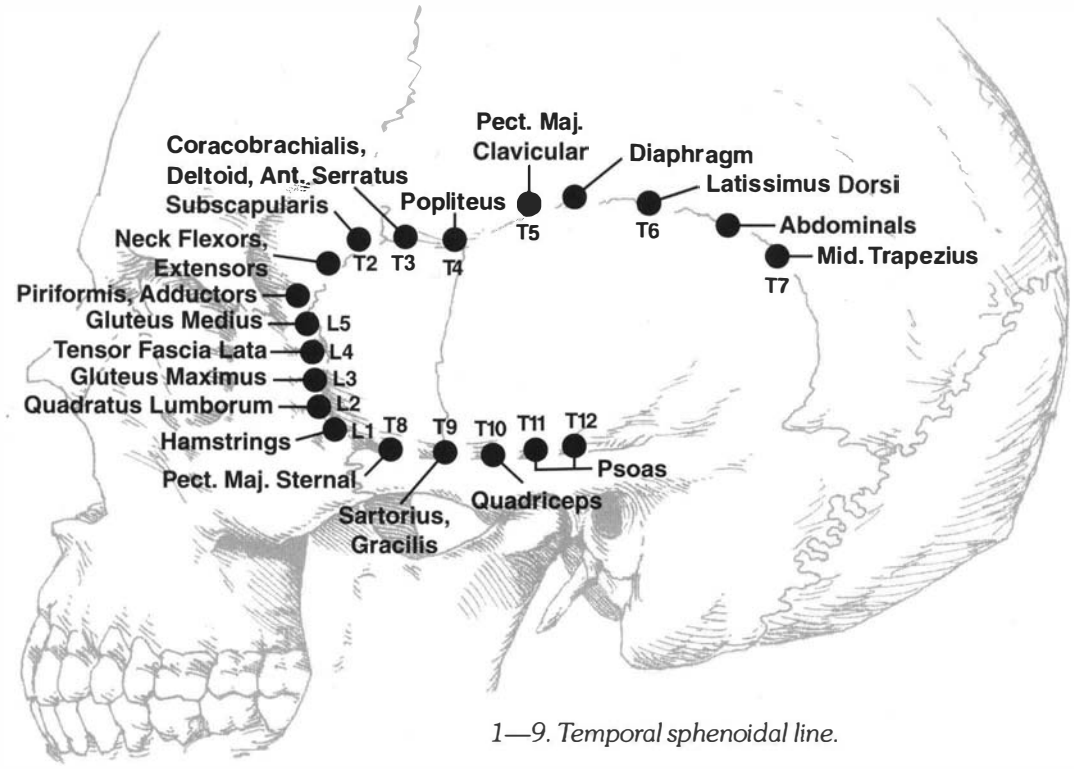
When a muscle tests weak with nothing being done to influence the test, it is called "weak in the clear." There are many AK examination techniques that, when applied, cause a strong muscle to test weak. If the muscle is not specifically associated with what is being tested with some AK examination technique, it is called an "indicator muscle." If the muscle is related with what is being tested and it is weakened by AK examination, it is a "subclinical condition."

Persistence in investigating an apparent lack of correlation finds that there is muscle association; however, it may be a hypertonic muscle or a subclinical involvement. A subclinical involvement is one in which body efforts to maintain homeostasis obtain enough functional balance for the muscle to test normal. Various methods have been developed in applied kinesiology to uncover hidden disturbance. Examples of this are the muscle stretch reaction in which a muscle fails to function properly after it has been stretched, and the strain/counterstrain technique wherein the muscle fails to function properly after it has been maximally contracted; yet in each case the muscle tests strong in the clear.

Active TS line points have primarily been used for diagnostic purposes. Recently they have been found effective in applied kinesiology as treatment points in the reticular activating system technique.

Examination

The TS line begins just anterior to the external acoustic meatus along the upper border of the zygomatic process. It progresses anteriorly along the superior surface of the zygomatic process to the temporal bone, then turns superiorly and courses along the temporal border of the zygomatic bone. Upon reaching the level of the frontal bone, it continues posteriorly along the superior edge of the great wing of the sphenoid and the temporoparietal



1—9. Temporal sphenoidal line.

suture, to approximately 1" behind the external auditory meatus. The diagnostic points distributed along the line are approximately 1/4" apart along the lower horizontal line and the vertical line. The upper horizontal line has points from 5/16" to 3/8" apart.

In addition to the organ or gland association, most points are associated with a vertebral level that indicates a possible subluxation in that vicinity.

An active TS line point is a nodular, tender area that shows positive therapy localization. The tenderness and positive therapy localization tend to spread out from the point, basically in the direction of the temporalis muscle fibers. An active point feels somewhat like a flattened BB under one or two slices of raw bacon. When first learning to use the TS line, confirm points located by palpation with tenderness on digital pressure and by positive therapy localization. Tenderness along the TS line does not necessarily mean there is an active point at that area; many times when cranial faults are present, the sutures where the TS line is located will be tender but will not actually have active points.

Palpation of the TS line is started anterior to the ear, palpating along the full length of the line. Palpation is done with a very light touch. It helps to use a somewhat rotary motion, sliding the integument over the TS line to improve sensation.

When appropriate treatment is administered to the associated muscle's five factors of the IVF, the positive TS line point will disappear. The more effective the treatment, the more quickly the nodule will disappear. If the nodule does not go away or it returns, all factors involved with the dysfunction have not been located and/or treated successfully.

Not all problems diagnosed in applied kinesiology will be revealed on the TS line. A muscle malfunctioning because its proprioceptors have been injured will not show on the TS line unless the condition persists long enough to cause more central problems to develop.

It is not known exactly why TS line points are present. Some associate them with cranial faults. This is supported by a respiratory quality to the positive TS line point. If an indicator muscle weakens when the point is therapy localized and is then strengthened with a phase of respiration, a cranial fault will usually be found. When it is corrected, the positive TS line point is often eliminated.

Duffy[16] considers the positive points to be trigger points in the temporalis muscle, which are secondary to postural distortions. He explains that the mandible changes position with the postural distortion. The mandible is suspended in position by the mandibular sling, which is made up by most of the muscles of mastication. Important among these is the temporalis muscle. Duffy's hypothesis is supported by analyzing postural distortion and then changing it to relieve the strained postural muscles. For example, in applied kinesiology a posterior ilium is associated with a weak sartorius muscle. When the TS line point for the sartorius is active, one can use DeJarnette blocks under the supine patient's pelvis — blocking toward correction. When properly done, the tenderness of the sartorius TS line point is immediately relieved.

Mastering the TS line requires persistent effort. When this is done, the applied kinesiologist has an excellent tool for more rapidly finding the problem areas; it also helps uncover hidden problems to which the body has adapted.

Hologram Model of Nervous System and Memory

As chiropractic continues to develop, some of the early hypotheses of why chiropractic works have changed. As knowledge expands, we are better able to apply therapeutic corrections with optimal effectiveness. Most of applied kinesiology has been developed in a clinical setting. The observation of body language, how the body reacts to different tests, and the subjective and objective improvement in health have been major factors in the development of AK. There is a constant effort to understand the mechanisms of examination and treatment methods and to apply current knowledge about physiology, anatomy, and the other sciences. There are some procedures that the current database of knowledge simply cannot adequately explain, or the appropriate association has not yet been made.

The hologram model of the nervous system and memory is an example of increasing knowledge that promises to improve our understanding of how some of the AK procedures work. Of primary importance is the attempt to explain the principles at work, i.e., develop alternate hypotheses and test them. As the hypotheses are tested, the strong will survive. Only by this method can a solid foundation be extended in this rapidly developing clinical science.

Understanding how the nervous system functions is an excellent example of the broadening scientific concepts. Even with the increased knowledge of recent years, much of the nervous system's function is yet to be discovered. One is reminded of the title of Restak's book, *The Brain: The Last Frontier*.[87] Hubel, in an article written for the public,[49] explains why brain research is slow, and he gives an overview of the extensive research that has been done to understand the brain's function. It has only been in recent years that man's knowledge has snowballed at incredible rates. In 1510, Copernicus pointed out that the earth revolves on its axis daily and around

the sun annually, with profound effects on succeeding scientific endeavors. Galileo used the newly developed telescope and mathematics to support the Copernican theory, for which the church condemned him in 1616 and again in 1633 as being "...vehemently suspected of heresy." Darwin, in 1856, demonstrated that man is related to all other living organisms. Einstein dramatically changed the path of research with his introduction of new concepts of time and space, mass and energy. Watson and Crick formulated the double helix molecular model of DNA to explain biological inheritance in physical and chemical terms. The last frontier of this scientific progression is improved understanding of what made all these discoveries possible: the brain, its nervous system, and memory.

Hubel[49] gives an overview of the extensive research conducted to understand the brain's function. He points out that the speed of research accelerated toward the end of the 19th century, and new techniques were developed during and after World War II that caused the study of neurobiology to become one of the most active branches in all science. Even with all this activity, brain research is just beginning.

Most neurobiologic research has been directed toward understanding the neuronal pathways, mapping activities of the brain,[74] and understanding transmission. The difficulty in doing this research can be appreciated when one considers there are approximately one hundred billion nerve cells in man's three-pound brain. The neurons and their adjacent branches are so intertwined and dense that the unstained tissue, when viewed through an optical microscope, is only a dense and useless smear.

Although there has been a great effort in mapping and understanding the connections of structures within the brain, it is quite a different thing from understanding the structure's physiology. Most of the understanding is at the receptor or input area of the nervous system and at the output section, such as motor neurons for muscle control. Far less is known about the workings of regions in between, which make up most of the nervous system.[49]

There is a tendency to liken the central nervous system — especially the brain — to a modern-day computer.[119] A computer is a machine, nothing more.[49] A computer's memory is clean, crisp, clear, and linear; the human nervous system is not. Pietsch[83] states, "Brains and computers operate on fundamentally different principles, and they mimic each other only when the task is trivial." The computer deals with input, the central processing unit (CPU), and output. Very simply stated, input to a computer is from a device that gives it information, such as typing at the keyboard. The information goes to the CPU, which is analogous to the sensory nervous system sending information to the central nervous system. Output is the movement of information that has been processed or stored in the computer so it can be viewed on a device, such as a monitor, or printed. Motor activity provides an example of output by the nervous system activating muscles, organs, and glands.

The analogy of the nervous system to a computer is acceptable as long as one is cognizant that the nervous system, in reality, does not function in the clean, crisp, clear, linear manner of a computer. Gevins et al.[25] point out that the human brain, unlike a fixed programmed computer, dynamically "programs" areas of the brain in anticipation of the need to process certain types of information. It has the ability to modify its processing to take certain types of action, depending upon the need. More is being learned about the dense, complex action taking place between input and output. The findings are consistent with numerous areas of the central nervous system having to function together for the body to properly perform certain actions. Referring to the brain's dynamic anticipatory programs, they state, "When these preparatory sets are incomplete or incorrect, subsequent performance is likely to be inaccurate."

The most attractive model of the nervous system today is the hologram theory. It gives the basic principles upon which memory is established. The principles of the hologram theory appear to spread throughout the body, relating with input (the sensory system), central nervous system, and output (functions of the body), similar to the way Einstein demonstrated the same relative principle in both the very small atom and the universe at large.

Before discussing the hologram theory, let's look at the wide range of memory usage and, in some cases, the location of memory. It has been established that memory is present in the simplest form of organisms, from protozoa, paramecia, bacteria, and roaches, becoming more complex on up to man.[83]

Bacteria (salmonella typhimurium and Escherichia coli) have a rudimentary memory that enables them to direct migration in their environment toward increasing concentrations of an attractant and away from increasing concentrations of a repellent. Koshland[62] uses the term "bacterial memory" not as being the long-term memory in the order of higher species, but real and useful to a bacterium, the same as memory is to humans in their survival. The memory of a bacterium enables it to direct its movement in a direction that aids in its survival. The effective memory span is calculated to be approximately the time it takes for a bacterium to swim twenty to one hundred body lengths. This gives the ability to detect concentrations of one part in 10^2 to one part in 10^3. Pietsch[83] helps in visualizing this concentration by giving it the rough equivalence of distinguishing a teaspoon of Beaujolais in a bathtub of gin, a "...formidable analytic problem," as Koshland[62] states.

This evidence of bacterial rudimentary mind can be explained by the holographic theory, according to Pietsch.[83] An example of the holographic theory active in lower life is given by Adler and Tso,[1] who investigated the movement of bacteria when presented simultaneously with an attractant and a repellent. When the attractant was higher, the E. coli's flagella had counterclockwise rotation

to move toward the substance. When the repellent was stronger, the flagella moved in a clockwise direction. Pietsch[83] explains, "In terms of holographic theory in its simplest form, the two opposite reactions are 180°, or pi, out of phase. By shifting from random locomotion to movement relative to a stimulus, the organism would be shifting from random phase variations in its flagella to the equivalence of harmonic motion, as if from cacophony to melody." This shift in phase is a key factor of holographic theory, which will be explained later.

With establishing memory in lower life forms comes the question, "Can it be used to make decisions?" Bacteria determining what movement in the environment is necessary for survival show an example of intelligence. As life forms become more complex, learning and decision-making improve.

Eisenstein[18] discusses the early work of Day and Bently (1911) wherein they demonstrated that paramecia are able to learn and remember. They placed a paramecium in a capillary tube with a diameter less than the animal's length. It swam the length of the tube and then, reaching the end, could only turn around by getting stuck, wriggling, bobbing, and — finally by accident — reversing direction. After numerous repetitions of the course, the animal learned the moves necessary to make an efficient turn. Pietsch[83] discusses Gelber's further evidence of the learning ability of the paramecium by conditioning it to go for food with the same basic approach Pavlov used in conditioning dogs to salivate at the ring of a bell. Other evidence that the paramecium can learn is that it takes non-foods, such as cramine particles (a staining agent), but soon "learns" to stop this; the change in behavior persists for some days.[76]

Pietsch[83] observed the ability of a protozoan to learn and remember its home environment so that it could return when separated briefly from it. When the protozoan would swim back to its apparent "home" but miss it, it would take a few pokes at a foreign strand and then test various areas until it found its original location. The foreign strands were not rejected because they were uninhabitable, since other protozoa were happily living there.

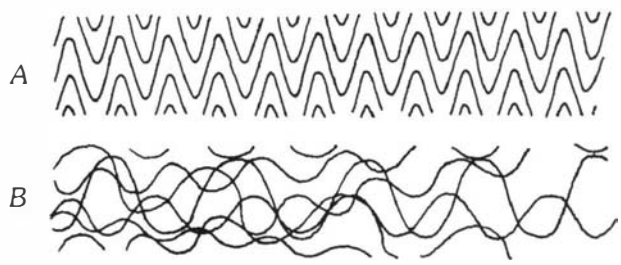
Many lower life forms have memory and decision-making ability without a brain, as such. We tend to think of higher life forms having memory and decision-making residing in the brain. To test this, Horridge,[47] in what has become known as the Horridge preparation, beheaded an insect suspended directly above a salt solution. With an electrical apparatus, the insect received a shock every time the leg relaxed and gravity pulled it down into the solution. The beheaded insect learned to keep the leg up by muscle contraction to avoid the shock. This experiment has been refined by Hoyle[48] making direct recordings from the specific nerves. He has found that "...debrained animals learned better than intact ones." After discussing the ability of decapitated insects to learn in the spinal cord and brainstem without the use of the brain, Pietsch states, "...the fact is that evidence of mind exists in some very strange places. Brain (in the sense of what we house inside our crania) is not a sine qua non condition of mind."

Various conditioning paradigms are applied to laboratory animals with portions of the brain removed to determine where learning takes place. The eye-blink reflex, conditioned with an auditory stimulus and a brief shock to the eyelid, has been demonstrated in a decerebrate cat in which the complete lower brainstem was transsectioned. The conditioning took place slower than in an intact animal, but it does demonstrate that decerebrate cats can learn the conditioned response.[78]

Finding specific memory locations has been a problem in neurobiology. Karl Lashley's experiments[64] showed the brain to be put together with exquisite anatomical precision, but they also showed that engrams and memory traces could not be localized. He found that a rat could continue to navigate a pre-learned maze with only insignificant errors when over 50% of its cortex was removed. The confusing aspect, in correlation with the anatomical precision of the brain, is that it did not matter which part of the cortex was removed, but how much of it. Karl Pribram, an associate of Lashley,[79] has suggested a model for brain and nervous system function based on the hologram.[85,86] The hologram model explains how the nervous system acts in order and pattern that is essential for life.

The hologram was discovered by Nobel laureate Dennis Gabor in 1947. Based on his principles, three-dimensional photography came into existence. The principles of hologram photography have been applied to many other aspects in science, providing a broader understanding and opening the door for many new discoveries. Gabor coined the word "hologram" from the Greek word "holos," meaning whole, to indicate that the hologram contains complete information about a wave. The term "holograph" means "written entirely in one's own hand."[112] Both hologram and holograph are used almost interchangeably in the hologram literature. Hologram will be used here except when quoting a source or discussing someone's work.

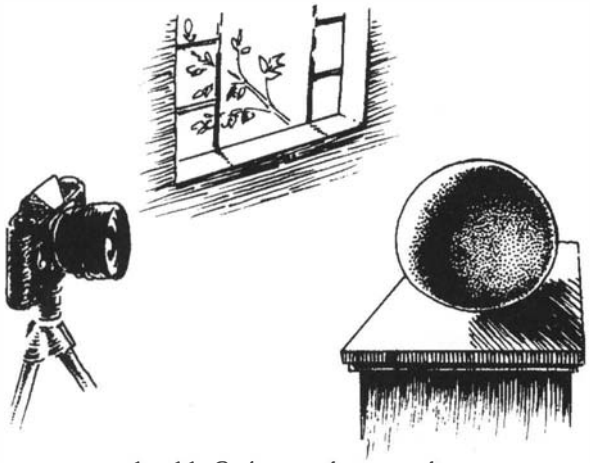
The hologram model of the brain and nervous system explains much that was previously enigmatic. A brief review of the discovery of the hologram, how a photographic hologram is made, and the hologram's general application in science and industry is appropriate here to better understand its application to the nervous system.



1—10. A – coherent, and B – incoherent light waves.

A light wave, as well as other types of waves, contains both intensity and phase. (figure 1-10) A standard photograph records only intensity and misses the phase, which carries the depth information.

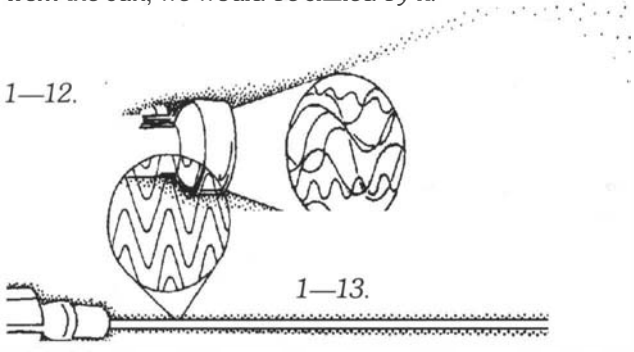
Standard photography is simple compared to holography. A regular camera uses a lens system to pick up light waves reflected off a subject. The waves are focused as an image, recording only the brightness and darkness of the reflected waves; thus only two dimensions are recorded because the important aspect of depth — the third dimension — is carried in the phase of the light waves, which is not recorded.[17]



1—11. Ordinary photography.

Standard photography records only the intensity of disturbed waves. It is a record made up of a mosaic of points of varying intensities. Westlake[114] states, "In some rough sense it can be said that the intensity (or amplitude squared) constitutes half of the information and that the phase differences constitute the 'other half' of the information. (In this sense normal photography should be re-named 'halfography.')"

Ordinary light — the light that makes a standard photograph — is incoherent. This type of light spreads as it travels from its source, becoming less intense with increasing distance. This is called the law of the square. Coherent light does not spread, and travels a great distance with little loss. Coherent light is a beam of photons that have the same frequency phase and direction. This does not occur anywhere in nature. If coherent light came from the sun, we would be sizzled by it.



A flashlight and a laser are examples of incoherent and coherent light waves, respectively.

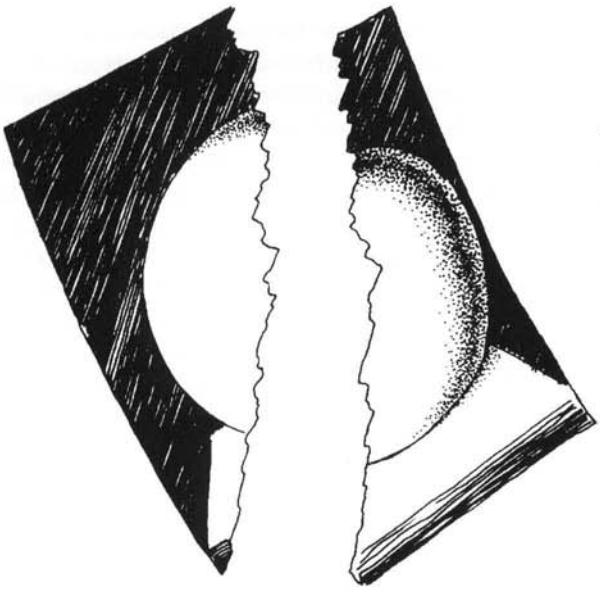
Gabor[24] acknowledges that before his own work Fritz Zemike had concluded that to record all the information — both the intensity and phase — of a wave, it would be necessary to record two beams from the same source. In photography, the beam directly reflected from the subject being photographed is called the object beam. It is this beam that carries the intensities that are recorded to make an ordinary photograph. The second beam is the reference beam, which is missed in ordinary photography. In Gabor's words, "The essential new step was the discovery of the *principle of reconstruction* which came to my mind one day at Easter, 1947." Since Gabor's area of interest was in improving the electron microscope to the point where individual atoms could be seen with it, it is ironic to read his report of holography twenty-five years after its discovery. "So for the time being one must admit the strange fact that holography was a success in all applications except in the one for which it was invented: for electron holography."[24] Since then Stroke, one of the current leaders in holography, has developed an electron microscope technique with which he has been able to take blurred, illegible electron micrographs and reconstruct them to view a virus resembling the double helix structure of the DNA molecule.[17]

When a standard photograph is torn in half, the picture separates; when each piece is observed, half of the picture is missing. In a hologram, since the entire information is recorded over the whole picture, tearing it in half produces two complete (only smaller) pictures of the subject. One can continue tearing the pieces of the picture apart and still have increased numbers of the complete picture; only the quality of the picture is reduced. The hologram model of the brain explains why Lashley[64] could cut out large portions of an animal's brain and have its learned tasks continue, regardless of the portion of brain removed. The important factor is how much of the brain is removed. The hologram model has redundancy of recording, but as quantity is removed, the quality is reduced; however, the total information is not.

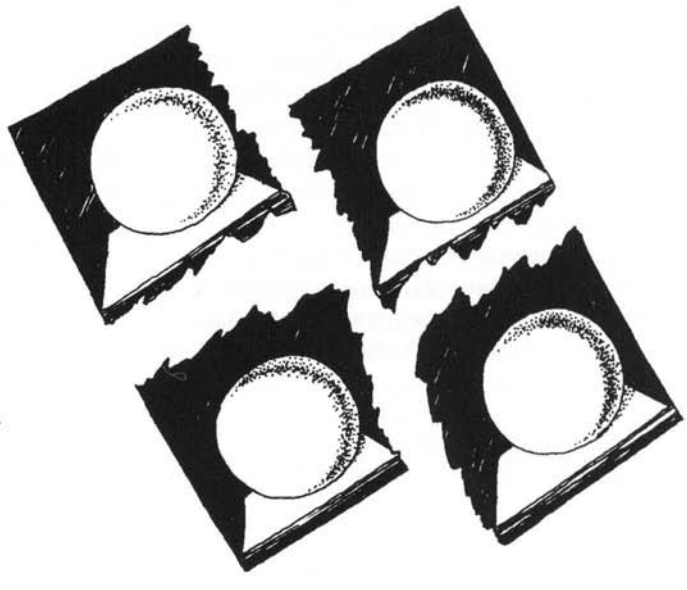
The source of coherent light is from a laser, which is an acronym for "Light Amplification by Stimulated Emission of Radiation." The first working laser was built in 1960. The advent of the laser opened the door for the continued development and improved understanding of the hologram.

In 1963 Leith and Upatnieks[69,70] made a major advancement in understanding the hologram by creating the first laser-produced hologram. They used special partially-coated mirrors to separate the laser light source into two beams. Diffusers were used to broaden the laser beam for larger holograms — an action that seemed to go against the requirement of a coherent light source. Regardless of the conventional reasons that it couldn't be done, they succeeded in bringing a new grasp to the undeveloped hologram.

A hologram is produced by two beams from the same source interacting. This is done by splitting a laser's



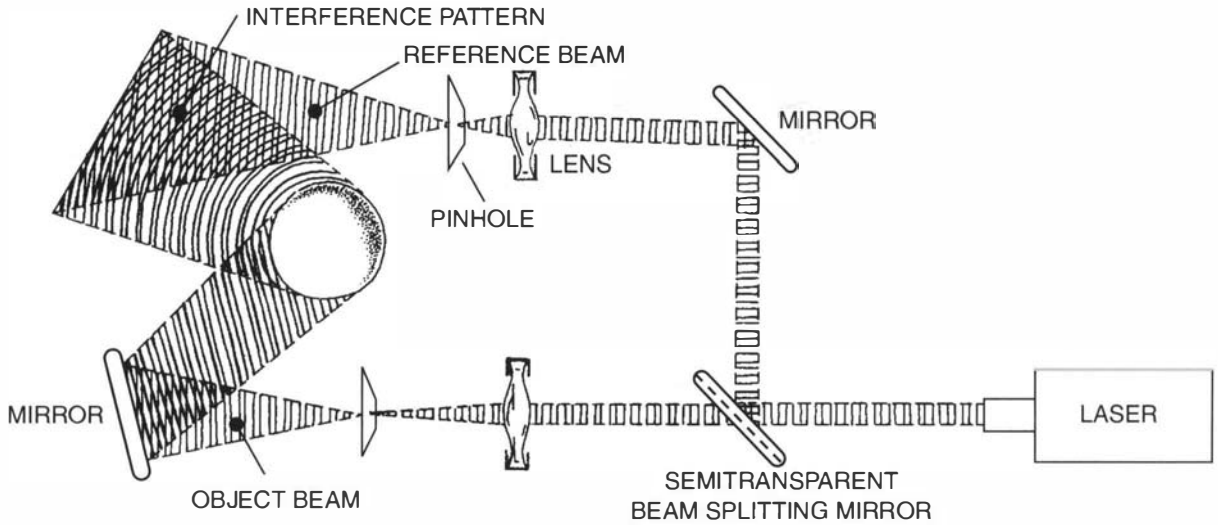
1—14. Tearing a standard photograph splits the image (information).



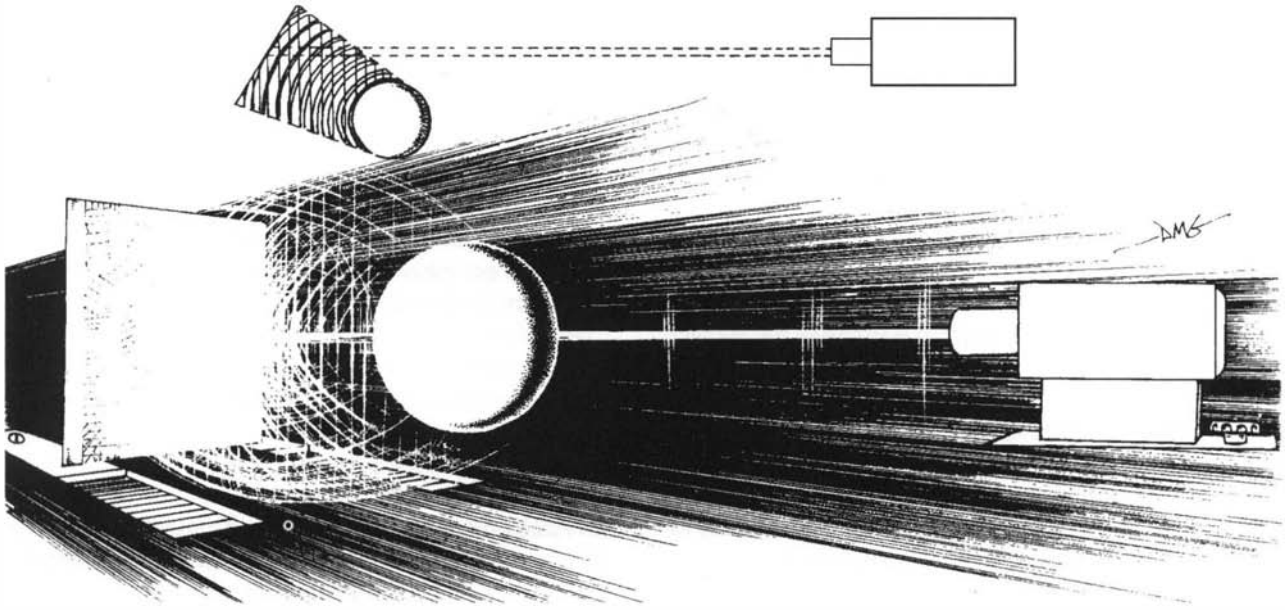
1—15. Tearing a hologram produces identical complete images (information), less only in quality.

coherent beam, with a portion of it passing through a semi-transparent mirror and the other portion being deflected to create the second beam. One beam travels to the subject and is called the object beam; the other beam is directed by way of mirrors to the film and is called the reference beam. When the object beam strikes the subject, it rebounds at various angles so the photons are no longer orderly or in phase. When the disturbed waves meet the regimented waves of the reference beam they interact, with some waves clashing and others reinforcing each other. This produces an interference pattern on the film, which is the "memory" of the hologram. When one looks at the picture, it is unrecognizable until it is reconstructed. This is done by illuminating it with the coherent waves of the laser. As the laser waves strike the interference pattern on the film they bend and alter, changing their intensity and phase to match those of the light waves that created the hologram. This creates a duplication of the out-of-phase light waves that originally came from the subject when it was struck by the object beam.

The image reconstructed from the hologram projects away from the storage medium (e.g., photographic film or plate) and appears to "hang" in thin air, presenting its three dimensions. The back, side, or front can be observed from different vantage points appearing as the original. Many individuals will reach out to touch a quality hologram, only to find a handful of thin air.



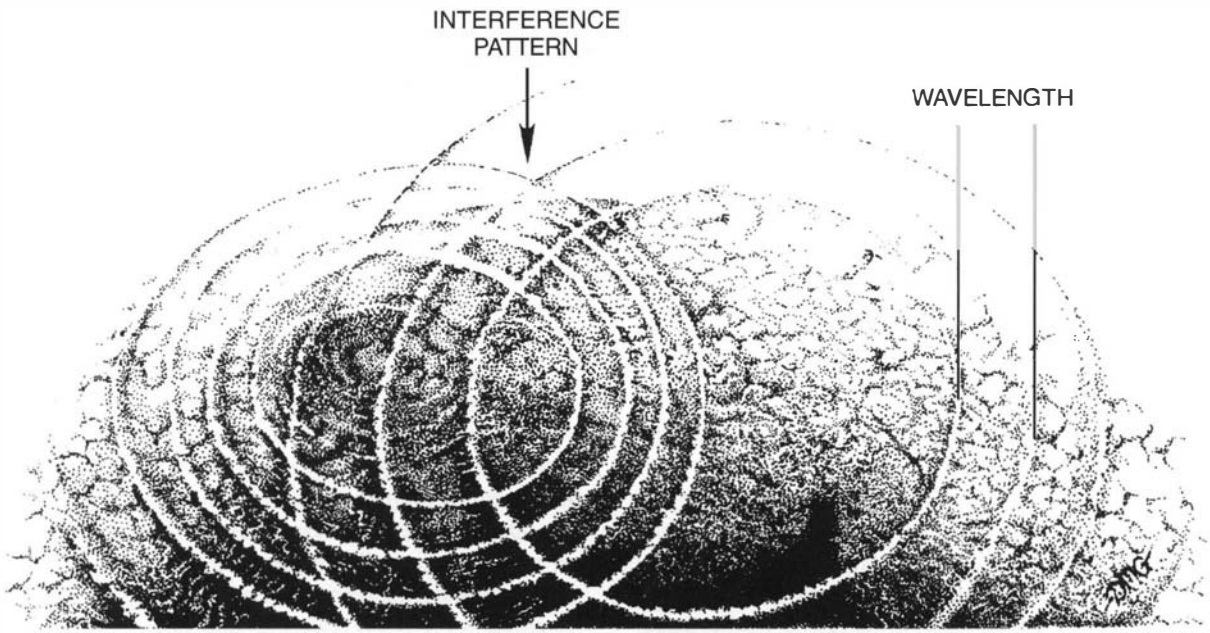
1—16.



1—17. The image is reconstructed by the coherent light striking the hologram at the same reference beam creation angle. The image appears where the object originally was.

The illustration of waves created when a rock is dropped into a still pool of water can be used to help understand waves and their interaction in a hologram. When waves from different sources collide, they interfere with each other. When the wave of one source is at its peak and the other at its lowest point, they cancel each other. This is similar to adding a positive and a negative figure together. If the waves meet when both are at their peak, there is an additive function creating a higher wave. An example is two rocks dropped into a pond to create the water waves that ultimately strike to form an interference pattern. The result is random because the rocks do not hit the water at exactly the same time; they are out of step, or incoherent.

Any spatial distribution of light can be analyzed into Fourier components, a form of calculus that transforms a complex pattern into its component sine waves. The super position of the fringe sets (called optical Fourier synthesis) is the image and records all the information of the original waves. In the Fourier-transformed record each



1—18. Two rocks dropped into a pool of water create waves which, when they meet, create an incoherent interference pattern.

point represents the amount of energy present in a waveform component of the entire array of light reflected from the object.[86]

The principles of holography are widely used in scientific endeavors, including physics, astronomy, medicine, and biology. The accidental discovery of "holographic interferometry" gives the ability to evaluate mechanical structural properties, such as in an automobile tire or airplane wing, in order to study the design of construction.[100] After a hologram is made of the subject to be studied, the material is stressed and a repeat hologram is made. If the stress causes structural change, the two holograms will not match and Newton rings are observed. The method measures deformations down to about 1/3,000 mm. For routine use there are methods to "desensitize" the measurement, reducing the accuracy to more practical levels.

Holography has come a long way since Gabor's discovery. At that time it was on a very small scale. The objects were microphotographs of 1 mm diameter. The holograms were about 1 cm.[24] Even with the great advancement of holograms since 1947, many experts feel the current hologram status is comparable to that of traditional photography in the mid-19th century. It is understood what holography is and how it works, but to use the system, improved lighting techniques to produce larger holograms at lower cost are necessary for continued advancement in its photographic use. A similar statement can be made for hologram principles being applied to biology.

Hologram principles, even in this immature state of development, have opened the door for many discoveries in the healing arts. One most notable discovery was by Allen Cormack and Godfrey Hounsfield, who shared the 1979 Nobel Prize for physiology or medicine for the development of the computerized axiotomography (CAT) scanner.[95] The reconstruction of x-ray images by this means of computer tomography is an extension of the principles of holography to x-ray images.

An even greater contribution to the healing arts appears to be in a better understanding of how the nervous system functions. We have looked at the hologram principle in photography to enable a better understanding of the hologram model of the nervous system. "In doing this, however, it must constantly be remembered that it is the mathematics of holography and brain function that needs to be compared and tested, not the optical or computer instantiations of holography."[86]

Pribram[28] recognized from the research of others that there is a common denominator in nervous system function, learning skills, and memory. The common denominator can be demonstrated by a Fourier analysis of neurologic wave forms. Whether one is dealing with the auditory, olfactory, gustatory, somatosensory, somatomotor, or visual system, all relate to the same mathematical principle that Gabor used to invent the hologram. Pietsch[83] states that "...relative phase is the birthmark of all holograms and thus the central issue in holographic theory."

The optical hologram helps in understanding the principle, but we must remember that it is the mathematics of holography applied to the nervous system that is important. There is considerable interconnection of the neurons in the neuronal nets, especially in the cortical regions. Each neuron has excitatory (positive) as well as inhibitory (negative) synapses. In neurophysiology, the "wave fronts" on the impulse level can be said to be basically coherent.[114] The coherent waves are capable of interfering constructively and destructively with each other. In the opinion of Mager et al.,[71] "...there is no necessity to have a reference wave in order to establish a holographic model of neuronal information storage. A coherent field, recorded on a suitable medium, can be reconstructed by a part of itself, as was shown theoretically at first by van Heerden."[105]

Biohholography is the application of hologram principles in nature.[38] These principles have been demonstrated mathematically in vision,[84,104] sound transmission,[4] and in the somatosensory,[105] somatomotor, and gustatory[109] systems.

E. H. Land,[63] the Polaroid developer, demonstrated that one area in the visual receptive field can create illusions in the perception of entirely different areas in the same receptive field, giving rise to color effects. This and other visual illusions are explained by the hologram.

The interaction of the nervous system at two locations appears to function like a hologram. Von Bekesy[108] studied the perception of "pitch" on the skin to relate to the function of hearing. He stimulated the skin on each forearm with a set of five vibrators to simulate the cochlea. When the phase of the vibration was properly adjusted, the source point of stimulation seemed to jump from arm to arm and then stabilize in the space just forward and between the two arms. As a hologram, this stimulus was "projected" away from the source, to hang there similar to the optical hologram.

A major breakthrough toward generalization of the holographic concept was made in 1964 when the first acoustic holograms were recorded, making evident that the holographic principle is also valid when the information-carrying waves are of non-electromagnetic origin.[38]

A particularly interesting hologram model is the way various creatures use echolocation to hunt and navigate. Among these are bats, whales, porpoises, and certain birds. These animals emit ultrasonic waves which, when reflected back, are interpreted to determine the environment. A bat can fly at high speed between fine wires and locate a tiny morsel of food far in front of it.[14] Until recently it has been an enigma that there can be thousands of bats in a cave, all emitting their ultrasonic impulse for navigation, yet each one seems to "own" its own impulses because there is no colliding with one another, or any apparent mixing of impulses. The answer comes from the hologram model. Greguss[38] reports that "...when a bat

emits an ultrasonic impulse, it also sends a stimulus to that part of its brain where the information carried by the wave reflected by the object is decoded and analyzed." The decoding is an interference pattern of the ultrasonic impulses that were sent out to bounce back, mixed with the reference background recorded in the brain. Because the brain record is kept within the bat, there is no other bat that will have a matching reference for the hologram, making each bat's signals its own. Similar observations have been made of other animals using echolocation.

There is much evidence to indicate that the holographic brain model is accurate. Dolgoff[14] gives an excellent overview of support for the model. To keep the model in perspective he states, "This analysis does not mean to imply that all brain and neural function can be reduced to a holographic process, but that certain processes are most accurately describable by analogy to specific, well-understood, holographic-related processes." An example of processing by two methods is in the auditory system, which functions both holoramically and non-holoramically.[4]

The computational and storage nature of the three-dimensional capability of an optical hologram adds another perspective to the neurologic model. The application of holography makes use of the associative storage of two light waves, A and B. A may be extracted from the hologram by illumination of the hologram with wave B. Stroke[100] states, "Perhaps most remarkable in its associative principle is the fact that wave B which may originate from a single point is by itself sufficient to produce wave A, which in turn may consist of millions of different picture points (i.e. of tens of millions of data 'bits'). This situation is somewhat similar to the recall of an entire book of thousands of pictures, each with hundreds of words, with the aid of a simple 'title' that perhaps consists of only a single word; say, 'Bible.' "

The massive storage capacity present in the hologram model helps explain the storage capacity of the brain. "Stimulus equivalence" is the ability to see an object as the same object, regardless of its position and apparent size. This is when we can recognize an individual's face, regardless of distance and orientation. If we had to have a separate memory for every size and position in which the face is viewed, it would take tremendous amounts of memory. Several have "...shown that although visual presentation of different shapes evokes different brain potential patterns, presentation of objects of the same shape but different size evokes similar patterns."[14]

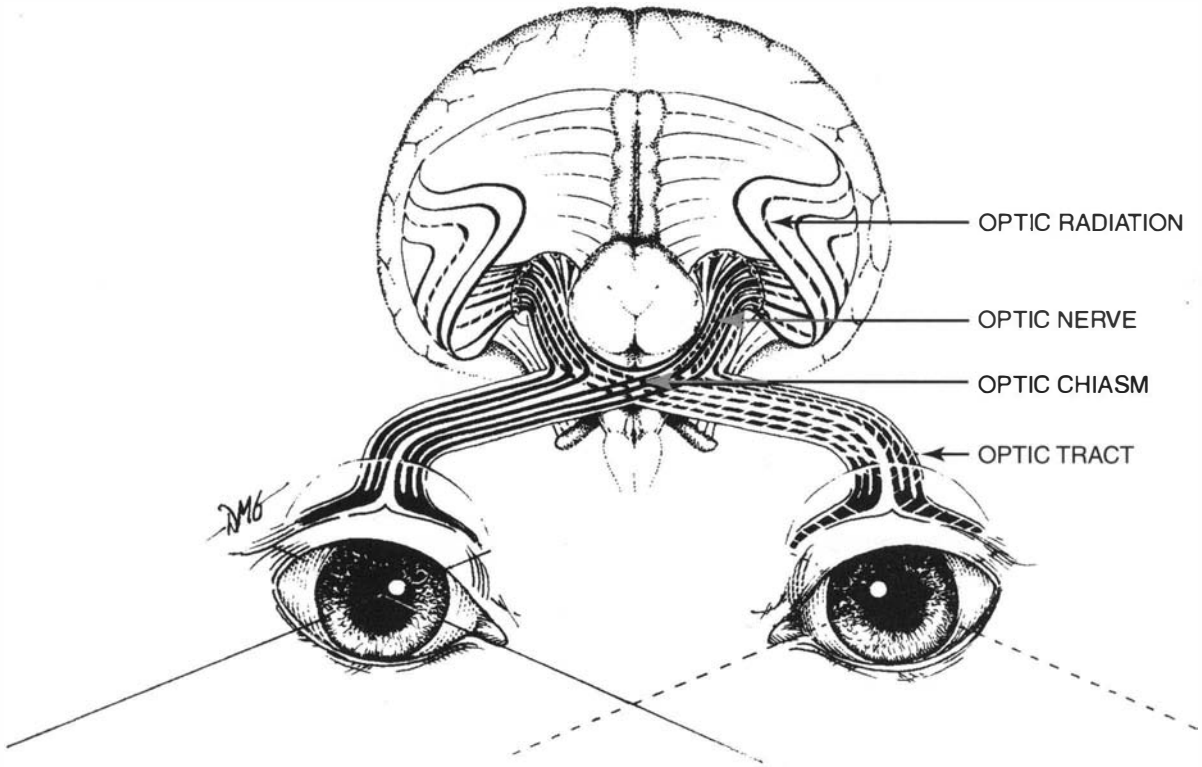
The brain appears to use the hologram process for massive storage. It is the interference patterns that have the facility for storing and distributing large amounts of information.[85] Many photographic holograms can be recorded on the same film or other media simply by changing the angle at which the laser beam hits the media, or by altering the wave length of the beam. The various images can be reconstructed by changing the laser beam to match that which was used in the initial recording. This gives the ability to record large quantities of data in very small areas. It is estimated that the information contained in a 50,000 volume library can be contained in a crystal cube only 1 cm in size. Moreover, any portion of that information can be delivered in twenty millionths of a second.[17] Pietsch[83] wonders if an individual who is trying to remember something is searching for the correct reconstruction angle for the hologram.

The wide range of material one perceives cannot be processed by feature detection and analysis by neural units and logic alone. Neural activity has junctional patterns with interactions that can display image-forming properties akin to those of optical information processing systems — properties of holograms.[85]

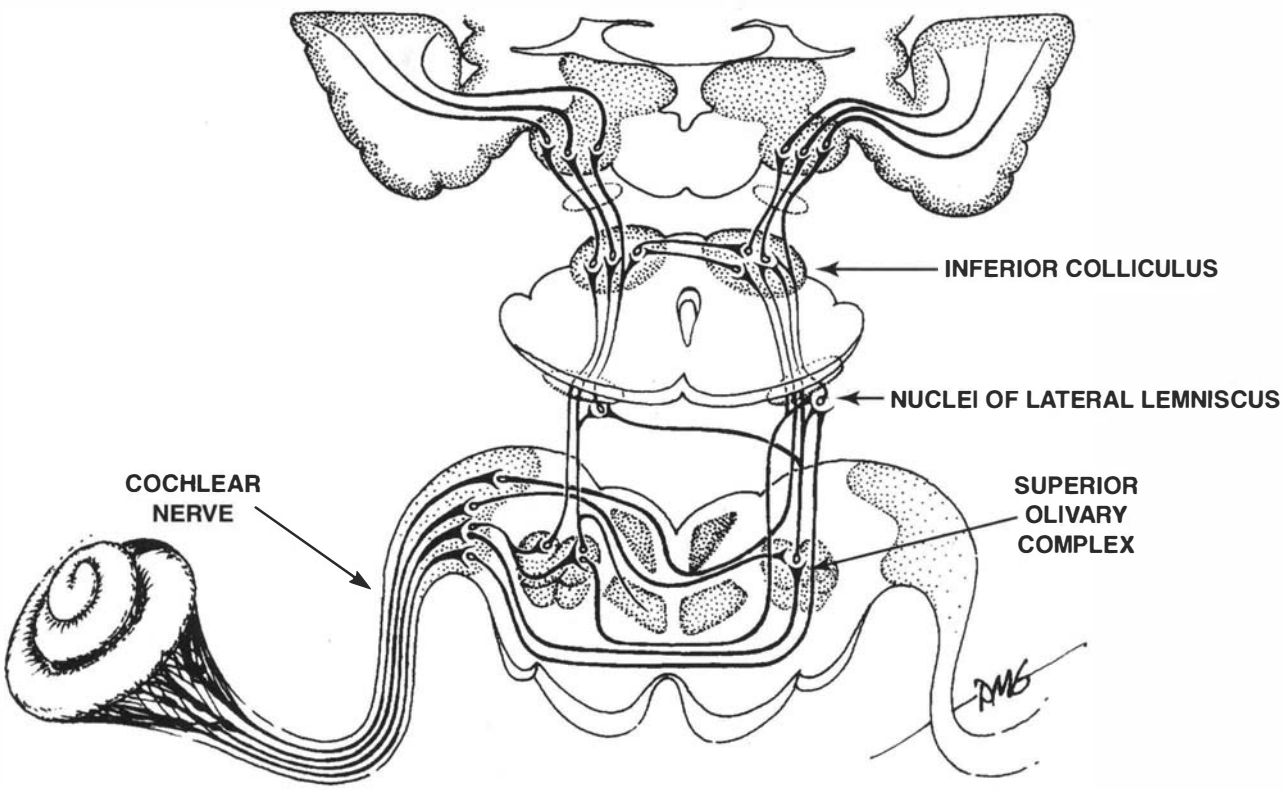
Does the holoramic theory reduce the nervous system to a unified function throughout? Keeping in mind Dolgoff's statement, "This analysis does not mean to imply that all brain and neural function can be reduced to a holographic process, but that certain processes are most accurately described by analogy to specific, well-understood holographic-related processes," we can consider that to some extent the holoramic model (interaction of waves) occurs throughout the body. We have seen how there is memory and learning capability in decerebrate animals, and memory in bacteria and other lower animals not possessing a brain. Hologram principles are applicable to light and vibration, and probably anything that produces waves. Maybe the holoramic theory even explains parapsychological phenomenon. Pribram[28] states, "The brain can instantly resonate to and thus 'recognize' wave forms. Once 'recognized' the inverse transform allows them to be implemented in behavior. We apparently need to get on the same wavelength — literally — before we can understand each other." Why have people long before the discovery of holograms said, "I get good vibes from him," or, "I don't understand you; we are simply not on the same wavelength."

With all the research that has been done on the nervous system, there are many unanswered questions. Goodheart often repeats, "Why is that?" This writer has never seen an explanation of why the right brain basically controls the left body and visa versa. Why is that? Sprieser[99] questions whether if we were to design the nervous system we would cross-wire it as it is. When considering the nervous system as a type of connected electrical or other transmission system, it appears that the design is a waste of energy, materials, and time. Sprieser wonders if the design might be for improved function.

The crossing of the nervous system may fit in with the holoramic memory concept. Vision fits this concept with the visual fields from both eyes being split. This is compared with the half-silvered mirror used as a beam splitter in making a hologram. The lateral visual field from both eyes goes directly to the ipsilateral brain, which can be likened to the reference beam. The medial visual field, by way of the optic chiasm, goes to the contralateral brain



1—19. Schematic illustration of the optic tract splitting to cross over at the optic chiasm; both eyes are then represented in the optic nerves.



1—20. Simplified illustration of the central auditory pathways showing the crossover fibers.

to form the object beam; thus the lateral visual field and the contralateral medial visual field join on the same side of the brain, perhaps forming the interference pattern needed to create a hologram.

The auditory system has similar nerve connections. A portion of the fibers goes to the ipsilateral brain and the rest to the contralateral brain, again forming an object beam and a reference beam. The separate inputs of a single sound source may join to form the interference pattern, giving the holographic representation.

Applying the holographic theory to the nervous system seems to explain the reason for the crossover design with some of the fibers remaining ipsilateral. This allows the reference and object beams to develop the interference pattern necessary for a hologram.

The holographic model of the nervous system appears to relate to much sensory and motor function throughout the body. What other reason explains the tracts crossing over to the opposite side of the body, with some tracts remaining ipsilateral throughout the body?

Another promising hypothesis put forth by Sprieser[98] is that the hologram model may be responsible for therapy localization, which is clinically observed in applied kinesiology. When one considers the evidence of the holographic model in the somatosensory and somatomotor systems, it seems that this may provide an understanding of this poorly understood but valuable clinical tool. Greguss[38] states, "The results of recent studies in chemical oscillations and oscillatory cellular dynamics strongly indicate that the holographic concept may exist not only at the neural, but also at the cellular, or even molecular, level." In addition, he states, "According to our model an organism is living and stays alive as long as it can handle — process — *all* the information patterns it receives independently of their form and origin." When there is lack of coherency of a given information pattern, the organism experiences pain or awareness. Goodheart[35] proposes that there is a perfect holographic image in the brain of all aspects of the body; if local holographic images do not match because of trauma or some other reason, pain or awareness develops. This may be likened to holographic interferometry where the two holograms do not match and Newton rings develop. If there is lack of coherency of the elbow image with the brain image there may be pain in the elbow, loss of strength, adaptation, or some other evidence of the dysfunction.

It seems probable that the interaction and quantity of information being processed is only now beginning to be understood. The brain functions in terms of interaction within and between masses of neurons.[5] The hologram model may give an insight into how the brain associates with the body in addition to the anatomical pathways. Thatcher,[103] in analyzing a hologram study on the retrieval of information, states, "...it would seem that holographic models must deal with constellations of holograms, coupled but distributed in space and confronting, specifically, an evolving space-time structure." Even with this mass of neurologic activity, the hologram model appears to simplify the nervous system's action. "One of the elegant things about the holographic domain is that memory storage is fantastically great. Storage is also simpler because all that is needed is to store a few rules rather than vast amounts of detail." With the concept of holographic nervous system function, Goodheart[35,36] has applied therapy localization to an interosseous vertebral subluxation and other factors. The effects of treatment based on the examination findings have proven clinically effective in improving various health problems. These applications are discussed elsewhere in this book.

To summarize, there are several factors of the hologram that are important in the neurologic analogy. Any portion of the hologram can re-create the original image on a smaller scale. This relates with Lashley's inability to remove an image by cutting out sections of the brain. The interference pattern that forms the hologram can be made from any form of energy outside the living organism. This is shown, among others, by light, sound, and electrical impulses. The reconstruction process is analogous to optical Fourier synthesis. In some area the waves undergo Fourier transformation. The signals that form a hologram can be superimposed on each other, and each can be reconstructed separately with each image's unique reference signal.[14] Pietsch states that answers to questions of the mind "...will not take the form of *the* physiological mechanism, *the* chemical reaction, *the* molecule, or *the* cellular response. Holographic theory denies the assumption implicit in questions that demand the answer as bits of brain." In addition, "...holographic theory provides a unified view of the subjective cosmos."[83]

CHAPTER 1 — REFERENCES

1. Adler, J., & W. Tso, "'Decision'"-making in bacteria: Chemotatic response of escherichia coli to conflicting stimuli," *Science*, Vol 184 (Jun 21, 1974).
2. American Diabetes Assoc, Endocrine Soc, & AMA, "Statement on hypoglycemia," *Metabolism*, Vol 22, No 3 (Mar 1973).
3. Barlow, D.H., S.C. Hayes, & R. Nelson, *The Scientist Practitioner — Research and Accountability in Clinical and Educational Settings* (New York: Pergamon Press, 1984).
4. Barrett, T.W., "The concept of holography in the auditory pathway," *in Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
5. Bartlett, F., "Quantification of neural processes reflecting readin and readout from memory," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
6. Bennett, Terence J., compiled by Sylvia I. Weigandt, *Neurological Reflex Technique* (Clinton, IA: privately published, 1956).
7. Burdinie, C., "The reactivity of muscle strength to visceral stimulation and neurolymphatic point pressure." Independent research, unpublished, U IL (Jun 1982).
8. Carpenter, S.A., J. Hoffman, & R. Mendel, "An investigation into the effect of organ irritation on muscle strength and spinal

mobility." Thesis, Anglo-European Coll Chiro, Bournemouth, England (1977).
9. Choiniere, R.J., ed, "Temporal sphenoid technique," *S.O.R.S.I. Dispatcher*, Vol 3, No 6 (Sep 1967).
10. Cleave, T.L., "The neglect of natural principles in current medical practice," *J Royal Naval Med Serv*, Vol XLII, No 2 (Spring 1956).
11. Cole, K.C., "Is there such a thing as scientific objectivity?" *Discover*, Vol 6, No 9 (Sep 1985).
12. Collin, H.B., "Research in chiropractic," *J Manip Physiol Ther*, Vol 3, No 1 (Mar 1980).
13. Corneal, J.M., & R. Dick, "An attempt to quantify muscle testing using meridian therapy/acupuncture techniques." Proceedings of Winter Meeting, ICAK, Palm Desert, CA, 1987.
14. Dolgoff, E.J., "A holographic brain model," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Ltd, 1975).
15. Douglass, W.C., "A call for research," *Today's Chiro*, Vol 16, No 3 (Jul/Aug 1987).
16. Duffy, D.H., "On the production of temporosphenoidal line indicators." Proceedings of Summer Meeting, ICAK, Detroit, 1979.
17. Editors, Time-Life Books, "Frontiers of photography," *Time-Life Books*, New York (1972).
18. Eisenstein, E.M., "The use of invertebrate systems for studies on the bases of learning and memory," in *The Neurosciences*, ed G.C. Quarton, T. Melnechuk, & F.O. Schmitt (New York: Rockefeller Univ Press, 1967).
19. Ellison, J.S., "The null hypothesis." Proceedings of Winter Meeting, ICAK, Acapulco, Mexico, 1981.
20. Ellison, J.S., "The use of the chi square test as an aid in clinical research." Proceedings of Summer Meeting, ICAK, Detroit, 1982.
21. Ellison, J.S., "ANOVA (analysis of variance)." Proceedings of Summer Meeting, ICAK, Detroit, 1984.
22. Esposito, V., G. Leisman, & Y. Frankenthal, "Neuromuscular effects of temporomandibular joint dysfunction," *Intern J Neuroscience*, Vol 68 (1993).
23. Friedman, M.H., & J. Weisberg, "Applied kinesiology — double-blind pilot study," *J Prosthet Dent*, Vol 45, No 3 (Mar 1981).
24. Gabor, D., "Twenty-five years of holography," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
25. Gevins, A.S. et al., "Human neuroelectric patterns predict performance accuracy," *Science*, Vol 235 (Jan 30, 1987).
26. Gin, R.H., & B.N. Green, "George Goodheart, Jr., D.C., and a history of applied kinesiology," *J Manip Physiol Ther*, Vol 20, No 5 (Jun 1997).
27. Goldstein, M., "Research in spinal manipulation — Reflections on processes, priorities and responsibility," *J Manip Physiol Ther*, Vol 3, No 1 (Mar 1980).
28. Goleman, D., "Holographic memory — interview with Karl Pribram," *Psych Today*, Vol 12, No 9 (Feb 1979).
29. Goodheart, G.J., Jr., *Applied Kinesiology* (Detroit: privately published, 1964).
30. Goodheart, G.J., Jr., *The Cranial Sacral and Nutritional Reflexes and Their Relationship to Muscle Balancing* (Detroit: privately published, 1968).
31. Goodheart, G.J., Jr., "Innate and the interdependent triangularity of man," *Chiro Econ*, Vol 16, No 4 (Jan/Feb 1974).
32. Goodheart, G.J., Jr., *Applied Kinesiology 1975 Workshop Procedure Manual*, 11th ed (Detroit: privately published, 1975).
33. Goodheart, G.J., Jr., *Applied Kinesiology 1977 Workshop Procedure Manual*, 13th ed (Detroit: privately published, 1977).
34. Goodheart, G.J., Jr., *Applied Kinesiology 1978 Workshop Procedure Manual*, 14th ed (Detroit: privately published, 1978).
35. Goodheart, G.J., Jr., "The interosseous vertebral holographic subluxation, Part I," *Chiro Econ*, Vol 29, No 2 (Sep/Oct 1986).
36. Goodheart, G.J., Jr., "The interosseous vertebral holographic subluxation, Part II," *ChiroEcon*, Vol 29, No 3 (Nov/Dec 1986).
37. Gowitzke, B.A., & M. Milner, *Understanding the Scientific Bases of Human Movement*, 2nd ed (Baltimore: Williams & Wilkins, 1980).
38. Greguss, P., "Holographic concept in nature," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
39. Grossi, J.A., "Effects of an AK technique on quadriceps femoris muscle isometric strength," *Phys Ther*, Vol 61, No 7 (Jul 1981).
40. Guyton, A.C. & J.E. Hall, *Textbook of Medical Physiology*, 9th ed (Philadelphia: W.B. Saunders Co, 1996).
41. Harper, W.D., *Anything Can Cause Anything*, 3rd ed (Seabrook, TX: privately published, 1974).
42. Harrower, H.R., *Practical Organotherapy — The Internal Secretions in General Practice*, 3rd ed (Glendale, CA: The Harrower Laboratory, 1922).
43. Hildebrandt, R.W., "The science of chiropractic." Palmer Coll Chiro, Davenport, IA (1967).
44. Hildebrandt, R.W., "Science, medicine and chiropractic theory," *J Manip Physiol Ther*, Vol 9, No 3 (Sep 1986).
45. Homewood, A.E., *The Neurodynamics of the Vertebral Subluxation* (Toronto: 1962).
46. Horn, M., "The mysterious genius of Leonardo," *U.S. News & World Report* (May 25, 1987).
47. Horridge, G.A., "Learning of leg position by headless insects," *Nature*, Vol 193, No 4816 (Feb 17, 1962).
48. Hoyle, G., "Learning of leg position by the ghost crab *Ocypode ceratophthalma*," *Behav Biol*, Vol 18, Abstract No 6139 (1976).
49. Hubel, D.H., "The brain," *Scientific Am* Offprint (1984).
50. Jacobs, G.E., T.L. Franks, & P.G. Gilman, "Diagnosis of thyroid dysfunction: Applied kinesiology compared to clinical observations and laboratory tests," *J Manip Physiol Ther*, Vol 7, No 2 (Jun 1984).
51. Jamison, J.R., "Educational preparation for chiropractic clinical research," *J Manip Physiol Ther*, Vol 7, No 2 (Jun 1984).
52. Kaminski, M. et al., "A model for the evaluation of chiropractic methods," *J Manip Physiol Ther*, Vol 10, No 2 (Apr 1987).
53 Katch, V., "'The burden of disproof...,'" *Med Sci Sports Exer*, Vol 18, No 5 (Oct 1986).
54. Keating, J.C., Jr., et al., "Toward an experimental chiropractic: Time series design," *J Manip Physiol Ther*, Vol 8, No 4 (Dec 1985).
55. Keating, J.C., Jr., & J.M. Nelson, "The chiropractic scientist-practitioner," *Today's Chiro*, Vol 15, No 2 (Mar/Apr 1986).
56. Keating, J.C., Jr., & R.D. Mootz, "Five contributions to a philosophy of the science of chiropractic," *J Manip Physiol Ther*, Vol 10, No 1 (Feb 1987).
57. Keating, J.C., Jr., & L. Calderon, "Clinical research preparation for chiropractors: Implementing a scientist-practitioner model," *J Manip Physiol Ther*, Vol 10, No 3 (Jun 1987).
58. Kendall, F.P., & E.K. McCreary, *Muscles — Testing and Function*, 3rd ed (Baltimore: Williams & Wilkins, 1983).
59. Kendall, H.O., & F.M.P. Kendall, *Muscles, Testing and Function* (Baltimore: Williams & Wilkins Co, 1949).
60. Kenney, J.J., R. Clemens, & K.D. Forsythe, "Applied kinesiology unreliable for assessing nutrient status," *J Am Diet Assoc*, Vol 88, No 6 (Jun 1988).
61. Klinkoski, B., & C. Leboeuf, "A review of the research papers published by the International College of Applied Kinesiology from 1981 to 1987," *J Manip Physiol Ther*, Vol 13, No 4 (May 1990).
62. Koshland, D.E., Jr., "A response regulator model in a simple sensory system," *Science*, Vol 196 (Jun 3, 1977).
63. Land, E.H., "Color vision and the natural image, Part I," *Proc Nat Acad Sci*, Vol 45, No 1 (Jan 1959).
64. Lashley, K.S., *Brain Mechanisms and Intelligence* (New York: Dover Pub, Inc, 1963).
65. Lawson, A., & L. Calderon, "Interexaminer agreement for applied kinesiology manual muscle testing," *Perceptual and Motor Skills*, Vol 84 (1997).
66. Leaf, D.W., "Nutrient testing evaluation." Proceedings of Summer Meeting, ICAK, Santa Monica, CA, 1985.
67. Leisman, G. et al., "Electromyographic effects of fatigue and task repetition on the validity of estimates of strong and weak muscles in applied kinesiology," *Perceptual and Motor Skills*, Vol 80 (1995).

68. Leisman, G., P. Shambaugh, & A.H. Ferentz, "Somatosensory evoked potential changes during muscle testing," *Intern J Neuroscience*, Vol 45 (1989).
69. Leith, E.N., & J. Upatnieks, "Photography by laser," *Sci Am*, Vol 212, No 6 (Jun 1965).
70. Leith, E.N., & J. Upatnieks, "Progress in holography," *Physics Today* (Mar 1972).
71 Mager, H.J., O. Wess, & W. Waidelich, "Associative information storage in a holographic system," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
72. Melzack, R., *The Puzzle of Pain* (New York: Basic Books, Inc, 1973).
73. Melzack, R., & P.D. Wall, *The Challenge of Pain* (New York: Basic Books, Inc, 1982).
74. Mishkin, M., & T. Appenzeller, "The anatomy of memory," *Sci Am*, Vol 256, No 6 (Jun 1987).
75. Motyka, T.M., & S.F. Yanuck, "Expanding the neurological examination using functional neurologic assessment. Part I: Methodological considerations," *Int Jn Neurosci*, Vol 97, Nos 1-2 (1998).
76. *New Encyclopaedia Britannica, The*, Vol 4 (1979).
77. Nordstrom, D.A., "Cranio Comments" (Letter to the editor), *J Craniomandib Prac*, Vol 4, No 2 (Apr 1986).
78. Norman, R.J., J.S. Buchwald, & J.R. Villablanca, "Classical conditioning with auditory discrimination of the eye blink in decerebrate cats," *Science*, Vol 196 (Apr 1977).
79. Orbach, S., "Students and colleagues of Karl S. Lashley," in *Neuropsychology after Lashley*, ed J. Orbach (Hillsdale, NJ: Lawrence Erlbaum Assoc, 1982).
80. Owens, C., interpreter/compiler, *An Endocrine Interpretation of Chapman's Reflexes* (Not given: nd).
81. Palmer, D.D., *The Science, Art and Philosophy of Chiropractic* (Portland, OR: Portland Printing House, 1910).
82. Perot, C., R. Meldener, & F. Goubel, "Objective measurement of proprioceptive technique consequences on muscular maximal voluntary contraction during manual muscle testing," *Agressologic* (Paris), Vol 32, No 10 (1991).
83. Pietsch, P., *Shufflebrain* (Boston: Houghton Mifflin Co, 1981).
84. Platzer, H., "Holographic modelling of the parallel information processing in the visual pathways of mammals," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
85. Pribram, K.H., *Languages of the Brain — Experimental Paradoxes and Principles in Neuropsychology* (Englewood Cliffs, NJ: Prentice-Hall, Inc, 1971).
86. Pribram, K.H., "Localization and distribution of function in the brain," in *Neuropsychology after Lashley*, ed J. Orbach (Hillsdale, NJ: Lawrence Erlbaum Assoc, 1982).
87. Restak, R.M., *The Brain: The Last Frontier* (Garden City, NY: Doubleday & Co, 1979).
88. Rose, J.C., "Research or practice?" *J Am Dietetic Assoc*, Vol 85, No 7 (Jul 1985).
89. Schmitt, W.H., Jr., "Muscle testing as functional neurology differentiating functional upper motorneuron and functional lower motorneuron problems," in *1986 Selected Papers of the International College of Applied Kinesiology*, ed W.H. Schmitt, Jr. (Shawnee Mission, KS: ICAK, 1986).
90. Schmitt, W.H., Jr., "But what if there's no water in the hose?" *Chiro Econ*, Vol 29, No 6 (May/Jun 1987).
91. Schmitt, W.H., Jr., "The functional neurology of pain and pain control clinical applications and new techniques." Proceedings of Summer Meeting, ICAK, Dearborn, MI, 1988.
92. Schmitt, W.H., Jr., & G. Leisman, "Correlation of applied kinesiology muscle testing findings with serum immunoglobulin levels for food allergies," *IntJn Neurosci*, Vol 96, Nos 3-4 (1998).
93. Schmitt, W.H., Jr., & S.F. Yanuck, "Expanding the neurological examination using functional neurologic assessment. Part II: Neurologic basis of applied kinesiology," *Int Jn Neurosci*, Vol 97, Nos 1-2 (1998).
94. Schwartz, J.P., "Some dangers in the development of new techniques in applied kinesiology." Proceedings of Winter Meeting, ICAK, St Maarten, 1980.
95. "Scientists of the year," in *1981 Yearbook of Science and the Future*, ed D. Calhoun (Chicago: Encyclopaedia Britannica, 1980).
96. Selye, H., *From Dream to Discovery — On Being a Scientist* (New York: Arno Press, 1975).
97. Selye, H., *The Stress of My Life* (Toronto, Ontario: McClelland & Stewart, 1977).
98. Sprieser, P.T., "Holographic memory and therapy localization." Proceedings of Summer Meeting, ICAK, Detroit, 1983.
99. Sprieser, P.T., "Holographic memory as a reason for the design of the nervous system," *Chiro Econ*, Vol 31, No 5 (Mar/Apr 1989).
100. Stroke, G.W., "Optical engineering," in *1981 Yearbook of Science and the Future*, ed D. Calhoun (Chicago: Encyclopaedia Britannica, 1980).
101. Sutherland, W.G., *The Cranial Bowl* (Mankato, MN: privately published, 1939).
102. Teller, E., *The Pursuit of Simplicity* (Malibu, CA: Pepperdine U Press, 1981).
103. Thatcher, R., "Electrophysicological correlates of information retrieval in rats," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
104. Tricoles, G. and E.L. Rope, "Two microwave experiments and the possibility of a holographic model of the retina," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
105. van Heerden, P.J., "Theory of optical information storage in solids," *Applied Optics*, Vol 2, No 4 (Apr 1963).
106. Vitelli, M., "A view on the fundamentals of chiropractic methodology research," *Today's Chiro*, Vol 8, No 3 (Jul/Aug 1979).
107. Vitelli, M., "A view on the fundamentals of chiropractic research methodology, Part II," *Today's Chiro*, Vol 8, No 4 (Oct/Nov 1979).
108. von Bekesy, G., "Synchronization of neural discharges and their demultiplication in pitch perception on the skin and in hearing," *J Acoustical Soc Am*, Vol 31, No 3 (Mar 1959).
109. von Bekesy, G., trans & ed by E.G. Wever, *Experiments in Hearing* (New York: McGraw-Hill Book Co, 1960).
110. Watkins, C.O., "The great mistake in chiropractic, Part 1,"*J CA Chiro Assoc* (Jun 1948).
111. Watkins, C.O., "Chiropractic must have promise to survive," *Am Chiro* (May 1987).
112. *Webster's Third New International Dictionary of the English Language* (1976).
113. Weissmann, G., "Hey, you 'ologists', don't put down the dabblers," *Discover*, Vol 7, No 2 (Feb 1986).
114. Westlake, P.R., "Status of neuroholographic concepts," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
115. Whatmore, G.B., & D.R. Kohli, *The Physiopathology and Treatment of Functional Disorders* (New York: Grune & Stratton, Inc, 1974).
116. Williams, R.J., *Nutrition Against Disease* (New York: Bantam Books, 1973).
117. Wiltse, L., "An amalgamation for progress," *Spine*, Vol 11, No 6 (Jul/Aug 1986).
118. Wing, T.W., "Applied kinesiology, related organs, meridians,and ear-ricular therapy," *Chiro Econ*, Vol 21, No 4 (Jan/Feb 1979).
119. Yates, F.E., Report on Conference on Chemically-Based Computer Designs. Crump Institute for Medical Engineering Report CIME, University of California, Los Angeles, CA 90024 (Aug 1, 1984).

THIS PAGE INTENTIONALLY LEFT BLANK

Chapter 2

General Examination and Treatment Procedures

Chapter two covers some of the basic examination and treatment techniques used in applied kinesiology. These relatively brief descriptions prepare the reader with some basic information and techniques that are necessary in further discussions of applied kinesiology techniques later in the text.

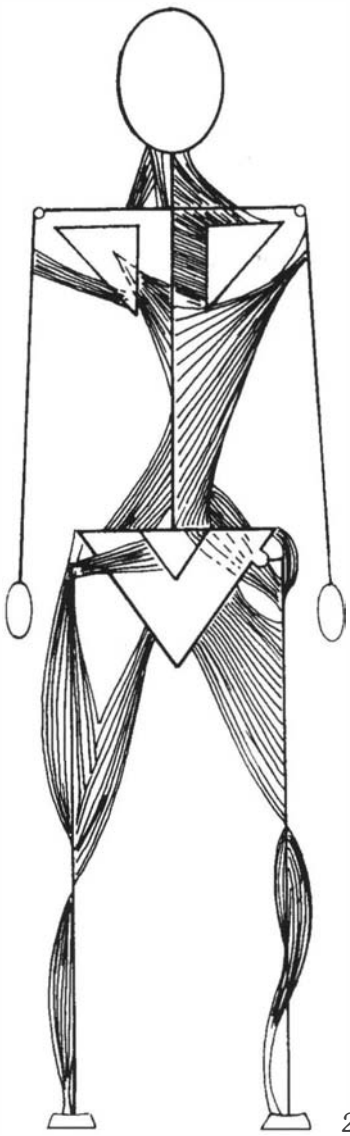
Postural Analysis

Postural analysis is a major source of information in applied kinesiology. It is one of three methods for quickly locating probable muscle dysfunction. It is used in combination with TS line evaluation and meridian therapy localization, discussed later. Use of these three sources of information conserves time in initial examination and helps locate major areas of disturbance.

The use of a plumb line is recommended for static evaluation. The accompanying stick figures schematically represent distortions that occur from single muscle imbalance. Evaluating single muscles is a simplistic way of evaluating posture; however, it is valuable when one is first observing the muscular correlation to postural imbalance.

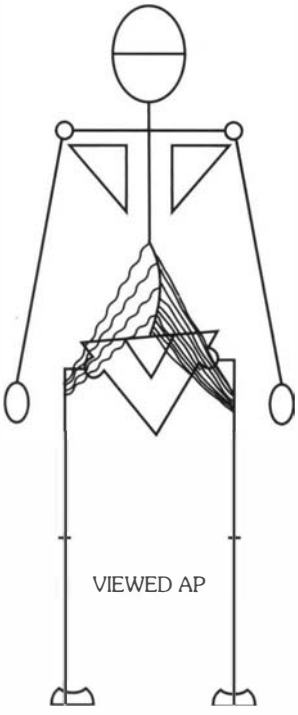
When there is postural evidence of muscular imbalance and manual muscle testing does not reveal it, consider antagonist muscles possibly being hypertonic, or the body's compensatory mechanisms attempting to regain balance. As one becomes more familiar with applied kinesiology, postural imbalances can readily be explained in nearly all cases.

The patient's movements often reveal muscle dysfunction. For example, if a patient has a weak right sternocleidomastoid and strong left one, he can get up from a supine position more easily if he turns his head to the right to align the strong left sternocleidomastoid to raise the head from the table. One may see a similar rotational motion of the patient when the oblique abdominal muscles are strong on one side and weak on the other. Many motions are very revealing, such as using the hands on the knees to aid weak quadriceps when rising from a chair.

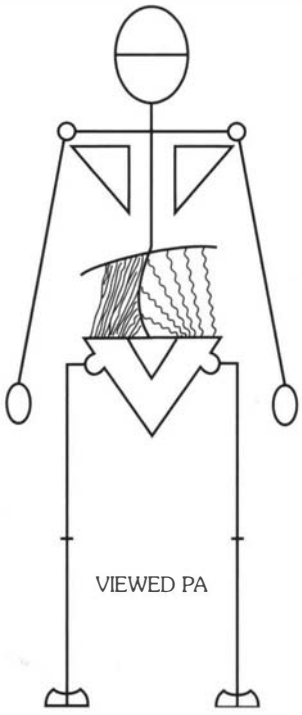


2—1.

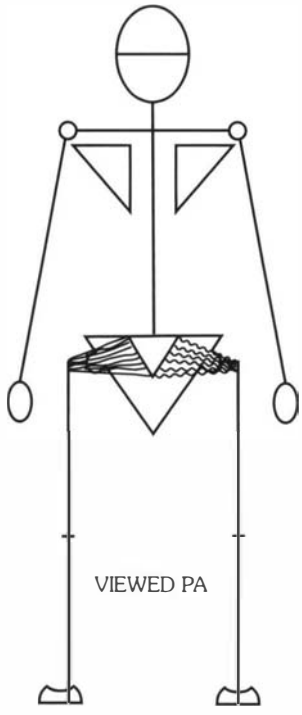
2—2.



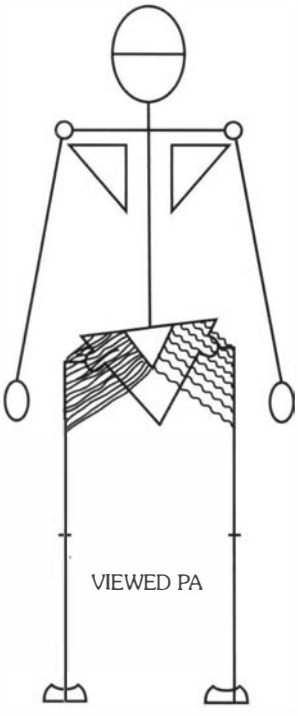
2—3. Toe turn-in on weak psoas side. Pronation of foot tendency. Pelvis raises and lumbars deviate to tight psoas side.



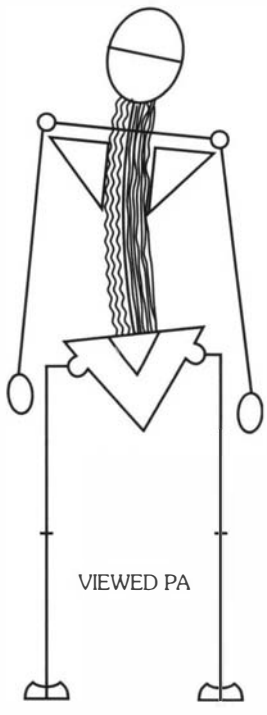
2—4. Right quadratus lumborum weak. Pelvis level, right 12th rib elevated and left lumbar curved.



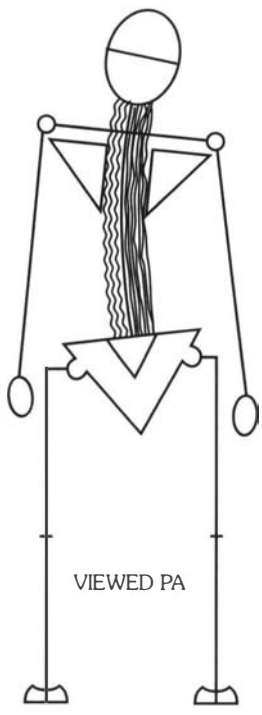
2—5. Right piriformis weak, left hypertonic. Left foot turns out.



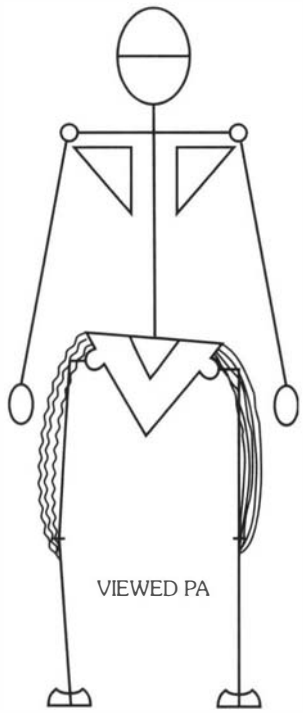
2—6. Pelvis elevation on side of gluteus maximus weakness. Leg and foot internally rotated; some loss of lateral knee stability.



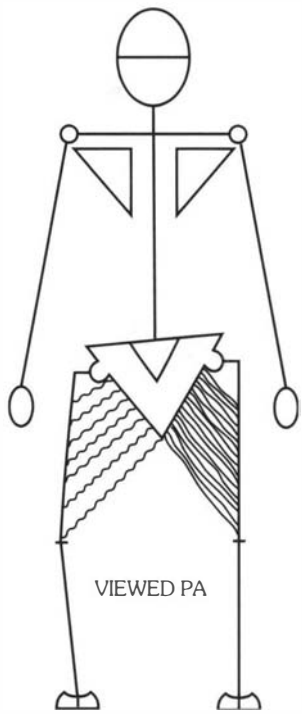
2—7. Right gluteus medius weak. Right pelvis, shoulder, and head all elevated.



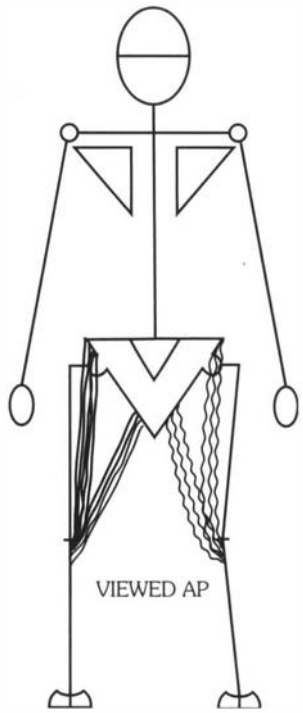
2—8. C-curvature on side of weak sacrospinalis. Shoulder, head elevation and low hip on side of weakness. In prone position, weak sacrospinalis is atonic.



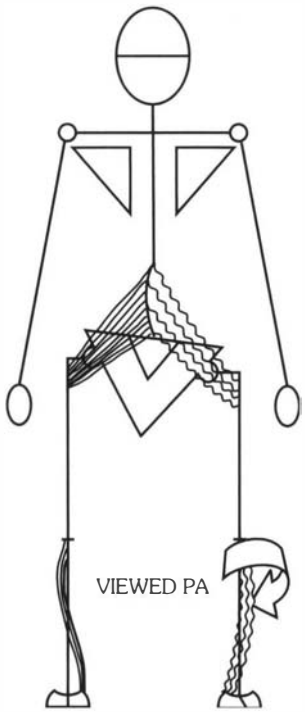
2—9. Left tensor fascia lata weak. Genu varus and pelvic elevation on weak side. Gluteus maximus also aids the knee support.



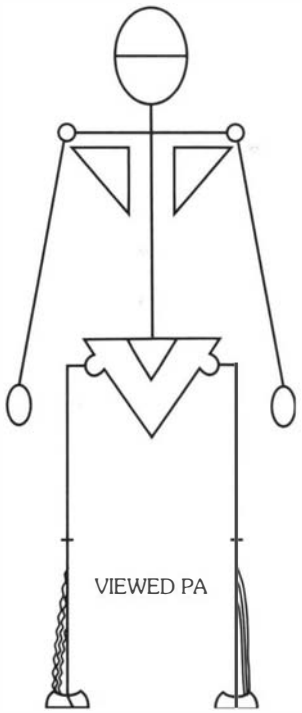
2—10. Left adductors weak. Genu varus on weak side. Pelvis elevation on opposite side.



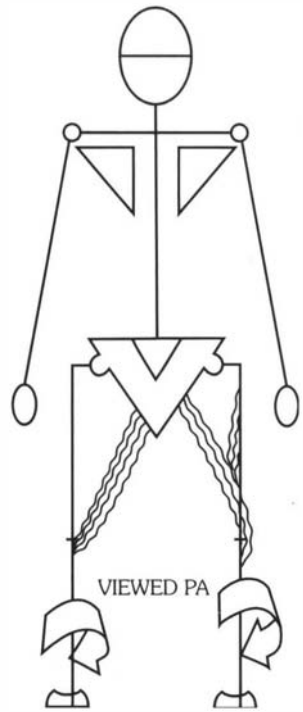
2—11. Weak sartorius and/or gracilis. Genu valgus — also affects AP balance of pelvis.



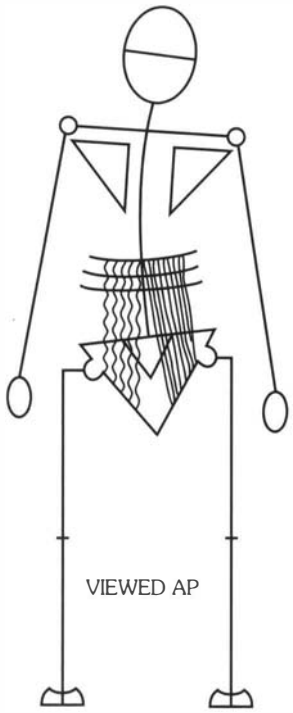
2—12. Tibialis anterior weak on right. Ankle pronation or pes planus. Problem compounded if psoas allows internal leg rotation.



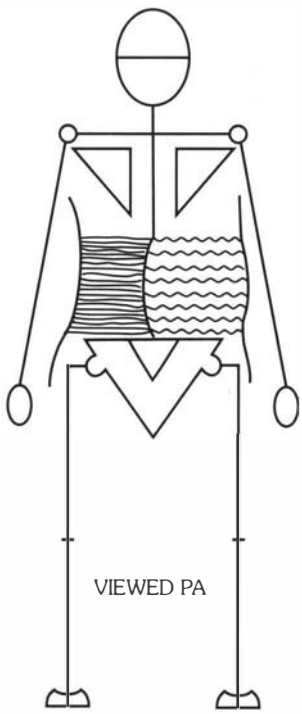
2—13. Weak peroneus group on left allows pes cavus or supination.



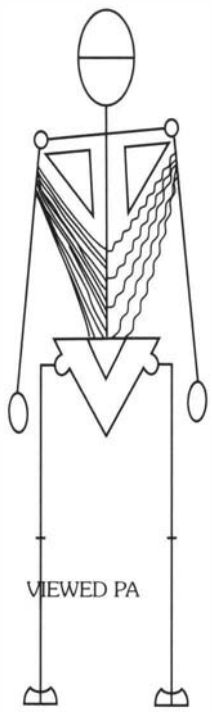
2—14. Medial hamstrings weak, allow external foot rotation. Lateral hamstring (biceps femoris) allows internal foot rotation.



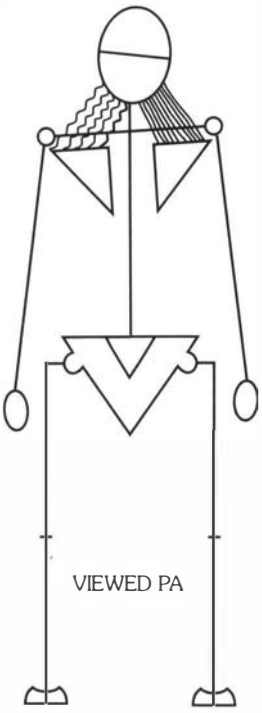
2—15. Weak rectus abdominis allows separation of pelvis and thoracic cage. If bilateral, a lumbar lordosis develops.



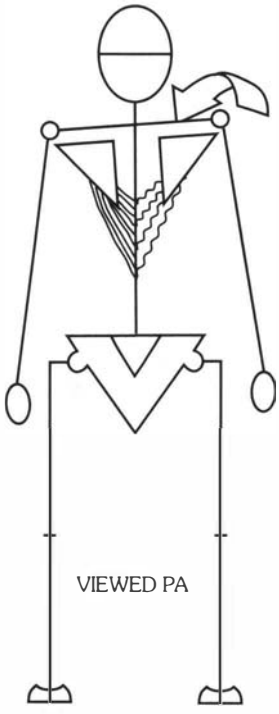
2—16. Weak right transverse abdominis. Lateral abdominal bulge and possible scoliosis. Abdominal bulge is best seen with patient doing sit-up.



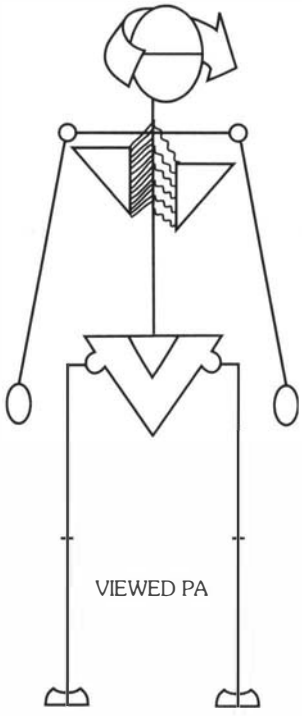
2—17. Weak latissimus dorsi on right. High shoulder and head level if other muscles are not involved. Upper trapezius involvement can easily confuse the pattern.



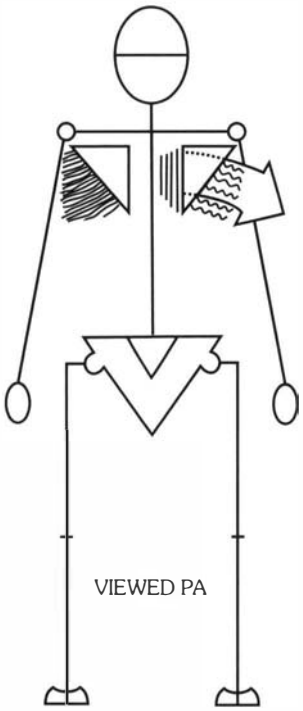
2—18. Weak left upper trapezius. Shoulder low on side of weakness. Head tilt away from side of weakness. Usually secondary tightness on opposite side.



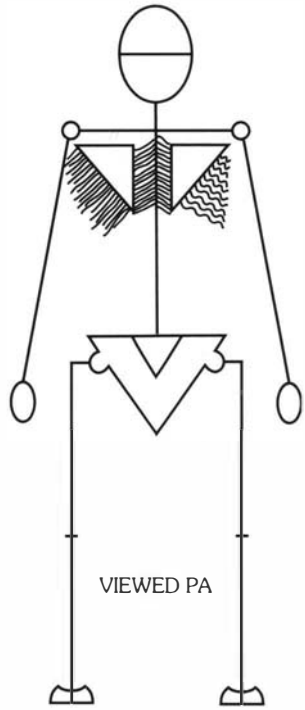
2—19. Weak right lower trapezius. Elevated scapula, kyphotic dorsal spine and forward roll of shoulder.



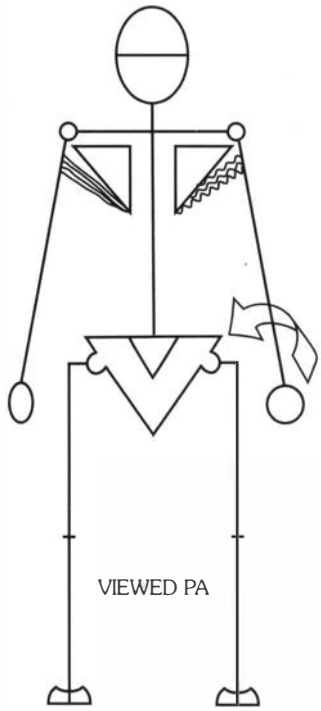
2—20. Weak rhomboids on right allow scapula to sag and head to rotate toward side of weakness.



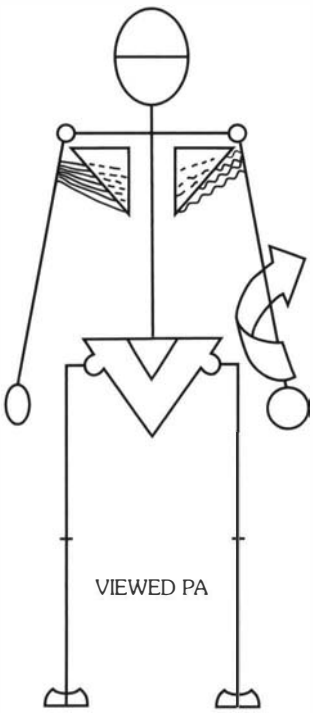
2—21. Weak serratus anticus on right allows scapula to wing away from thoracic cage.



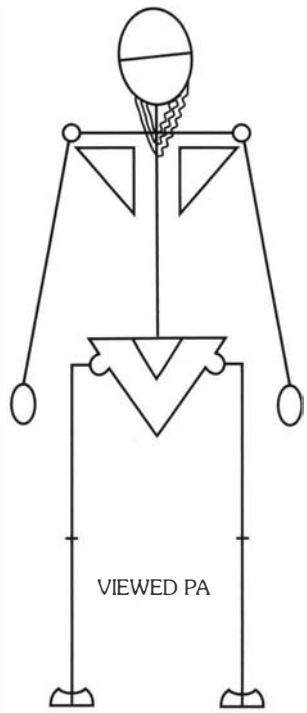
2—22. Weak serratus anticus with secondary rhomboid contraction. Less winging of the scapula.



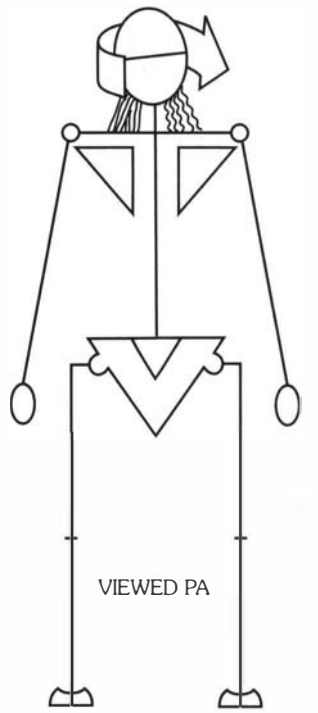
2—23. Weak right teres minor and/or infraspinatus with other external rotators (posterior deltoid, supraspinatus) allow internal rotation with hand facing palm posteriorly.



2—24. Subscapularis and other internal rotators (teres major, anterior deltoid, pectoralis major, latissimus dorsi) when weak allow external rotation or the palm to face forward.



2—25. Neck externsor and/or flexor group weakness causes lateral flexion of neck.



2—26. Sternocleidomastoid weak on right. If tilt is due to SCM only, head rotation will be to side of weak SCM.

2—27. Weak abdominals fail to keep pubes and anterior thoracic cage approximated. Lordosis of lumbar spine and facet jam result.

2—28. Hamstrings if weak allow anterior tilt of pelvis, lumbar lordosis, and facet jam. Correlate with possible posterior ischium subluxation.

2—29. Gluteus maximus provides posterior pelvic, lateral knee support. Weakness contributes to lumbar lordosis and facet syndrome, plus knee instability.

2—30. Weak sartorius and/or gracilis fails to support anterior pelvis. Posterior pelvic imbalance results. Correlate with possible posterior ilium subluxation.

2—31. Rectus femoris weakness allows posteriority of pelvis and loss of lumbar curve.

2—32. Forward lean is present in soleus weakness due to poor posterior tibial support.

2—33. Bilateral psoas weakness allows loss of lumbar curve.

2—34. Weak lower trapezius fails to support thoracic spine, and kyphosis results.

2—35. Forward head position from weak cervical extensors.

2—36. Lack of anterior support of knee by weak quadriceps causes knee hyperextension and posterior pelvic tilt.

2—37. Knee hyperextension when popliteus is weak.

2—38. Hyperextension of knee is compensatory for weak gastrocnemius.

2—39. Weakness of triceps brachii causes elbow to be in excessive flexion. Evaluation must consider possibility of overdevelopment of biceps brachii. Illustration exaggerated.

2—40. Weak biceps brachii causes elbow to be straight or in extension. Illustration exaggerated.

Therapy Localization

Science is at a loss to understand and explain completely the many types of energy and control mechanisms within the body. When the mass of energy and regulating mechanisms are balanced, health results. D.D. Palmer spoke frequently of the energy patterns of the body.[40] He admonished the profession, "I wish that all chiropractors could take in this basic principle of our science — *that too much or not enough energy is disease.*"

Progressively new observations and discoveries are made about energy patterns. Shafica Karagulla, M.D.,[25] has studied the aura and vortices of force as seen by people with higher sense perception. Records of the energy field in health and disease have been made using Kirlian photography,[29] which is understood on only a limited basis. Electromagnetic balance and imbalance have been studied by Davis and Rawls.[6] The electrical resistance of the meridians of the body changes under different aspects of health, and can be measured by the Ryodoraku method.[50]

Instruments for measuring the electromagnetic energies of the body and evaluating the nervous system are becoming more sophisticated. Even with the increased interest in evaluating various forms of body energy and control, we are still just on the edge of comprehensive understanding.

A major development in applied kinesiology occurred when Goodheart[13] observed that if a patient touched an area of dysfunction, the results of manual muscle testing changed. A muscle that previously tested weak became strong when the patient touched an area of dysfunction. The system is called therapy localization (TL). It finds the location of a problem, but it does not necessarily tell you what is wrong.

There are numerous applications for therapy localization. The various reflexes, subluxations, meridian points, and nerve receptors show positive therapy localization when there is dysfunction. An example of therapy localization is seen when the pectoralis major (clavicular division) tests weak. One can have the patient place the fingertips over the neurolymphatic reflex area for the muscle, and the muscle will test strong if that reflex is involved with the weak test. Stimulation of the NL reflex, in the normal treatment manner described later, will then cause the muscle to test strong without the therapy localization. Therapy localization can further be used to determine if the reflex was adequately stimulated. If the muscle again weakens when the patient therapy localizes the neurolymphatic reflex area, further stimulation of the reflex is needed. Therapy localization is used in this manner to determine when effective treatment has been applied to the various reflexes, subluxations, or other factors.

If a previously strong muscle weakens when a re-

flex point associated with it is touched by the patient, it is a subclinical involvement; that is, the reflex is active but apparently not involved enough to cause an associated muscle to test weak without the TL.

There is a generalized effect on the muscles of the body when there is positive therapy localization. When a muscle not known to be associated with a reflex or other factor is used to evaluate therapy localization, it is called an indicator muscle. Using an indicator muscle to evaluate therapy localization is a generalized approach that is often valuable in screening a patient for various types of problems. First, a muscle is tested to determine if it is strong without therapy localization. This is known as testing a muscle "in the clear." An example of using an indicator muscle is evaluating for vertebral subluxations. Vertebral subluxations can cause many or only a few muscles to test weak. In other words, the muscles that will test weak as a result of a vertebral subluxation are somewhat unpredictable. To therapy localize for a vertebral subluxation, one should find a muscle that tests strong in the clear. This muscle is then tested to evaluate for positive therapy localization. The patient touches the skin over a vertebral level, and the examiner tests the indicator muscle. This may be repeated over several areas of the spine until the indicator muscle tests weak, indicating positive therapy localization.

There is a difference between the palmar and dorsal surfaces of the fingers. Therapy localization is usually best done with the palmar surface of the patient's fingertips touching the skin of the area to be evaluated. It is hypothesized that therapy localization either adds energy to or subtracts it from the skin area. There is probably a neurologic aspect to the phenomenon.

Therapy localization is best done with the patient's fingers touching the skin. Less powerful TL is present when cloth is interposed between the skin and fingers. For example, if there is a cotton garment such as a patient gown between the fingertips and the skin being evaluated, there will usually be the same therapy localization findings as without the interposing cloth; however, some positive therapy localization may be missed. In general, synthetic fabrics interposed between the fingers and skin reduce the effect of TL more than natural fabrics. Therapy localization is also less effective when there are many layers of cloth between the fingertips and skin.

Certain materials completely block the effect of therapy localization. A thin layer of lead foil between the fingertips and skin being evaluated will completely block therapy localization, as will some ceramic materials.

As noted, therapy localization is usually done with the palmar surface of the fingertips. There is a difference in polarity between the palmar and dorsal surfaces of the hand.[6] In some instances therapy localization will be negative with the palmar surface, but it will be positive with the dorsal surface of the fingers. It is not completely understood why this occurs, but it is suspected that it correlates with ion balance and/or neurologic disorganization in the body.

In addition to helping determine areas in need of functional correction, therapy localization is generally positive over pathologic areas. For example, an individual with a gastric ulcer will usually have positive therapy localization over the stomach. Trauma, such as a fracture or torn ligament, almost always has positive therapy localization.

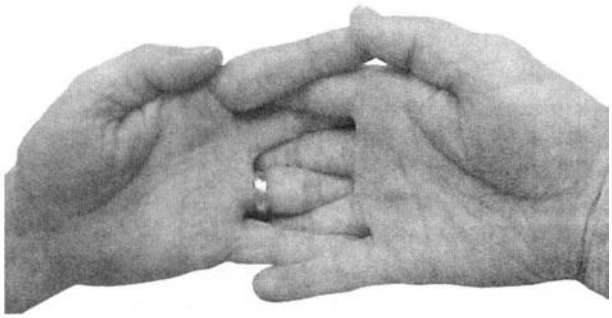
Although therapy localization is a valuable examination tool, one must take care to keep it in proper perspective. Therapy localization only tells that something is wrong in an area; it does not tell what is wrong. One must use the various methods of differential diagnosis to arrive at a final conclusion.

Therapy localization should be limited to the patient touching the area to be evaluated. When the doctor or another individual touches the area, there may be a change in the muscle test, either testing stronger or weaker. Another individual touching the area for therapy localization introduces variables that are difficult, if not impossible, to evaluate. This can readily be observed by having several individuals therapy localize to the same area on another individual. The results of the test will often vary when different individuals touch the area to be tested. It appears that some individuals with higher energy levels can add energy to the area being tested, while others with low energy levels subtract energy. The additional variables of the doctor or another individual touching the area for therapy localization may cause errors in interpretation. This procedure is not recommended.

Enhancing Therapy Localization

There are various methods for enhancing therapy localization. Wetting the patient's fingertips gives a better contact between fingers and skin. This is often used when the patient is severely dehydrated. A "high-gain" type of therapy localization has been used; the patient touches the thumb and little finger together while therapy localizing with the index, middle, and ring fingers. This is thought to call higher centers relating to man's unique characteristic of thumb apposition.

Finger Interlink. Interlinking the fingers of the two hands is another "high-gain" therapy localization. Goodheart[18] notes that traditional therapy localization utilizing both hands alerts both left and right brain activity. Interlinking the fingers adds a possible "mass action effect," since the right hand-left brain and the left hand-right brain sequential pathways are effectively doubled. This therapy localization method appears to effectively neutralize neurologic disorganization, also known as "switching," which is often a left brain-right brain confusion. Neurologic disorganization is discussed in Chapter 5.



2—41. Interlinked finger therapy localization provides a "high-gain" therapy localization and may override neurologic disorganization.

Two-Point Therapy Localization. In certain types of conditions one can use a second point of therapy localization to determine if it cancels the initial positive therapy localization. For example, if there is positive therapy localization at a thyroid reflex point, one can have the patient continue to therapy localize at that point and, with the other hand, therapy localize various endocrine reflex points. If therapy localizing to a gonad reflex cancels the weakening of the indicator muscle, it is possible that the gonads are overreacting in their function of inhibiting thyroid action. Two-handed therapy localization can also be applied to structural factors. If there is a knee subluxation that continues to recur, have the patient therapy localize to the knee; if positive, use the other hand to touch the ankle, sacroiliac, or other structure that might be related with the recurrent knee subluxation. If, for example, therapy localization to the ankle cancels the positive knee therapy localization, evaluate the ankle and possibly the foot for contributing to the recurrent knee subluxation.

Numerous variations of therapy localization are discussed with various therapeutic and examination approaches throughout this text and others on applied kinesiology.

Right and Left Brain Function

The brain appears to be a mirror image of itself; however, there is significant evidence that each hemisphere has a special function. It is well-known that the right hemisphere of the brain basically controls the left side of the body, while the left hemisphere controls the right side. While the majority of the nerve fibers decussate, there are some that do not; thus there is some control of the right body by the brain's right hemisphere, and of the left body by the left hemisphere. This is important because it enables one hemisphere to take control of both sides of the body in case there is damage to the other hemisphere.

In most individuals the left hemisphere has verbal function, and spatial activity is centered in the right hemisphere. This right-left specialization is based on right-handedness. According to Ornstein,[38] about 5% of the population is left-handed. These persons have less consistency in the development of the two sides of the brain. In some there is reversal of left-brain verbal function and right-brain spatial function. In others it appears that both sides are equally developed for both specialties. The usual left-brain verbal and right-brain spatial orientation is present in still others.

There has been considerable study of right-left brain activity.[1,27,28,37,42,45] Much of the research indicates that there is a set pattern for most people organized with right-hand dominance. Bogen[2,3,4] and Ornstein[38] each present a set of dichotomies of the characteristics of the two sides. Ornstein states that he presented the table "... only for purposes of suggestion and clarification in an intuitive sort of way, not a final categorical statement of the conception. Many of the poles are, of course, tendencies in specializations, not at all binary classifications." Here we, too, will consider a tentative dichotomy for the functions of the two sides. This list was developed by observing research on bilateral brain function and from clinical observation of applied kinesiology findings.

A Tentative Dichotomy

Left Brain	Right Brain
Logical	Non-logical
Mathematical	Tonal
Rational	Nonsensible
Reasonable	Unpredictable
Practical	Non-practical
Linear	Spatial
Masculine	Feminine
Intellectual	Intuitive
Negative	Positive
Time/history	Eternity/timelessness
	Under some circumstances, clairvoyant, clairaudient, clairessential

2—42.

Therapy Localization with Emphasis on Unilateral Brain Activity

Occasionally involvement of an organ or gland can be observed on a clinical basis, but not evidenced in associated muscular weakness or hypertonicity. It is assumed that compensating factors are overriding the muscle dysfunction. The muscle dysfunction can sometimes be brought out by having the patient therapy localize to a reflex associated with the dysfunctioning organ or gland. In some cases even this fails to show an asso-

ciation. When the individual is requested to either hum (activating right brain) or say the multiplication tables (which activates the left brain), there may be an immediate and dramatic weakening of the muscle that previously failed to give evidence of associated involvement. The same tonal or mathematical activity under these conditions does not cause any weakening when there is no therapy localization, nor does it if other random, non-involved areas are therapy localized.

The attention of the "mind" seems to be directed to one portion of the brain at a time.[21] Hypothetically, the point being therapy localized is being put in closer contact with the brain by its one-sided action.

The factor that is therapy localized and found positive during right or left brain activity is treated as usual. It may be a reflex, an area that needs manipulation, an acupuncture point, or some other factor. After treatment has been administered, the area is again therapy localized with right or left brain activity to determine if correction has been obtained.

It is important when an individual hums that he not mentally activate the words for the tune. A popular tune is "Happy Birthday," but the lyrics are so well-known that they may go through the person's mind as he hums the tune. This activates both sides of the brain and is not effective in accessing a single side.

Temporal Tap

The temporal tap is an applied kinesiology mechanism that appears to penetrate the filter of the sensory system. What we are capable of conceiving depends upon innate body mechanisms that filter our ability to sense the external and internal stimuli constantly presented to the nervous system. Prior experience also influences our perception of sensory stimuli. The temporal tap apparently deals with the filter of the sensory system, as well as with bilateral brain function.

The nervous system is bombarded with stimuli, much of which is unnecessary for the survival and immediate requirements of the organism. If man were aware of all the stimuli in his environment, there would be much unnecessary processing and the system would probably be overwhelmed. Various animals have different filters to their sensory systems. The filters basically work on a "need to know" basis. The frog's visual response is for defense, such as observation of a bird of prey, and for obtaining food by detecting insects when they come close enough to capture.[30] The human likewise filters out most of the electromagnetic spectrum by special sensory capability. Visual light is only a very small portion of the spectrum, ranging approximately between 380-760 billionths of a meter.

The nervous system filters out low priority sensory input by adaptation. The large afferent (group II) nerve fibers adapt to constant low velocity or low intensity stimulation. When first putting on clothes we are aware of the pressure. After wearing them for a short time, the fibers adapt so that we are no longer aware of this stimulus unless attention is drawn to it. When first sitting in a chair, pressure of body weight on the chair is apparent, but the body soon adapts. Sudden changes of pressure, temperature, and other factors are easily recognized, but adaptation is rapid if the stimulus is non-noxious.

Because we must discriminate between continuous safe stimuli and survival-related ones, we have evolved sensory systems that respond primarily to alterations in the external environment. When there are no changes in the environment, the stimulus becomes subliminal and the body does not react. When some new stimulus enters the environment, it is interpreted; based on the change, action may or may not take place. Our perception is governed by previous experience.[24] A conceptualization of sensory input is from what we believe to be true. Many of the concepts that humans hold are limiting. The comment to the butterfly is appropriate: "Of course you can fly, but first that cocoon must go."

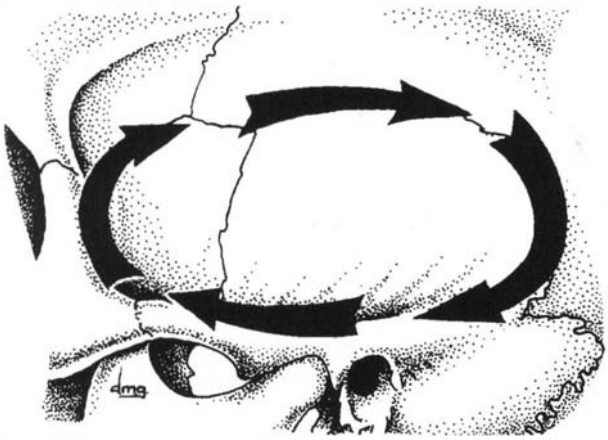
Ornstein[38] postulates and supplies experimental evidence that the autonomic nervous system, which has control of the innate physiologic systems of the human body, may not be completely involuntary. The autonomic nervous system is subject to voluntary control if the situation is set up appropriately. Yogis can alter their heart rates to 300 beats per minute, or significantly change body temperature. Other alterations include those of blood flow to various limbs, increasing or decreasing kidney urinary production, pancreatic secretion, heart rhythms, and removal of blocks in an ECG that were produced by morphine.

The applied kinesiology temporal tap technique appears to penetrate the sensory nervous system that is ordinarily blocked, as long as the input is in keeping with the body's needs and can be accepted by the individual. It was introduced into applied kinesiology when Goodheart[14] became intrigued by the reportedly good results of a Czechoslovakian doctor who helped individuals reduce smoking by giving positive affirmations while manipulating the temporal bone. When Goodheart attempted to contact the doctor, he received a letter stating that the man was deceased. Since the technique was not described, there was no further information from that source. Goodheart's first attempts at penetrating the sensory system by giving positive and negative thoughts and other sensory stimuli, while manipulating the temporal bone and oth-

erwise stimulating the area, were unsuccessful. After much experimenting, a successful method was found.

The temporal tap consists of tapping on the temporal sphenoidal diagnostic line (page 15), beginning just in front of the ear at the point associated with the psoas. The tapping is continued along the zygomatic process, upward at the anterior margin of the temporalis muscle, proceeding around the temporal sphenoidal line to the superior and posterior of the ear. Sensory input of some type is provided while this is done. The input can be a suggestion given by the physician or patient, visual suggestions, therapy localization, or other stimuli.

When temporal tapping, there is a positive and a



2—43. Begin anterior to the ear and tap sharply around the temporal sphenoidal (TS) line.

negative side to the head. Temporal tapping the left side of the head causes acceptance of positive statements; tapping the right side will result in accepting negative statements. This appears to correlate with the different dominance of the two hemispheres of the brain. The positive-negative acceptance may be reversed in a left-handed individual.

The procedure for temporal tapping is for the physician to tap the head, beginning in front of the ear and proceeding around the TS line. Tapping is done with the palmar surface of the fingertips of the physician's right hand on the left side of the patient's head. The tapping should be quick and sharp so that the fingers bounce off the skull, but not so hard that it causes discomfort to the patient. While tapping, some type of sensory input is presented to the patient. It can be in the form of a suggestion that will change function. For example, in the presence of a strong pectoralis major (sternal division), the physician can say, "Your muscle is weak," while tapping around the TS line. It is best to tap around the line three or four times while repeating the statement each time. After the temporal tap procedure, the previously strong muscle will test weak. This suggestion is for demonstration purposes only. The muscle weakness is temporary; it will recover spontaneously. It is recognized that different people have different levels of suggestibility. The muscle will test weak on highly suggestible people without the temporal tap procedure. In the usual situation, the temporal tap will cause the muscle to test weak; it will not when done improperly. For example, if the tapping is done in the opposite direction, it will not cause the muscle to test weak.

To make a similar suggestion regarding muscle weakness when tapping the right side of the subject's head, the physician uses the palmar surface of the fingertips of his left hand. In this case the statement must be negative, such as, "There is no need for your muscle to be strong." The statement, "Your muscle will be weak," will not be effective when temporal tapping is applied to the right side of the patient's head, since it only accepts negative statements.

In addition to positive and negative sensory input being applicable to only one side of the head, there appears to be an electromagnetic factor applicable in the tapping mechanism. The temporal tap is only effective when the physician uses the palmar surface of his right fingertips on the left side of the patient's head. The palmar surface of the right hand is electrically positive; the dorsal surface of that hand is negative.[6] If the physician uses the palmar surface of his left fingertips to tap on the patient's left side, or uses the dorsal surface of the knuckles of the right hand on the left side of the head, neither will be effective in temporal tapping.

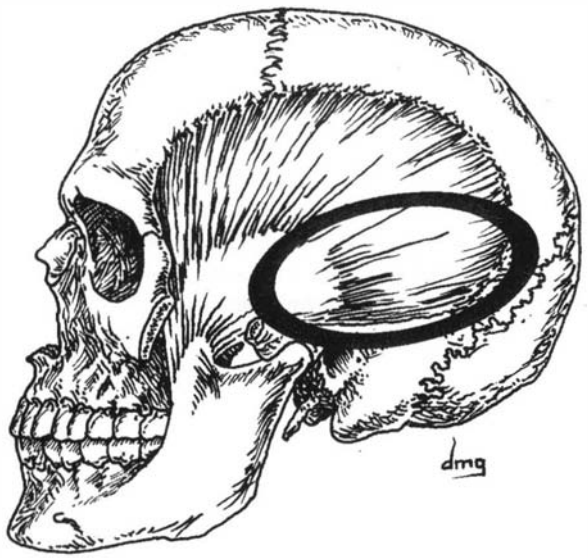
The patient can tap his own TS line; however, the handedness is reversed. The patient uses the palmar surface of his left fingertips on the left TS line, and the palmar surface of the right on the right. If using his knuckles (dorsal surface), the patient will use his right hand on his left TS line, and vice versa.

A simple way to evaluate the effectiveness of TS line tapping is to simply make a statement to the patient that is true or false and observe the reaction in a previously strong indicator muscle. For example, if the patient has brown hair, tap the left TS line with the palmar surface of your right fingertips and say several times, "You have blond hair." The usual response is for the indicator muscle to weaken. It will not weaken with the statement, "You have brown hair." Applying temporal tap to the right side of the patient's head requires the statement, "You do not have brown hair," to cause the indicator muscle to weaken.

A large percentage of left-handed individuals will respond exactly the opposite. This is probably because of genetic reversal of the right- and left-brain dominance, i.e., left-brain spatial and right-brain verbal. In some cases there is poor differentiation between the temporal tap characteristics of the two sides. This usually relates with neurologic disorganization and probable failure of proper development. These individuals are sometimes significantly improved by cross patterning, discussed on page 179.

Finding and correcting the cause of neurologic disorganization is important to restoring health in many individuals. (This subject is discussed in detail in Chapter 5.) Often when there is neurologic disorganization the person has mixed dominance, e.g., right-handed and left-eyed. The temporal tap can help determine if the bilateral brain function is as usual or is genetically reversed. If tapping on the left side of the head results in response to positive statements, brain organization is as usual. If tapping on the right side elicits response to a positive statement, the individual is organized to be left dominant. In this case the left hand, eye, and foot should be dominant. Determination of bilateral brain organization may be important in developing training protocol designed to organize one-sided dominance.

There is some relation between mandibular movement and the temporal tap. If an individual retrudes the jaw, temporal tapping will not be effective until the jaw is returned to its normal position. Retrusion of the jaw is done by the posterior fibers of the temporalis muscle. If these fibers are hypertonic there will be no effect from the temporal tap, even though the mandible is not held in a retruded position. The posterior fibers of the temporalis muscle can be evaluated by palpation and therapy localization. Treatment for hypertonic posterior



2—44. Ellipse indicates the probable area of muscle spindle cell involvement if the posterior fibers of the temporalis muscle are hypertonic. Retrusion of the mandible renders the temporal tap ineffective.

fibers of the temporalis muscle is usually directed to the neuromuscular spindle cell (page 62), after which the effect of temporal tapping will be as usual.

There are several uses for the temporal tap. It can be used to control some involuntary actions. It has been used by dentists and others as an effective method of controlling an overactive gag reflex.[8] When a dental procedure or cranial therapy cannot be effectively accomplished because of an overactive gag reflex, one can temporal tap the individual with the statement, "You will be fine without gagging" to the left side of the head, and "There is no need for you to gag" to the right side.

Dentists have also reported effective results in controlling excessive salivation and bleeding during oral surgery. Initially the procedure was not effective with the sensory input, "You will get along fine without bleeding" for the left side and "There is no need to bleed" for the right. After changing the sensory input to "You will get along fine without as much bleeding" for the left, and "There is no need to bleed so much" for the right, the procedure was successful. The sensory input given to the patient during temporal tap must be acceptable. It is not acceptable that one would not bleed during surgery, as it is a normal process of infection control and healing. Similar modified statements will be appropriate for excessive salivation.

Control of involuntary activities, such as the overactive gag reflex, bleeding, and salivation, gives evidence that the temporal tap is effective for entering suggestions to the nervous system which help bring involuntary activity under control. The effects of the temporal tap are not permanent, usually lasting thirty minutes or so in the control of involuntary actions. The procedure is not effective for regulating conditions such as blood pressure, circulation, or digestive function. The usual therapeutic approaches must be completed to permanently bring physiologic function under normal control.

The temporal tap has been successfully used to help individuals eliminate problematic habits, if the subject desires the change. Among other things, it has been used to help stop smoking, control excessive drinking, diminish unrealistic fears, and control drug use. The temporal tap is incapable of overcoming what an individual believes to be true. If an individual has made numerous attempts to control his alcoholism without success, his belief will be that it is impossible to stop drinking. For the temporal tap to be effective in this type of habit change, there must be additional supportive activity to change the individual's basic belief. The doctor specializing in natural health care has additional understanding of the relationship of the adrenal gland, nutrition and nutritional absorption, and other factors contributing to the alcoholic problem. When the doctor explains the additional supportive factors to the patient, it helps him develop a new belief that success may be possible. In this case, the temporal tap supports the effort.

The mechanics of the temporal tap for habit change are the same as for other uses. A positive statement is administered to the left side, while either the doctor or the patient taps. Remember, it is necessary for the doctor to use the palmar surface of his right fingertips on the left side of the patient's head, or for the patient to use the palmar surface of his left fingertips

on the left side. For the right, the doctor uses the palmar surface of his left fingertips; the patient uses the palmar surface of the right. If, for some reason, the opposite hand from the one indicated above is to be used, the dorsal surface or knuckles are used. For stopping a habit, sensory input for the left side of a normally organized individual is, "You (I) will get along fine without smoking." For the right side, "There is no need to smoke." The patient repeats the temporal tap whenever there is a desire to smoke or indulge in whatever habit is being altered. The patient must desire to break the habit, and he must believe that it can be accomplished.

Temporal tap augments therapeutic processes. For example, an individual can be placed on DeJarnette pelvic blocks to correct a category I or II, and then temporal tapped. The temporal tapping is done on the patient's left temporal sphenoidal line with the doctor's right fingertips, palmar surface. There is no sensory input in the form of a verbal suggestion. The sensory input appears to come from the mechanical stimulation of receptors resulting from the torque position of the pelvis, created by the pelvic blocks. In most cases the pelvic correction will be obtained immediately.

Eyes Into Distortion (EID)

There is an optimal body posture for efficiency and comfort.[9] Patients should first be evaluated in the standing position for distortion, and then throughout the examination for movement efficiency. The initial plumb line analysis, as discussed earlier in this chapter, indicates what muscles may test weak. It also gives information to further evaluate the patient on a neurological adaptation basis. The important aspect here is the modular distortion seen at the plumb line.

First, observe the patient from the posterior; record the head, hip, and lateral tilting position. Next record the AP posture. With the plumb line slightly anterior to the lateral malleolus the line should be slightly anterior to the midline of the knee, through the greater trochanter of the femur, approximately midway between the anterior and posterior body at the abdomen and chest level, and finally through the center of the shoulder joint and ear lobe.[26]

A common postural fault is the head forward of the plumb line. When the posterior thorax touches a wall, there should be no more than 6 cm distance between the wall and the curve of the neck.[17]

Body posture is intricately organized with the equilibrium proprioceptors.[19] One often thinks of the labyrinthine, visual righting, and head-on-neck reflexes as the equilibrium proprioceptors. Proprioceptors throughout the body, probably concentrated in the sacroiliac and spinal ligaments, must organize with these reflexes. Applied kinesiology techniques of equilibrium proprioceptor synchronization, PRYT, gait, and dural tension provide considerable information about modular interaction within the body.

When there is body distortion, the muscles try to correct or adapt to the distortion. In some cases improper stimulation to the equilibrium proprioceptors causes the distortion. An example is an atlas subluxation. The receptors for the head-on-body reflexes are located in the ligaments of the upper cervical region.[35] When they are distorted by the subluxation, the central nervous system may receive a head-tilt signal that conflicts with the actual head position as reported by the labyrinthine reflexes. If self-correction is not possible, the body adapts to the disturbed head position with other body distortions. A major emphasis in applied kinesiology is to maintain organization of the equilibrium proprioceptors. This often requires correction of the stomatognathic system, an important part of which is leveling the head.[48]

There is an automatic adaptation of eye position to body activity, postural positions, and postural distortions. Parker[41] provides an excellent demonstration of the relationship of the eyes with the vestibular apparatus. As you look at this text, rotate your head back and forth about three cycles per second. The words in front of you remain stationary, and you can continue reading. Move the book back and forth at the same rate and the words blur; reading becomes impossible. In the first instance the semicircular canal signals the oculomotor muscles to rotate your eyes; this organization is lacking in the second instance.

Goodheart[16] noticed that muscles test differently when a patient's eyes are oriented in different directions, according to his postural distortion. This apparently is because of an adaptation of the oculomotor muscles to the individual's distortion. When the eyes are oriented with the distortion, subclinical faults appear; that is, an individual may have symptoms and other indications of a condition — such as a category II sacroiliac subluxation — but positive therapy localization is not present. When the patient turns his eyes in the direction of the postural distortion, positive therapy localization is present. This method of testing is called "eyes into distortion" (EID). The eyes are oriented in the direction of major postural distortion. For example, if a patient's head is tilted down on the right, the EID position is for the eyes to be down and to the right. There are six primary eye positions: (1) eyes down

and to the right, (2) right, (3) eyes up to the right, (4) eyes up to the left, (5) left, and (6) eyes down and to the left. Occasionally the distortion indicates that the eyes should be moved directly up or directly down. This occurs when the distortion is strictly in the sagittal plane, with no rotation. Often there is a combination of tilt and rotation. If the head is tilted to the right and rotated to the left, one would have the patient turn his eyes down and to the right and then turn them slightly to the left, toward center. If the head and pelvis are tilted in opposite directions, pick the major distortion.

In patients who have adapted to their condition, numerous weak muscles may appear when the eyes are put into the direction of distortion. The EID position takes away the adaptation the extrinsic eye muscles have made to the distortion.

Elsewhere in applied kinesiology literature[48] there is considerable discussion of the stomatognathic system's integrated activity in body orientation and equilibrium. The hyoid muscles are an intricate part of this organization. Their role can be observed by putting the hyoid bone in the same position as EID. The findings that were positive when the eyes were into distortion will be negative when the hyoid is moved into a position that parallels the EID. This indicates a parallel adaptation of the hyoid muscles and extrinsic eye muscles to the patient's distortion. It may be that much of the eye position adaptation is secondary to the hyoid muscles because correction of the hyoid muscles, described on page 418, eliminates many of the EID findings.

Ocular lock, discussed on page 172, is a part of neurologic disorganization as revealed by applied kinesiology testing. It does not appear to be part of the EID mechanism. Ocular lock seems to relate to whether the two sides of the body function well together, and to cranial faults. EID appears to be an adaptation to postural distortion. With ocular lock almost any indicator muscle will weaken, whereas with EID, muscles that weaken are associated with the patient's specific conditions. Also, muscle weakness from the ocular lock test is not cancelled by paralleling the hyoid bone to the eye position, as in EID.

In addition to finding subclinical faults, determining a patient's EID position at the beginning of each examination helps evaluate whether therapeutic efforts were effectively applied. Often one can strengthen a muscle by neurolymphatic reflex, subluxation correction, or any of the many other treatment methods. Sometimes when the eyes are moved into distortion, evidence of the active reflex or subluxation returns, indicating further therapeutic efforts are needed. One simply reapplies the treatment that was effective before retesting with EID.

It may be difficult to determine the major postural distortion. For example, is the head low and to the right or high and to the left? When body modules are distorted in opposite directions, which is the major distortion? The head may be low on the right, and the pelvis high on the right. Schmitt[43] found that when the eyes move in the direction opposite EID, i.e., "eyes out of distortion" (EOD), there is strengthening of an associated muscle that is weak in the clear. An associated muscle is a postural muscle that is part of the distortion. The strengthening is hypothesized to result from increasing the compensatory action of the eye position that occurs because of the distortion. This assumes that EID position takes away the body's pattern of eye compensation for the distortion, and is the cause of muscles weakening with eyes into distortion. Simply stated, EOD improves the body's eye compensation to the distortion, and EID takes away the body's adaptation.

The EOD method for determining the position in which the eyes should turn for EID is best done using the temporal sphenoidal line indicators to find the muscles probably involved. Schmitt uses the example of TS line indicators for psoas and latissimus dorsi weakness on the left and a pectoralis major (sternal division) weakness on the right. Testing all the muscles yields strength, with the exception of the latissimus dorsi on the left. Using the left latissimus dorsi that is weak in the clear, the eyes are turned into the various positions until one is found that causes the muscle to strengthen. If the muscle strengthens with the eyes up and to the left but is weak in every other position, eye position is the compensatory factor for improved muscle function (EOD). The direction of EID, then, would be eyes down and to the right, moving the eyes away from the compensatory position. When the eyes are in this EID position, the psoas and pectoralis major (sternal division) weaken and the latissimus dorsi returns to weakness.

In the example presented, all the muscles exhibited as positive on the TS line changed with eye movement; this does not always occur. There may be a local involvement causing a muscle to test weak; thus a muscle may not necessarily be a part of the EID pattern. It may require treatment to the neuromuscular spindle cell, origin and insertion, or other factor(s).

EID is a valuable asset in applied kinesiology diagnosis in uncovering subclinical problems. It is often used when a patient has responded well to treatment but reaches a certain plateau where results begin to level off. When there is still an obvious need for additional correction as observed by the symptomatic complex and/or structural distortion still present, evaluate with EID to find hidden conditions.

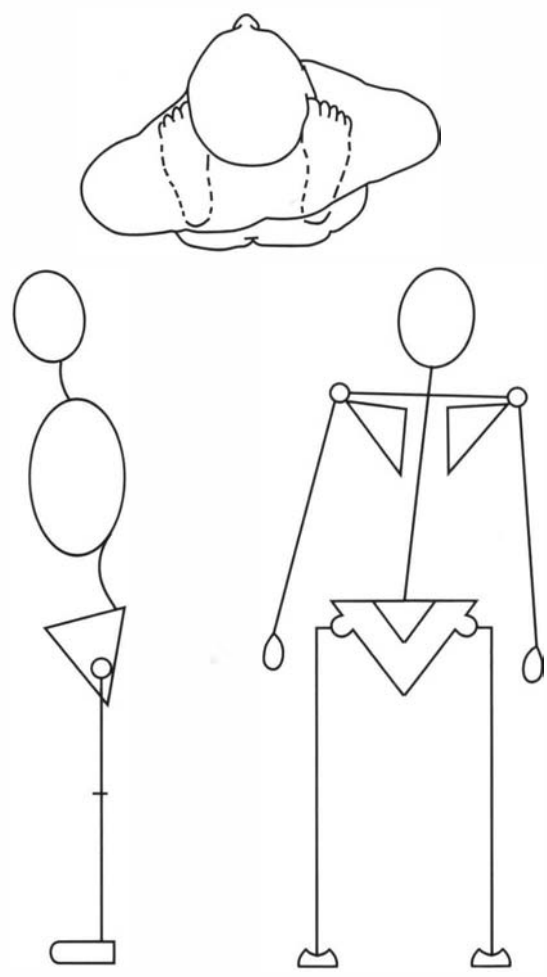
EID is routinely used when testing for certain conditions, such as applied kinesiology's beginning and ending meridian technique. Its use in examining specific conditions is discussed with the condition.

Body Into Distortion (BID)

An examination technique similar to eyes into distortion is "body into distortion" (BID). It has long been noted in applied kinesiology that examination findings may be different when the patient is standing or sitting than when lying prone or supine. This has been related to weight-bearing factors, such as subluxations in the feet and pelvis. A basic examination principle is to examine the person in the way in which he lives and in the manner in which symptoms develop. People do not live lying prone or supine on an examination or adjusting table.

Goodheart[17] observed that disorganization, structural faults, active reflexes, and other factors become evident when a patient is examined in the same postural distortion that is present when he stands. Examining him in this manner is called "body into distortion." It is a relatively simple matter to observe the postural distortion of an individual while standing, then exaggerate that position when he is lying prone or supine. The distortion may be a lateral tilt of the pelvis with opposite tilt of the head. There may be contralateral rotation of the pelvis and shoulders. When the patient is on the examining table, the standing posture of tilt and rotation is exaggerated. The physician passively flexes the patient at the pelvis or neck to exaggerate the standing posture on the table. DeJarnette blocks or a pillow can be placed under one side of the pelvis or shoulder girdle for rotation. It may be necessary to put a pillow under the head if it is in front of the plumb line when the patient stands.

The usual procedure is to first examine and treat the patient without BID. The method is effectively used when examination findings are negative or if there is lack of continued improvement when a patient reaches a plateau. Another indication to examine with BID is lack of improvement of the patient's postural distortion, even though therapy localization, challenge, and other examination procedures are negative. Pitch, roll, yaw and tilt (PRYT), discussed later, are common findings when there is postural distortion. The physician may correct one or more factors with the PRYT technique and still find the patient symptomatic and posturally distorted. Re-examining the patient for PRYT with BID may disclose additional corrective efforts necessary for what has just been corrected; there may be additional positive findings in PRYT.



2—45. A patient with the above distortion is placed on the table angled toward the right from the pelvis. Pillows under the right shoulder and head exaggerate the distortions.

The BID position supplies information about positioning the patient when spinal adjusting is indicated. A basic rule is to always adjust toward the plumb line. When the spine is left of the plumb line, adjust from left to right. Use vertebral challenge, discussed in Chapter 3, to determine the best vertebral contact.

Origin/Insertion Technique

The first technique used in applied kinesiology to change muscle function was stimulation of the muscle's origin and insertion. In 1964, Goodheart[12] was using manual muscle testing to identify muscle dysfunction involved with poor structural integrity. On one patient he observed a weak serratus anticus muscle. The weakness was paradoxical, because there was no observable atrophy of the muscle in comparison to the opposite side. On closer examination, palpation revealed discrete painful nodules at the origin of the muscle at the ribs. This finding was not present on the opposite serratus anticus, which had normal strength. To determine if these nodules

were possible trigger points affecting the muscle's strength, Goodheart deeply massaged the nodules. Upon immediate re-testing, he found the muscle had gained strength to approximately 70% of the opposite side. This was the birth of applied kinesiology and the ability to immediately change muscle function.

When muscle weakness was found on manual muscle testing of other patients, the nodules at the origin or insertion were often present. When the nodules were deeply massaged, the muscle usually returned to normal strength, with lasting results. When the nodules were present, the patient often revealed recent or chronic trauma to the area. Goodheart hypothesized that the weakness was due to a microavulsion of the tendon from the periosteum. It is possible that some of the early results from use of origin/insertion technique came about from manipulation of the Golgi tendon organ, discussed later. The heavy massage used in origin/insertion technique possibly strengthens the muscle by vibrotactile stimulation. The alpha motor neuron is facilitated by vibrotactile stimulation as indicated by increase of the F wave,[47,51] which is an antidromic volley that excites the alpha motor neurons following peripheral nerve stimulation. The well-established effects of vibrotactile stimulation develop from stimulation of less than 100 Hz, which is in the range of digital stimulation.

Origin/insertion technique continues to be a viable therapeutic approach. It is applicable when nodules are present. There will usually be a history of trauma to the area. The point requiring heavy digital stimulation will have a positive therapy localization, which will be absent after effective treatment. The muscle should test strong following treatment; if it does not, further evaluation and treatment are necessary.

Many early techniques in applied kinesiology have been refined and found to have therapeutic benefits not recognized earlier. So it is with the origin/insertion technique. It is part of the correction when a person's strong muscle becomes weak after repeated muscle contraction, discussed in Chapter 6.

Neurolymphatic Reflexes

Frank Chapman, D.O., discovered the "Chapman reflexes" in the 1930s.[39] He correlated the reflexes with specific organs and glands, and with different types of health problems. Most of the reflexes are as described by Chapman and are now called neurolymphatic reflexes (NL) in applied kinesiology. Locations and associations of additional reflexes have been added to those described by Chapman.

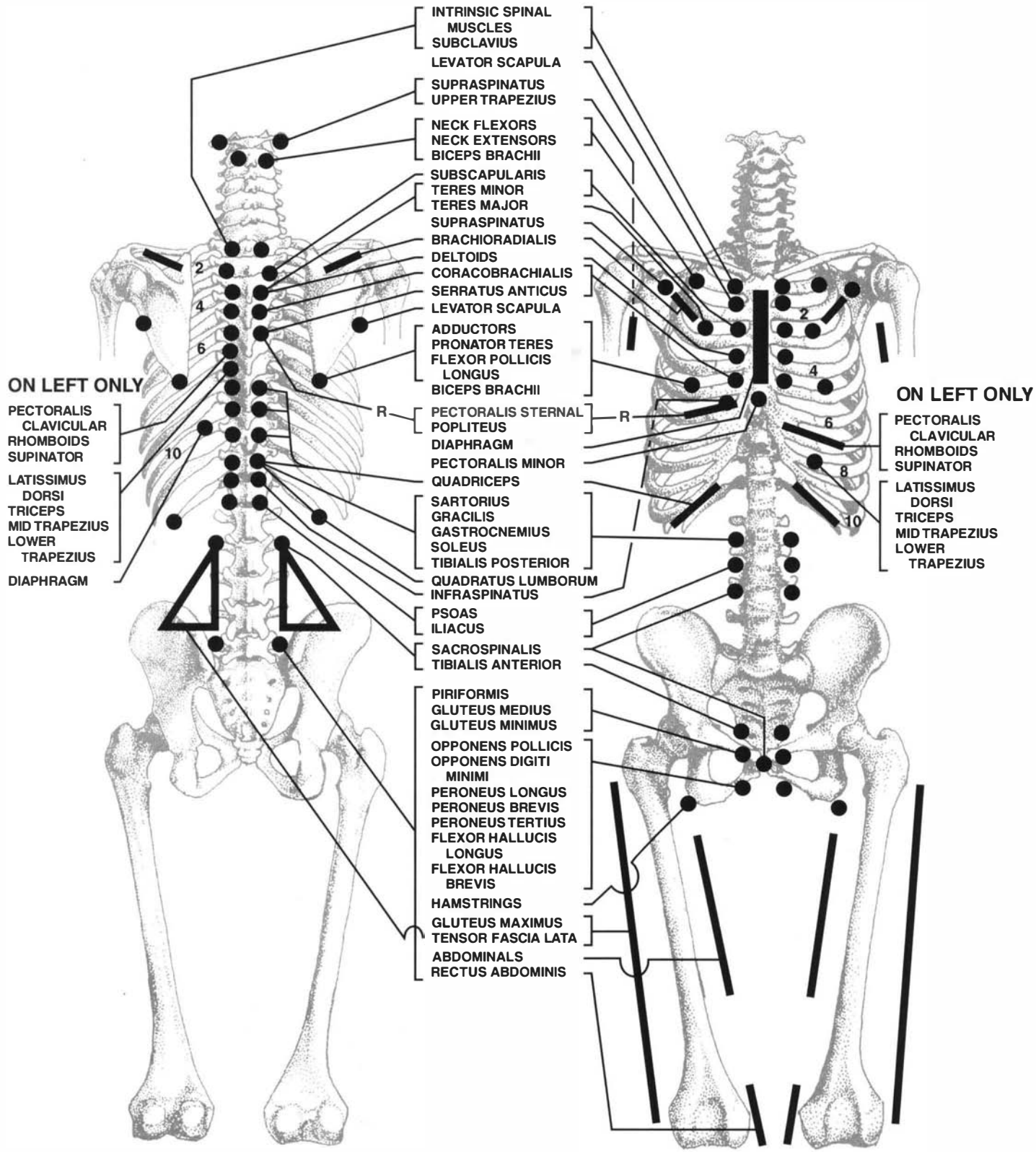
The reflexes are located primarily along the anterior intercostal spaces, on the anterior abdomen down to the pubis, and posteriorly along the spinal column. There are some reflexes located on the legs and arms (figure 2—46). Active neurolymphatic reflexes can usually be palpated and are quite tender on the anterior. The tenderness is usually in direct ratio to the chronicity and severity of the condition. Most reflexes are approximately 3 cm in diameter; some of the reflexes are linear.

Palpatory evidence of the neurolymphatic reflex changes with chronicity. The less chronic active neurolymphatic reflex has a puffy, doughy feeling over the entire reflex area. With more chronicity, the doughiness concentrates into globules the size of small lima beans. The most chronic active reflex feels like many small BBs located in the subcutaneous fat. The posterior reflexes are usually less tender and more difficult to determine a change in tissue consistency.

Treatment of the neurolymphatic reflex is accomplished with rotatory massage by the physician's fingertips. Originally the pressure used was described as light, approximately the amount you can stand on your eyeball. It has since been found that heavier and deeper stimulation, sometimes for a prolonged time, is necessary to clear the reflex. Generally the more pressure used, the less time of stimulation necessary.

Chapman's original observation of the reflexes was associated with organ and gland function. A major breakthrough in the use of the neurolymphatic reflex was accomplished by Goodheart when he correlated the neurolymphatic organ association with specific muscle association. Muscle testing provides an objective method for determining the need for and success of NL stimulation. Upon successful stimulation of the NL reflex, there will be a dramatic improvement of the associated muscle on manual muscle testing. The improvement is a lasting one if all corrections are made and there is no other factor in the patient's health causing reactivation of the reflex. For example, if a patient is on a diet harmful to good colon function, there will be a repeated weakening of the tensor fascia lata muscle, associated with the colon, on subsequent patient visits. The recurrence of active NL reflexes gives the doctor indication to look deeper for the causative factor.

Review the section on therapy localization on page 37 for additional information on determining when effective neurolymphatic treatment has been accomplished.



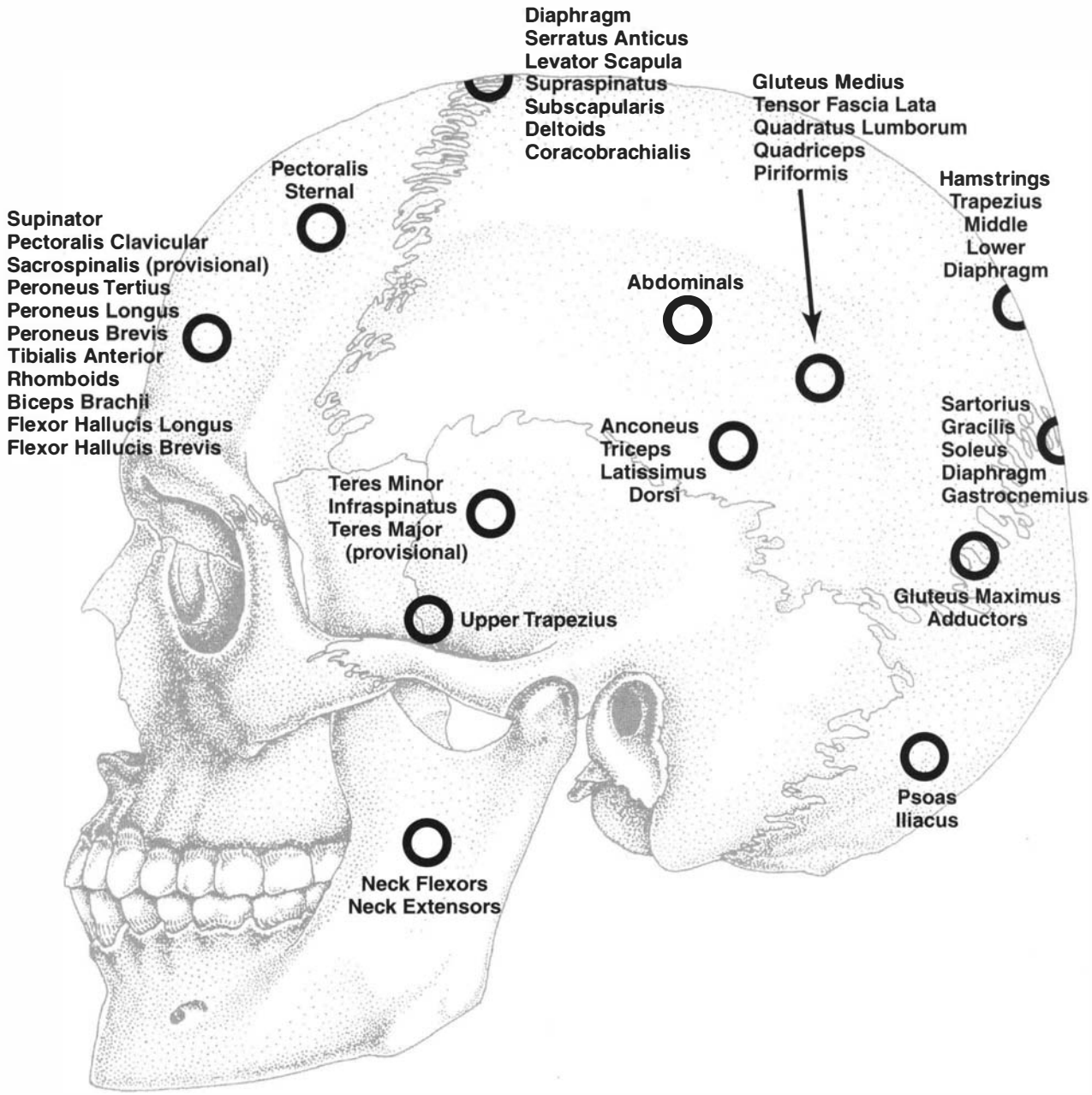
2—46. Neurolymphatic reflexes.

Neurovascular Reflexes

In the early 1930s a chiropractor in California, Dr. Terence Bennett,[31,32] discovered locations about the head that he felt influenced the vascularity of different organs and structures.

During the mid-1960s, Goodheart found that he could improve muscle function, as determined by manual testing, by stimulating the Bennett reflexes. These reflexes became known in applied kinesiology as neurovascular reflexes (NV). A specific muscle responded only to one reflex, but most reflexes influenced more than one muscle. Bennett's reflexes are primarily on the anterior surface of the trunk and on the head. The reflexes on the head are those used in applied kinesiology, with few exceptions.

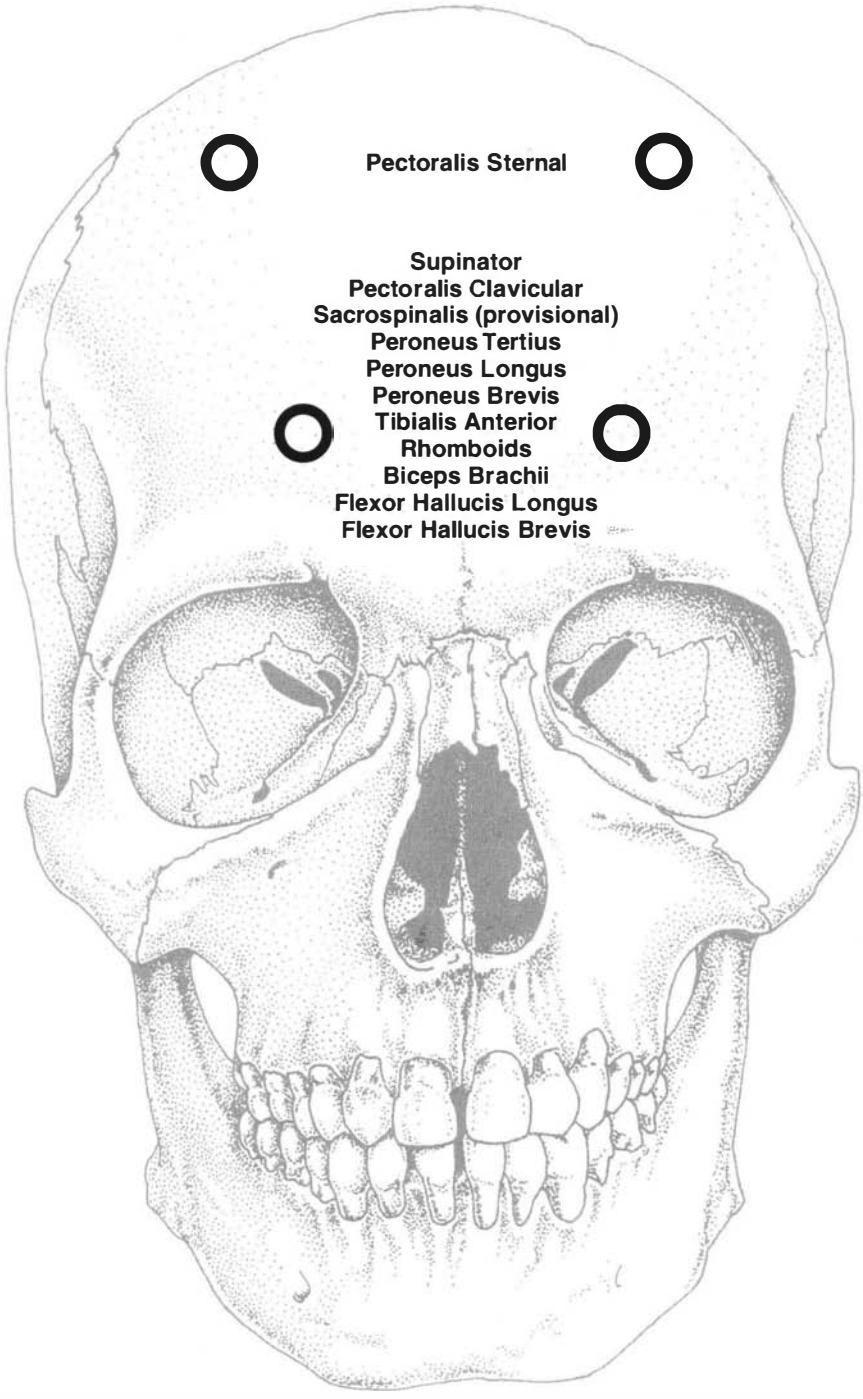
Goodheart's association of muscle dysfunction with active NV reflexes added an objective evaluation of when the NV reflexes needed treatment, and whether the treatment was effective. The development of therapy localization in 1973 enabled doctors to further diagnose activity of the neurovascular reflexes and enhanced Bennett's and Goodheart's work.



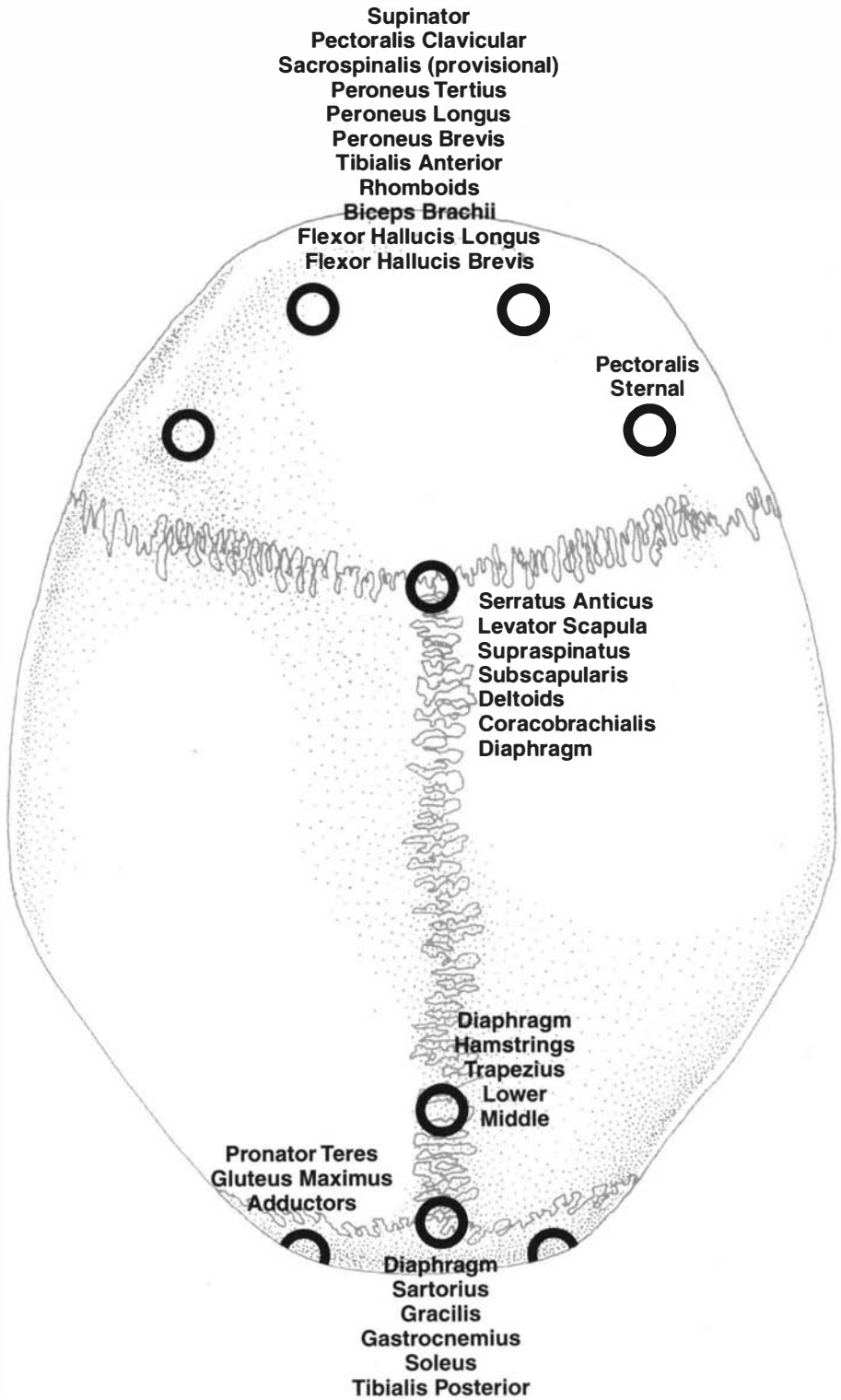
2—47. Lateral view.

The association of the NV reflex appears to pertain to the ectodermal unfolding of the embryo, relating the general nervous system with the skin receptors.

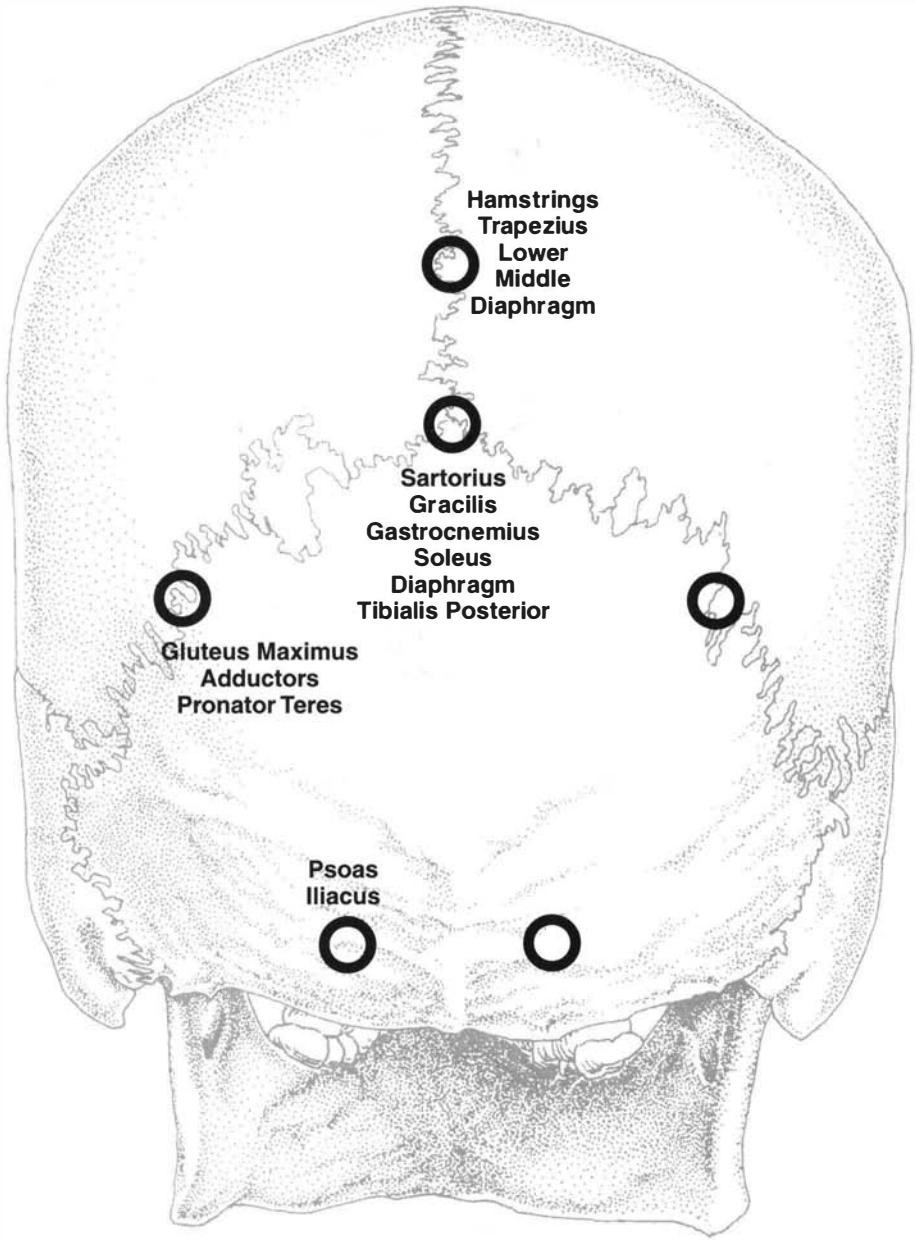
The neurovascular reflex is stimulated by a very light touch. The physician uses his fingertips to contact the NV point and gives a slight tug to the skin. When the reflex is actively treated, skin pulsation is felt. If the pulsation is not readily felt, change the vector of tissue tug until it is. Once pulsation is felt, the reflex is usually held for approximately twenty seconds. Improvement of the associated muscle function on manual muscle testing and therapy localization is evidence of effective treatment. (See therapy localization, page 37, for additional information on length of stimulation and diagnosis.) With some cases it may be necessary to hold the stimulation for up to five minutes before evidence of effective treatment is present.



2—48. Anterior view.



2—49. Superior view.



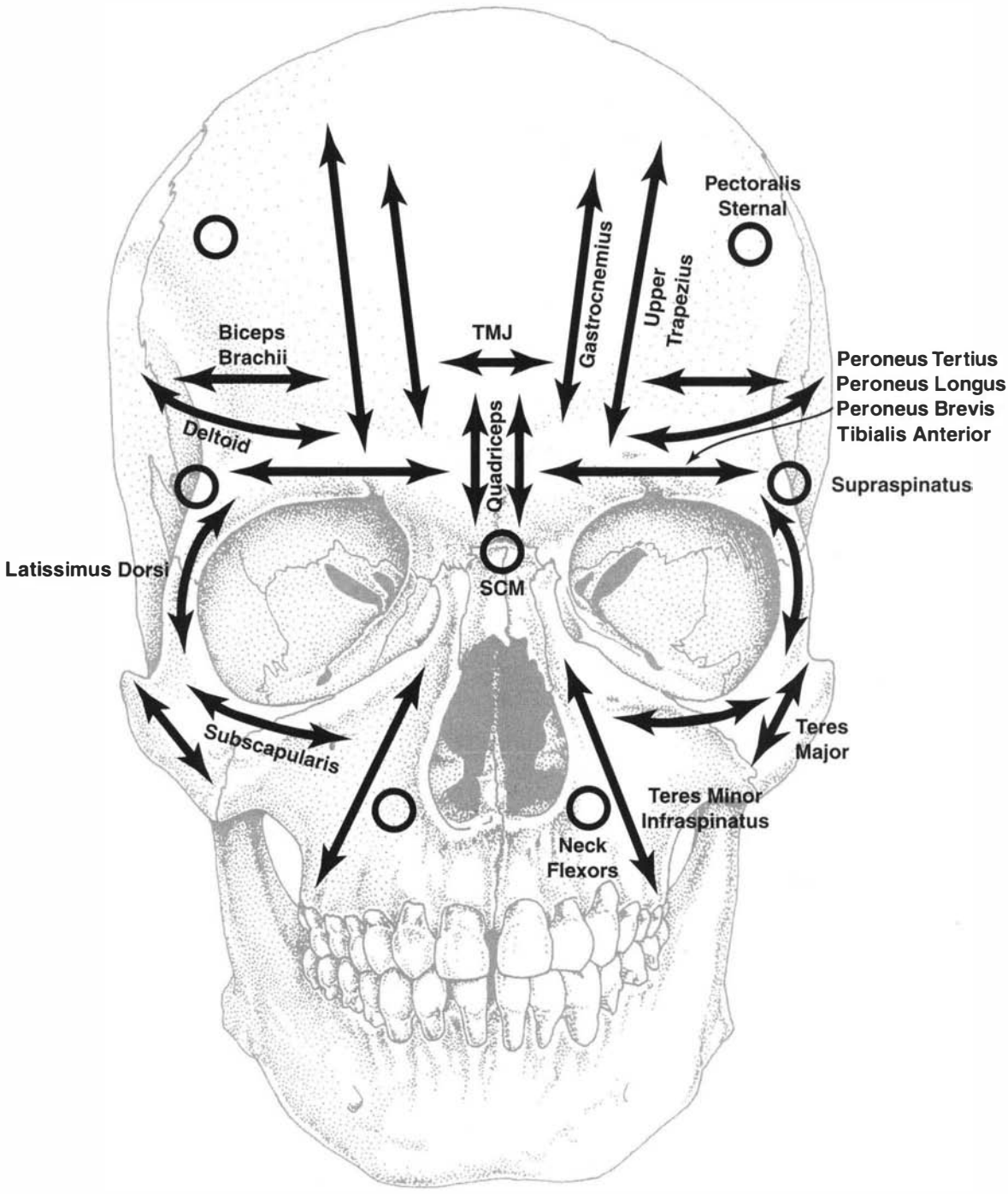
2—50. Posterior view.

Stress Receptors

Skin reflexes located about the cranium are known as "stress receptors" in applied kinesiology. These reflexes respond to the physician's linear digital stimulation while the patient takes a certain phase of respiration.

These reflexes can improve muscles that test weak and, in addition, can reduce activity in hypertonic muscles that tend to cramp or are actually in spasm. These were the first reflexes in a therapeutic approach used in applied kinesiology that dealt with the hypertonic muscle. Besides improving muscle function, there is also influence on organs and glands through a somatovisceral reflex. Each reflex is associated with a muscle; the organ or gland influenced is one associated with the muscle.

A stress receptor may be active for no apparent

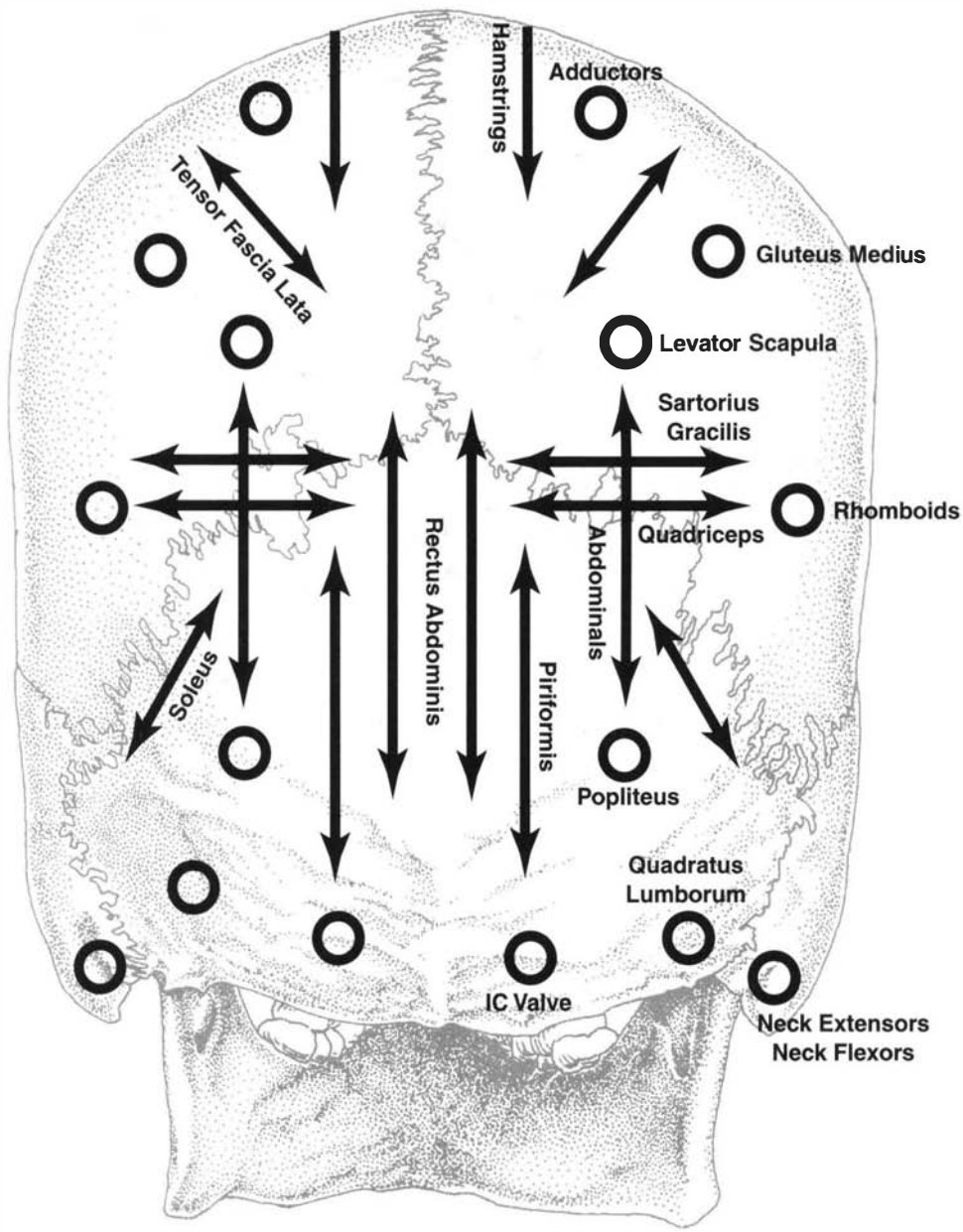


2—51. Anterior view.

reason, but there is usually history of trauma to the reflex area. The history may be recent or chronic.

Active stress receptors can be located with therapy localization. The usual approaches to therapy localization are followed. A muscle weak as a result of an active stress receptor will strengthen; a hypertonic muscle will weaken when the receptor is therapy localized.

The direction of linear stimulation to a stress receptor is determined by challenge. Challenge simply means applying digital pressure over the stress receptor and testing the associated muscle for change. Pressure is applied by the physician sliding his finger over the skin of the stress receptor. Some stress receptors are longitudinal, as indicated on the charts, and typically

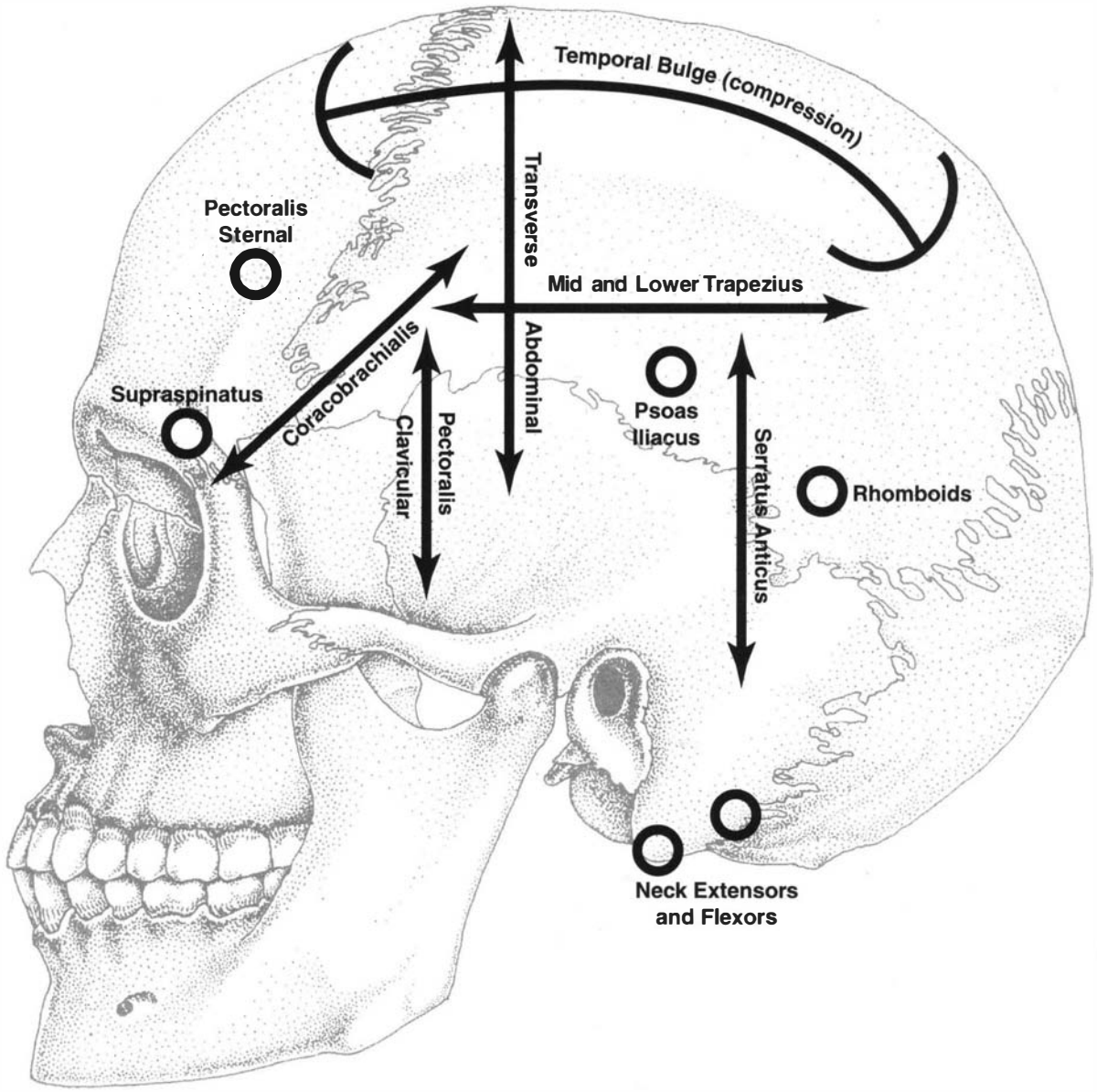


2—52. Posterior view.

respond to stimulation in only two directions. Some receptors are represented by circles on the charts. These respond to a shorter linear stimulation that can be in any direction.

A positive challenge occurs when the digital stimulation changes the muscle function; that is, a hypertonic muscle weakens or a weak muscle tests strong.

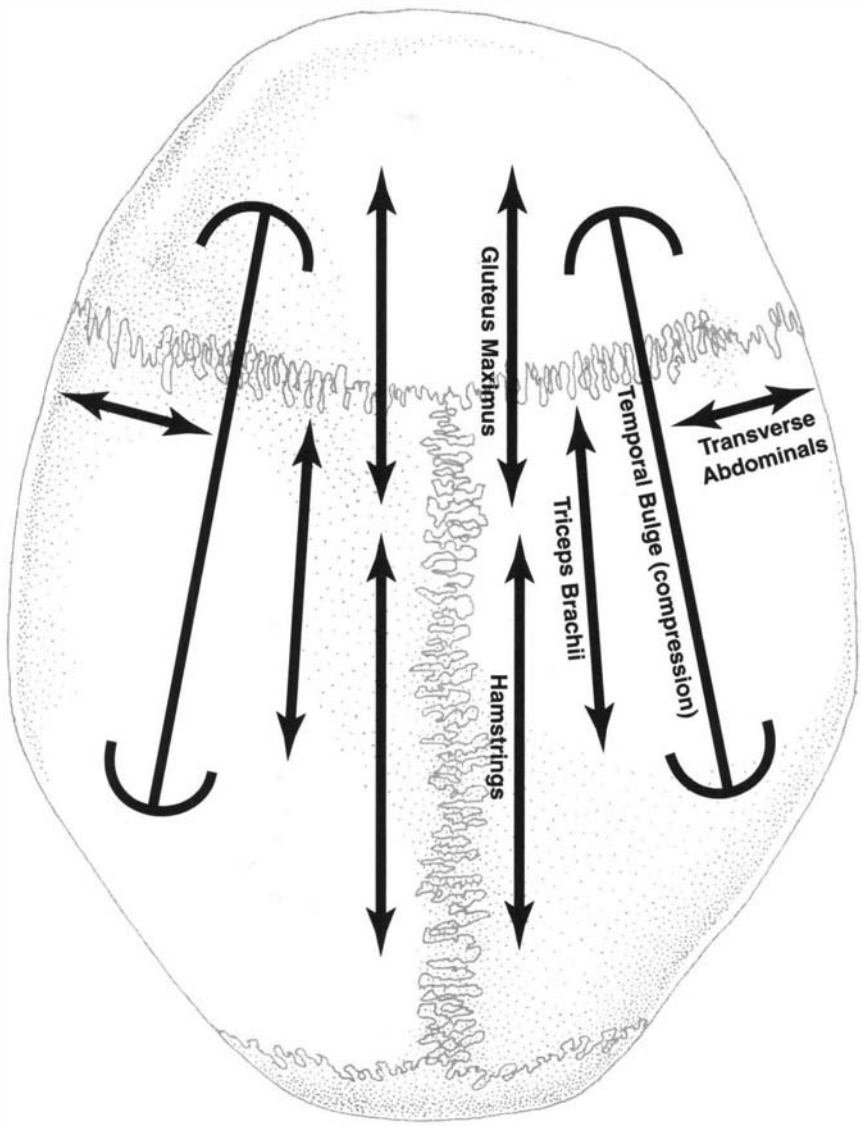
There is a respiratory quality to the stress receptor that should be applied during the therapeutic digital pressure. To determine the phase of respiration, find the respiration that abolishes the results of the challenge. For example, if a muscle tests weak and is then strengthened by stress receptor challenge, have the patient take a deep inspiration. If the muscle again tests weak, that is the phase of respiration that should be taken during digital stimulation of the receptor. On the other hand, if a muscle is hypertonic and the challenge causes the muscle to weaken, find the phase of respiration — either



2—53. Lateral view.

inspiration or expiration — which causes the muscle to again test strong immediately after it weakened from the challenge.

Apply digital pressure in the direction of positive challenge with the phase of respiration that abolished the challenge; repeat with four or five respirations, using 1-7 kg of pressure. After this treatment the muscle should test normal, and there should be no positive therapy localization or positive challenge over the stress receptor. There is one stress receptor that differs from the others; it is associated with the temporal bulge cranial fault and bilateral pectoralis major (clavicular division) weakness. This stress receptor is located bilaterally, mostly parallel to the sagittal suture. Rather than a single linear contact, it requires contact on both ends of the stress receptor, with compression directed toward the center of the reflex area.



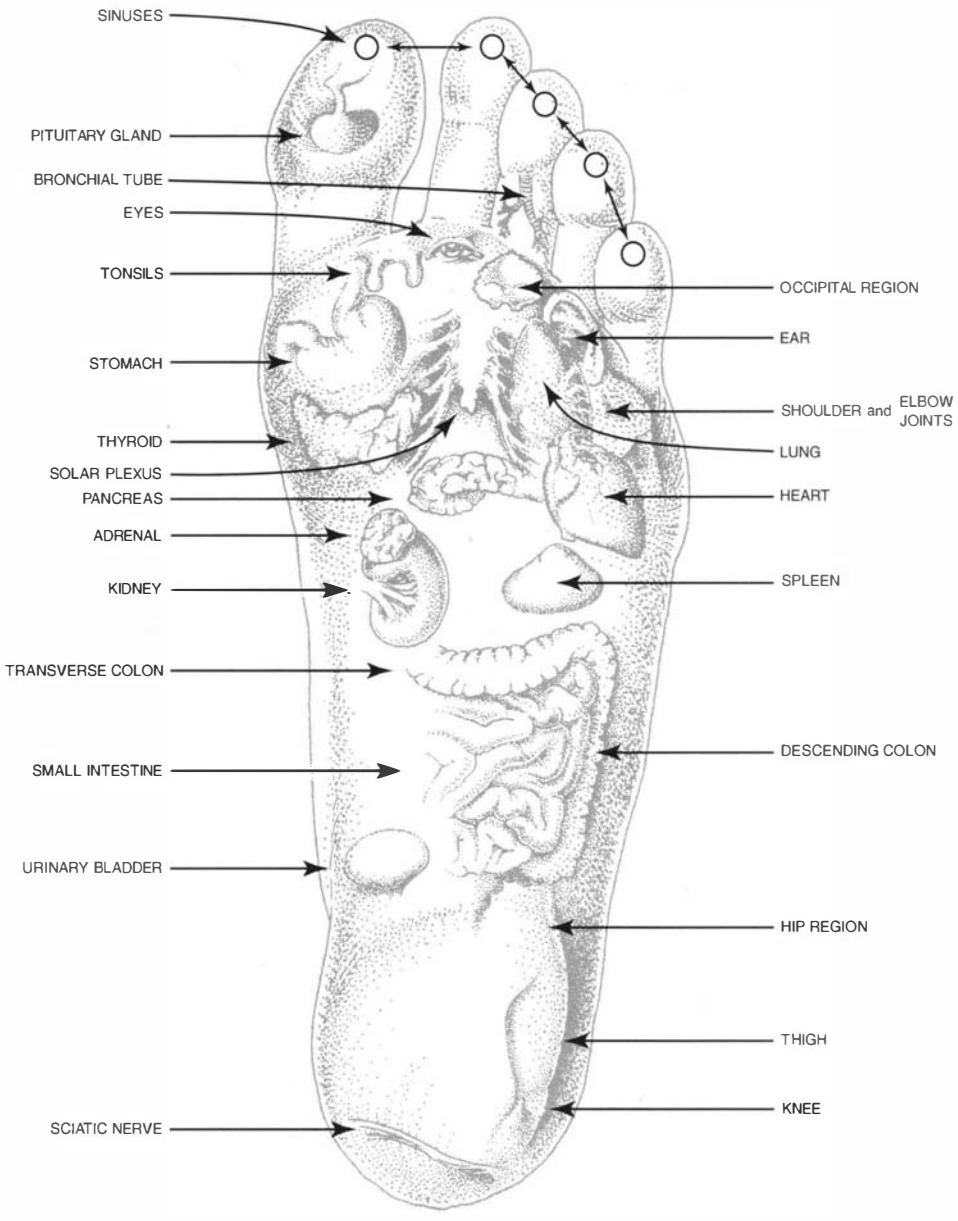
2—54. Superior view.

Foot Reflexes

In several areas of the body there are homuncular (little man) representations of the body. These are seen in the brain, ear, foot, and perhaps elsewhere such as some of the little understood reflexes used in applied kinesiology. Initially this author totally disbelieved in the association of these representations with body function. As more knowledge is gained, better explanations are bound to be developed. We have seen what appears to be a correlation with the colon reflex of the foot, which is in the vicinity of the cuboid bone. When this is subluxated, it causes the tensor fascia lata muscle to test weak. In any event, active foot reflexes are often associated with foot dysfunction, with or without foot subluxations.

The foot is a complex, dynamic mechanism subjected to considerable abuse. There are in-depth examination and treatment procedures in applied kinesiology for foot dysfunction. Discussion here is limited to what appear to be neurologic reflexes that affect muscle function, as observed by manual muscle testing; they may also influence organs or glands.

Many foot reflex charts have been published by

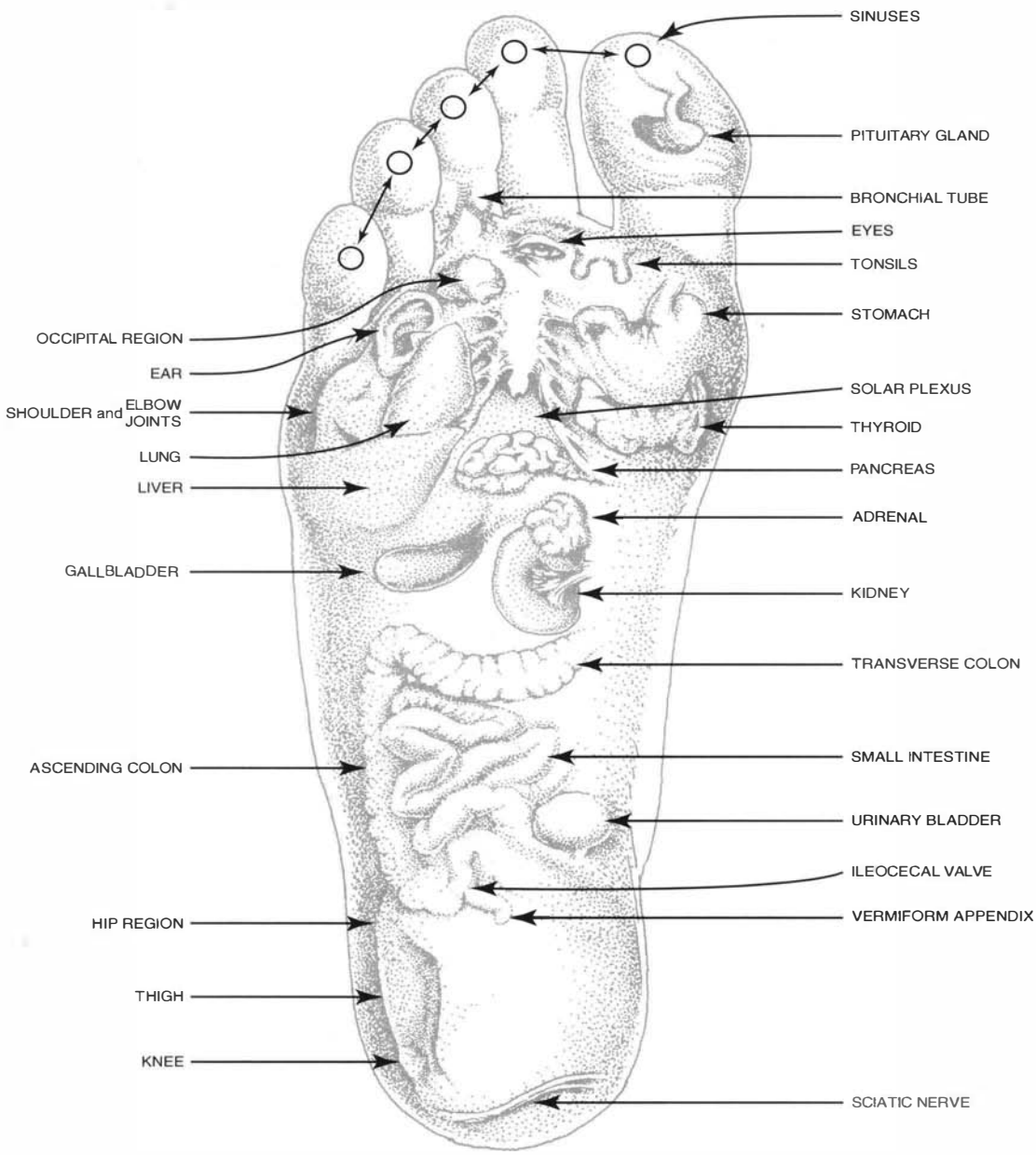


2—55. Left foot.

various authors. The explanation of foot reflexology varies from author to author. Generally the continued use of foot reflexes has been on an empiric basis. Foot reflexes listed here have been found to associate on a muscle-organ/gland basis with muscle dysfunction.

Although stimulation of foot reflexes will change muscle function, one should thoroughly evaluate the feet for subluxations, extended pronation, and intrinsic and extrinsic muscle dysfunction. If there are active reflexes and there is foot dysfunction, treatment of the reflex by stimulation will probably not provide lasting benefit. The key is to correct the foot subluxation, extended pronation, or other foot dysfunction. After the foot correction, there will most likely be no active foot reflexes.

Active foot reflexes have positive therapy localization. The best way to evaluate the foot reflexes is to stimulate the reflex and re-test a previously weak muscle thought to be associated with it. For example, if the psoas muscle is weak, one can stimulate the kidney reflex on the sole of the foot by digital goading and then re-test the psoas. If the psoas is associated with the active foot reflex, the muscle will now test strong. Evidence of adequate stimulation is obtained by negative therapy localization and adequate response of the associated muscle.



2—56. Right foot.

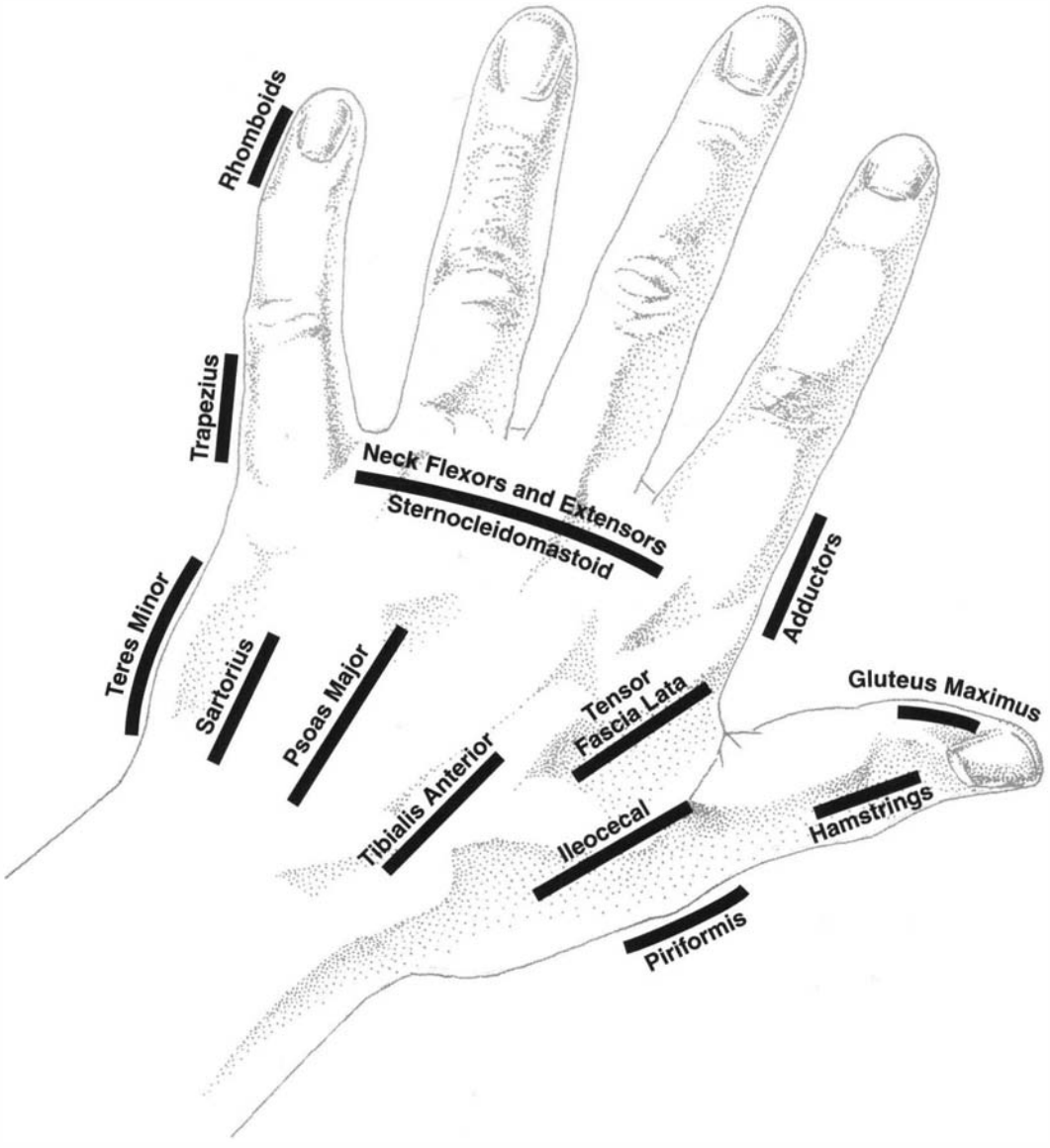
Hand Reflexes

There are reflexes on the hands that relate with most of the major muscles of the body. When active, the reflexes show positive therapy localization and challenge.

Frequently hand reflex treatment is applicable for individuals who use their hands in a manner that subjects them to trauma, such as carpenters hammering or mechanics pulling hard on wrenches.

Body language indicating the need to evaluate hand reflexes is seen when an individual develops symptoms in association with using the hand in a strenuous manner. A mechanic may complain of shoulder pain when pulling on wrenches, or a carpenter may have neck pain develop from hammering.

When a hand reflex is active, therapy localization will cause an associated muscle to strengthen or a general indicator muscle to weaken. Challenge is done to determine the direction of treatment. There is one lin-

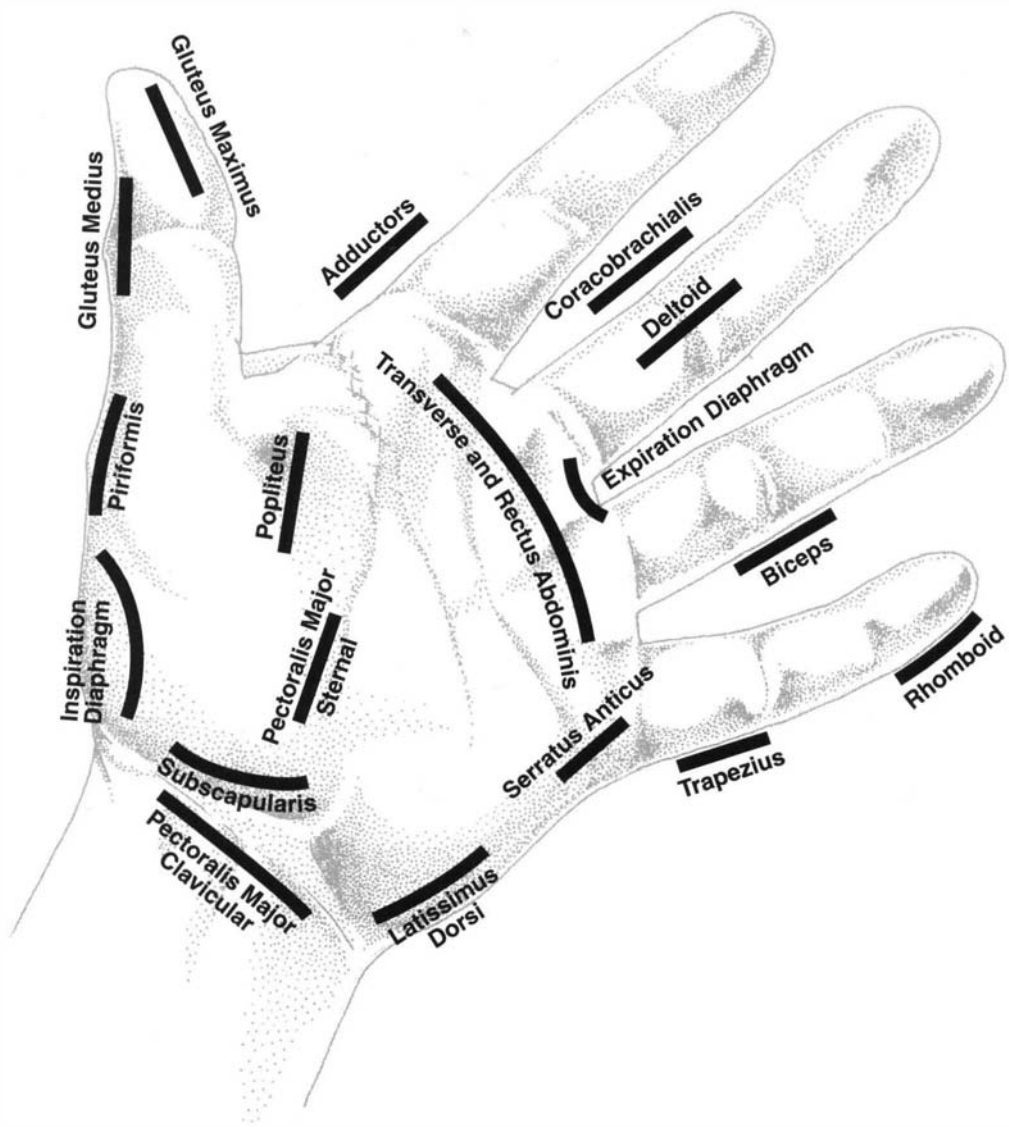


2—57. Dorsal surface.

ear direction of stimulation that will be applicable. Challenge is accomplished by digitally stimulating the length of the reflex. Pressure applied in the sliding stimulation is 2-7 kg. Positive challenge will cause an associated weak muscle to temporarily test strong.

There is a phase of respiration applicable to the positive hand reflex that is determined by having the patient take a phase of respiration and hold it immediately following a positive challenge that caused an indicator muscle to weaken, i.e., an indicator muscle weakens with the challenge and then tests strong when deep inspiration or expiration is held. Treatment is accomplished by stimulating the length of the hand receptor in the direction that caused positive challenge, while the patient takes the phase of respiration that abolished the challenge.

Treatment to the reflex will improve muscle function; however, if there is hand dysfunction, such as subluxations or intrinsic or extrinsic muscle dysfunction, the reflex will again become active with use of the hand. The hand should be thoroughly examined using applied kinesiology techniques, and functional disturbances should be corrected.



2—58. Palmar surface.

Proprioceptors

Sherrington[44] classified proprioceptors as nerve endings in the organs that are stimulated by actions of the body itself. Their afferent input to the central nervous system is responsible for the organization of continuing body actions. As one moves, there is facilitation and inhibition of muscles as a result of the initial action. Normally these muscle changes are predictable, as described in the facilitation and inhibition of the shoulder muscles during gait.

Manual muscle testing may reveal that a muscle fails to function properly under certain conditions. Further evaluation may indicate that proprioceptors are being improperly stimulated, thereby supplying the central nervous system with inappropriate information. The central nervous system can only interpret the information provided by the afferent system; if that information is false, it cannot provide the proper facilitation or inhibition to muscles.

There are techniques in applied kinesiology that affect muscle, joint, skin, and equilibrium proprioceptors. Treatment is designed to eliminate inappropriate stimulation to or by the receptors; when it is effective, normal muscle function is restored as determined by manual muscle tests.

Joint Receptors

Nerve receptors involved with position sense reside in the joints and their ligaments. In addition to position sense, Freeman and Wyke[10] demonstrated in cats that stimulation to the joint receptors affects muscle function. To eliminate stimulation to other receptors, they resected the skin over the area, separated the muscles by tenotomy, and — in some cases — divided the posterior deep fascia. They also used various methods of anesthesia in the ankle joint capsule to determine the source of neurologic activity. They found that when dorsiflexion is passively applied to the ankle joint, there is EMG activity of the gastrocnemius muscle with coincident depression of tibialis anterior activity. With plantar flexion at the ankle, gastrocnemius activity is progressively abolished as the tibialis anterior activity is augmented. The investigation to determine that the activity comes from the ankle ligaments led them to believe "...that physiological and pathological alterations in the articular mechanoreceptor input are capable of reciprocal facilitation and inhibition of the *gamma* motor neurons related to muscle spindles in the extensor and flexor muscles of the limbs...." They go on, "This may explain why patients who have recovered from the structural effects of injury to individual joint capsules may show persisting abnormality in the postural reflex activity of the muscles operating over the affected joint." From an applied kinesiology point of view, subluxations of bones in the feet or ankles, as well as other extraspinal subluxations, can improperly stimulate the mechanoreceptors and cause muscle facilitation or inhibition that is not in keeping with body needs. This closely parallels the thinking of Freeman and Wyke.

In applied kinesiology it is clinically observed that there is more dysfunction of muscles closely associated with an extraspinal subluxation than with those muscles remote from it; however, it is observed that remote muscles may improve their function after the subluxation is corrected.

Joint position change affecting remote muscles is demonstrated in a study by Nashner.[36] The tibialis anterior, quadriceps, gastrocnemius, hamstrings, and sacrospinalis were evaluated by electromyography with the subject standing on a platform. The platform was tilted to induce dorsiflexion or plantar flexion of the ankle. This influenced the joint receptors and also the muscle and skin receptors. Body sway thus induced caused activation of the muscles evaluated. There is organization in the temporal pattern of muscle activation, depending on the status of the body and the purpose required. Sherrington[7] points this out, stating that the action taken "...is in part determined by the posture already obtaining in the limb at the time of the application of the stimulus." This means that facilitation or inhibition of muscles may be in different order or of different muscles, depending on the limb's position at the time of stimulus. In Nashner's study,[36] when the body sway was induced by the tilting platform there was distal-to-proximal activation of the muscles to maintain postural balance. On the other hand, when AP sway was initiated voluntarily by the subject, the sequence of activation reversed, to proximal-to-distal muscle pairs. This shows that when making an applied kinesiology evaluation before and after a therapeutic attempt, one must have the patient in the same position for both tests. Also, the dependable function of muscles changes with different body positions.

Under normal conditions, when stimulation is applied to extraspinal articulations by motion or a mechanical shock, muscles are stimulated or inhibited in keeping with the body's needs, and homeostasis rapidly returns. If the articulation is in subluxation, motion may cause inappropriate stimulation to the receptors, with unpredictable muscle facilitation or inhibition. Applied kinesiology uses two methods to evaluate extraspinal joint function and its relation to muscle function.

Shock Absorber Test

The shock absorber test is administered by applying a mechanical shocking stimulus to the articulation, and then testing a previously strong indicator muscle for weakening. The shock absorber test can be applied to any diarthrodial joint of the body. An example is for the examiner to strike the sole of the patient's foot with his closed fist, then test a previously strong muscle for weakening. When there is normal joint function, the muscle will remain strong; if there is a subluxation, such as the talus in the ankle mortise, the indicator muscle will test weak. The weakness will last from several seconds to minutes; then it will spontaneously recover. After the subluxation is evaluated and corrected, the shock absorber test will no longer cause the indicator muscle to weaken. The best indicator muscle to test is one associated with the articulation. It may cross the articulation or be remotely associated. An example of remote muscle association to the foot is shoulder flexors and extensors, which are intricately involved with gait motion, as is ankle motion.

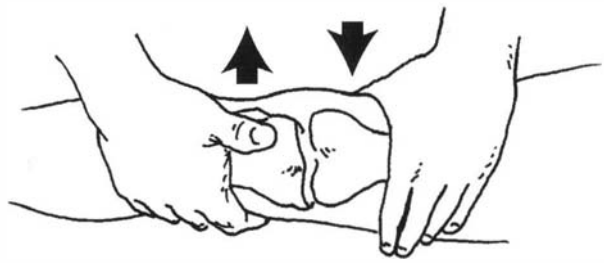
Usually the striking force can be applied in a general manner. A quick, shocking-type force offers the best evaluation. When there are numerous articulations potentially involved, shocking forces in various vectors may be necessary to elicit a response. If the shock absorber test is used as a screening device, a subluxation may be missed by improper application. For example, the foot has many articulations that could potentially be subluxated. A general shock to the plantar surface may not cause a shock into the metatarsals, calcaneus, or other subluxated joints. The cuneiforms, navicular, and talus — which are not subluxated — may receive the brunt of the shock. The test would fail to reveal the subluxation.

Extraspinal Subluxation Challenge

When an extraspinal articulation is subluxated, there will be muscles that test weak as a result. Frequently the involved muscles are directly associated with the articulation, but they can be remote from the subluxation. A positive challenge for an extravertebral subluxation occurs when force applied to the articulation reduces the subluxation. When the correct vector of force is applied, the dysfunctioning muscles will temporarily test strong.

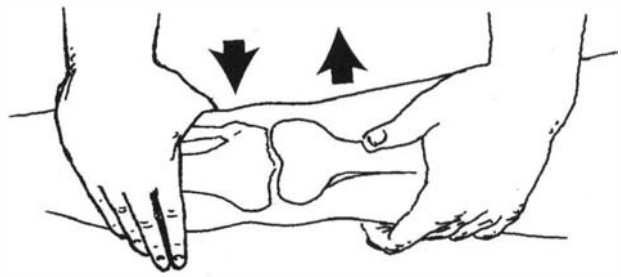
The challenge force is designed to push one portion of the articulation into a different relationship with the other aspect. If the change of relationship reduces the subluxation, the associated muscles will strengthen; if the relationship worsens, previously strong indicator muscles will weaken.

Extraspinal subluxations are adjusted in the direction of challenge that caused associated weak muscles to test strong. This is opposite the rebound challenge of spinal subluxations, discussed later. There will be one specific vector of force that causes the maximum strengthening of the associated muscles. Because subluxations are specific, the adjustive thrust should be specific. It is possible that the usual manipulative techniques will not fit with the optimal challenge. In this case, one must improvise and develop techniques to meet the patient's needs. The patient's subluxations do not always conform to the available techniques.



2—59. Pressure in the direction of the arrows will improve the position of the subluxation and strengthen a muscle which is weak because of the subluxation.

It is best to find a muscle that tests weak as a result of the subluxation and evaluate with challenge, as indicated above. There are times when pain or some other factor interferes with the testing process. In these unusual situations, the articulation may be challenged to find the vector causing the greatest weakening of an indicator muscle. The articulation is then adjusted in the direction opposite the challenge. The basic rule is to adjust a non-spinal subluxation in the direction of challenge causing an associated weak muscle to strengthen, or opposite the direction that causes a strong non-associated indicator muscle to weaken.



2—60. Pressure in the direction of the arrows will cause an increase in the subluxated position, causing a previously strong indicator muscle to weaken.

Muscle Proprioceptors

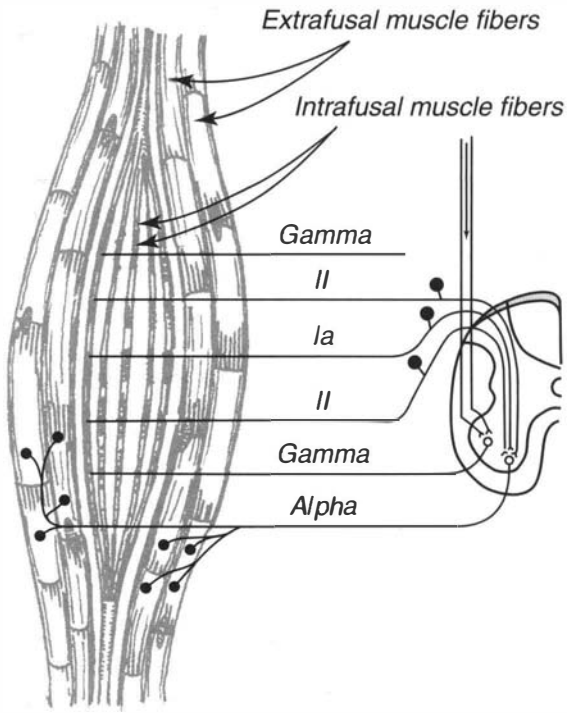
Muscle proprioceptors consist of Golgi tendon organs and neuromuscular spindle cells. They are instrumental in regulating the muscle in which they reside, and associated muscles; in addition, they augment the joint receptors in signaling body position sense. Muscle contribution to proprioception is demonstrated by experiments in which the joints and skin of a finger or the whole hand are locally anesthetized.[33] Since the muscles that move the fingers of the hand are not involved because of their remote position, their afferent supply remains intact. The remaining proprioceptive ability of the subject indicates that stretch-sensitive receptors, such as the muscle spindle cells, contribute to joint position sense.

Another experiment that has contributed to the knowledge of a muscle's role in position sense is pulling on the muscle's tendon with the joint immobilized.[34] This has been done experimentally during surgical procedures where structure, such as a finger or hand, is immobilized while the tendon of the muscle that normally moves the digit is pulled. Even though the structure normally moved by the muscle remains stationary, stretching the muscles gives the subject the sensation that the finger or other structure being studied has moved. Since some patients at surgery fail to observe this sensation, McCloskey[34] had his colleagues assist him with an experiment on himself "...in which the tendon of (his) extensor hallucis longus...was exposed under local anesthetic, cut across, and pulled so as to stretch the muscle. This was done while the foot and the joints of the toes were totally immobilized. Without visual or other feedback (he) could readily detect pulls of less than 1 mm imposed on the muscle (at about 2.5 mm/sec), and these felt like plantar flexions of the terminal joint of the big toe; that is, like movements which would normally impose similar stretches on the tested muscle." Further evidence that joint capsule and ligament receptors are not the only factors giving position sense is that when an individual has a complete joint replacement (the capsules and ligaments are removed along with the nerves), there is joint position sense. This surgical procedure leaves the skin basically intact. Studies by Gandevia and McCloskey[11] show that proprioceptive acuity is superior when all receptors are allowed to contribute, and diminished when the muscles are disconnected. Initial applied kinesiology examination and treatment will be directed toward the muscle in which the proprioceptor resides.

Neuromuscular Spindle Cell

Neuromuscular spindles are located throughout the muscle, with a higher concentration in the central belly. Concentration of neuromuscular spindles depends upon the type of muscle in which they are located. In the postural (tonic) muscles they are less concentrated, while they have higher concentration in muscles with more precise control (phasic).

The muscle spindle varies in length from 2-20 mm and is enclosed in a sheath, making a fluid-filled cavity. Within the cavity are three to ten small muscle fibers, called intrafusal to differentiate them from the larger skeletal or extrafusal muscle fibers. The intrafusal muscle fibers are small and do not contribute to the strength of the skeletal muscle. The intrafusal fibers attach to the extrafusal muscle sheath so that the intrafusal fibers stretch or shorten with the extrafusal fibers. The purpose of the neuromuscular spindle cell is to act as a comparator between the extra- and intrafusal fibers. This is accomplished by the efferent and afferent nerve supply to the intrafusal fibers. The



2—61. Neuromuscular spindle cell.

length of the intrafusal fibers is set by the gamma motor neurons. Tension on the intrafusal fibers is reported by the afferent Ia and II fibers. In general, spindle afferents exert an excitatory effect upon the muscle in which they reside, a facilitatory effect upon synergistic muscles, and an inhibitory effect upon antagonist muscles.

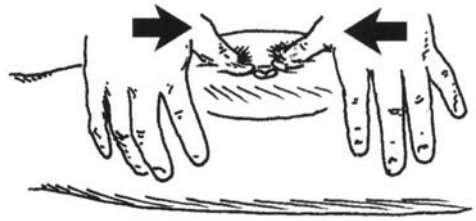
The effect of the neuromuscular spindle cell on muscle strength can be observed when the examiner

stimulates the neuromuscular spindle cell on a normally functioning subject. The thumbs are applied approximately 2" apart over the belly of the muscle and the apparent location of a neuromuscular spindle cell. Rather heavy digital pressure is applied by moving the thumbs toward each other, parallel with the muscle fibers. The muscle is then tested for weakness. The digital maneuver appears to take pressure off the intrafusal muscle fibers, causing a decrease of the afferent nerve impulse and, in turn, causing temporary inhibition of the extrafusal fibers. The experiment may need to be repeated because there is no way of precisely locating a normal neuromuscular spindle cell. Inhibition of the muscle will last long enough to test it two or three times.

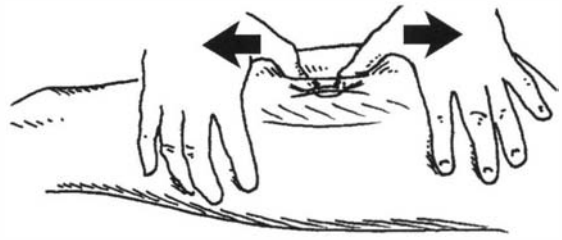
The effect of neuromuscular spindle cell stimulation can be immediately reversed by the examiner pulling the neuromuscular spindle cell apart. It must be pointed out that a normal muscle is not expected to get stronger when the neuromuscular spindle cell is pulled apart. This would be trying to make normal more normal. Research done to test the AK neuromuscular spindle hypothesis on this erroneous premise failed.[20] Treatment by applied kinesiology methods is directed only toward a dysfunctioning neuromuscular spindle cell that is causing abnormal muscle function. When manipulation of the neuromuscular spindle cell is successful in returning normal function, it is suspected that trauma has in some way disturbed the neuromuscular spindle cell function. Hypothetically this can develop from adhesions between the intra- and extrafusal fibers so that they cannot slide between each other for comparative purposes. Another possibility is edema causing pressure and stimulation at the nuclear bag area. These mechanical disturbances appear to cause the neuromuscular spindle to become either hyper- or hypoactive, transferring erroneous information through the simple oligosynaptic loops into the neuronal pools, affecting the homonomous or other muscles.

Examination and Treatment. Therapy localization and palpation are used to determine if a neuromuscular spindle cell may be dysfunctioning, thus causing hypertonicity or weakness of a muscle. Evaluation is easier in the case of a weak muscle. The muscle will test strong when the patient therapy localizes over the neuromuscular spindle cell. A dysfunctioning neuromuscular spindle cell can usually be located with palpation. It feels like a fibrous area in the muscle tissue, and is generally very sensitive to heavy digital pressure. One usually palpates for the fibrous area first, then therapy localizes to reduce the time of searching the muscle with therapy localization.

To treat the neuromuscular spindle cell causing a muscle to test weak, place the thumbs over the area located by palpation and therapy localization and pull apart with 1-7 kg of pressure; occasionally a harder pressure may be necessary. As stated, the area will be tender to the patient. Traction on the spindle cell is done



2—62. Digital pressure toward ends of neuromuscular spindle to weaken muscle.



2—63. Direction of digital pressure to strengthen muscle which is weak from apparent neuromuscular spindle malfunction.

several times in the general area. After treatment the muscle should test strong, and the treatment should not need to be repeated.

To treat a muscle that is hypertonic as a result of neuromuscular spindle cell dysfunction, reverse the direction of pressure application. Contact both ends of the area located by palpation and therapy localization, and apply the same type of pressure to both ends of the spindle cell toward the center. It is more difficult to determine if adequate treatment has been provided to a hypertonic muscle because the muscle — if returned to normal — will exhibit normal strength on manual muscle testing. The best way to determine whether the neuromuscular spindle cell has been adequately treated is to see if positive therapy localization has been abolished. If the hypertonic muscle was responsible for limited range of motion it will increase.

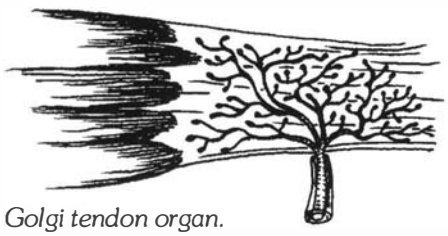
The optimal pressure application to a dysfunctioning neuromuscular spindle cell is either separating or approximating pressure over the spindle cell, as described. In small muscles, a two-finger contact is not possible. Apply the digital stimulation with one finger in a similar directional characteristic as best possible. It is generally relatively easy to treat neuromuscular spindle cells, even in small muscles where contact is difficult.

Neuromuscular spindle cell dysfunction that influences a remote muscle is called a reactive muscle pair. This requires a different type of examination that will be discussed later.

Golgi Tendon Organ

The Golgi tendon organs are located in the tendon close to the musculotendinous junction. A few to many muscle fibers — an average of ten to fifteen — are attached to each Golgi tendon organ. The Golgi tendon organ is situated in series with the muscle, whereas the neuromuscular spindle cell is parallel to the muscle. The neuromuscular spindle cell monitors the length of the muscle, while the Golgi tendon organ monitors its tension. Contraction of the muscle stimulates the Golgi tendon organ, which inhibits the homonomous muscle.

The primary purpose of the Golgi tendon organ is



2—64. Golgi tendon organ.

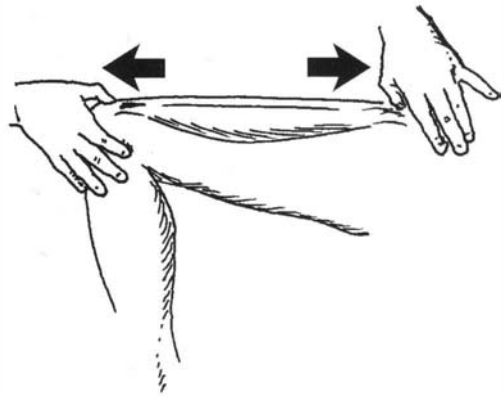
to protect the homonomous muscle. An example of Golgi tendon organ function is seen in arm wrestling. Typically a match continues until one of the opponents gives out completely — all at once — when impulses from the Golgi tendon organ overpower the alpha motor neuron impulses and shut the muscle down. Individuals who train to build strength develop a learned response that overrides the Golgi tendon organ. Some competitors in arm wrestling have succeeded in overriding the Golgi tendon organ so well that the humerus is fractured rather than the muscle giving out.

Action of the Golgi tendon organ can be demonstrated in a way similar to that of the neuromuscular spindle cell. In the normally functioning rectus femoris or other strong muscle, digitally stimulate the musculotendinous junction of both ends of the muscle away from the belly. The stimulation consists of a rather heavy pressure over a wide area that probably contains the Golgi tendon organ. If successful the muscle will immediately test weak, and will usually remain so long enough for several tests. The better organized an individual's nervous system, the shorter time the weakness will remain. If the muscle fails to weaken, re-stimulate in a little different area. Under normal conditions it is difficult to determine the exact location of the Golgi tendon organs.

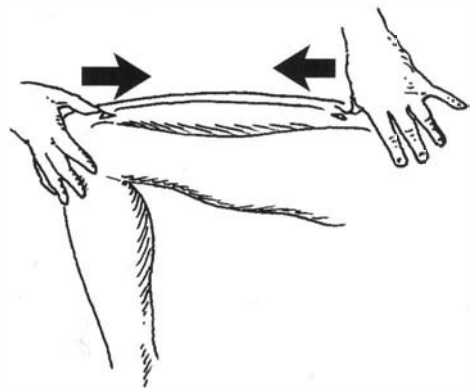
Examination and Treatment. Like the neuromuscular spindle cell, the Golgi tendon organ appears to dysfunction as a result of trauma. The muscle will most often test weak when the Golgi tendon organ dysfunctions. Occasionally it may appear that the muscle is hypertonic because of a dysfunctioning Golgi tendon organ; however, the neurologic mechanism for this is speculative.

When a muscle is weak, the Golgi tendon organ can be evaluated by therapy localization which, when positive, will cause the muscle to strengthen. Therapy localization to the Golgi tendon organ, when positive, will also cause indicator muscles to weaken; however, the best evaluation is by testing the homonomous muscle. The dysfunctioning Golgi tendon organ is first located by palpation. It will usually palpate as a discrete nodular area at the musculotendinous junction, and will be exquisitely tender to digital pressure. There may be Golgi tendon organ involvement at either the origin or insertion, or both ends of the muscle. Both ends should be evaluated if the anatomical location of the origin and insertion allows inspection.

The manipulative pressure for treating the Golgi tendon organ is rather heavy digital manipulation over the palpated nodules that had positive therapy localization. The manipulation is done in alignment with the muscle fibers toward the belly to strengthen the muscle, and away from the belly to weaken it. Immediately after manipulating the receptor, the muscle should test strong and remain so without further treatment.



2—65. Direct pressure over Golgi tendon organ away from belly to weaken.



2—66. Direct pressure over Golgi tendon organ toward belly to strengthen.

Nutrition

If the neuromuscular spindle cell or Golgi tendon organ must be treated again, raw bone in the form of concentrate or nucleoprotein extract will often prevent return of the dysfunction. Goodheart feels that phosphatase, which is present in these substances, is the factor that improves the condition.

Reactive Muscles

A reactive muscle is one that tests weak only after another muscle has previously been contracted. The muscle which tests weak is known as the "reactive muscle," and the initial muscle contracted is the primary one. The two muscles are referred to as a reactive pair, or a portion of a reactive group.[46] The weakening of the reactive muscle is apparently due to improper signaling from the neuromuscular spindle cell, or possibly Golgi tendon organ, of the primary muscle.

In Goodheart's original discussion of reactive muscles,[15] he hypothesized that the neuromuscular spindle cell of the primary muscle is "set too high"; thus, when there is activation of the primary muscle, the Ia afferent impulses cause an overabundant inhibition of an antagonist muscle through the inhibitory interneuron.

Triano and Davis[46] studied the reactive muscle phenomenon with electromyography. The study was of reactivity in the deltoid secondary to contraction of the rhomboid muscle. The study established the reactivity of deltoid weakening after rhomboid contraction. The deltoid no longer weakened after the rhomboid was treated to set down the neuromuscular spindle cell (spindle cell together treatment). To determine if the apparent spindle cell manipulation related with Hagbarth's studies[22,23] on muscle reflex activity from cutaneous touch and pressure, they applied generalized pressure into the muscle belly; it did not have the same effect.

Reactive muscles are discovered by analyzing the patient's type of problem. Often the primary muscle is an antagonist of the reactive one, such as the hamstrings being reactive to the quadriceps muscles. As the reactive pair becomes more remote from each other, the evaluation becomes more difficult. The muscle pair may be associated as in gait or the sequential aspects of sports, or it may be remote, with association difficult to understand.

The reactive muscle problem can be present in any type of structural condition. It is often associated with athletic injuries and should always be suspected when there is no apparent reason for joint strain. An example is an athlete who is having knee or ankle problems, which may include recurrent injuries for no apparent reason. Upon examination, the muscles supporting the knee or ankle test strong and orthopedic tests, x-rays, and other testing procedures reveal no problem. There will often be a history of running with or without cutting, and the knee or ankle seems to "give out." Under these circumstances, analyze the muscles that function during the activity, and then have the patient contract a muscle; immediately afterward test the muscle(s) that acts sequentially. A positive test is when both muscles test strong in the clear, but one tests weak after the primary muscle contracts.

A pattern of reactive muscles is known as "muscle interlink." This is reactivity between upper and lower contralateral extremity muscles that have a comparable function in gait. For example, the right knee and left elbow flex together during gait; thus there is facilitation of the hamstrings and contralateral biceps muscle. To evaluate for muscle interlink, first determine that the muscles are strong in the clear. Muscle interlink is present when one of the muscles or muscle groups tests weak immediately after the contralateral muscle was contracted by muscle testing. (For further information about the organization of muscles and joints in gait function, see "Ligament Interlink," page 211.)

Treatment is nearly always applied to a dysfunctioning neuromuscular spindle cell in the primary muscle. Locate the spindle cell by therapy localization and palpation, as previously described under "Neuromuscular Spindle Cell Treatment." Manipulate the two ends of the spindle cell toward each other to "set down" the spindle cell. Immediately after treatment, the reactive muscle should test normal after contraction of the primary muscle.

Occasionally, Golgi tendon organ dysfunction appears to be responsible for a reactive muscle pair. If muscle reactivity is found and no neuromuscular spindle cell dysfunction can be located, evaluate the musculotendinous junction for possible dysfunctioning Golgi tendon organs. Treat as indicated under "Golgi Tendon Organ Treatment" and re-test for reactivity. Clinically this is occasionally effective, although the neuromuscular function is not understood.

Reactive Muscle Chart

The left column of Table 2—67 represents the muscles suspected of being reactive; the right column is the muscle that requires muscle spindle cell or Golgi tendon organ sedation. Note that all muscles listed on the right are also listed on the left, and vice versa. The reactive muscle may be in either sequence.

No chart is all-inclusive; additional examination for muscle interlink, as described, may be necessary. Other combinations not as frequently observed may also be found by analysis of the patient's problem. If a particular joint is involved, the prime mover, synergists, antagonists, and fixator muscles should be evaluated. Contralateral muscles should also be evaluated; they may be involved on the basis of cross-reciprocal innervation. Generally the patient can provide clues about activities that appear to make the condition worse. This is especially true in sports. All preparatory motions to the difficult activity should be evaluated for possible contribution on a reactive muscle basis.

The chart is organized by body sections, beginning at the cervical spine and proceeding to the shoulder, elbow, trunk, pelvis, hip, knee, and ankle. Note the overlapping of muscles that influence two articulations.

Table 2—67.

Suspected Reactive Muscle	Sedation Required
Neck flexor	Contralateral psoas
Splenius capitis	Contralateral piriformis
Upper trapezius	Latissisimus dorsi Biceps Contralateral upper trapezius
Deltoid	Rhomboid Pectoralis minor
Supraspinatus	Rhomboid Pectoralis minor
Rhomboid	Deltoid Serratus anticus Supraspinatus
Latissimus dorsi	Contralateral hamstring Upper trapezius
Pectoralis minor	Serratus anticus Supraspinatus Deltoid
Pectoralis major (clavicular division)	Gluteus maximus
Serratus anticus	Rhomboid Pectoralis minor
Biceps	Triceps Upper trapezius
Triceps	Biceps Supinator
Sacrospinalis	Transverse abdominals Gluteus maximus Hamstrings
Diaphragm	Psoas
Rectus abdominis	Quadriceps Contralateral gluteus medius
Upper rectus abdominis	Lower rectus abdominis
Lower rectus abdominis	Upper rectus abdominis
Transverse abdominals	Sacrospinalis
Psoas	Adductors Contralateral anterior neck flexor
Gluteus medius	Contralateral rectus abdominis
Piriformis	Contralateral splenius capitis
Gluteus maximus	Sacrospinalis Pectoralis major (clavicular division)
Hamstrings	Sacrospinalis Contralateral latissimus dorsi Quadriceps Popliteus
Tensor fascia lata	Adductors Peroneus tertius
Adductors	Tensor fascia lata Psoas
Quadriceps	Gastrocnemius Hamstrings Rectus abdominis Sartorius
Sartorius	Tibialis anterior Quadriceps
Popliteus	Gastrocnemius Hamstrings Upper trapezius
Gastrocnemius	Popliteus Quadriceps
Tibialis anterior	Sartorius
Peroneus tertius	Tensor fascia lata

Cutaneous Receptors

Cutaneous exteroceptors are classified as mechanoreceptors, thermoreceptors, and nociceptors. Mechanoreceptors are classified[5] as position, velocity, or transient detectors. Position detectors signal displacements of the skin and are sometimes referred to as touch or pressure receptors. They also have the ability to signal velocity. The nerve terminates at Merkel's cells. Stretch, or a movement of the skin adjacent to the corpuscles, has no effect. This localized sensibility gives discrete localization to the stimulus. The type II ending is the Ruffini ending, which is found in skin both with and without hair. Adequate stimulus is displacement of the skin directly over the receptor, and stretching of adjacent skin.

Velocity detectors are found in skin both with and without hair. There are several types that associate directly with hair follicles. Adequate stimulation is movement of the hair. In skin without hair, the Meissner corpuscle is important in velocity sensation. The Meissner corpuscles are most numerous in the skin over the fingertips. They are sensitive to low frequency vibration in the range of 5-40 Hz.[49] Vibration can be produced by the fingertips or other skin moving across the surface.

Pacinian corpuscles are transient detectors. They are present not only in the skin, but also at fascial planes around joints and tendons and in the mesentery. A mechanical stimulation causes an on/off response in the Pacinian corpuscle. Animal experiments have shown that stimulation of a single Pacinian corpuscle can readily be detected in recordings from the cerebral cortex. Adequate stimulation consists of vibration or a tap, and the frequency range is 60-300 Hz.[49]

The cutaneous receptors are stimulated with joint motion. For example, when the knee flexes, the skin over the quadriceps and anterior knee stretches. When the knee extends, the skin over the hamstrings and popliteal surface stretches. The muscle underlying the area of skin stretch is normally inhibited with the joint motion. The contribution of the cutaneous receptors to the neurologic organization of muscles in joint movement can be observed by manual muscle testing in applied kinesiology. The examiner stretches the skin over the quadriceps muscle in alignment with the muscle fibers. This is done by simply taking a pinch of skin between the thumb and forefinger of each hand and pulling the skin apart, taking care not to heavily pinch the skin with the grasping fingers. Immediately after stretching the skin, test the quadriceps muscle group. Under normal circumstances, the muscles will test weak for a variable length of time. Stretching the skin in this manner simulates knee flexion, as if the hamstrings had contracted. This would cause reciprocal inhibition of the quadriceps muscles, which is what is observed when the muscle tests weak.

This basic function is present throughout the body. Some additional examples are stretching the skin over the dorsum of the foot and anterior ankle to cause the tibialis anterior muscle to weaken, as if the gastrocnemius and soleus muscles had contracted. Stretching the skin over the anterior, medial, and posterior axillae causes the latissimus dorsi to test weak. This skin stretch simulates the arm being placed over the head, which requires inhibition of the latissimus dorsi.

Examination and Treatment

Clinical evidence in applied kinesiology indicates that sometimes the cutaneous receptors are inappropriately stimulated or react inappropriately to stimulation, sending information not in keeping with the joint motion. When a muscle tests weak as a result of improper signaling from the cutaneous receptors, it can immediately be made to test normal by skin stretching. For example, if the hamstring muscles test weak, stretch the skin over the quadriceps group in the direction in which it would normally be stretched if the hamstrings were contracting. The amount of stretch is the amount the skin will yield without damage or severe pain. If dysfunction of the cutaneous receptor is associated with dysfunctioning hamstring muscles, they will test strong immediately after the skin stretch. Since no structure other than the skin is manipulated or stimulated, it appears that the improved muscle function results from stimulation of the cutaneous receptors. Making a muscle strong in this manner is only a diagnostic test for apparent cutaneous receptor dysfunction. The strengthening of the hamstrings will only last for twenty or thirty seconds, regardless of how vigorous or long-lasting the skin stretch is. The diagnostic approach to the cutaneous receptor is limited to stretch, followed by manual muscle tests. Therapy localization does not appear to reveal any disturbance.

There is an additional factor that can be added to the skin stretch that will make the correction long-lasting or permanent. In reviewing adequate stimulus to the cutaneous receptors, Goodheart[17] associated vibration as a possible added requirement. Both the Meissner and Pacinian corpuscles respond to frequency stimulation. Ranges are 5-40 Hz and 60-300 Hz, respectively.[49] When Goodheart added a vibration with his fingertips to the skin stretch, the improvement in muscle function became long-lasting. This is accomplished simply by stretching the skin as previously described, and adding as quick a vibration as possible with the fingers. Skin stretch and vibration are continued for twenty to thirty seconds for adequate treatment. Both the stretch and vibration must be done simultaneously; either done alone will not produce lasting results.

Goodheart[17] found that 300 Hz electrical stimulation produced the same results as stretching and vibrating the skin. The 300 Hz electrical stimulation by itself, without stretch, will produce the results. The electrical generator is an acupuncture-type instrument capable of producing selected Hz stimulation.

In most instances, the treatment described will be long-lasting, with repetition not needed. In case dysfunction returns, clinical evidence has shown a low potency, full complex of vitamin B makes the correction long-lasting.

CHAPTER 2 — REFERENCES

1. Bakan, P., "Dreaming, REM sleep and the right hemisphere: A theoretical integration," *J Altered States Conscious*, Vol 3, No 4 (1977-78).
2. Bogen, J.E., "The other side of the brain I: Dysgraphia and dyscopia following cerebral commissurotomy," *Bull Los Angeles Neurol Soc*, Vol 34, No 2 (Apr 1969).
3. Bogen, J.E., "The other side of the brain II: An appositional mind," *Bull Los Angeles Neurol Soc*, Vol 34, No 3 (Jul 1969).
4. Bogen, J.E., & G.M. Bogen, "The other side of the brain III: The corpus callosum and creativity," *Bull Los Angeles Neurol Soc*, Vol 34, No 4 (Oct 1969).
5. Burgess, P.R., & E.R. Perl, "Cutaneous mechanoreceptors and

nociceptors," in *Handbook of Sensory Physiology, Vol II — Somato-Sensory System*, ed A. Iggo (New York: Springer-Verlag, 1973).
6. Davis, A.R., & W.C. Rawls, Jr., *Magnetism and Its Effects on the Living System* (Hicksville, NY: Exposition Press, 1974).
7. Denny-Brown, D., ed, *Selected Writings of Sir Charles Sherrington* (New York: Oxford Univ Press, 1979).
8. Eversaul, G.A., *Dental Kinesiology* (Las Vegas: privately published, 1977).
9. Feldenkrais, M., *Awareness through Movement — Health Exercises for Personal Growth* (New York: Harper & Row, 1972).
10. Freeman, M.A.R., & B. Wyke, "Articular reflexes at the ankle joint: An electromyographic study of normal and abnormal influences of ankle-joint mechanoreceptors," *Br J Surg*, Vol 54, No 12 (Dec 1967).
11. Gandevia, S.C., & D.I. McCloskey, "Joint sense, muscle sense, and their combination as position sense, measured at the distal interphalangeal joint of the middle finger," *J Physiol* (Lond), Vol 260, No 2 (Sep 1976).
12. Goodheart, G.J., Jr., *Applied Kinesiology* (Detroit: privately published, 1964).
13. Goodheart, G.J., Jr., *Applied Kinesiology 1974 Research Manual*, 10th ed (Detroit: privately published, 1974).
14. Goodheart, G.J., Jr., *Applied Kinesiology 1976 Workshop Procedure Manual*, 12th ed (Detroit: privately published, 1976).
15. Goodheart, G.J., Jr., "Reactive muscle testing," *Chiro Econ*, Vol 21, No 4 (Jan/Feb 1979).
16. Goodheart, G.J., Jr., *Applied Kinesiology 1981 Workshop Procedure Manual*, Vol 1, 17th ed (Detroit: privately published, 1981).
17. Goodheart, G.J., Jr., *Applied Kinesiology 1986 Research Manual*, 22nd ed (Geneva, OH: AK Printing, 1986).
18. Goodheart, G.J., Jr., *Applied Kinesiology 1992/93 Workshop Procedure Manual* (Grosse Pointe Woods: privately published, 1993).
19. Gowitzke, B.A., & M. Milner, *Understanding the Scientific Bases of Human Movement*, 2nd ed (Baltimore: Williams & Wilkins, 1980).
20. Grossi, J.A., "Effects of an AK technique on quadriceps femoris muscle isometric strength," *Phys Ther*, Vol 61, No 7 (Jul 1981).
21. Guyton, A.C., and J.E. Hall, *Textbook of Medical Physiology*, 9th ed (Philadelphia: W.B. Saunders Co, 1996).
22. Hagbarth, K.E., "Excitatory and inhibitory skin areas for flexor and extensor motoneurones," *Acta Physiol Scand*, Vol 26, Supp 94 (1952).
23. Hagbarth, K.E., "Spinal withdrawal reflexes in the human lower limbs," *J Neurol Neurosurg Psych*, Vol 23 (1960).
24. Ittelson, W.H., & F.P. Kilpatrick, "Experiments in perception," *Sci Am*, Vol 405 (Aug 1951).
25. Karagulla, S., *Breakthrough to Creativity — Your Higher Sense Perception* (Santa Monica, CA: DeVorss & Co, Inc, 1967).
26. Kendall, H., F. Kendall, & D. Boynton, *Posture and Pain* (Baltimore: Williams & Wilkins, 1952).
27. Kinsbourne, M., ed, *Asymmetrical Function of the Brain* (Cambridge: Cambridge Univ Press, 1978).
28. Kinsbourne, M., & J. Cook, "Generalized and lateralized effects of concurrent verbalization on a unimanual skill," *Q J Exp Psych*, Vol 23 (1971).
29. Kirlian, S.D., & V.K. Kirlian, "Photography by means of high-frequency currents," in *The Kirlian Aura*, ed S. Krippner & D. Rubin (Garden City, NY: Anchor Press/Doubleday, 1974).
30. Lettvin, J.Y. et al., "What the frog's eye tells the frog's brain," in *Embodiments of Mind*, ed W.S. McCulloch (Cambridge, MA: The M.I.T. Press, 1965).
31. Martin, R.J., ed, *Dynamics of Correction of Abnormal Function — Terrence J. Bennett Lectures* (Sierra Madre, CA: privately published, 1977).
32. Martin, R.J., *The Practice of Correction of Abnormal Function — "Neurovascular Dynamics" (NVD)* (Sierra Madre, CA: privately published, 1983).
33. McCloskey, D.I., "Kinesthetic sensibility," *Physiol Rev*, Vol 58, No 4 (Oct 1978).
34. McCloskey, D.I., "Signals of proprioception in man," in *Proprioception, Posture and Emotion*, ed D. Garlick (Kensington, NSW: Univ New South Wales, 1982).
35. McCouch, G.P., I.D. Deering, & T.H. Ling, "Location of receptors for tonic neck reflexes," *J Neurophysiol*, Vol 14 (May 1951).
36. Nashner, L.M., "Fixed patterns of rapid postural responses among leg muscles during stance," *Exp Brain Res*, Vol 30 (1977).
37. Nicholas, J.M., ed, *Images, Perception and Knowledge* (Boston: D. Reidel Publishing Co, 1977).
38. Ornstein, R.E., *The Psychology of Consciousness* (San Francisco: W.H. Freeman & Co, 1972).
39. Owens, C., interpreter/compiler, *An Endocrine Interpretation of Chapman's Reflexes* (Place, publisher not given: no date).
40. Palmer, D.D., *The Science, Art and Philosophy of Chiropractic* (Portland, OR: Portland Printing House, 1910).
41. Parker, D.E., "The vestibular apparatus," *Sci Am*, Vol 243, No 5 (Nov 1980).
42. Restak, R.M., *The Brain: The Last Frontier* (Garden City, NY: Doubleday & Co, 1979).
43. Schmitt, W.H., Jr., "A screen test for eyes into distortion problems: Eyes out of distortion (E.O.D.) technique." Proceedings of Summer Meeting, ICAK, Detroit, 1982.
44. Sherrington, C., *The Integrative Action of the Nervous System* (Cambridge: Cambridge Univ Press, 1952).
45. Sperry, R.W., "The great cerebral commissure, *Sci Am*, Vol 174 (Jan 1964).
46. Triano, J., & B.P. Davis, "Reactive muscles: Reciprocal and cross-reciprocal innervation phenomenon." Proceedings of the 7th Annual Biomechanical Conference on the Spine, University of Colorado at Boulder, 1976.
47. Upton, A.R.M., A.J. McComas, & R.E.P. Sica, "Potentiation of 'late' responses evoked in muscles during effort," *J Neurol Neurosurg Psychiatry*, Vol 34 (1971).
48. Walther, D.S., *Applied Kinesiology, Volume II — Head, Neck, and Jaw Pain and Dysfunction—The Stomatognathic System* (Pueblo, CO: Systems DC, 1983).
49. Willis, W.D., Jr., & R.G. Grossman, *Medical Neurobiology*, 3rd ed (St. Louis: The C.V. Mosby Co, 1981).
50. Wong, E., *Wong's A.P.P. Ryodoraku System* (Not given: A.P.P. Technique Seminars, 1975).
51. Wong, P.K.H., M. Verrier, & P. Ashby, "The effect of vibration on the F wave in normal man," *Electromyogr Clin Neurophysiol*, Vol 17 (1977).

Chapter 3

Spinal Column

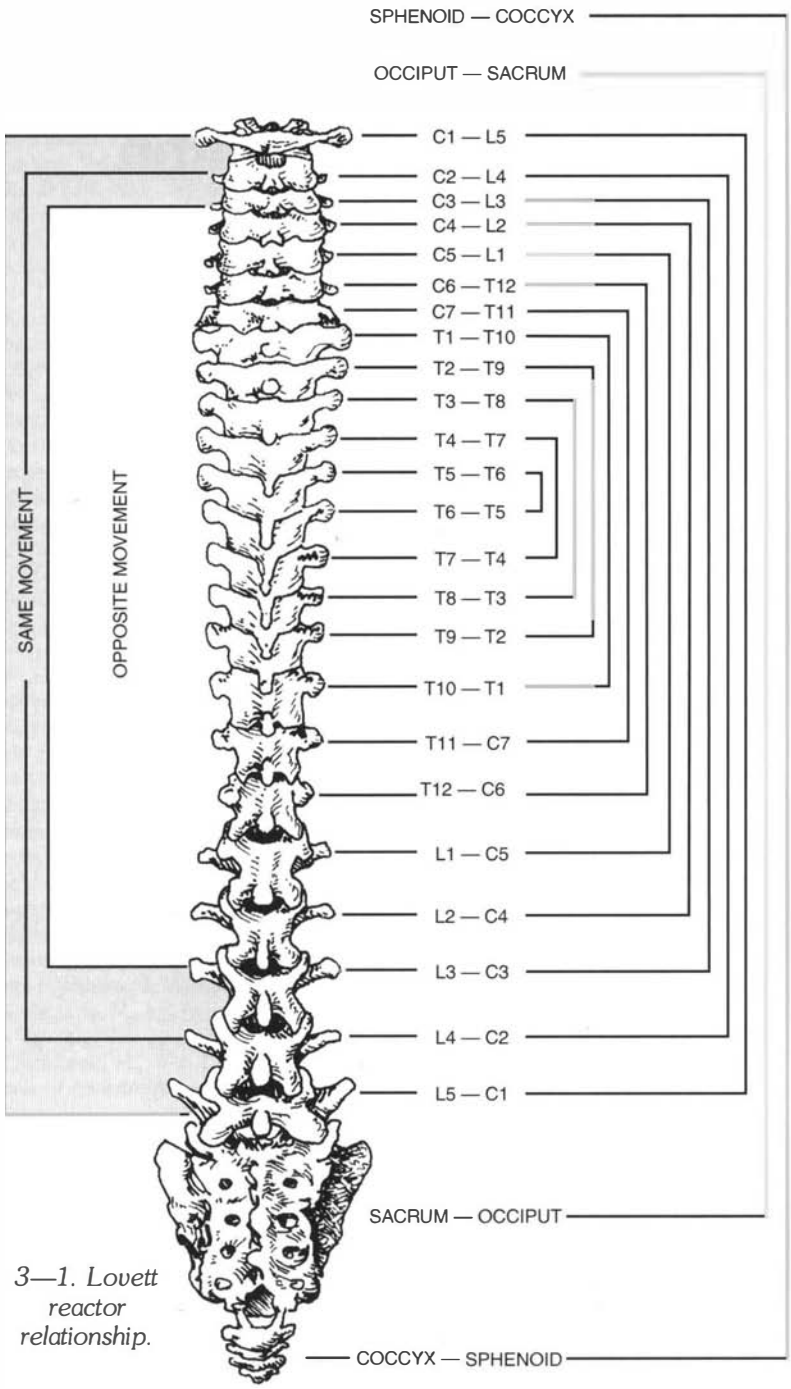
Lovett Reactor

A relationship between vertebral motions has been described in applied kinesiology[36] in which the atlas and 5th lumbar vertebrae rotate in the same direction when an individual walks, giving a relationship of motion to the atlas and 5th lumbar. The relationship continues throughout the spine, with C2 rotating in the same direction as L4 and C3 with L3. At this point, the movement shifts to counterrotation as C4 counterrotates with L2 and C5 with L1. The counterrotation continues throughout the rest of the spine until the upper half of the spinal column meets the lower half at the 5th and 6th thoracic vertebrae. Prior to being termed the Lovett reactor, this was called the "halfwit brother" or "Lovett brother reactor." Clinical evidence indicates that the Lovett reactor vertebrae are often related in primary and compensatory subluxations. If there is a primary subluxation at L4, there may be a compensatory subluxation at C2. If the subluxation at C2 is corrected without correcting L4, the C2 subluxation will frequently return.

The relationship of movement has been somewhat supported by the studies of Inman et al.[70] Metal pins were inserted into the spinous processes of human subjects and their motion studied. Minimal rotation was found at the 7th thoracic vertebra, with a gradual increase of counterrotation toward the shoulders and pelvis.

Analysis of the motion of the pelvis, thorax, shoulders, and head reveals the reason for the counterrotation or same rotation of the Lovett reactors. As the leg moves forward in the swing phase of gait the pelvis rotates forward, contributing to the length of the stride. The shoulder girdle moves posteriorly on that side, while the head counterrotates with the shoulder girdle to maintain forward head position during gait. This, then, requires opposite rotation of the lumbar and lower thoracic vertebrae with the upper thoracic vertebrae. The upper cervical vertebrae must rotate in the same direction as the lumbar vertebrae as the head counterrotates with the shoulder girdle. The relationship is extended as the sacrum reacts with the occiput and the coccyx with the sphenoid.

There may initially be evidence by vertebral challenge and therapy localization that there are subluxations of the associated vertebrae. Sometimes correcting one vertebra will eliminate evidence of the subluxation at its reactor; in other cases it may be necessary to adjust both vertebrae. It is good general practice to evaluate the Lovett reactor when a subluxation is located.



3—1. Lovett reactor relationship.

Generally the Lovett reactor subluxation will be compatible with the motion illustrated in the chart. If the subluxation is one of the top three or bottom three vertebrae, the subluxations will often be in the same direction. In other words, if there is an axis right subluxation, there will probably be a 4th lumbar right subluxation. The general pattern described is not always applicable. There may be congenital or muscular anomalies, or other factors influencing the relationship. The vertebra should be adjusted in the direction of positive challenge after having ruled out neurologic disorganization.

Vertebral Subluxations

Unless there is bone deformity, muscle imbalance is basic to structural distortion and dysfunction. For spinal subluxations to be maintained, muscle imbalance must be present. Spinal examination should include a method for locating vertebral levels that have muscle imbalance capable of maintaining the subluxation.

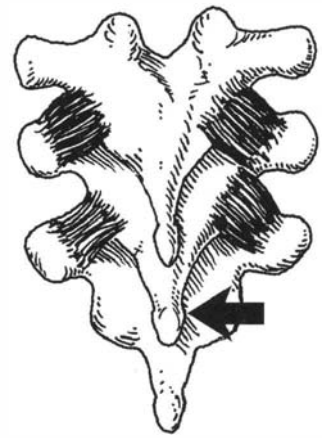
Applied kinesiology examination reveals a difference between spinal subluxations and spinal fixations. A fixation is lack of movement between two or more vertebrae, whereas a subluxation is aberrant movement from hyperirritable intrinsic muscles of the subluxation complex. It is the hyperirritable intrinsic muscles that maintain the subluxation complex.

Using needle electromyography, Denslow and Clough[26] studied the intrinsic muscles of vertebral levels considered to be in lesion. There was reflex muscle activity in the lesion area, but none in control areas. Further study by Denslow[25] revealed that when mechanical stimulation was applied to move the vertebra in lesion, there was reactivity of the intrinsic muscles. Similar stimulation in non-lesion control areas indicated that the hyperreactive intrinsic muscles were unique to areas of lesion.

The hyperreactive muscles of a subluxation complex are in keeping with the principle that abnormal structural function is maintained by muscular imbalance. The body is a self-correcting, self-maintaining mechanism; with normal vertebral structure and muscle function, the body corrects its own subluxations.

Therapy localization can help locate areas of vertebral subluxation. Not all areas showing positive therapy localization along the spinal column are subluxations, because acupuncture associated points on the bladder meridian and posterior neurolymphatic reflexes are adjacent to the spinal column and also show positive therapy localization when active. Generally, therapy localization for a vertebral subluxation can be evaluated using any strong indicator muscle of the body. In some instances, there may be positive therapy localization only when muscles of the subluxation's neurologic level are evaluated.

Vertebral challenge[37,104] differentiates a subluxation from other factors that show positive therapy localization. In addition, vertebral challenge provides information regarding how the subluxation complex should be corrected.



3—2. Imbalanced rotatory brevis. Arrow points in direction of positive challenge.

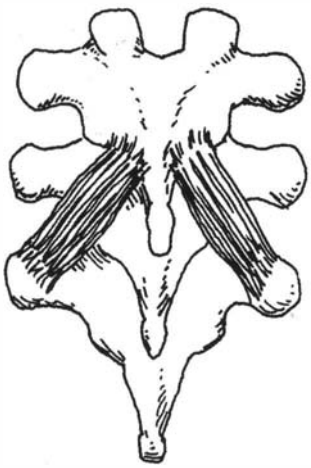
To challenge a vertebra for a subluxation, apply digital pressure to a transverse or spinous process in a direction that rotates or tips the vertebra; then release the pressure. This stimulates the intrinsic muscles; under normal conditions, they will quickly return to homeostasis. When the muscles are hyperirritable, the stimulation of challenge will cause them to overreact and hypothetically increase the subluxation complex. In this situation, a strong indicator muscle will immediately test weak. The weakness usually lasts for five to thirty seconds, and sometimes up to several minutes. The amount of weakness perceived in the manual muscle test depends upon the vector of challenge to the vertebra. One specific vector will cause maximum weakening. As one deviates from that vector, weakening will be perceived, but not to as great an extent. The object of the challenge is to find the contact point and vector causing maximum weakening. Clinical evidence indicates that the optimal vector and contact point for correction of the subluxation is exactly the same as the challenge that caused maximum weakening of the indicator muscle. Adjustment of the subluxation can be done in any manner to which the physician is accustomed. Obviously the effectiveness of the adjustment will vary with the expertise and accuracy of application.

When an effective adjustment has been accomplished, there will no longer be positive therapy localization or challenge. It is not necessary to obtain an audible release for an effective correction; attempting to obtain one may cause trauma to the soft tissues.

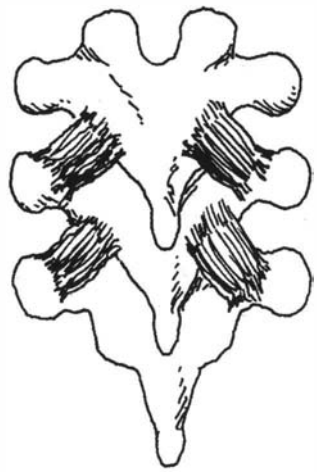
Intrinsic Spinal Muscles

The intrinsic spinal muscles that appear to be most important to vertebral subluxations are those that bridge two or three vertebrae, such as the rotatores brevis and longus, respectively. Palpation of the intrinsic muscles is an excellent method for screening areas of probable subluxation. Along with therapy localization and challenge, palpation is a valuable tool for finding subluxations and evaluating the corrective effort.

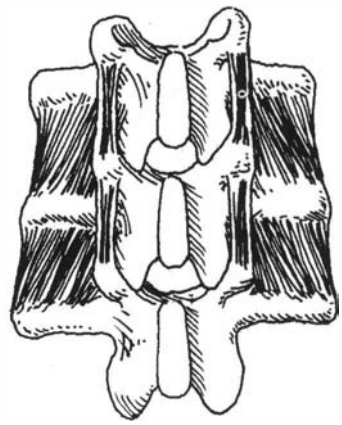
If muscles fail to balance after a subluxation has been corrected, it is necessary to apply therapy directly to the muscles. This is done with origin/insertion technique and deep massage to influence the muscle proprioceptors. When there is involvement of the intrinsic muscles, there will be a very specific positive therapy localization over the involved muscle. One often finds the offending weak muscle opposite the hyperirritable (contracted) muscle. For example, if there is a positive challenge on the spinous process from right to left, the vertebra should be adjusted by spinous contact on the right, with the adjustive thrust directed left. In this case a hyperirritable muscle would be on the right and a possibly weak muscle on the left. Positive therapy localization to the left transverse process and lamina of the vertebra above indicates that origin/insertion technique to the rotatores brevis should be applied. A hyperirritable or contracted muscle secondary to a weak antagonist is consistent with early observations of general skeletal muscles in applied kinesiology.



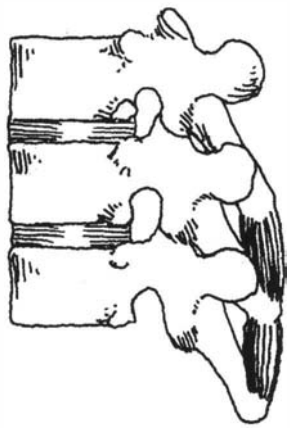
3—3. Rotatores longus



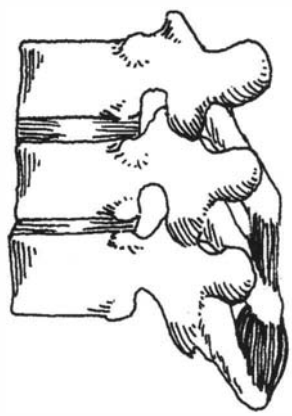
3—4. Rotatores brevis



3—5. Intertransversarii



3—6. Balanced interspinalis

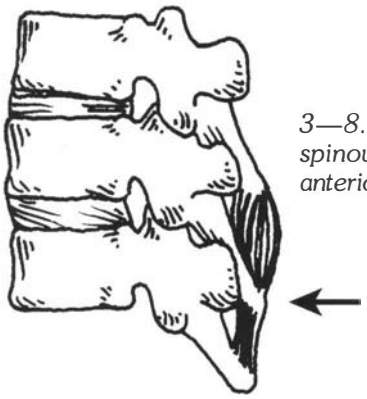


3—7. Hypertonicity of lower interspinalis muscle pulls vertebra into posterior subluxation, especially if there is thinning of the intervertebral disc.

Anterior Thoracic Subluxation

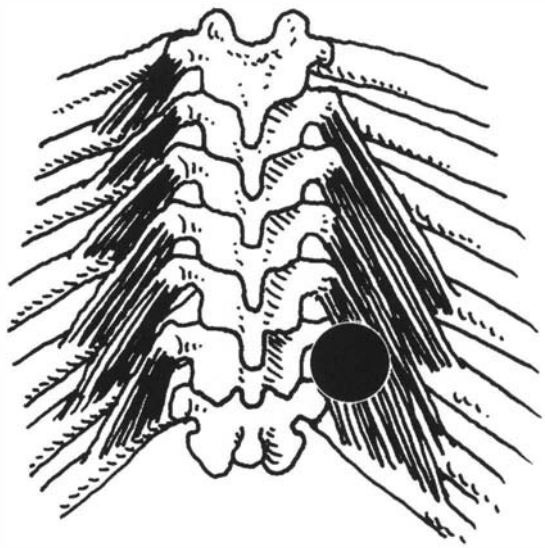
Anterior thoracic subluxations often recur, even though adequate manipulation has been accomplished. Normal function of the interspinalis and/or levator costarum muscles is important in maintaining the correction. In the thoracic spine, the interspinalis muscles are at the 1st and 2nd vertebrae and sometimes between the 2nd and 3rd and the 11th and 12th vertebrae.

The anterior thoracic subluxation, like others, has positive therapy localization, although it is often difficult for the patient to therapy localize in the area of potential subluxation. The subluxation will cause exquisite tenderness at the spinous process where the interspinalis muscle and/or the interspinalis ligament attaches. This appears to result from the stretching of the muscle and/or ligament and its inherent dysfunction associated with the subluxation.



3—8. Tenderness at the spinous process present in anterior subluxation.

The interspinalis and levator costarum muscles hold the vertebra posteriorly and inferiorly. Weakness of these muscles allows the vertebra to move into a flexion subluxation along the facet planes. Prior to adjusting the subluxation, the muscles should be evaluated and treated when necessary. The interspinalis muscles are usually treated with origin/insertion technique. The levator costarum muscles can be challenged and, if positive, treated with a respiratory assist. The levator costarum brevis muscles arise from the transverse process of the vertebra and insert into the outer surface of the rib immediately below. The levator costarum longus muscles are associated with the four lower levator costarum brevis. Their origin is from the transverse process; they insert into the rib two below. To challenge the levator costarum muscle for weakness, press in an anteromedial direction on the rib immediately below (or two below) the anterior thoracic subluxation. A positive challenge is seen when a strong indicator muscle weakens. Correction is made while the patient takes a certain phase of respiration. While the indicator muscle is still weak, determine the phase of respiration that immediately abolishes the weakness; it will usually be inspiration. Correction is accomplished by pressing in the same direction of positive challenge while the patient takes the phase of respiration that abolished



3—9. Press at dot in a medial and anterior direction for challenge.

the weakness. Repeat with four or five respirations, using four to five pounds of pressure. Re-therapy localize and/or challenge to make certain the correction is obtained. If it is not, repeat the procedure and re-test. Correction of the muscles will sometimes correct the subluxation and no further action is necessary. In most cases it will be necessary to adjust the thoracic vertebra. There are two common methods of adjusting anterior thoracic vertebrae. The procedural choice is determined by the patient's position on the plumb line when viewed laterally. The principle is to always adjust toward the plumb line; i.e., if the patient is anterior, use an anterior thrust, moving spinal alignment back toward the center of gravity. If the patient is posterior to the plumb line, use a posterior-to-anterior thrust. The standing anterior thoracic subluxation adjustment is applicable when the patient is posterior to the lateral plumb line. The doctor places a block or pad on the vertebra below the anterior one. It is held in place with the physician's chest. The patient crosses his arms across his chest and the physician puts his arms around the patient, grasping the crossed arms. From this position, a lifting traction with anterior pressure on the blocked vertebra is applied with a quick motion.

The supine adjusting technique is applied when the patient is anterior to a lateral plumb line. The physician places his closed fist or a small block between the patient and the table on the vertebra inferior to the anteriority. A light thrust is applied to the patient's arms crossed over his chest as he attempts to curl up, lifting his head and shoulders from the table.

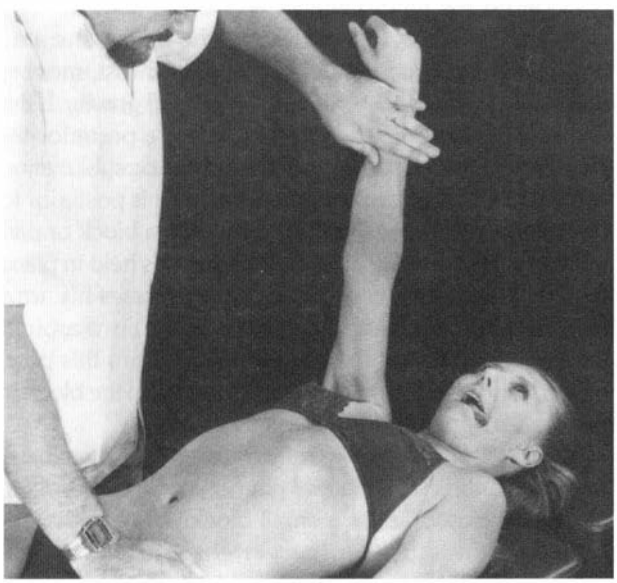
Frequently an audible release is observed in both techniques, but it is not necessary. Re-evaluate with therapy localization and palpation to determine effectiveness of the correction.

Occipital Subluxation

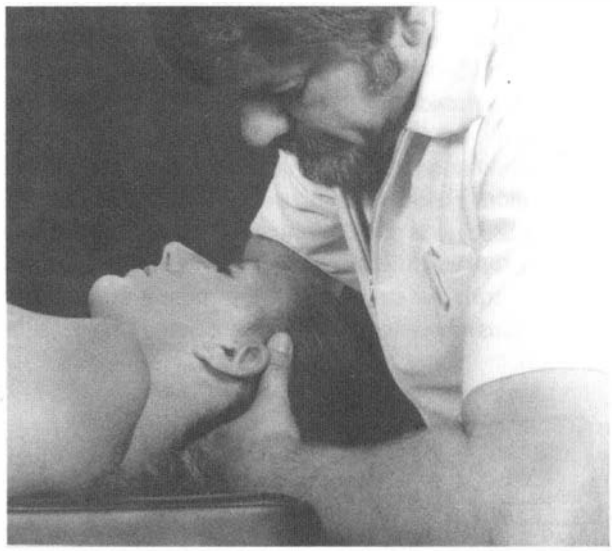
When there is head tilt it may be primary to neurologic disorganization, resulting in considerable structural distortion and dysfunction. This appears to be due to the interrelationship of the head-on-neck reflexes, located in the ligaments of the upper cervical vertebrae, with the visual righting and labyrinthine reflexes. An occipital subluxation will have positive therapy localization over the occipitoatlantal articulation.

When an occipital subluxation is the cause of neurologic disorganization, therapy localization to the occipitoatlantal articulation will cancel positive therapy localization at KI 27, an indicator for neurologic disorganization. Neurologic disorganization should be cleared before general examination and treatment are done. When therapy localization to the occipitoatlantal articulation cancels positive KI 27 therapy localization, correct the occipital subluxation; if no other causes of neurologic disorganization are present, there will no longer be positive therapy localization at KI 27. The basic procedures for correcting neurologic disorganization are discussed in Chapter 5.

Since there are many factors that might show positive therapy localization in this area, one needs to differentiate an occipital subluxation. An easy method for doing this is a unique factor observed in applied kinesiology. When there is a lateral occiput, a strong indicator muscle will weaken when the patient protrudes his tongue to the side of occipital laterality; it will not weaken when the tongue is extended straight out or to the side opposite laterality. It is believed the reason the indicator muscle weakens is that the laterally extended tongue pulls on the hyoid by way of the hyoglossus muscle. Hyoid position is considered in applied kinesiology to be intricately involved with structural orientation.



3—10. A strong indicator muscle will test weak when the tongue is protruded to the side of occiput laterality. After correction of the occipital subluxation, no weakness will occur with tongue protrusion to either side.



3—11. Occiptal adjustment. Note that the head is kept straight with the sagittal plane.

The direction of adjustive thrust for an occipital subluxation is determined by challenge. Various vectors of force are applied to the occiput, and a strong indicator muscle is tested for weakening. The optimal vector is in the direction of maximal indicator muscle weakening. The optimal contact on the occiput for the adjustive thrust is on the nuchal line, where an extremely sensitive point is palpated. The vector of correction is from this point through the glabella (bridge of nose). This vector should correspond with the vector found by challenge. A metacarpal contact is made, and at the time of thrust the physician's other hand lifts the occiput in a superior direction. This allows the occipital condyle on the side of laterality to rotate down and medially along the plane of the lateral mass of the atlas, while the opposite condyle is lifted out of the lateral mass. The patient's head is kept in alignment with the sagittal plane so that no rotation is put into the cervical spine. Care must be taken to avoid excessive stress to the cranium that might cause cranial faults.

Re-evaluate with therapy localization and challenge. The sacrum is the Lovett reactor to the occiput and may be in secondary subluxation. Failure to correct the sacrum will often cause the occipital subluxation to recur.

Upper Cervical Subluxation

The upper cervical area, especially the atlas, has an integrative motion with many specific directions of subluxation. Subluxation of the area will show positive therapy localization and challenge. Challenge should be directed to the anterior, lateral, and posterior aspects of the transverse process. Challenge is applied in all directions of possible subluxation. Several directions of challenge may be positive. Choose the vector of challenge that causes the greatest amount of indicator muscle weakness as the vector of correction.

A lateral atlas has a unique relationship with leg imbalance. When the supine patient's short leg changes to a long leg when prone, there will probably be a lateral atlas subluxation. The atlas will be lateral on the side of the short leg when the patient is prone.

The occiput-upper cervical complex of muscles regulates the small motion within this area and influences how the mechanoreceptors are stimulated in this very important area of spinal function. The head-on-neck receptors reside within these joints,[90] and recently what may be a very important relationship has been brought to light. There is a connection from the rectus capitis posterior minor muscle to the dura called the posterior atlanto-occipital membrane-spinal dura complex by Hack et al.[53] The connection has been further studied by the U.S. National Library of Medicine's Visible Human Project.[55] There is dural thickening at the point of contact, indicating traction is applied to the dura by this connection.

Knowledge of connection to the dura from the upper cervical complex is not new, but it is just now getting attention to reveal the possible ramifications. Rutten et al.[101] report that Von Lanz described extensive connective tissue bridges between spinal dura and surrounding structures in the craniocervical region in 1929. Rutten's team did further dissections that support the findings of these connections.

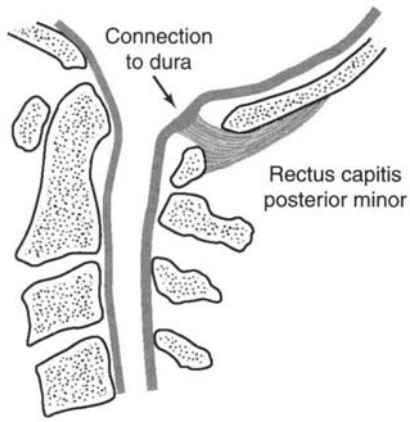
The high levels of mechanoreceptors in the rectus capitis posterior minor muscle emphasize the probable importance of its connection to the dura. "It has been suggested that the function of the RCPM [rectus capitis posterior minor] is to provide static and dynamic proprioceptive feedback to the central nervous system, monitoring movement of the head and influencing movement of the surrounding musculature.[1,72,73] Therefore, it seems plausible the RCPM muscle may act, at least in part, to

3—12. Obliquus capitis inferior

3—13. Obliquus capitis superior

3—14. Rectus capitis posterior major

3—15. Rectus capitis posterior minor



3—16. Connection to the dura from the rectus capitis posterior minor.

3—17. Rectus capitis lateralis

'monitor' stresses on the spinal dura mater by way of this connective tissue (muscle-dural) bridge."[54]

In addition to the dural role as it applies to the cranial-sacral primary respiratory system, there may be a role in the so-called "tension headache." Haldeman,[56] in a "Point of View" following the paper by Hack et al.,[53] states: "The observation in the present report that muscle may have a direct influence on the dura mater, a pain sensitive structure, suggests an alternative mechanism for pain generation for cervical headaches."

Intrinsic muscles of the upper cervical area may need to be treated with origin/insertion or muscle proprioceptive techniques. Analyze the subluxation direction and look for muscles that may be weak or hypertonic to perpetuate the subluxated position.

If, after correcting muscle imbalance, the subluxation remains, adjust in the vector that caused the greatest amount of indicator muscle weakening. Almost any specific method of adjusting is satisfactory to obtain an effective correction. Limit the adjustive force to the subluxated vertebra, avoiding general rotatory manipulation of the entire cervical spine.

A positive posterior challenge on the anterior portion of the atlas transverse process indicates atlas anteriority, which is best adjusted by occipital contact. Re-challenge with a broad contact on the occiput, applying a generally anterior vector of force. Find the specific vector that causes maximal weakening of an indicator muscle. This vector is usually from the contact point to the glabella, with the patient's head rotated away from the side of atlas anteriority. Apply a slightly superior lift to allow the atlas to move posteriorly. When the adjustive force is applied it should be limited to the occiput, avoiding contact to the temporal bone that might result in cranial faults.

3—18. Broad skull contact for anterior atlas adjustment

Primary Atlas Technique (PAT)

Maintenance of proper upper cervical function is paramount to normal function of the nervous system. The techniques of examining and treating this area are constantly being upgraded in applied kinesiology. Rather than strictly evaluating structural position, there are methods of determining when there is dysfunction, how to correct it, and how remote problems affect the maintenance of proper upper cervical function.

A frequent comment in applied kinesiology is "Above all level the head." The first observation that should be made in postural analysis is whether the head is level as viewed anterior to posterior and laterally. A level head, proper cervical vertebral movement, and muscle function are of primary importance because of the intricate relationship of the head-on-neck, visual righting, and labyrinthine reflexes. Muscle balance and function, as well as proper vertebral movement, are responsible for head position.

Insights into body organization come from the writings of Raymond Dart, anatomy professor at Witwaterstrand University in Johannesburg, South Africa.[21,22,23] His thoughts were published in the 1940s and republished in a dental orthopedic paper collection.[100] Dart's understanding of the body's integration has been a guiding light in advancing applied kinesiology integration of examination and treatment techniques.

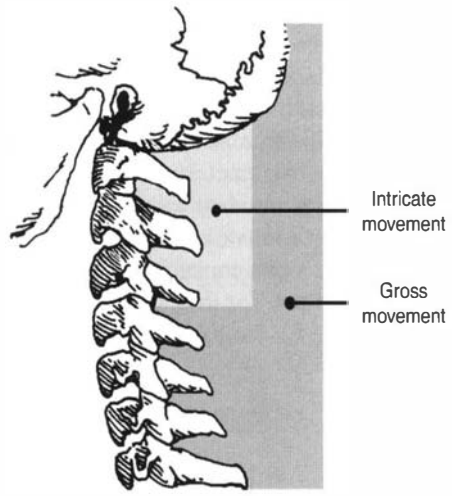
Dart[21] notes that Douglas "... claimed: (1) that the primary function of muscle is the 'relationing' of the various parts of the body to one another; (2) that their function as movers of body-parts upon body-parts is secondary; (3) that, of such 'relationing' in the body, the head-neck relation, brought about by the suboccipital group of muscles (atlas-occipital, axis-occipital, atlas-axis), is of paramount importance, and that it is worthy of the distinction of being recognized as ***'the primary relation upon which all more ultimate relations depend.'*** " (Emphasis added.)

The suboccipital muscles are unique in their nerve supply, all being supplied by the dorsal ramus of the 1st cervical spinal nerve,[119] providing only motor and no sensory supply. This group of small muscles is responsible for controlling the head-to-neck relationship, which provides small amounts of movement between the head and upper neck.

The general motion of the head-to-shoulder girdle relationship is provided by the sternocleidomastoid, upper trapezius, and the deep flexor and extensor muscles. There is a relatively small positioning mechanism in the limited upper cervical region to the head that is contained within the grosser movement mechanism of the head-on-shoulder girdle. The former small intrinsic muscles have a distinct motor supply without sensory supply, and the latter has a relatively wide nerve supply.

The relationship of the upper cervical vertebrae, their muscles, and head position is paramount to or-

ganization throughout the body. Dysfunction here causes disorganization between the head-on-neck, visual righting, and labyrinthine reflexes. The tonic neck reflexes, later in life called the head-on-neck reflexes, have their recep-



3—19. Intricate movement of the upper cervical area is contained within the gross movement of the head-to-shoulder girdle.

tors in the occipitoatlantal and atlantoaxial joints without any contribution from the intrinsic muscles or skin.[90] The medial longitudinal fasciculus binds the three motor nuclei (oculomotor, trochlear, and abducens) together anteriorly, supplying the eyeball muscles. Posteriorly it connects the anterior horn cells supplying the musculature that links the head to the trunk.[21]

Finally, the important sensory link in determining the body's postural adjustment evoked by the longitudinal bundle is formed by the intersegmental fibers running from the vestibular nucleus to the eye muscle nuclei anteriorly, and to the anterior cervical segments and posterior co-extensive spinal nucleus of the spinal accessory nerve. The intersegmental fibers coming from the brain segment supplied by cranial nerve VIII (vestibular) cause those simultaneous modifications in tension in the musculature that controls the position of the eyes relative to the head, and of the head relative to the trunk and forelimbs. These are occasioned reflexly by the ever-changing positions of a mobile head (supplied by a single pair of tactual nerves).[21]

Most of this description is from Dart's writings. A recent overview of the somatosensory system of the neck points out three posture-related reflexes.[11] 1). The cervicocollic reflex activates neck muscles when head position change stretches the muscles. Normally the cervicocollic reflex is integrated with other reflexes, such as the vestibulocollic and optokinetic reflexes, to assist in the maintenance of head position. 2). The tonic neck reflex alters limb muscle activity when the body moves in relation to the head acting to maintain a stable posture. The tonic neck reflex is integrated with other reflexes, such as the vestibulo-spinal reflex, to achieve postural stability. 3). The cervico-ocular reflex, with the vestibulo-ocular and optokinetic reflexes, assists in maintaining eye position. Bolton[11] summarizes his review by stating, "It is clear that in addition to signaling nociception, the somatosensory system of the neck may influence the motor control of the neck, eyes, limbs, respiratory muscles and possibly the activity of some preganglionic sympathetic nerves."

The importance of the head-on-neck relationship has been recognized by many. Several in chiropractic have given special attention to the area, and some have even devoted exclusive attention to the analysis and correction of upper cervical problems. Alexander,[5] an actor and teacher, taught proper body use with concentration on proper head carriage. A quick, simple method of detecting dysfunction, determining correction needed, and monitoring the functional improvement should be part of every physician's skills.

For many years this author has observed that there is often a positive vertebral challenge of the atlas when there is not positive therapy localization over the vertebra. Goodheart,[49] by a chance observation, noted that there is often positive TL over the atlas when the skin is touched with the patient's thumb but not with a finger or fingers. He rationalized that this occurs because the atlas has no sensory root; therefore the skin over the atlas is less sensitive to TL. In some manner — possibly because of a higher quantity of receptors in the thumb — there is a close, if not exact, relationship of positive atlas challenge to thumb TL over the atlas transverse process, indicating an atlas subluxation. Therapy localization should be done over the transverse process because there could be positive TL to the upper cervical muscles, as previously described.

When an atlas subluxation is identified by this TL, the atlas is challenged and adjusted in the usual manner. Because this is such an important area to have at zero default, Goodheart[50] described an extensive examination and treatment protocol, a portion of which is presented here. He has named this the Primary Atlas Technique (PAT) because of the numerous findings cleared by the treatment. In addition to being paramount in organizing the structure, the correction usually corrects chronic and active visceral faults. Treatment of the additional procedures outlined below helps eliminate recidivism of an atlas subluxation.

1. Note high occiput side.
2. Test temporomandibular joint with ipsilateral and contralateral therapy localization and note involvement.
3. Test for cerebellar involvement and note involvement (page 229).
4. Following each step of correction, test with sagittal suture tap technique and treat as applicable (page 132).
5. Relieve most painful TMJ side by ligament interlink

technique (page 211). Hold hyoid toward least painful side and digitally manipulate least painful side until there is a reduction of pain on the contralateral side.
6. Treat pterygoid muscle pain with spondylogenic reflex T2-4 tap technique (page 416).
7. Establish EID direction, which is usually down on the low occiput side (page 43).
8. Establish active versus passive cervical rotation and lateral flexion range of motion. Limitation of cervical rotation indicates a probable anterior atlas on that side because the atlas anteriority has already taken up a portion of the rotation.
9. Correct with cervical compaction technique (page 108) with simultaneous EID, jaw opening and closing, and the patient deeply inspiring and expiring. This usually corrects the cerebellar, TMJ, and cervical range of motion.
10. Re-test the atlas transverse process with thumb therapy localization ipsilaterally and contralaterally. If still positive, challenge the atlas and correct. There may also be subluxations of the axis and/or C3.
11. Test and treat the abdominal muscles for weakness, including stretch, contraction, and repeated muscle action (pages 192, 201, 192, respectively).
12. Test sternocleidomastoid and deep neck flexor muscles; if weak, treat with 5 IVF factors. In addition, test muscle stretch reaction and strain/counterstrain for need of treatment.
13. Test sternocleidomastoid, upper trapezius, deep neck flexor, and rectus capitis posterior minor muscles with the pinch test for involvement of myofascial gelosis (page 198). Treat each involved muscle with percussion.

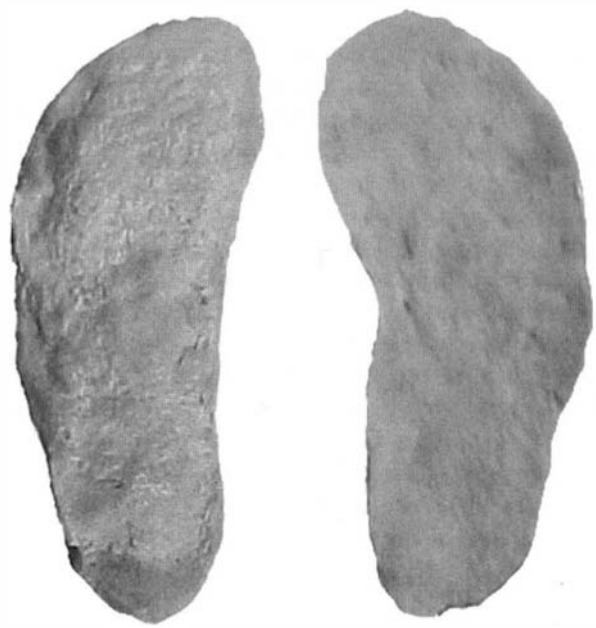
Sacral Distortion

Sacral distortion or dysfunction like the upper cervical area can cause wide-ranging adverse effects on body function. The importance of these areas can be recognized by the fact that in the past large numbers of chiropractors have devoted their entire practice to examination and treatment of the upper cervical vertebrae or to the sacrum.

Tension of the dura mater can cause many neurologic problems as described by Breig.[13] The important vertebrae in relation to the dura are the upper cervical vertebrae and the sacrum. There is firm dural connection with the upper cervical vertebrae with no further firm connection until the 2nd sacral segment.[119] This provides a unique type of therapy localization that will be seen as we proceed.

The sacrum is the foundation for the spinal column and has specific movement between the innominate bones that must be present for normal function. During gait the sacrum moves posteriorly, and the 5th lumbar rotates anteriorly about a vertical axis on the forward leg side. When this motion becomes aberrant, the entire spinal organization can be disturbed. Goodheart[50] comments: "The sacral subluxations come nearer explaining the persistent subluxation lesion than does any combination of lesions."

The sacrum can be subluxated inferiorly on one side or both. Sacral subluxation is improper positioning of the sacrum between the ilia; it is not a sacroiliac subluxation. When the sacrum is inferior on one side, it is impossible to adjust the ilium to the sacrum. If it is attempted, the patient will probably be made worse than before help was sought. Usually either the sacrum or innominate bone is subluxated, i.e., the sacrum is subluxated and the innominate bones are in normal position, or an innominate bone is subluxated and the sacrum is in normal position. If there is a complex of dysfunction between the sacrum and innominate bones, it is usually a pelvic category I that is found by examination and corrected after the sacrum is corrected.



Sacrum *Innominate*

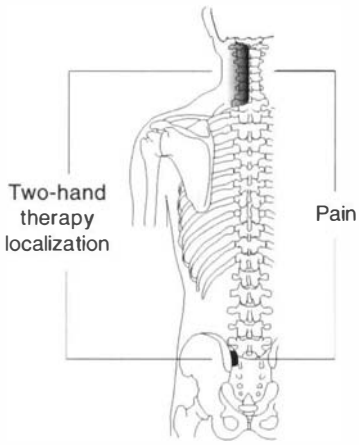
3—20. Matching surfaces of the sacroiliac articulation. The surfaces are slightly curved with the sacrum convex and the pelvic surface concave.

Sacral Subluxation Examination

Unilateral or bilateral palpatory pain at the nuchal ligament indicates probable sacral subluxation. Palpate the nuchal ligament from the external occipital protuberance to 7th cervical vertebra, comparing discomfort bilaterally. Have the patient grade the pain on a scale of

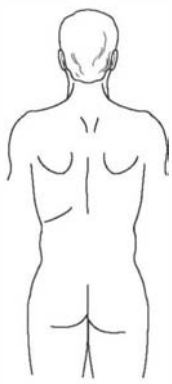
0-10 for later comparison. Nuchal ligament pain is due to the spondylogenic reflex of the sacrospinous and sacrotuberous ligaments.[27] (See page 129.)

There will not be positive therapy localization at the nuchal ligament or at the sacrum. If positive TL is present in the cervical region, check for and correct cervical subluxations and/or any other factor that could be responsible for the finding, e.g., neuromuscular spindle cell or Golgi tendon organ muscle involvement. There will be positive therapy localization when the nuchal ligament and sacrum ala are therapy localized simultaneously. The two-handed therapy localization appears to reflect the relationship of the firm dural contacts to the upper cervical vertebrae and sacrum.



3—21. Positive therapy localization is only present when both the cervical vertebrae and sacrum are therapy localized together.

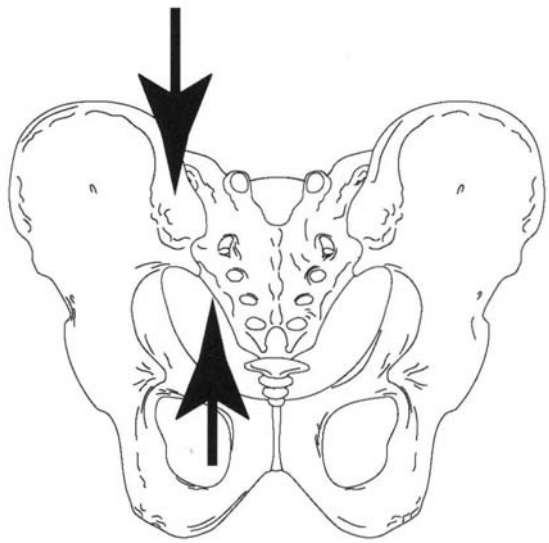
The nuchal ligament involvement may or may not be observed on postural analysis. Rely more on the pain and tension of the nuchal ligament observed by palpation. The gluteal fold will be lower on the inferior sacral side. There is usually a skin crest fold under the scapula contralateral to the inferior sacrum, unless the patient is thin. The iliac crests will be level, and the lumbar spine will deviate toward the inferior sacral side from the L5 level. The contralateral arm will be flexed.



3—22. Nuchal ligament pain and lower gluteal fold on right and deeper skin crest fold under left scapula indicate right inferior sacrum.

Pain at the sacral ala indicates sacral lesion. Palpate for the pain just medial to the posterior superior iliac spine. Again have the patient rate the pain on a scale of 0-10 for further comparison.

Direct challenge to the sacrum will often be negative with the patient prone, but a superior challenge on the lateral aspect of the sacral apex while keeping the ilium from moving superiorly will be positive for an inferior sacrum on that side. It may be necessary to change the vector of challenge to observe positive results.



3—23. Press on the posterior superior iliac crest and sacrum to challenge for an inferior sacrum. The vector of simultaneous pressure may need to be varied to obtain a positive challenge.

When there is a sacral fault, prone leg length indicates the sacral position. Leg length measurement is done by the examiner placing his thumbs inferior to the internal malleoli for comparison. In a unilateral sacral fault, the sacrum is inferior and anterior on the long leg side when there is unilateral nuchal ligament pain on that side.

A bilateral sacral fault will have bilateral nuchal ligament and sacral alae pain, positive two-handed therapy localization bilaterally, and the legs will be even. There will be a flattening of the lumbar curve.

An oblique sacral fault will have bilateral nuchal ligament and sacral alae pain, positive two-handed therapy localization bilaterally, and the legs will be uneven. The long leg is the anterior inferior sacral side, and the short leg is the posterior superior sacral side.

Correction

The piriformis muscles are important in sacral stabilization and function. Examine for piriformis weakness or stretch reaction before treating the sacral subluxation. Other muscles involved in pelvic support and function should also be evaluated and treated with the 5 IVF factors, when appropriate.

A nuchal ligament-sacral reflex needs to be broken before adjustment. This reduces muscle tension and makes correction easier and more complete. Contact the tenderness at the nuchal ligament with one hand and the sacral ala with the other. Hold the contacts until tissue relaxation takes place, which indicates the reflex is broken. This may take up to thirty seconds. If the condition is bilateral, both sides need to be treated with the same double contact. There will be reduction of palpatory pain, but it may not be as great as after the subluxation is corrected. Although breaking this reflex reduces tissue tension and relieves pain, it does not correct the condition. Without correcting the sacral position, improvement from breaking the reflex will be lost as soon as the patient walks.

Anterior-Inferior Sacrum. With the patient prone, the physician stands on the side of lesion and makes a thumb contact on the sacral ala close to the posterior superior iliac spine. The thumb contact is held firmly but not with a heavy pressure. Stabilize the posterior iliac spine with the rest of your hand. With the other hand grasp the patient's ankle and flex the knee to stretch the rectus femoris, which rocks the ilium anteriorly. If the patient complains of excessive knee or thigh pain, reduce the knee flexion to tolerance. Hold the knee flexion until you feel the sacrum move superiorly. The movement is very subtle and may feel more like tissue relaxation.

In some cases, tension on the rectus femoris is inadequate to rock the ilium anteriorly because of knee pain or very loose knee range of motion. In this case maintain knee flexion at a comfortable position for the patient, and extend the hip by lifting the thigh from the treatment table. If the position needs to be held very long, the physician can place his thigh under the patient's leg by hip and knee flexion.

Re-evaluate the two-handed therapy localization, leg length balance, and tenderness at the nuchal ligament and sacral ala. In most cases, the pain will be greatly reduced. Sacral correction should relieve 95-100% of nuchal ligament pain. If nuchal ligament pain remains but the two-handed therapy localization is negative and the legs are balanced, treat the sacrospinous and sacrotuberous spondylogenic reflexes (page 129).

Bilateral Anterior-Inferior Sacrum. Raise the patient's pelvis by table adjustment or placing a roll under the pelvis. Flex the patient's knees and hold them in position with your chest. Contact the sacral alae with bilateral thumb contacts just medial to the posterior superior iliac spines. The posterior ilia are stabilized with the rest of your hand. Slowly increase flexion of the patient's knees by pressing on his legs with your chest. Sacral movement is monitored by your thumb in relation to the rest of your hand. Relaxation and eventual sacral movement take time, and patience is important. When both sides have moved up, the correction is made. The correction may not be effective on the first effort. Finally, re-evaluate the pain level at the nuchal ligament and sacral alae, two-hand therapy localization, and leg length. If the legs are now uneven, only one side of the bilateral sacral inferiority has been corrected.

There is frequently an anterior thoracic subluxation with the bilateral sacral inferiority (see page 73). It is usually best to correct these before the sacral correction is made.

Oblique Sacrum. The anterior-inferior sacrum side is corrected in the same manner as when it is a unilateral condition. The posterior superior side is challenged in the usual manner of vertebral challenge and corrected with an adjustive thrust in the vector of maximum positive challenge.

Respiratory Adjustment

A non-forceful technique has been developed in applied kinesiology for vertebral correction when force is contraindicated. This is applicable when treating recent trauma such as a hyperflexion-hyperextension (whiplash-type dynamics) cervical sprain. Forceful adjustment of the vertebra would probably cause further soft tissue damage. This method can be used when there is contraindication to the usual adjustive techniques, e.g., pathology, osteoporosis.

The vertebra is challenged as usual to find the vector that causes maximum weakness of an indicator muscle. While the muscle is still weak, the patient is asked to take a deep inspiration. If the indicator muscle immediately becomes strong, this is the phase of respiration on which to make the correction. If there is no strengthening of the indicator muscle with inspiration, re-challenge and have the patient deeply exhale; then test the indicator muscle for strengthening while the patient holds the exhalation.

The respiratory adjustment is made by pressing with four to six pounds on the spinous or transverse process of the vertebra in the vector that caused maximum indicator muscle weakness, while the patient takes the phase of respiration that abolished the challenge. Repeat six or seven times, then re-evaluate the patient with challenge and therapy localization to determine that the effort was effective.

Persistent Subluxation

Ideally, when a subluxation is corrected it does not recur. When the same subluxation must be corrected numerous times, there is either an intrinsic or a remote problem causing the recurrence. The intrinsic muscles of the sacrospinalis can be evaluated with therapy localization and treated with origin/insertion and muscle proprioceptive techniques. If the therapy localization is very specific, one can usually determine which of the intrinsic muscles is at fault and apply accurate treatment to the muscle.

The neurolymphatic reflex for all intrinsic spinal muscles is at the junction of the clavicle, sternum, and 1st rib. This is the site of acupuncture point KI 27, which is an indicator in applied kinesiology for neurologic disorganization.

Evaluating the spine with manual muscle testing finds subluxations and provides information about how to correct them. More important, it tells whether the corrective effort is effective; more important still, it tells whether the correction is lasting. Many individuals lose their chiropractic corrections while walking from the treatment room to the front office. Obviously, no matter how good a correction is, if it doesn't last it is of no value.

Areas corrected during a previous treatment should always be evaluated for recurrence of the problem. If the correction must be repeated, further evaluation should be made to determine why the original correction was lost. The additional evaluation often must be of remote areas not directly relating with the subluxation or its intrinsic muscles.

Sometimes subluxations appear to be secondary to imbalance of the meridian system. Along the spine on the bladder meridian are acupuncture points called associated points. When these points are active there is often a subluxation of the adjacent vertebra. The meridians must be balanced and the subluxation corrected.

Extrinsic muscles may cause general body distortion that influences the subluxation. Often a correction is lost immediately when the patient stands, in which case the weight-bearing mechanism must be evaluated. Sometimes the correction holds until the patient walks, chews some food, or has some type of chemical or mental stress.

Neurologic disorganization is often the cause of persistent subluxations. Proper facilitation and inhibition of muscles attaching to the vertebrae must be present for organization during walking and other movement. As one becomes familiar with applied kinesiology, it is obvious that many factors can cause an improper temporal pattern of spinal muscles.

There are several procedures in applied kinesiology that test groups of muscles together. The group tests usually evaluate how the modules of the body (e.g., pelvis and shoulder girdle) work together. When there is disorganization of the module, the spine is often strained and subluxations result. If the disorganization is not corrected, the subluxations will quite likely return.

Walking activates many factors that cause subluxations to recur. Consideration should be given to foot dysfunction, dural tension, equilibrium reflex synchronization, and category I and II pelvic faults.

A key factor in evaluating persistent subluxations is to have the patient do the things he normally does every day. These may be activities at work, sports, and exercise. When the factor causing the recurrent subluxation is found, the physician can then analyze it to determine the type of corrective effort needed. It can be on any side of the triad of health, and it may require any of the therapeutic approaches used in applied kinesiology.

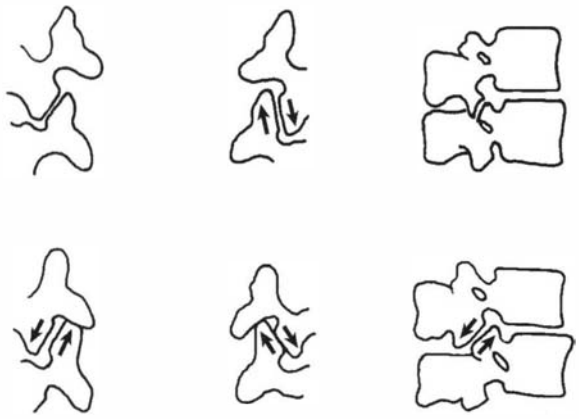
Imbrication Subluxation

Facet imbrication[61] is a subluxation wherein the articulations overlap in a shingle-like manner. This type of subluxation is more prevalent in the lumbar spine but it can occur elsewhere, especially in the cervicals. The etiology is usually chronic postural strain or the sudden trauma of a compressive force to the spine, such as jumping from a roof and landing on one's feet. The structural strain type is the slow, progressive, insidious development of the condition due to hyperlordosis and/or hyperextension of the lumbar spine. Loss of intervertebral disc space from disc degeneration will also change the articular facet relationship, causing an imbrication subluxation.

Diagnosis. Objective evidence of an imbrication subluxation is seen on oblique x-rays of the lumbosacral spine. X-rays taken in a PA projection provide less distortion because the rays travel with the concavity of the lumbar lordotic curve.

Imbrication may be unilateral or bilateral. It is seen as a slipping of the superior facet upon its mate, and is an interruption of Hadley's S-curve or line. The inferior portion of the superior facet slides down into the intervertebral foramen, compromising the space. The imbrication may be so severe that it forces the superior facet into the pars interarticularis of the vertebra below. The impingement may erode the bone structure, causing a painful irritation and weakening of the pars interarticularis.

Clinical orthopedic tests should support the x-ray diagnosis. Local lumbar pain is accentuated by extension of the lumbar spine. Kemp's sign of oblique exten-



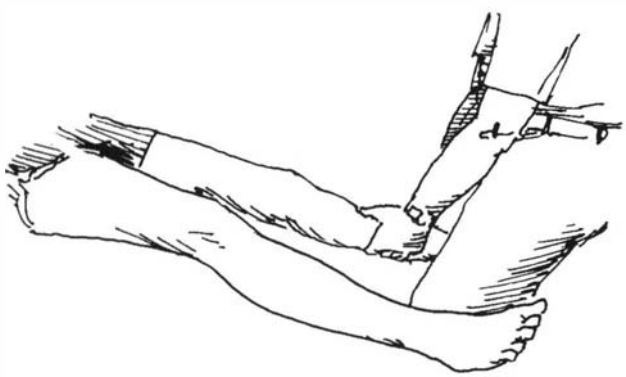
3—24. Facet imbrication as observed on oblique and lateral x-ray projections.

sion accentuates the pain at the level of lesion, but it does not cause a radicular radiation of pain into the lower limb, as does an intervertebral disc condition.

There will be positive therapy localization over an area of facet imbrication. In combination with the x-ray, orthopedic, and therapy localization findings, challenge will confirm the presence of an imbrication subluxation. The subluxation will not show a positive challenge when the usual methods of pressing on the spinous, transverse, or mamillary processes in various vectors are used, with the possible exception of an inferior-to-superior challenge on one of the structures. The specific challenge for facet imbrication is separation of the vertebrae. This is accomplished by stabilizing the area above or below the imbrication, and separating the vertebrae with traction on the leg or arm.

To challenge for a facet imbrication, the facets are separated and a previously strong muscle is tested for weakness. Convenient muscles to test when the leg is tractioned are the tensor fascia lata and piriformis. The lumbar spine can also be evaluated by tractioning on the arm and testing for an indicator muscle weakening. The usual muscle evaluated is either division of the pectoralis major.

The level of facet imbrication is usually diagnosed on x-ray. Correction is accomplished by stabilizing the patient's vertebra above or below the imbrication, depending on whether traction is applied to the leg or arm for deimbrication. Stabilization adjacent to the imbrication is accomplished by having the patient lie on a block that is approximately 4" x 12" x 1-1/2". DeJarnette blocks are readily available in most offices and can be used. The tapered ends of the blocks approximate each other at the patient's spinous processes.



3—25. Traction on the leg applies a rebound challenge for imbrication subluxation.

If the level of facet imbrication is not known, it can be determined by placing the stabilizing block(s) at the L5 level and applying traction to the leg. If challenge is positive, the facet imbrication is between L5 and S1. If challenge is negative, reposition the block(s) to L4 and re-challenge. If negative, proceed up the spine until the challenge becomes positive.

Good stabilization of the vertebra above or below the imbrication is important. Poor stabilization allows the adjustive force to dissipate, often precluding an effective correction. If the examination table is excessively soft, a fairly large board should be placed on the table and the block(s) placed on the board.

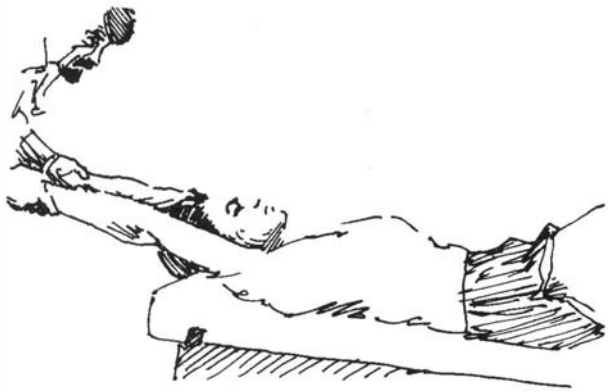
Treatment. To correct lumbar imbrication, stabilize the patient's lumbar vertebra above the facet imbrication level as indicated above. Apply traction to the leg on the side of facet imbrication. Move the patient close to that side of the table so that the leg can be abducted and slightly extended. Flex the contralateral leg at the hip and knee, with the patient's foot resting flat on the table. This position reduces some of the lumbar lordosis and aligns the tractional force with the facet plane. The patient holds the edges of the table to further stabilize himself. This is particularly necessary for lightweight individuals. Apply a quick, sharp traction force to the leg by contacting above the knee. An audible release at the level of the facet imbrication will often be obtained, but it is not necessary. Knee trauma is precluded by the tractional force being applied above it. Re-evaluate with challenge and therapy localization to determine if the adjustive attempt was successful.

Holmes[64] describes an alternate method for stabilizing the patient. The patient's position is the same as described above. The physician contacts above the knee with one hand only, using his opposite hand to stabilize the patient's contralateral, flexed knee. To use this method, the physician must be able to apply adequate traction above the knee with only one hand while his other hand stabilizes the patient at the flexed knee.

When imbrication is bilateral or at several levels, it is necessary to successively apply the correction. If there are many levels of imbrication, one begins by correcting the uppermost level first if traction is applied to the leg. The initial corrective effort may correct all the levels. If challenge and therapy localization indicate that all levels were not corrected, lower the block(s) to the appropriate level and repeat the procedure. If the imbrication is bilateral and the initial effort does not correct both sides, move

the patient to the opposite side of the table and repeat the procedure.

When there is hip pathology or some other condition that may be traumatized by traction on the leg, the physician has the option of applying the traction through the arms. In this case, the block(s) is placed on the lower vertebra of imbrication. The patient's hips and knees are flexed so that the feet are flat on the table. This reduces the lumbar lordosis and improves the facet alignment for correction. Corrective traction is applied in a quick, sharp manner to the 180°-flexed arm for unilateral imbrication, or to both arms for bilateral imbrication. Again, re-evaluate with challenge and therapy localization to determine that the corrective effort was effective.



3—26. Traction can be applied to the arms for imbrication correction. The block stabilization is below the subluxation.

Prior to adjustment, evaluate the muscles responsible for maintaining pelvic and lumbar alignment. There is often hypertonicity of the lower sacrospinalis, weakness of the abdominals and gluteus maximus, and poor hamstring function. Intrinsic muscles of the spine may also be involved. Of particular interest is possible hypertonicity of the intertransversarii muscles. Correct any muscle weakness or hypertonicity before adjusting the facet imbrication.

To effectively correct facet imbrication, it is necessary to apply traction in axial alignment with the spinal column. Side posture rotary lumbar adjustments are ineffective, as are mamillary or spinous contacts with the patient prone.

Facet imbrication may be present in combination with acute intervertebral disc conditions. Treatment of the facet imbrication is contraindicated in the presence of an acute disc condition. First, treat the disc involvement. When it is resolved, the imbrication technique can be applied. One will often see symptomatic improvement as the disc involvement is resolved, though localized pain remains in the lumbar area. At this time treat the facet imbrication; there will often be a rapid resolution of the remaining pain.[63]

Facet imbrication can also occur in the cervical spine.[87] It is differentiated from other cervical subluxations by negative challenge when done by the usual spinous or transverse process contact. There will be a positive challenge when the physician applies cephalad traction to the occiput. This is done with the patient supine. The physician applies traction with a broad contact on the occiput, releases it, and tests for weakening of an indicator muscle. The corrective force is a rapidly applied traction in the vector of positive challenge. The physician must take care to have a broad contact on the occiput and avoid applying force to the mastoid processes, as this might cause iatrogenic cranial faults.

Intraosseous Subluxations

Therapy localization and challenge have been invaluable assets in determining when a subluxation is present, how to adjust it, and whether the corrective effort is effective. Furthermore, these examination tools have helped determine the cause of recurring subluxations. As mentioned, one should ideally have to adjust a subluxation only one time. Sometimes subluxations recur almost immediately when a patient stands or walks. The recurrence of a subluxation and efforts to determine why it has done so have been responsible for many advances in applied kinesiology examination and treatment.

Goodheart[44,45] has observed on a clinical basis several consistent findings regarding vertebral therapy localization, challenge, and therapeutic efforts. He has associated the hologramic model of the nervous system with some of the AK findings regarding vertebrae. Goodheart's method of investigation has been to find physical measures that eliminate the positive therapy localization and/or challenge to a vertebra. He then tests for improved range of motion, muscle function, and other factors to determine if they improve from the therapeutic effort.

Weight-bearing Change

When therapy localization and challenge give evidence that a subluxation has been corrected, re-evaluate it with therapy localization with the patient standing. If

positive therapy localization returns, sharply tap the spinous process approximately ten times. Frequently the positive therapy localization or any other indication of a subluxation is removed by this process. It appears to aid in the maintenance of the vertebral correction. Goodheart[40] attributes the improvement to a piezoelectric effect in the bone, probably associated with hologramic memory in the vertebra.

Under normal circumstances, one can cause a strong muscle to weaken by temporal tapping with the suggestion, "The muscle will be weak" on the left side, and "There is no need for the muscle to be strong" on the right side. After adjusting a vertebra and tapping the spinous process as indicated above, there will no longer be a weakening of muscles supplied by nerves at or below that vertebral level with the temporal tap, but there will continue to be a weakening with temporal tap of muscles supplied by nerves exiting above the vertebral adjustment level.

Intraosseous Subluxation

An intraosseous subluxation, sometimes called a hologramic subluxation, is explained by microscopic stress within the bone's crystalline structure, which is considered to distort the hologramic memory of the local area.

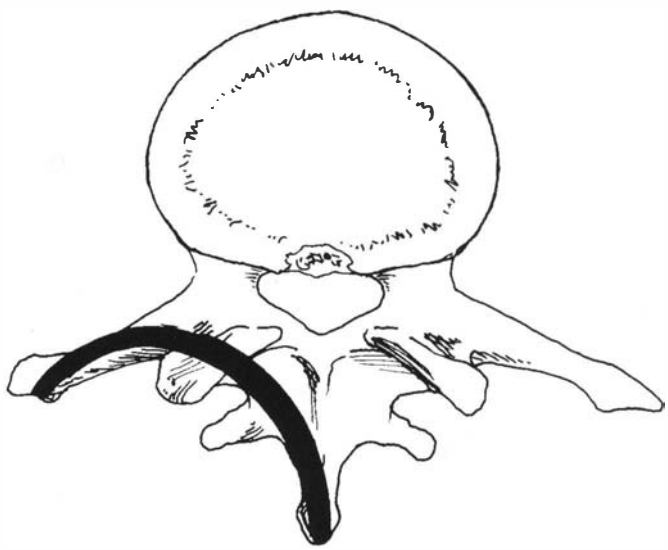
Goodheart attributes changes observed in function due to an intraosseous subluxation to two aspects of the hologramic memory model. (1) The body's image is not in one place; it is all over, and (2) there is a perfect three-dimensional image of the total body in the brain. As long as the local image of body structure parallels that perfect hologramic image in the central nervous system, balance is present. When the structure is distorted, the brain's perfect hologram does not match. One neurologically becomes aware of the distortion and/or disturbance.

This fits well with various aspects of the hologramic model. Greguss[52] states, "According to our model an organism is living and stays alive as long as it can handle — process — all the information patterns it receives independently of their form and origin." When there is failure of coherency of a given information pattern, the organism experiences pain or some other dysfunction.

Perhaps drawing an analogy with an industrial use of holograms will help clarify this concept. The hologram has become very valuable in non-destructive testing. This is called hologramic interferometry, and it is used in product research and design. A hologram is produced of some structure, such as an automobile tire or an airplane wing. Force is then applied and another hologram is made. When the hologram is reconstructed, if there has been any change between the first hologram and the second, there will be a lack of coherency and Newton rings will develop. The ability to measure deformations down to 1/3,000 mm makes this technique too precise for ordinary use. A method to reduce the sensitivity has been developed for practicality.[35] Hologramic interferometry is not limited to industrial use for research and design. It has been applied to how physical stress affects bone structure. An example is applying stress to a vertebral body (*in vitro*) before and after two holograms are taken, but exposed on a single photographic plate. The reconstruction of the interferogram with laser light yields an image covered with interference fringes, which highlights regions of deformation of the surface of the vertebra.[110]

Goodheart's model — a perfect body image in the central nervous system being compared with the images throughout the body — seems to relate with hologramic interferometry. When there is lack of coherency between the two, an awareness develops for the nervous system to attempt adaptation. Pain or dysfunction may result.

Goodheart postulates that various portions of the bony structure of the skull and framework of the spine act as wave sources, similar to the object and reference beams in a photographic hologram. The waves may



3—27. It is hypothesized that there are waves from the transverse and spinous processes that form the object and reference beams of a hologram.

emanate from the bone as piezoelectric waves.

In the case of a vertebra, it is hypothesized that the waves from the spinous and transverse processes become incoherent due to a microcompaction or separation of the bone's matrix. The therapeutic approach is to apply pressure toward separation or compaction of the matrix.

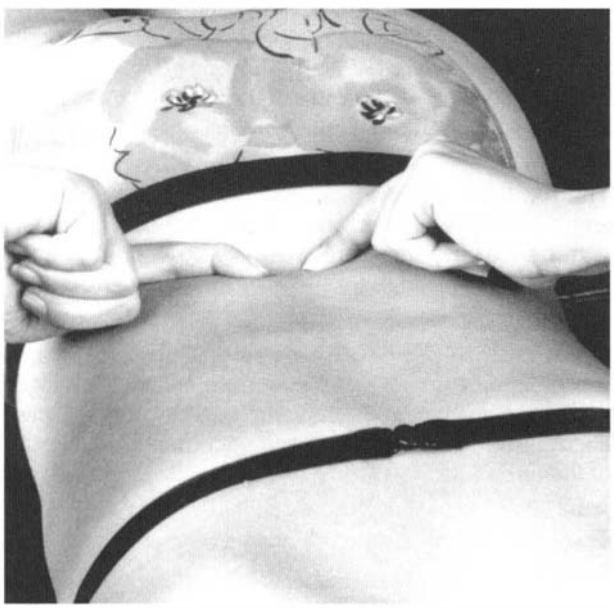
In applied kinesiology there are predictable ways the body reacts to various challenges and therapy localization under normal function. The intraosseous vertebral subluxation does not show positive therapy localization in the usual manner; consequently, this type of dysfunction has been overlooked for many years.

The intraosseous vertebral (holographic) subluxation is located by characteristic therapy localization. First, the vertebra is generally therapy localized. If there is a positive therapy localization, evaluate for subluxations, involvement of the intrinsic muscles, active posterior neurolymphatic reflex, or active associated acupuncture points. If any of these are present, they should be corrected before evaluating for an intraosseous subluxation.

In the presence of negative one-handed therapy localization to the vertebra, therapy localize with one finger on the spinous process, and with the other hand very accurately therapy localize the transverse process. Weakening of a previously strong indicator muscle indicates a probable intraosseous subluxation. There will be no weakening with only one hand on the spinous process, or one hand on the transverse process; it requires the two-handed therapy localization.

The intraosseous subluxation will also be revealed by the interlaced finger method of therapy localization, probably because of bringing the two sides of the brain into analysis.[48] It is better to use the two-handed, two-finger method because it helps specifically isolate the contact points for adjustment

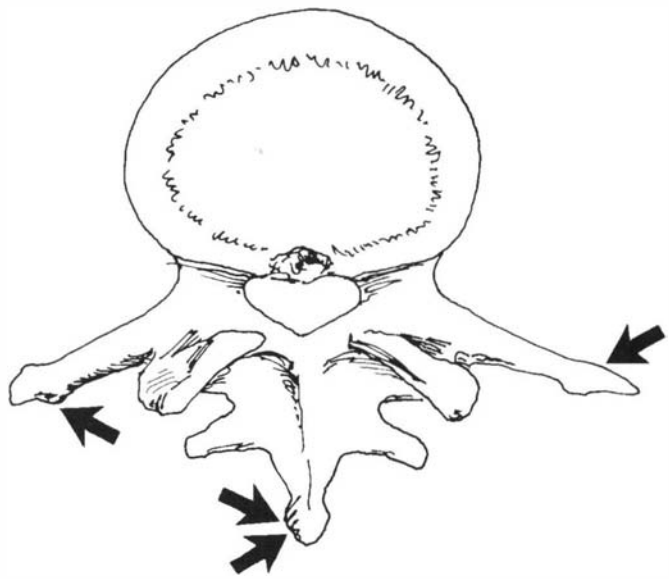
When there is two-handed therapy localization,



3—28. In an intraosseous subluxation there is positive therapy localization with one hand on the spinous process and the other on a transverse process, but none with single-handed therapy localization.

the physician challenges the vertebra by contacting the transverse process with one thumb and the spinous process with the other. Pressure is directed toward separating the spinous and transverse processes, and a previously strong indicator is tested for weakening. The spinous and transverse processes are also challenged by approximating them. The positive challenge is usually in a direction toward separation. When there is a positive challenge, the indicator muscle will remain weak long enough to test it several times. In the presence of a muscle weak from challenge, determine the phase of the patient's respiration that immediately abolishes the weakness. Holding a deep breath will usually eliminate the weak indicator muscle test.

Correction of the intraosseous subluxation is made by simultaneously contacting the spinous and transverse processes in the same manner as when challenging. Two moderate simultaneous thrusts are applied to the spinous and transverse processes in the direction of positive challenge while the patient holds the phase of respiration that abolished the challenge. Successful correction is indicated by a negative challenge and negative two-handed therapy localization. No further treatment is required for the intraosseous subluxation. It does not appear to have a consistent muscle imbalance pattern. It is unusual to have to repeat the treatment to the vertebra.



3—29. Challenge of an intraosseous subluxation is made by spreading or approximating the spinous process from or with a transverse process and testing a strong indicator muscle for weakening.

More study is needed to expand the concept of the hologram and its relation to the nervous system. The intraosseous vertebral subluxation is a useful technique that will probably become more widely used as it is better understood. It is advantageous in that it is effective and non-traumatic, and no undesired effects of the treatment have been documented. It has been reported effective in conditions that were treated unsuccessfully with other AK techniques.[30] Goodheart recommends that the vertebra be evaluated for an intraosseous subluxation after the spine has been adjusted for a subluxation, especially when there is an occipital fault.[43]

Vertebral Fixations

Many in chiropractic have used the terms "subluxation" and "fixation" interchangeably, and there have been numerous definitions and descriptions of both. We will not attempt to define either a subluxation or a fixation here; rather, we will explain the functional differences between the two as observed by manual muscle testing in applied kinesiology.

A fixation involves at least two vertebrae, and usually three. The triple vertebrae involvement is probably due to the muscular attachment of the rotatores longus and brevis, with the longus spanning a three-vertebrae complex. The differences between vertebral subluxations and fixations are listed below.

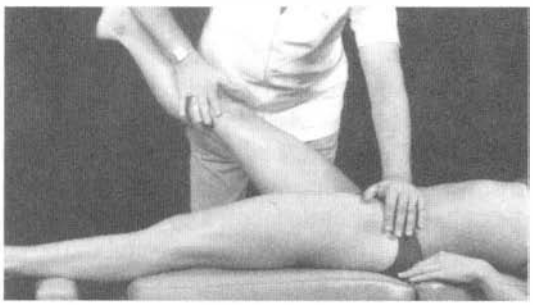
Subluxations	Fixations
Structures Involved	
One specific structure of the spinal column is involved in a subluxation. It can be a vertebra, a portion of the pelvis, or the occiput that is out of normal function with the rest of the spinal column.	In a fixation complex a minimum of two structures will be involved, and they will have restricted movement between them. Usually three vertebrae are involved in a fixation; however, there may be two or up to five (and possibly even more).
Muscle Weakness	
There is no consistency of muscle weakness associated with vertebral subluxations. The wide range of neurologic ramifications can cause almost any muscle associated with a specific spinal subluxation to be weak.	There are specific bilateral muscular weaknesses commonly associated with vertebral fixations (discussed on the next page).
Therapy Localization	
Therapy localization over a subluxation will cause a strong indicator muscle to weaken, or a muscle weak as a direct result of the subluxation to strengthen.	When therapy localizing over a vertebral fixation, a previously strong indicator muscle will not weaken unless there is an attempt to introduce motion into the fixation complex. This is done by therapy localizing over the suspected fixation complex while the patient actively moves the spine in that area. In the presence of a fixation, an indicator muscle will weaken. Therapy localization over a fixation will strengthen the bilateral muscle weakness associated with the fixation.
Challenge	
The vertebra or other spinal structure is challenged with a single point of contact.	There will usually be no reaction to a single-point challenge. Challenge is accomplished by challenging two vertebrae at the same time, usually by pressing in opposite directions on the spinous or transverse processes.
Static X-ray	
A subluxated vertebra is usually observable as misaligned on a static x-ray film.	Generally no misalignment between fixed spinal structures is observed on x-ray. The mechanism at fault is a lack of motion between contiguous vertebrae, rather than a misalignment causing apparent encroachment on the radix of the nerve.
Motion X-ray	
Serial static x-rays or cineroentgenography will usually show aberrant movement of the subluxated vertebra. This is apparently due to stimulation of the hyperactive intrinsic muscle or muscles involved with the subluxation complex.	There will usually be hypokinesis of the spinal fixation complex.
Correction	
A subluxation can be adjusted with a single point of contact.	A fixation requires a two-handed contact or some other method of stabilizing one of the structures while the other is manipulated, because a single-handed contact just moves the entire complex rather than unlocking the mechanism. Occasionally a fixation complex is unlocked with a single-handed contact; however, such results are due more to luck than calculated correction.

Bilateral Muscle Weakness of Fixations

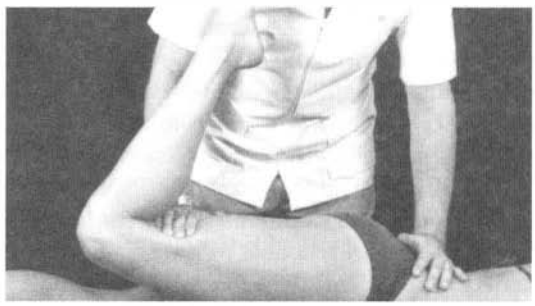
When a fixation complex is present in the spinal column, it has a specific bilateral muscle weakness. The bilateral weakness observed by Goodheart[38] reveals many spinal fixations that would otherwise be overlooked.

Fixation levels associated with muscles that test weak bilaterally are as follows:

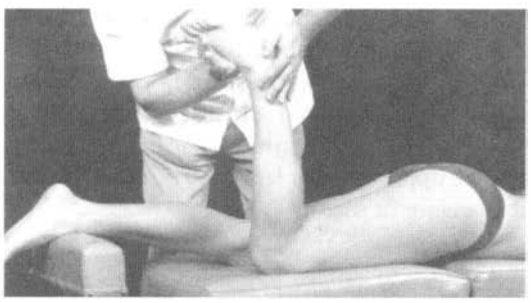
1. Occipital fixation — bilateral psoas muscles.
2. Upper cervical fixation — bilateral gluteus maximus muscles.
3. Lower cervical fixation — bilateral popliteus muscles.
4. Cervicodorsal fixation — bilateral deltoid muscles (rarely, bilateral serratus anticus muscles).
5. Thoracic fixations — bilateral teres major muscles.
6. Dorsolumbar junction fixation — bilateral lower trapezius muscles.
7. Lumbar fixation — neck extensors test weak when tested together bilaterally.
8. Sacral fixation — neck extensors test weak bilaterally when tested individually or as a group.
9. Sacroiliac fixation — neck extensors test weak on one side only, and no other factor is found for the dysfunction.



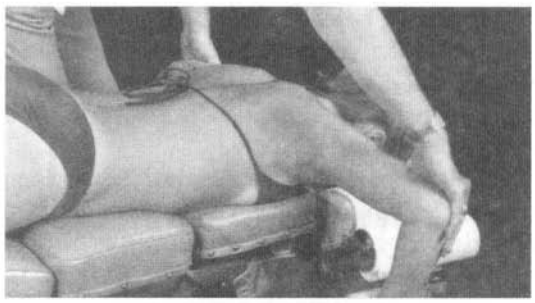
3—30. Psoas



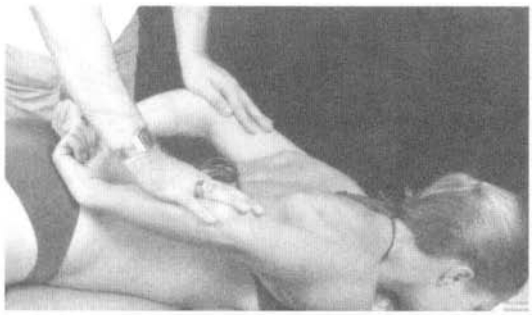
3—31. Gluteus maximus



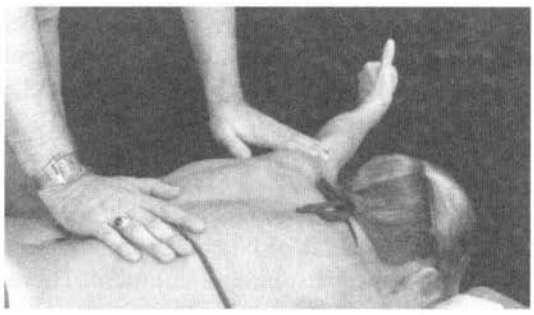
3—32. Popliteus



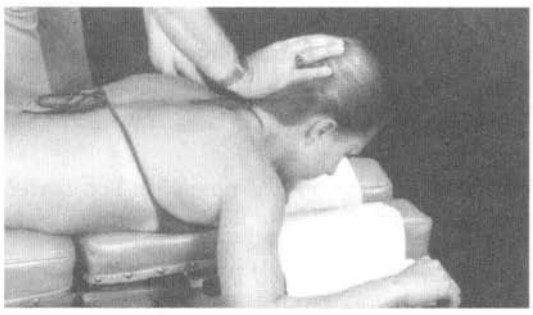
3—33. Middle deltoids



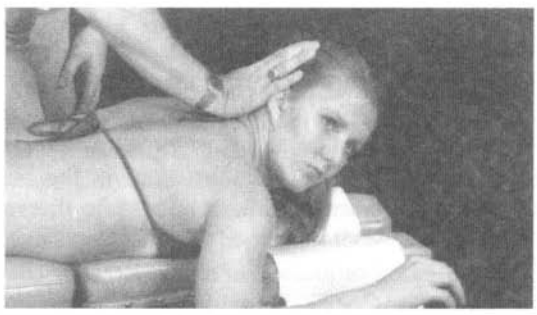
3—34. Teres major



3—35. Lower trapezius



3—36. Neck extensors — group



3—37. Neck extensors — right

The bilateral weakness appears to relate with the equilibrium proprioceptors located in the ligaments along the spine. Schmitt[103] designed a model to demonstrate this hypothesis. The most thoroughly studied spinal equilibrium proprioceptors are the tonic neck receptors in the infant or head-on-neck receptors in the adult. Their location relates with the upper cervical fixation and its associated bilateral gluteus maximus weakness. Schmitt demonstrated that contraction of the neck extensor muscles to forcefully extend the head on the neck causes weakness in previously strong bilateral gluteus maximus muscles as long as the contraction is held.

In individuals who can localize contraction of the extensor muscles, a similar demonstration can be performed. Contraction of the mid-cervical extensor muscles causes weakness of the bilateral popliteus muscles. Contraction at the cervicodorsal junction causes bilateral deltoid muscle weakness. The demonstration can be carried on down the spine with the appropriate muscles weakening with contraction, as long as the subject is capable of localizing contraction to a specific area. This demonstration is applicable in some subjects, but not all. The bilateral muscles do not weaken in those who have very organized nervous systems, apparently because their bodies recognize that weakening is not necessary under these conditions. Others who do not demonstrate the bilateral weakening as described above may be incapable of isolating the muscle contraction to the localized area.

Another method to demonstrate a fixation complex causing bilateral muscle weakness is to stimulate the intrinsic and paravertebral muscles of an area with sine wave current. While the localized muscles are contracting from the stimulation, the associated bilateral muscles will test weak. In a similar manner, a belt can be tightly placed around the innominate bones to create an artificial sacroiliac articulation fixation. In this case, the cervical extensor muscles will test weak while the belt is in place.

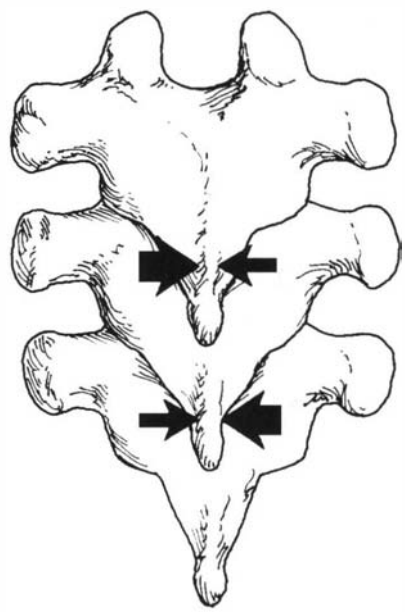
Evaluation and Correction

The general localization of a fixation complex is done in applied kinesiology by finding bilateral muscle weakness. Not all cases of bilateral muscle weakness will have an associated fixation complex. There are occasions when the bilateral weakness is due to some other aspect of the five factors of the IVF. To determine that the bilateral weakness is associated with a spinal fixation, have the individual therapy localize over the spinal area. If a fixation is responsible, the muscles will no longer test weak as long as the therapy localization is held. It may be necessary to have the individual therapy localize with the dorsal surface of the hands instead of the palmar surface to eliminate the weak tests.

The method of fixation analysis and correction is a modification by Goodheart of Martindale's[88] approach to evaluating the spinal column. The original system was a complex method of motion palpation to find structures of the vertebral column that were in fixation, and the key motion that would unlock the vertebrae. Goodheart's adaptation of the system, combined with bilateral muscle tests, simplified the procedure considerably, but it is still somewhat more complicated than some other approaches. Its advantage is that it consistently unlocks vertebral fixation complexes in a specific manner; other more generalized approaches frequently fail to do this.

The procedure consists of three basic steps. The first step finds the vertebrae involved in the fixation complex. The second step determines the direction in which the vertebral motion is limited. The third step locates the vertebrae of the complex that are the keys to restoration of mobility.

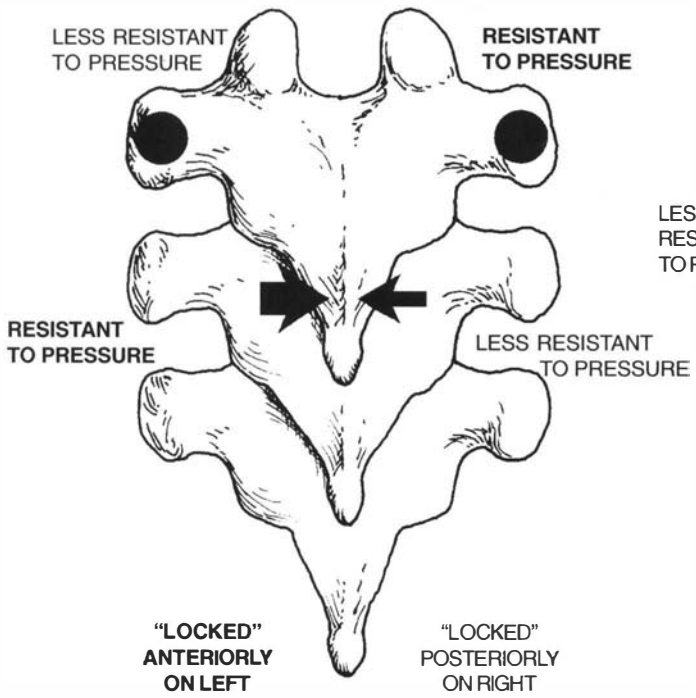
Step 1. To generally locate a fixation complex, test for bilateral weakness of the muscles associated with fixations. The fixation will be in the general area indicated previously for bilateral muscle weakness. The fixation complex is further delineated by motion palpation. Palpate for motion between adjacent vertebrae by pressing on the spinous or transverse processes of the vertebrae to rotate them in opposite directions. Judge the motion available between the vertebrae; then reverse contacts to rotate the vertebrae against each other in the opposite direction. By progressively evaluating the motion between vertebrae, establish the upper and lower limits of the fixation group. When outside the fixation group there will be a soft, yielding motion in both directions. Of course, the upper or lower limit may be defined by reaching the occiput or sacrum.



3—38. Step 1. Pressing alternately on two vertebrae in opposite directions, locate fixation complex by resistance. Evaluate large arrows together. Continue until freedom of motion is found in both directions with a vertabra or structure above or below. This locates top and bottom of the fixation complex.

Step 2. The fixation complex will be able to rotate easily in one direction, but it will resist movement in the opposite direction. The top vertebra of the complex — found in step 1 — is the key in determining the direction in which rotation is locked, and which is freely movable. This is usually accomplished by pressing the spinous process both right and left, observing the direction in which it moves easily and has resistance. The motion of the top vertebra can also be evaluated by pressing on the transverse or mamillary processes and observing for resistance on one side.

The complex is considered locked posteriorly or anteriorly. Reference to posterior and anterior relates only to movement ability, not to the directional misalignment considered in vertebral subluxation analysis. If the right transverse process resists anterior movement, it indicates that the vertebra is locked posteriorly on that side. This is listed as a right posterior fixation. This, then, would indicate that the left side is locked anteriorly, so the complex would be a left anterior fixation and a right posterior one.



3—39. Step 2. Evaluate which direction vertebra can and cannot rotate. List which side is locked posteriorly and which is locked anteriorly.

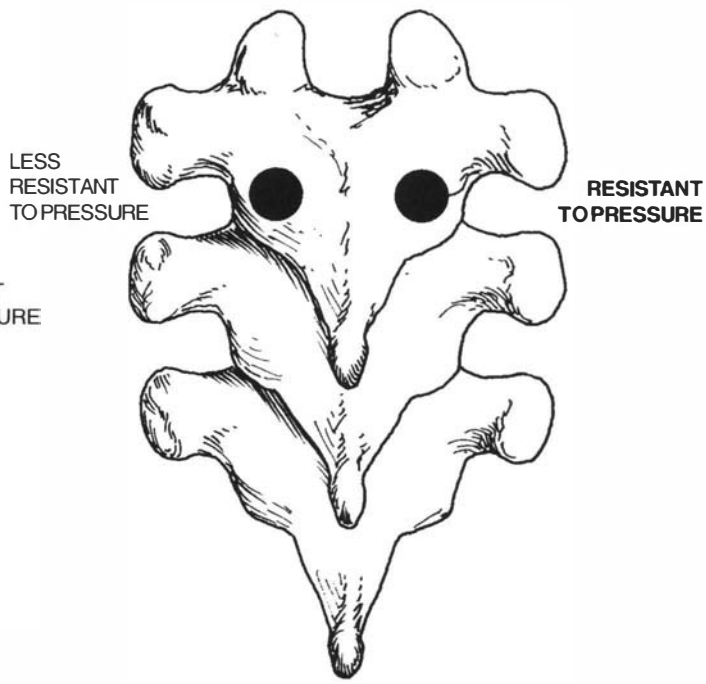
The same type of analysis is made if the motion palpation is applied to the spinous process. If the spinous process is pushed from left to right and resists movement, the right side is locked posteriorly and the left side anteriorly.

Step 3. In step 1 the vertebrae involved in the fixation complex are located, giving top and bottom definition to the complex. In step 2, the direction in which the fixation complex is locked is established and nomenclature assigned. Step 3 determines which side of the fixation is primary. The combined information from steps 1, 2, and 3 is used to determine which two vertebrae will be adjusted.

The primary side of fixation is found by comparing bilaterally the resistance to digital pressure applied by the examiner over the facet articulations of the top two vertebrae in the complex. First, press anteriorly on one articulation and then on the other, making comparison. One side will resist more than the other, indicating the primary side of fixation.

The illustration given in step 2 shows the right side of the top vertebra not rotating anteriorly, indicating it is locked posteriorly, with the left side locked anteriorly. This information combines with the third step to give a final listing of the fixation complex. If resistance is felt at the right facet articulation, the fixation is on the right; thus it would be listed as a right posterior fixation. If the resistance is found on the left, it is a left anterior fixation.

At this point a rule is applied. If the fixation is on



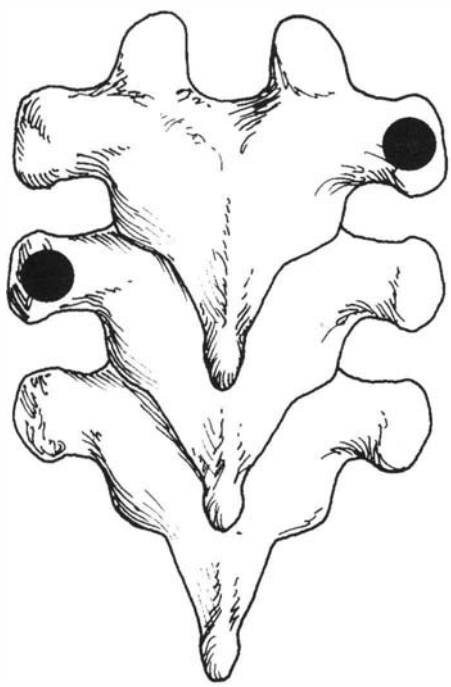
3—40. Pressure over facet articulations of top two vertebrae will have more resistance on one side than the other. Greater resistance is found on the side of primary fixation. Combine data of side of greater resistance with direction of rotation in which the top vertebra is locked, as was determined in Step 2. In these illustrations, right posterior fixation.

the posterior side, the top vertebra of the complex is adjusted on the vertebra immediately below. If the fixation is an anterior one, the bottom vertebra of the complex is adjusted on the vertebra above. Only the top two or bottom two vertebrae are manipulated, but the entire complex will unlock regardless of the number involved.

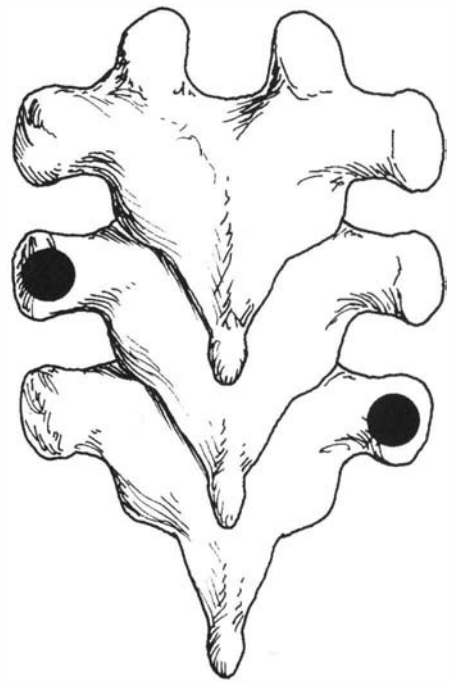
The contact points for unlocking a fixation are the transverse processes in the thoracic spine, mamillary processes in the lumbar spine, and laminae in the cervical spine. The exception to this is the atlas; the contact point, of course, is the transverse process or lateral aspect of the posterior arch. The contact point for the vertebra adjacent to the top or bottom one is on the side opposite the fixation. The top or bottom vertebra is contacted on the side opposite the adjacent vertebra. The pattern, then, is to contact the top vertebra in a posterior fixation on the side of fixation, or the bottom vertebra on the side opposite fixation.

Contact for an anterior fixation described above is opposite that described by this author in previous publications.[115,116] Although the contact described for an anterior fixation in the previous publications has been effective for the author, it is opposite that described by Goodheart.[38] This was pointed out by Conable,[19] who made an analysis and hypothesis of how fixations are unlocked. The change here is to maintain consistency on the subject.

The manipulation to unlock a fixation is a two-step thrust. The first thrust is on the top or bottom vertebra of the complex, as indicated. Almost immediately following there is a quick thrust from the opposite hand on the adjacent vertebra. There is usually an audible release; however, it is not necessary for effective correction. Effectiveness is indicated by strengthening of the bilateral muscle weakness, and no positive therapy localization combined with spinal movement of the area.



Example — Right Posterior Fixation
3—41. Contact top vertebra on side of fixation and vertebra below on opposite side. Give a quick one-two type adjustment, with first movement to top vertebra.

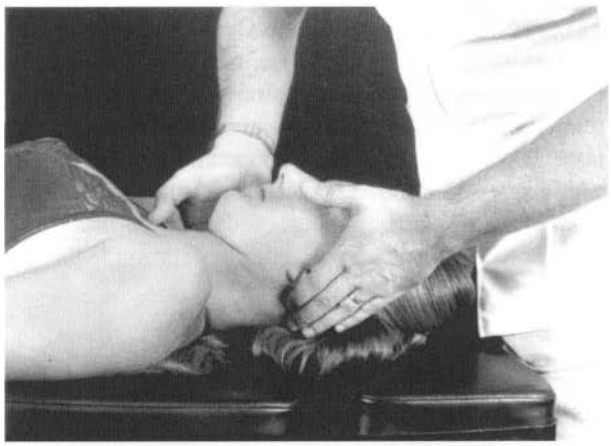


Example — Left Anterior Fixation
3—42. Contact bottom vertebra opposite side of fixation and the vertebra immediately above on the side of fixation. Give a quick one-two type adjustment, first movement to bottom vertebra.

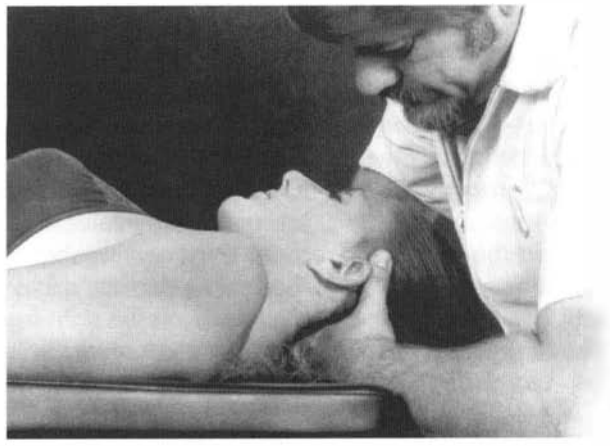
Occipital Fixation

An occipital fixation is comprised of the occipitoatlantal articulations, sometimes referred to in applied kinesiology as occipital jamming. It is characterized by bilateral psoas weakness, which can also be caused by bilateral foot involvement. Evidence of a fixation causing the bilateral weakness is bilateral psoas weakness strengthening when the patient therapy localizes the occipitoatlantal articulation.

To challenge for an occipital fixation, the cervical spine — including the atlas — is stabilized with one hand, while the physician moves the occiput in relation to the atlas. A positive challenge is a previously strong indicator muscle weakening. The challenge should be done in various vectors to find the maximum indicator muscle weakening. This is the optimal vector of correction.



3—43. The atlas must be stabilized, as the occiput is challenged on it for an occipital fixation.

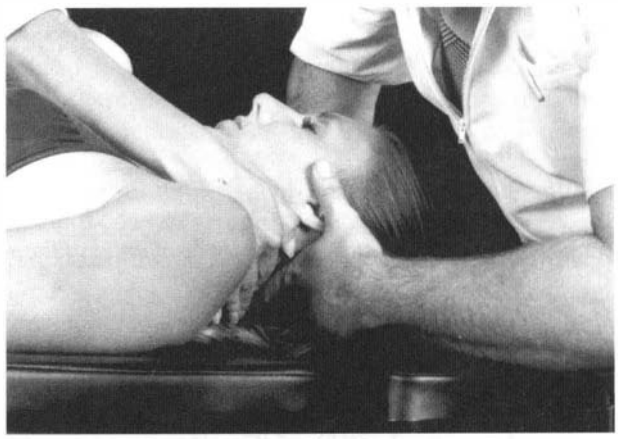


3—44. Avoid rotation of the cervical spine and any contact with temporal bone, as cranial faults may be created.

The contact point for unlocking an occipital fixation is found by palpating along the inferior nuchal line on the side of positive challenge for an extremely sensitive point. This is the optimal point of contact for correction. The line of drive for correction is from this point to the glabella. This vector should agree with the challenge vector. A 1st metacarpal contact is made at this point. Without rotating the neck, remove the slack and thrust from the point of contact directly to the glabella.

In other areas of the spine, correction of a fixation requires a two-handed contact. One hand makes the adjustive thrust while the other one stabilizes the adjacent bone. The atlas cannot be stabilized by the physician, since both hands are needed to contact and stabilize the head. In some instances, it is necessary to have a support person stabilize the cervical spine, including the atlas, to effectively unlock an occipital fixation.

The contact on the skull should be on the occiput only. Contacting the mastoid process or some other area of the skull may create iatrogenic cranial faults.



3—45. It may be necessary for a support person to stabilize the cervical spine.

Sacral Fixation

A sacral fixation is a bilateral sacroiliac fixation. Correction is directed to only one side, which is determined by use of motion evaluation similar to that of the rest of the spine.

Indication of a probable sacral fixation is weak deep neck extensor muscles when both the right and left groups are tested separately and found weak. When a group tests weak on only one side, there is a probable unilateral sacroiliac fixation on that side.

Confirmation of weakness associated with a sacral fixation is obtained by therapy localization to the articulation that eliminates the weak muscle tests. Therapy localization to a sacral fixation, like others, will not cause a general indicator muscle to weaken unless the TL is held while attempting to introduce motion into the articulations.

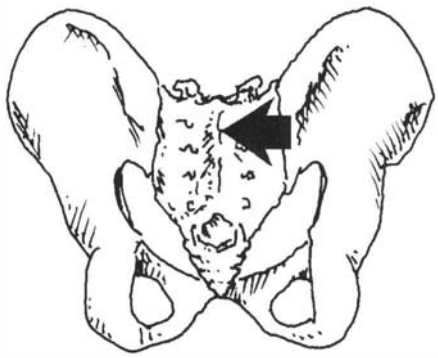
A sacral fixation will challenge if the innominate is stabilized while the challenging pressure is applied to the sacrum. When the patient is prone, challenge to the sacrum only is usually effective because the patient's weight on the anterior superior iliac spines and pubis adequately stabilizes the innominate bones, in effect causing a two-handed challenge.

Hypertonicity of the piriformis or psoas can cause sacroiliac or sacral fixations because the muscles cross over the articulations. If the muscle stretch reaction is present, the involved muscle should be treated with fascial release or intermittent cold with stretch technique. Schmitt[106] points out that the fascial release technique applied to the hypertonic muscles often eliminates the fixation without manipulation.

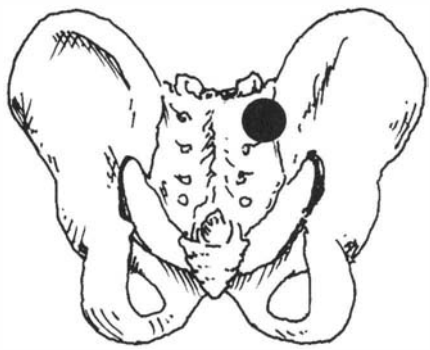
The sacral fixation is analyzed in a manner similar to vertebral fixations. Since the fixation is already defined as consisting of the two sacroiliac articulations, the usual first step — localizing the fixation — is unnecessary.

First contact the tubercles of the sacrum and attempt to move the sacrum both right and left. It will move more easily in one direction, with greater resistance in the opposite direction. If the sacral tubercle moves more easily toward the left, the sacrum is fixed anteriorly on the left and posteriorly on the right because the sacrum can move into fixation more easily than out of it.

Only one sacroiliac articulation need be adjusted,



3—46. Step 1. Press both right and left on sacral tubercle; determine movement and resistance. Tubercle moves more easily to side of anterior fixation, and there is resistance to side of posterior fixation.

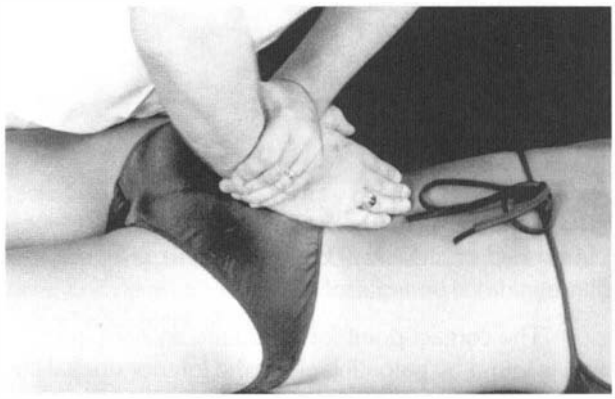


3—47. Step 2. Press at the location of the dot, first on one side and then on the other; determine which has the greater resistance. This is the side of major fixation. Combine this information with that from Step 1 to delineate posterior or anterior fixation.

even though both are fixed. One side is primary and is the side on which the adjustment is made. Find the side by pressing anteriorly, just medial to the posterior superior iliac spine. Compare the resistance of the two sides. The side with greater resistance will be the one to correct. If the sacral tubercle moves more easily to the left and has greater resistance to digital pressure on the right, it is a right posterior sacral fixation.

Posterior Fixation Correction

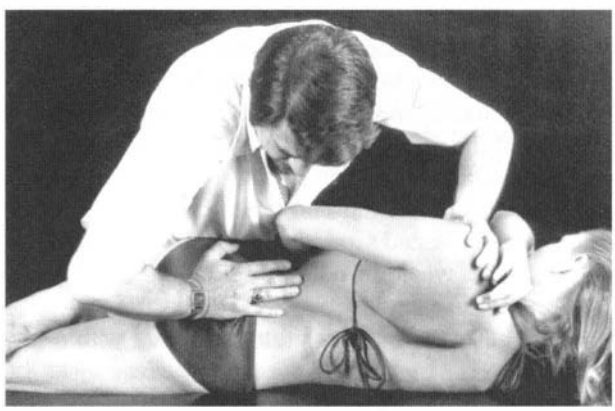
With the patient prone, contact the sacrum medial to the posterior superior iliac spine and adjust in an anterolateral vector along the plane of the sacroiliac articulation. The ilium is stabilized by contact with the table, allowing a one-handed contact for fixation correction.



3—48. Contact for right posterior sacral fixation adjustment

Anterior Fixation Correction

If the resistance in step 2 is found on the anterior fixation side, the correction is made in a side-lying position. The anterior fixation side is farthest from the table. The patient's leg close to the table is straight, while the other is flexed at the hip and knee, with the toes tucked behind the knee of the straight leg. The doctor stabilizes the posterior fixation side of the sacrum (closest to the table) and the flexed leg and pelvis, while the shoulder away from the table is rotated posteriorly. This places torsion through the spinal column into the sacrum, moving the anterior fixation side of the sacrum posteriorly. The key to obtaining this correction easily is good stabilization of the opposite side of the sacrum, and of the pelvis and leg on the side of anterior sacral fixation.



3—49. Doctor's right hand is stabilizing posterior side of fixation next to table, while the sacrum is brought posteriorly on anterior fixation side by torque throughout the spinal column.

Sacroiliac Fixation

A sacroiliac fixation is indicated when the unilateral neck extensors test weak with the other side strong. This muscle weakness, of course, can be caused by any of the five IVF factors. Evidence that a sacroiliac fixation is causing the neck weakness is when it strengthens with therapy localization to the sacroiliac. One must also differentiate the sacroiliac fixation from a category I or II.

The sacroiliac fixation is usually readily unlocked by an adjustive thrust to separate the innominate from the sacrum. Often, correcting hypertonicity of the piriformis or psoas is all that is necessary to eliminate a sacroiliac fixation.

There may be a subclinical fixation; i.e., the unilateral cervical extensors are strong in the clear and weaken when the sacroiliac is therapy localized. If this is a true subclinical fixation, only the cervical extensors will weaken with the therapy localization. In this case, there is often a respiratory pattern to the fixation. While the patient is therapy localizing to the sacroiliac, have him hold a deep phase of respiration to determine if it abolishes the positive therapy localization. If so, correct with a medial vector of force on the posterior superior iliac spine in the case of inspiration assist and a lateral vector for expiration assist. (For further information on respiratory sacroiliac correction, see page 112.)

Fixation-Masking Patterns

The bilateral muscle weakness associated with fixations is a consistent pattern. In some instances, the pattern will not reveal itself until another correction is made. Schmitt[105] observed patterns wherein, after one fixation was corrected, another appeared. This occurs because there are two fixation complexes present, but one bilateral muscle imbalance covers the other in an agonist-antagonist relationship. If one fixation has exceptionally weak muscles, the antagonist muscles contract because of lack of opposition; thus they test strong until the first fixation has been corrected. Its correction eliminates the interplay between the two sets of muscles, and the second set tests weak.

The dorsolumbar fixation is associated with bilateral lower trapezius weakness, and the cervicodorsal fixation is associated with bilateral deltoid weakness. When the arm is abducted to 90° for deltoid testing, the scapula rotates to begin facing the glenoid cavity superiorly. This somewhat aligns the middle and posterior fibers of the deltoid to be antagonistic to the lower trapezius fibers. Clinically it has been observed that when the dorsolumbar fixation is corrected, the cervicodorsal fixation may be revealed by bilateral deltoid weakness that was not previously present.

Cervical and occipital fixations may be present simultaneously. Bilateral gluteus maximus weakness is indicative of an upper cervical fixation, while bilateral psoas muscle weakness indicates an occipital fixation. The pelvis is extended on the thigh by the gluteus maximus and flexed on the thigh by the psoas. The gluteus maximus muscles may test weak bilaterally while there is normal function of the psoas muscles. After correction of the upper cervical fixation and strengthening of the gluteus maximus muscles, there may be bilateral psoas weakness indicating an occipital fixation. Fixations may mask subluxations. There may be no therapy localization or challenge indicating a spinal subluxation until a fixation in the area has been corrected. After correction of the fixation, evaluate for subluxations as indicated previously and correct, if present.

In applied kinesiology, bilateral pectoralis major (clavicular division) weakness appears to indicate dysfunction of the cranial primary respiratory mechanism. In the event that clinical evidence indicates a digestive dysfunction relating with hydrochloric acid production and the bilateral pectoralis major (clavicular division) muscles are strong, evaluate for a dorsolumbar fixation. If present, correct it and re-evaluate the bilateral pectoralis major (clavicular division) muscles. They will often be weak when tested bilaterally after the dorsolumbar fixation has been corrected.

Weight-Bearing Fixations

Fixations may not be revealed unless an individual is weight-bearing. To evaluate for a weight-bearing fixation, simply test the bilateral muscles associated with the fixation with the patient sitting or standing. When there are recurring fixations with the patient in a weight-bearing position, he may need octacosanol, which has clinically been found to help eliminate recurring fixations of the weight-bearing type. Often insalivation of octacosanol will cause the bilateral muscles that tested weak on weight bearing to return to normal without further adjusting. This will not happen unless the fixation was corrected prior to the weight-bearing test.

Weight-bearing fixations can also be evaluated by pressing caudally on the vertex of an individual's head while he is prone or supine. This simulates weight bearing by directing axial pressure into the spinal column. Pressure can be applied to the lower area of the spinal column by having the patient push with his feet on the footrest of a table while supine or prone.

Flexion and Extension — Atlas and Occiput

Disturbance of flexion or extension between the atlas and occiput has been known as "rocker motion involvement." Generally, disturbances of this nature are corrected with the usual evaluation and correction procedures for the upper cervical region and the occiput. Occasionally normal motion cannot be obtained in this manner, probably because of the inaccessibility of the anterior muscles of the region. Flexion and extension of the region can be further tested in various positions of flexion and extension of the cervical spine. All the tests are done with the patient supine and the physician testing an indicator muscle.

Occiput on atlas flexion

The patient flexes the cervical spine, beginning with the occiput on atlas and continuing until maximum flexion is reached. The posterior aspect of the cranium maintains contact with the examination table. The position is one in which the patient attempts to touch his chin to his chest, but he is prevented from doing so by keeping his head on the examination table. A positive test is observed when a previously strong indicator muscle weakens.

Correction is obtained by the doctor stabilizing the patient's head while the patient attempts to return to the test position. The doctor allows no movement, so the muscles make a maximum isometric contraction. This is repeated three or four times. There should be no weakening of the indicator muscle on re-testing.

Occiput on atlas extension

In a manner similar to the flexion test the patient extends his neck, beginning with the occiput on atlas, until maximum extension is obtained. The head remains on the examination table. While the patient is in this position, a previously strong indicator muscle is tested for weakening.

Correction is made in a manner similar to that for occiput on atlas flexion. The examiner holds the patient's head while he makes a maximum effort to return to the test position. The motion, limited by the doctor, allows maximum isometric contraction of the patient's muscles. Repeat three or four times and re-test to determine if correction was obtained.

Atlas on occiput flexion

The patient maximally flexes the neck, taking his head from the table to touch his chin to his chest. While this position is held, a previously strong indicator muscle is tested for weakening.

To correct, the doctor flexes the patient's neck to the maximum, with the patient passive throughout the procedure. Repeat three or four times and re-test to determine if correction was obtained.

Atlas on occiput extension

The patient extends his neck the maximum amount while keeping his head off the examination table. In this position, a previously strong indicator muscle is tested for weakening.

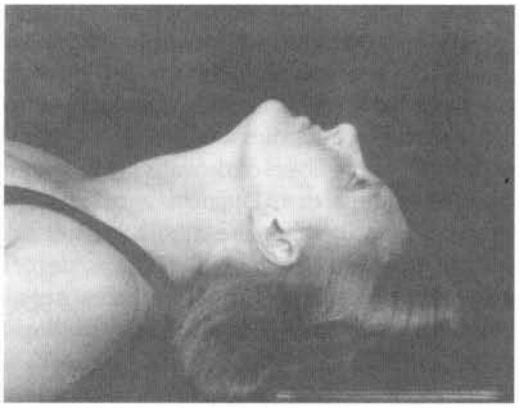
As in the atlas on occiput flexion, the correction is actively done by the doctor. The patient's neck is extended to the maximum three or four times, with the patient passive throughout the procedure. Re-test to determine if correction was obtained.

It is unusual for more than one of the four possible types of rocker motion involvement to be present. The correction is a lasting one that should not need repeating. If the involvement does return, additional factors — such as gait mechanism, foot, and pelvis — should be evaluated to determine the cause of the secondary stress in this region.

3—50. Test — head on table

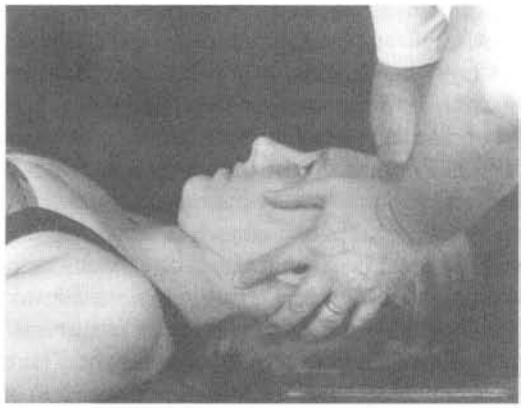
Occiput on atlas flexion

3—51. Correction

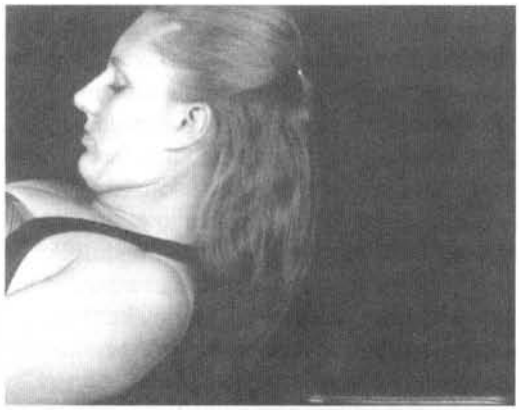


3—52. Test — head on table

Occiput on atlas extension

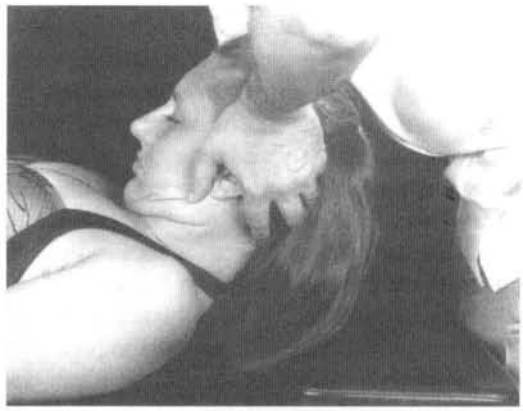


3—53. Correction



3—54. Test — head off table

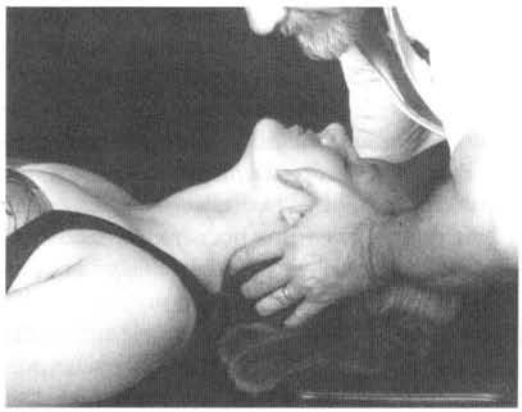
Atlas on occiput flexion



3—55. Correction

3—56. Test — head off table

Atlas on occiput extension



3—57. Correction

Chapter 3

Lumbar Intervertebral Disc

An intervertebral disc is the cause of back pain in only a very small percentage of cases.[118] When it is the cause, conservative treatment can be very valuable to the patient in reduced time of disability and future well-being. On the other hand, inappropriately applied conservative treatment can increase the disability.

The literature abounds with books and papers on the intervertebral disc syndrome. A comprehensive discussion of proper diagnosis and treatment of the condition is beyond the scope of this text. The discussion here will be limited primarily to unique applied kinesiology techniques to add to standard examination procedures and treatment. A limited presentation of standard examination needed to correlate with applied kinesiology examination and treatment will be given.

Often a chiropractor sees an individual in the early stages of disc degeneration. In addition to proper treatment, it is important that the physician educate the patient on proper spinal maintenance, including lifting and general spinal health care. It is inevitable that the intervertebral disc will mature and degenerate with age, but the degeneration should progress normally and not be accelerated because of improper treatment by a physician or structural abuse by the patient. The nutritional status of the patient is also important in maintaining optimal disc health.

Macnab[86] states, in a classic description of degeneration, "An intervertebral disc presenting features commonly found in the sixth decade could be described as a 'degenerate disc' when found in a man age thirty." He describes disc progression throughout the decades of life. During the first two decades the annulus fibers become more clearly demarcated, and the nucleus pulposus maintains its gelatinous consistency. In the third decade the nucleus becomes firmer, but it is still pliable and soft. During the fourth decade the nucleus loses its gelatinous character, and the annulus fibers begin hyaline degeneration. During the fifth decade the nucleus is a homogeneous, firm mass, with its size decreased by encroachment of the annulus fibers. In the sixth decade the nucleus is dry and friable, with further reduction in its size. The nucleus pulposus of the seventh decade gives the appearance of condensed cream cheese. It is brownish, dry, and friable, and merges indistinctly into the annulus. Similar changes continue throughout the additional decades of life.

The nucleus pulposus is made up of a gel that gives it the properties of hydration, solvation, or imbibition. As the gel becomes hydrated, it swells to provide the strength, resilience, and flexibility necessary for the nucleus pulposus. In comparing normal discs from cadavers with abnormal discs obtained from a laminectomy, Hendry[58] found a reduction of imbibition pressure as a constant feature of the abnormal disc. He relates this breakdown to failure of the hydrated nucleus pulposus to support its weight, placing greater strain on the annulus fibrosis.

During the period from twenty to fifty years of age, a disc is more likely to prolapse because of the high fluid content. It is under constant pressure, as evidenced by its bulging outward when sectioned transversely.[112]

A classification of intervertebral disc disturbances is of value in developing a treatment plan and making a prognosis. In 1955 Charnley[15] wrote a paper outlining the causes and treatment for low back pain and sciatica. His thorough work has been reviewed and updated by White and Panjabi.[118] They list seven types of disc involvement.

Type 1 — acute back sprain. Trauma caused by a sudden increased load, such as a slip, twist, fall, or lift, may cause tearing of the annulus fibers, posterior ligaments, or musculotendinous structures. These are richly endowed with nerve fibers that cause pain. In addition, as Charnley suggests, deep layers of the annulus may be torn. One can easily recognize that this might cause a weakening of the disc, allowing repeated episodes of strain to further damage it. Type 1 disc involvement responds to rest and may recover more rapidly with various types of electrotherapy. Forceful manipulation immediately after the trauma is contraindicated, since it increases soft tissue damage and promotes further swelling. Use non-forceful techniques, described later.

Type 2 — fluid ingestion. Charnley[15] and Naylor[97] hypothesize that there may be an increased passage of fluid into the nucleus pulposus for some unknown reason. This may stretch the peripheral annular fibers, causing pain, and may be the precipitating factor in a chain of events that can lead to further disc trauma. White and Panjabi[118] point out that there is little to discredit this hypothesis. One factor that raises some question is that astronauts returning from outer space have a heightened disc space but no back pain. This may be due to a slow increase in the fluid pressure with no combatting force of gravity, giving adequate time for adaptation of the tissues and nerves. White and Panjabi go on to state, "We suggest that [fluid ingestion] may be the explanation for spontaneous idiopathic organic spine pain (cervical, thoracic, or lumbar) unrelated to trauma, which accounts for a significant number of the many cases of spine pain."

In applied kinesiology, low back pain, neck pain, and many other types of pain located throughout the body have been associated with an active ileocecal valve syndrome. It is hypothesized that toxicity resulting from the valve dysfunction causes the body to retain fluids to dilute the toxicity. The fluid retention may cause an increased fluid uptake in the intervertebral disc, accounting for the "unknown reason" to which Charnley alludes. Clinically, various conditions of peripheral nerve entrapment — such as a carpal tunnel syndrome — are observed to become symptomatic with

an active ileocecal valve syndrome, recovering when the valve condition is corrected. Increased hydration of the intervertebral disc increases the internal pressure, making the annulus more vulnerable to rapid change of load such as is present in forceful vertebral manipulation. Adjustment of the spine under this condition should be non-forceful, as described later.

Type 3 — posterolateral annulus disruption. Because the external annulus fibers are prodigiously endowed with nerve fibers, disruption or failure can cause back pain or referral into the sacroiliac region, the buttock, or the back of the thigh. The characteristic differentiating this type of "sciatic" pain is that there will be no neuromuscular deficit or positive nerve stretching tests, such as the straight leg raise.

Type 4 — bulging disc. This is often called a disc protrusion. The nucleus pulposus remains intact but bulges into the annular fibers, causing some breakdown and stretching to bulge the annulus fibrosis. This may cause pain from irritation to the annular nerve fibers or bulge upon the nerve root, causing true sciatica. The nucleus is contained by the annulus and possibly the posterior longitudinal ligament. A true picture of disc herniation will probably be present, with Dejerine triad, positive nerve stretch tests, and motor and sensory involvement. A bulging disc responds well to proper manipulation and rest, but it may recur with activity.

Type 5 — sequestered fragment. A portion of the nucleus pulposus separates from the main mass and is sometimes called wandering disc material. True prolapse has not developed because the sequestered fragment is still contained by the annular fibers and posterior longitudinal ligament. The clinical picture will be similar to that of type 4. A therapeutic trial of proper manipulation is applicable. The sequestered fragment may move about and locate where there is minimal stretching of annular fibers and no nerve root irritation. Healing by scarring may contain the fragment, providing long-lasting relief. This condition may have frequent exacerbations and remissions, and may ultimately require surgery.

Type 6 — displaced sequestered fragment. A portion of the nucleus has separated from the main mass and developed into a true prolapse with the fragment external to the annulus fibrosis. The clinical picture is the same as for types 4 and 5, and usually includes the Dejerine triad, positive nerve stretch tests, and motor and sensory loss. The prolapsed disc is least likely to respond to manipulation and rest, but again a therapeutic trial is justified because of the difficulty in differentially diagnosing types 4, 5, and 6. A true surgical emergency exists when there are cauda equina symptoms. The problem may be indicated by visceral disturbances, such as difficulty in urination or incontinence and bowel difficulties. Turek[112] indicates that there is rarely a disturbed bladder function. The most common finding is a temporary depression of both patellar or ankle reflexes brought on by exercise and restored after rest. Cauda equina symptoms indicate that there is a large central protrusion, since there is ample space available for the cauda. Because of the central location of the displacement, there is a lack of positive nerve traction tests and limited painful lateral bending tests.

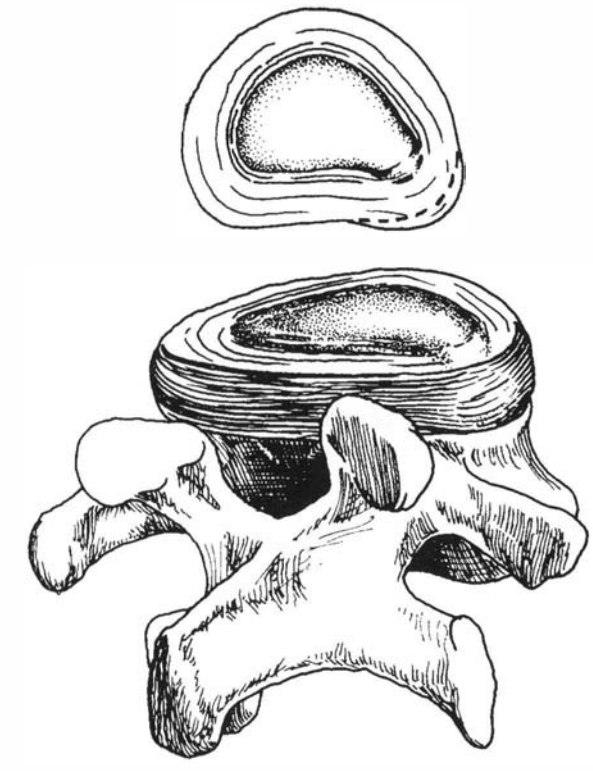
Type 7 — degenerating disc. The degenerated disc occurs in later life, when the annulus fibrosis and the nucleus no longer serve their mechanical functions. The degenerated disc is often associated with degenerative joint disease of the vertebral bodies and facets. The symptomatic picture varies. There may be chronic pain, exacerbations of pain, or an individual may be asymptomatic. In the presence of degenerated discs it is unlikely that a herniation or prolapse will occur; for this reason, herniation and prolapse are infrequent from the fifth decade of life on.

To maintain optimal tissue integrity, a disc depends on its surrounding tissue for nutrition and elimination of metabolic products because of its lack of blood supply. At birth several small blood vessels penetrate the disc from the vertebral and lateral borders, but they all disappear after twelve years of age, leaving the disc with no direct blood supply. The disc obtains its nutrients by diffusion from the capillary beds at the disc margins. The ligaments that surround the periphery of the disc are well supplied with vessels and capillaries in the child and in the adult. In addition, the disc receives nutrition from the capillary beds in the vertebral marrow spaces lying next to the cartilaginous end plates. As one ages these capillaries grow sparser, which supports the idea that a restricted flow of nutrients to the nucleus and inner annulus may contribute to, or even underlie, disc degeneration.[31]

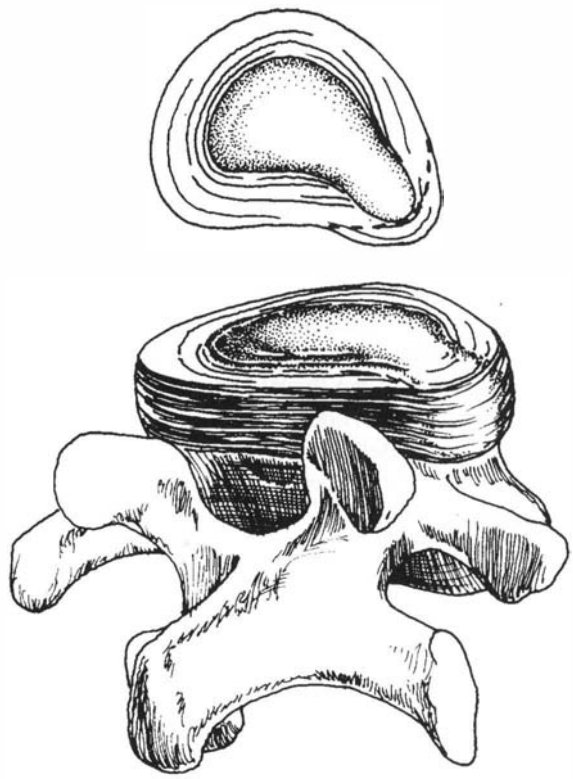
The nucleus pulposus is made up of a mucal protein gel that contains various glycosaminoglycans, supported by a dense collagen fiber network. Because of the avascular nature of a disc, it depends on imbibition for its nutrition. Glucose and oxygen enter the disc mainly via the end plates, while sulfate ions — important for the production of new glycosaminoglycan — generally enter via the annulus fibrosis. The posterior boundary of the annulus receives fewer negative recharged ions since its area for available fusion is smaller. The glycosaminoglycan turnover is very slow, around 500 days. As Nachemson says, "No wonder ruptured discs take a long time, if ever, to heal."[94]

Bourdillon[12] hypothesizes that there is an "imbibition pump" responsible for removing metabolites and bringing nutrients into the disc. The movement mechanism of the vertebral body changes pressure on the disc to exchange fluid, much like holding a sponge in water and repeatedly squeezing and letting go of it. He postulates that lack of movement, such as that present in a vertebral fixation complex, deactivates the imbibition pump, ultimately causing nutritional deficiency and degeneration of the disc.

It is clinically observed in an applied kinesiology practice that individuals with disc degeneration nearly

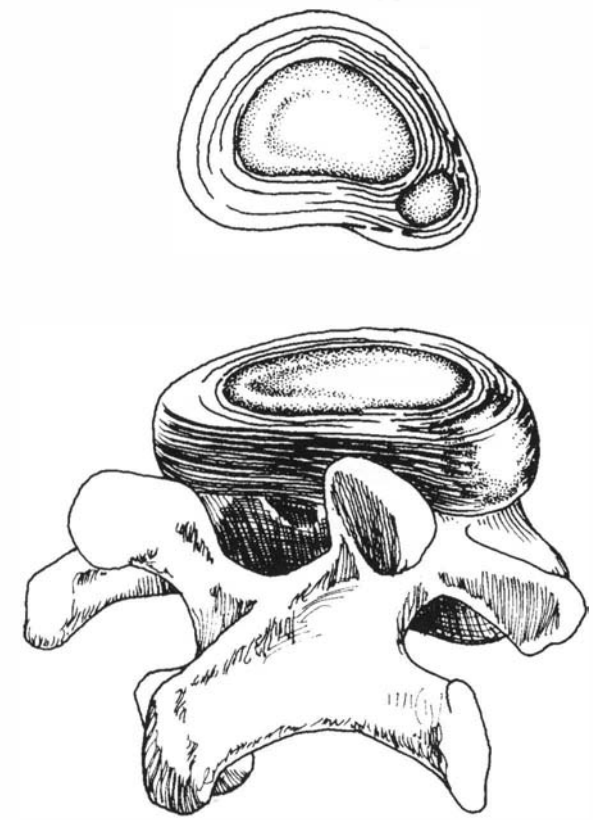


3—58. Type 3 — posterolateral annulus disruption.

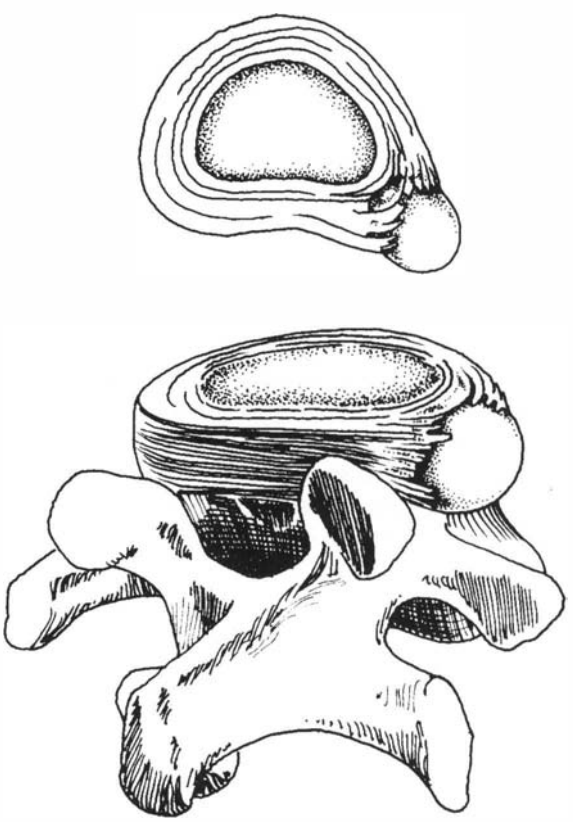


3—59. Type 4 — bulging disc.

Figures 3—58 through 3—62 after White and Panjabi, Clinical Biomechanics of the Spine *(Philadelphia: J.B Lippincott Co., 1978). Redrawn with permission.*

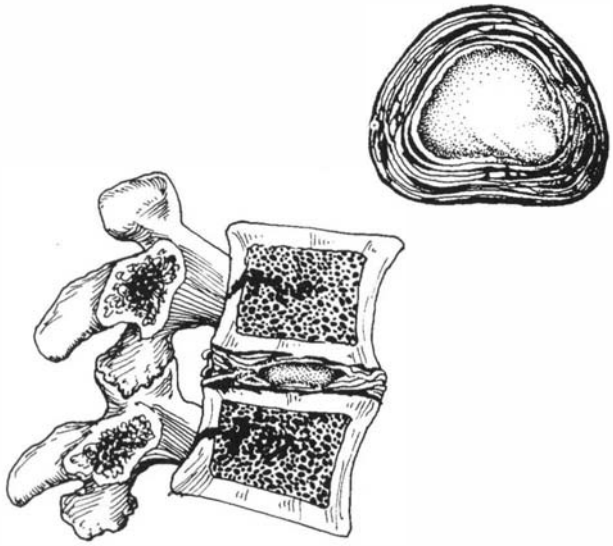


3—60. Type 5 — sequestered fragment.



3—61. Type 6 — displaced sequestered fragment.

always have a vertebral fixation complex at that level. It seems reasonable that lack of motion resulting from the fixation may be the cause of accelerated disc degeneration. A recent *in vitro* study by Katz et al[81] questions this hypothesis. They compared the transport of small solutes into the disc between a group of laboratory specimens that were flexed for one hour with those that remained static. There was no increase in the movement of small solutes into the disc of the flexed group over the one that was static. To help answer this question, an *in vivo* study needs to be done. In any event, the vertebral fixation complex should be corrected. It may be an important factor in retarding disc degeneration.



3—62. Degenerated intervertebral disc. The disc depends on movement for nutrition and removal of waste products. The vertebral fixation complex may contribute to more rapid degeneration. Applied kinesiology testing often indicates a fixation complex at the level of disc degeneration.

Muscular Relation to Disc and Lower Spine Involvement

A basic tenet in applied kinesiology is to balance muscle function around joint dysfunction. In an acute intervertebral disc condition, it may be impossible to test the muscles that support and control the low back and pelvis. Treatment is first applied to the acute condition. As soon as possible, muscle function should be evaluated and corrected if improper.

The psoas muscles are frequently involved with an intervertebral disc condition. Psoas muscle origin is generally considered to be from the lumbar spine; however, in movement of the lumbar spine, the origin is at the lesser trochanter of the femur and the insertion is to the anterior surface of the transverse processes, lateral borders of the vertebral bodies, and corresponding intervertebral discs from T12 through L5. In some instances there is failure of the psoas to attach to L5, which may be responsible for unusual movements between L4 and L5 as observed on lateral flexion x-rays. Illi[68] points out the importance of lumbar spine rotation during flexion.

By measuring intradiscal pressure, Nachemson[95] found it increased with a person sitting and standing. In a further study,[93] he recorded psoas activity by electromyography while a person was in the sitting and standing positions. He confirmed the studies of Basmajian[7] and others that the psoas is an active postural muscle. The intradiscal pressures increased with sitting and standing above the level that could be explained by gravitational forces alone. Nachemson postulated that the downward pull of the psoas major during weight bearing contributes to the increased intradiscal pressure.

It is important to evaluate the length of the iliopsoas muscles.[116] First evaluate hip extension with the patient's knee flexed. If hip extension is limited, allow the knee to extend. If the hip extension increases to normal, the shortness is in the rectus femoris rather than in the iliopsoas.

One or both of the psoas muscles often have a muscle stretch reaction, indicating the need for fascial release (page 192); this usually lengthens the muscle and gives a greater range of motion in extension and external thigh rotation. The psoas muscle(s) also frequently needs strain/counterstrain technique (page 201), indicated by it testing weak immediately after being maximally contracted for three seconds when it previously tested strong.

Eie and Wehn[29] studied intra-abdominal pressure during weight lifting. The diaphragm contracts and abdominal muscles tighten to increase intra-abdominal pressure, providing additional support for the anterior lumbar spine. This flattens the spine and, because of the change of the movement of forces, reduces the compressive force on a disc.

Bartelink[6] measured the increased abdominal pressure with lifting by implanting a gastric balloon. He found that stronger, more athletically fit individuals had a higher increase in intra-abdominal pressure with lifting than those of lighter build and in poor condition. Electromyography revealed that the increase in abdominal pressure comes primarily from the transverse, and somewhat the oblique, abdominal muscles. The rectus abdominis does not contribute to the increase in abdominal pressure.

It has been clinically observed in an applied kinesiology practice that individuals who take a deep breath prior to lifting have cranial faults. It is suspected that an individual obtains an improved function by taking the phase of respiration that improves the cranial position. Obtaining optimal function without having to take a phase of respiration helps limit additional pressure on the disc. This is supported by a study Nachemson et al.[96] did on the effects of the Valsalva maneuver on lumbar trunk loads. They found by measuring intradiscal pressure that the Valsalva maneuver increased loads on the spine, apparently because of the effects of the muscle activity antagonistic to the maneu-

ver, which were measured by electromyography. Correction of cranial-pelvic-respiratory dysfunction usually allows an individual to cease holding a phase of respiration prior to lifting and, in essence, performing the Valsalva maneuver that increases intradiscal pressure. Sometimes, although the cranial faults have been corrected, an individual will continue taking a phase of respiration because of a developed habit pattern. Training in proper lifting should include elimination of performing a Valsalva-type maneuver when lifting.

The abdominal muscles should be evaluated. When they dysfunction there is poor anterior support to the spinal column. Often the correction of cranial faults that caused an individual to take a phase of respiration will improve abdominal muscle function. The jammed sagittal suture is the most important cranial fault in influencing function of the abdominal muscles. The diaphragm should also be evaluated because its integrity is necessary to maintain intra-abdominal pressure.

Examination

The examination for an intervertebral disc syndrome includes a complete orthopedic and neurologic study. This will be only briefly outlined here, with emphasis on applied kinesiology additions to the examination. The reader is encouraged to review texts that thoroughly cover the subject, such as Cox,[20] Hoppenfeld,[65,66] and Turek.[111,112]

Analysis of the antalgic position, if the patient has one, and bending tests reveal much about the intervertebral disc syndrome. The antalgic position is one the patient assumes voluntarily or involuntarily to alleviate nerve root irritation. The antalgic position may be toward or away from the side of sciatic radiation. A common correlation is that if the antalgic position is away from the side of sciatic radiation, the herniation is lateral to the nerve root; if sciatic involvement is on the side of antalgic lean, the herniation is medial to the nerve root.

If there is little or no lateral antalgic position, one can have the patient flex the spine laterally and use the same criteria of evaluation as those for an antalgic position. If the pain increases on flexing toward the side of sciatic pain, the disc involvement is lateral to the nerve root; if the pain increases by flexing away from the side of sciatic pain, the herniation is medial to the nerve root. If there is no lateral antalgic lean, there may be a central disc herniation.

Lateral flexion x-rays can help localize the level of disc herniation.[99] Normally there should be narrowing of the intervertebral disc space between the vertebrae on the side of lateral bending. There will be limited motion at the level of disc involvement when lateral bending encroaches upon the nerve root, with resumption of wedging at the disc level above.

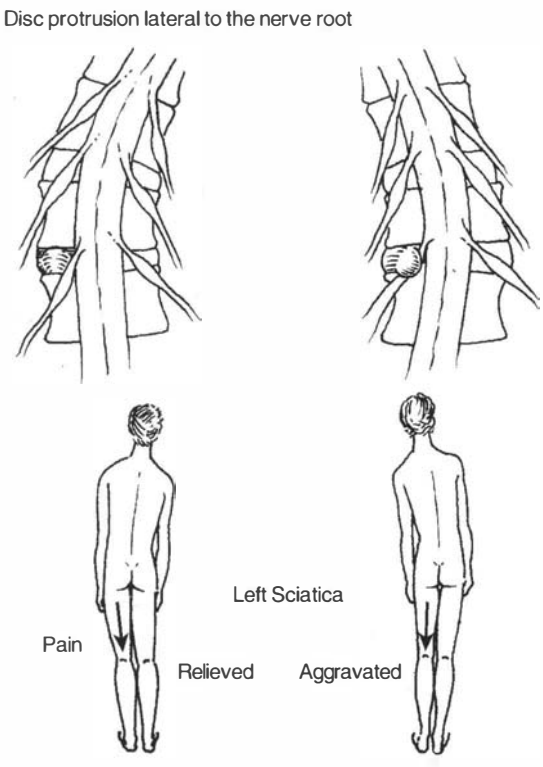
The patient's dermatomes are evaluated for sensory deficit. Loss should be confined to one dermatome area. It is unusual for more than one nerve root to be involved. Light touch — tested by a cotton swab — is lost first, followed by pain sensation loss. During recovery, sensation of pain returns before the light touch.

Testing the myotome level of the nerves should be of particular interest to the applied kinesiologist because of the routine use of manual muscle tests. Muscles generally listed for the neurologic levels are as follows: L4 nerve root, L3-4 disc, tibialis anterior; L5 nerve root, L4-5 disc, extensor hallucis longus, extensor digitorum longus and brevis, and gluteus medius; S1 nerve root, L5-S1 disc, flexor hallucis longus, peroneus longus and brevis, gastrocnemius, soleus, and gluteus maximus.[20,66,89] There is some overlap of the myotomes. Muscle tests must be correlated with other tests to reach a final diagnosis.

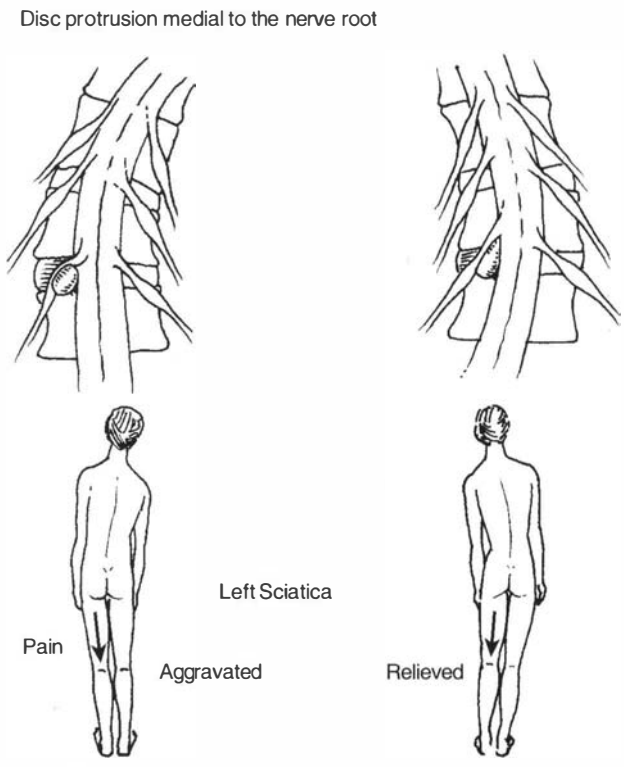
The lower two disc levels are the most common areas of herniation. Shapiro[108] reviewed the literature on 3,000 cases of single lumbar disc herniation. He found that 43% involved the L5-S1 disc, and 47% involved the L4-5 disc. The remainder were protrusions at higher lumbar levels. When examination reveals confusion of disc level involvement, one must remember that there may be more than one herniation. In two of the studies Shapiro reviewed, there were multiple prolapses in 10-15% of the cases.

Muscle testing as used in standard diagnosis can be enhanced by adding applied kinesiology therapy localization and challenge to determine the cause of the muscle weakness. Bandy[4] proposes that weakness of specific muscles not associated with the five factors of the IVF indicates disc involvement. Muscle weakness associated with peripheral nerve entrapment of the intervertebral disc is different from the "weakness" of applied kinesiology, where a muscle fails to lock on initial testing. The type of muscle weakness is further differentiated by failure of the nerve entrapment weakness to strengthen when therapy localized to neurovascular, neurolymphatic, and other of the five factors of the IVF. The muscles Bandy associates with disc involvement for applied kinesiology testing are S1 nerve root, L5-S1 disc, gastrocnemius; L5 nerve root, L4-5 disc, tibialis anterior; and L4 nerve root, L3-4 disc, rectus femoris (straight head). The gastrocnemius[9] and rectus femoris (straight head)[8] are tested with Beardall's methods. The test for the gastrocnemius is modified from Beardall's to test both the medial and lateral heads together. The leg is held in neutral, with no rotation as is done in the medial and lateral gastrocnemius tests. (These tests are described on page 329.) The test for the rectus femoris (straight head) is with the patient supine. He flexes the hip to lift the leg 30° from the table, with the knee kept in extension. The examiner stabilizes the contralateral ankle to the table and directs testing pressure against the ankle toward the table.

Positive indication of disc involvement in Bandy's procedure is seen when the muscles strengthen when the lumbar spine is flexed laterally, but they do not strengthen with other of the five factors of the IVF. Almost always the lateral flexion will be in the direction that relieves the nerve root from disc encroachment, that is, away from



3—63a.



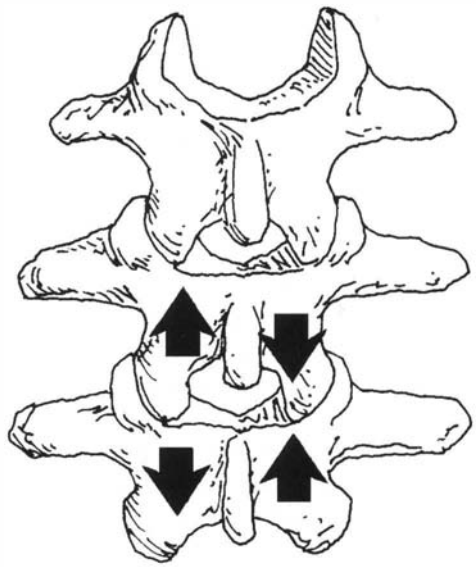
3—63b.

Increased sciatic pain with lateral flexion of the spine indicates medial or lateral disc protrusion. From B.E. Finneson, Low Back Pain, *2nd ed (Philadelphia: J.B. Lippincott Co, 1980) with permission.*

the side of sciatic involvement for a lateral protrusion and toward the side of involvement for a medial protrusion.

Generally an intervertebral disc involvement does not show positive therapy localization to the lumbar vertebrae in the usual manner, which is touching the area with one hand and having a previously strong indicator muscle weaken. When the vertebra above the disc lesion is therapy localized with one hand and the vertebra below with the other hand, therapy localization will be positive.[39] This method of localizing disc involvement is added to the routine diagnosis of disc lesions and should help confirm the level of involvement. If there is any discrepancy, further evaluation should be done to determine a final diagnosis.

An additional applied kinesiology diagnostic approach to disc involvement is the two-handed challenge of the vertebrae above and below the disc lesion.[39] The spinous or transverse processes are contacted, and a separating force is applied. The muscles usually tested with this challenge are the hamstrings. The piriformis is also a convenient indicator muscle, but it may cause problems because the patient may rotate the pelvis to change the test parameters. When there is wedging of the vertebrae, as indicated by the patient's antalgic position on neutral and lateral flexion x-rays, there will usually be a more pronounced positive challenge by contacting the transverse processes on the side of the open wedge to separate the intervertebral disc space. When vertebral subluxation challenge is positive, correction is made in the direction of challenge that caused a previously strong indicator muscle to weaken. The same basic principle is present in intervertebral disc challenge. The greatest weakening of the indicator muscle may be with separation of



3—64. The vertebrae above and below the possible IVD involvement are challenged by separating or approximating at the spinous, transverse, or mamillary processes and testing a strong indicator muscle for weakness.

the spinous processes, mamillary processes, or transverse processes. Occasionally the indicator muscle will weaken more on approximation, which is usually of the transverse processes on one side. A positive challenge when opening the wedge is more common and indicates that the optimal therapeutic maneuver is to separate the vertebrae, which agrees with Cox's[20] distraction therapy.

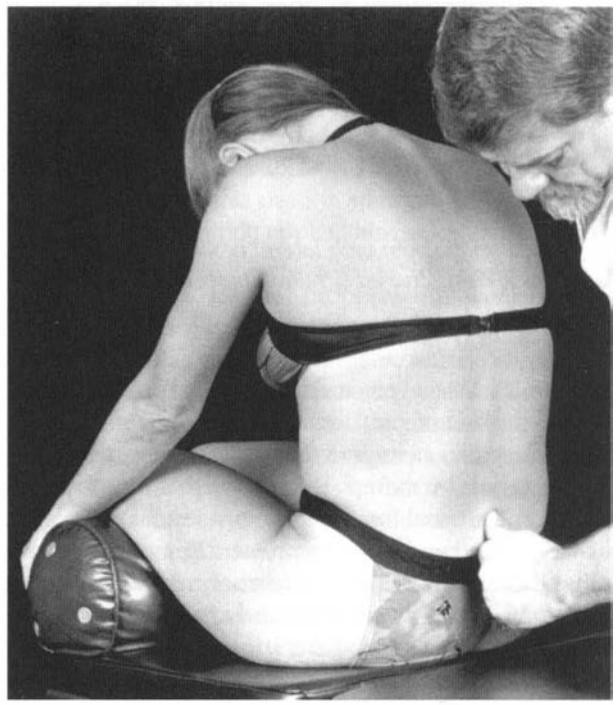
Obersteadt[98] evaluated fifty patients with indication of lumbar disc involvement to determine if there was a correlation between the two-handed therapy localization described by Goodheart's[39] and Bandy's[4] specific muscle test procedures. In this study the gastrocnemius was weak in twenty-one of the fifty patients tested, indicating S1 nerve root involvement. A comparison of the results of the lateral bending test with two-handed therapy localization and challenge revealed a 98% correlation between the two techniques. The tibialis anterior was weak in five of the fifty patients, indicating L5 nerve root involvement. There was 100% correlation between the two techniques. The rectus femoris (straight head), indicating L4 nerve root involvement, was weak in twenty-four of the fifty cases studied. There was 100% correlation between the simultaneous challenge of the spinous processes of L3-4. In only six was there strengthening of the rectus femoris with lateral spine bending. In the eighteen who didn't strengthen with lateral spine flexion, there was no positive two-handed therapy localization to L3-4. All the patients not showing a positive lateral bending test and two-handed therapy localization were found to have facet imbrication. Challenge for facet imbrication caused the rectus femoris (straight head) to strengthen; it did not strengthen with lateral lumbar flexion. Adjustment by imbrication technique corrected these eighteen cases.

Treatment

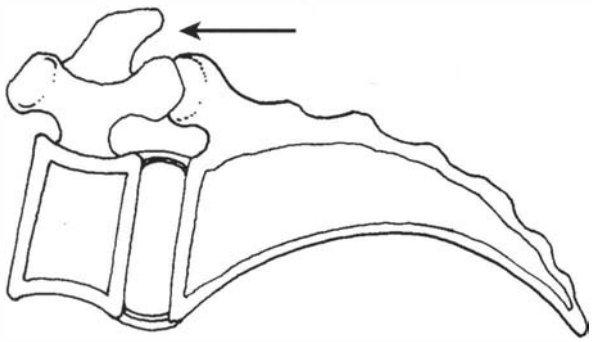
The original applied kinesiology treatment developed by Goodheart[39] for intervertebral disc involvement is application of non-traumatic pressure to the vertebra above the involved disc level in the direction of maximum positive challenge. Treatment of the intervertebral disc involvement can be done with the patient prone or seated; more often it is done prone. First the therapeutic pressure is applied to the vertebra above the lesion. Contact is made on the spinous or transverse process that causes the greatest amount of positive challenge in a cephalad direction. Pressure is applied as the patient slowly exhales to the maximum. As the patient inhales, caudal pressure is applied to the vertebra below the lesion at the contact point of maximum challenge; this is repeated four or five times. Challenge and therapy localization will be negative if the therapeutic effort was effective.

The AK technique can also be applied to the vertebrae with the patient seated, hands on his knees, and a cushion (such as a Dutch roll) placed under the distal thigh. The head and neck are flexed so that the chin rests on the chest. This position reduces the lumbar lordosis. The vertebrae are challenged the same as with the patient prone, and pressure is applied to the spinous or transverse process with the same respiratory correlation as that described for the prone patient.

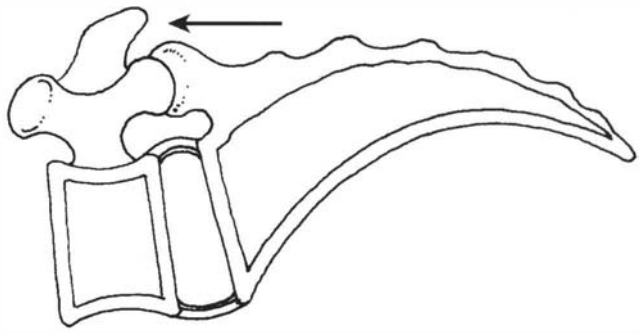
Holmes[62] combines applied kinesiology and Cox[20] techniques for intervertebral disc conditions. Using the AK diagnostic approach of therapy localization and challenge with other standard disc diagnostic techniques, the area of involvement is located. The patient is placed on the distraction table in the standard Cox manner, with or without ankles strapped, and the caudal portion of the table is gently moved downward as the patient inhales. Slight to moderate resisting pressure is applied to the vertebra above the disc lesion. As the patient exhales, the caudal portion of the table is allowed to come toward the ceiling, and greater cephalad pressure is applied to the vertebra above the lesion. The patient's respiration should be slow and deliberate to full phases of inhalation and exhalation. To repeat, the cycle is with full inspiration with the caudal portion of the table moving toward the floor, while modest pressure is applied to the spinous process above the disc lesion. With full exhalation the caudal portion of the table moves toward the ceiling, and more pronounced pressure is applied in a cephalad direction to the vertebra above the lesion. The cycle is repeated ten to twelve times



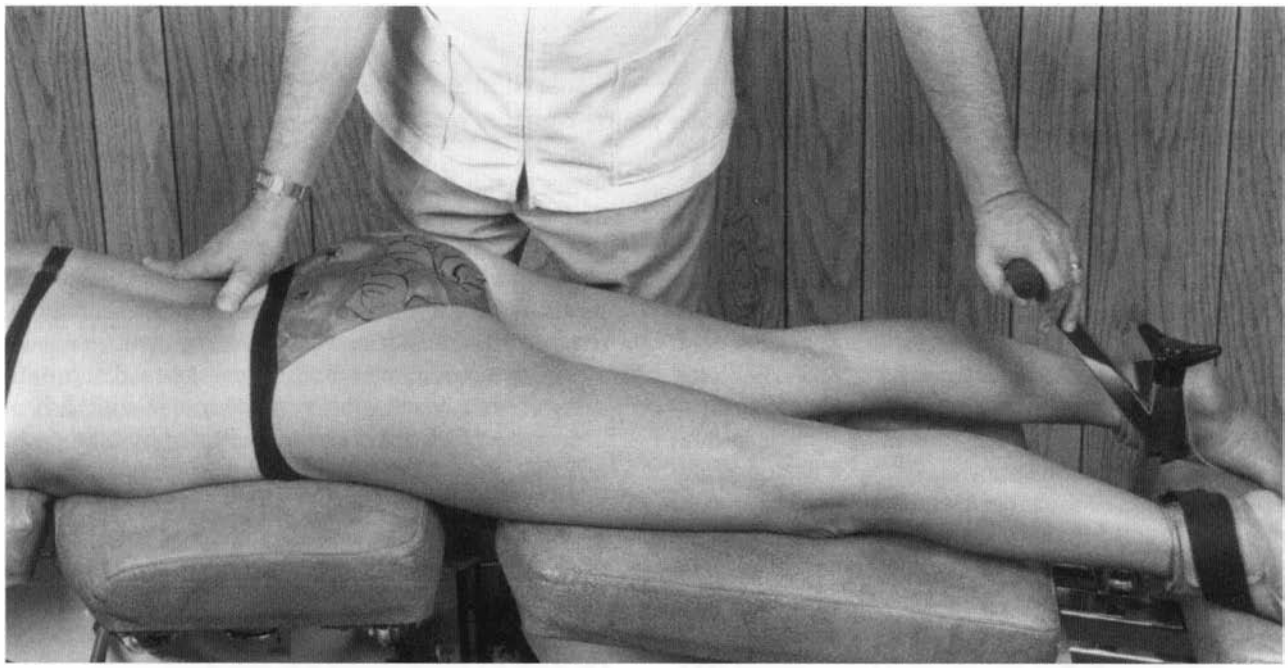
3—65. Sitting position for IVD treatment reduces the lumbar lordosis. Pressure is applied in direction of greatest positive challenge. First apply cephalad pressure on vertebra above disc involvement with patient expiration, then apply caudal pressure on vertebra below disc involvement on inspiration.



3—66. Inspiration. Slight pressure on spinous process as caudal portion of table drops.



3—67. Expiration. Modest pressure on spinous process as caudal portion of table elevates.



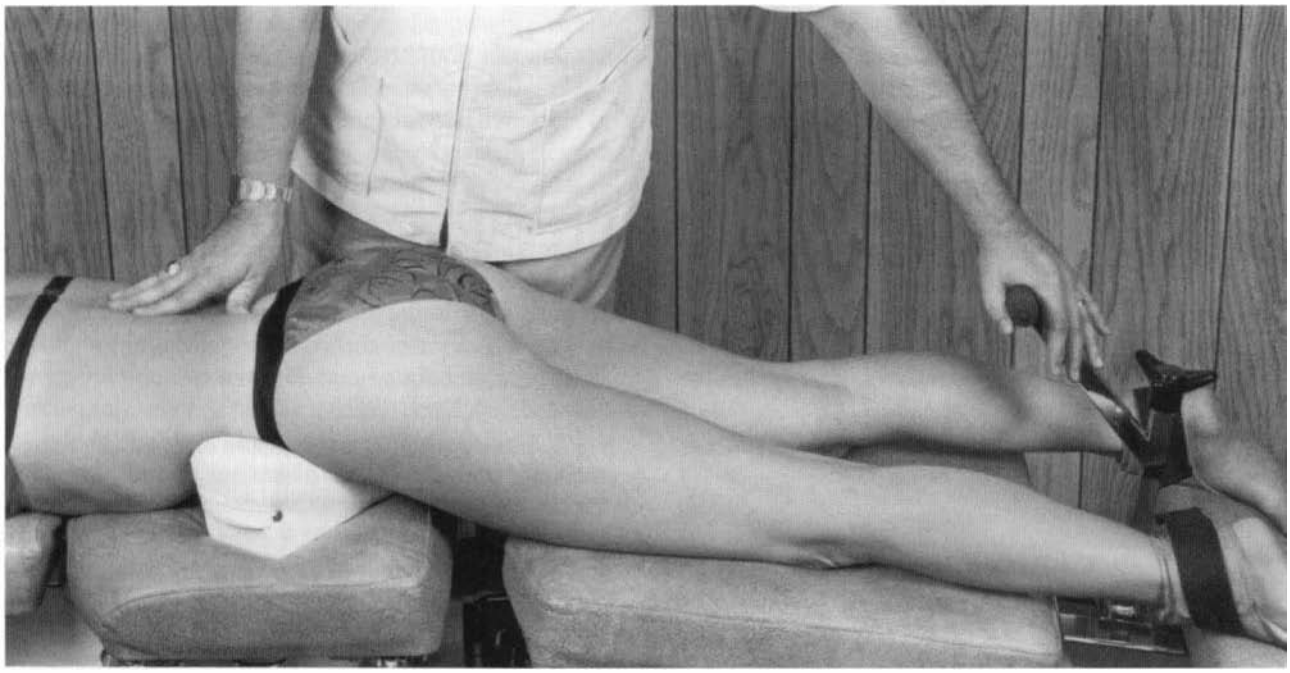
3—68. Hold spinous process as caudal portion drops. Lift the spinous process toward patient's head as table comes up.

to complete a treatment. Again the vertebrae are evaluated by therapy localization and challenge to determine treatment effectiveness.

Modifications and additions can be applied to the vertebral maneuvers previously described. Feder[32] recommends the use of DeJarnette blocks to put a torsion in the pelvis, similar to treating a category I pelvic fault. If therapy localization and challenge indicate the pelvis is involved in the complex, the blocks are placed at an angle of 45° with the points facing each other. One block is placed under the ischium on the short-leg side, and the other under the ilium on the long-leg side. Proper placement of the blocks is indicated by elimination of positive therapy localization at the sacroiliac articulations.

If the flexion distraction table is equipped with rotation capabilities of the caudal and thoracolumbar portions, they can be rotated to twist the pelvis in a manner similar to placement of the DeJarnette blocks. The caudal portion of the table contacts the pelvis at the acetabular level, and the thoracolumbar portion contacts the ilium. Care should be taken not to put torsion into the lower lumbar segments because they have limited rotational capability.

If the flexion distraction table is so equipped, the



3—69. DeJarnette blocks can be added to rotate pelvis.

caudal piece of the table can be laterally flexed to enhance opening of the wedge on a lateral disc protrusion. The caudal portion of the table is laterally flexed to approximately 30°, contralateral to the side of involvement.

Disc protrusion medial to the nerve root is indicated by increased pain when the patient laterally flexes away from the side of involvement. It is more difficult to treat and is, overall, less responsive. If lateral flexion of the caudal portion of the table is used, it should be toward the side of involvement. Contact on the mamillary or transverse process is on the side showing maximum weakening of an indicator muscle when challenged. If approximating the mamillary or transverse process on the side of involvement causes marked maximum challenge, pressure is applied with movement of the table on the side of involvement. If separation of the mamillary or transverse process contralateral to the side of involvement shows maximum challenge, pressure is applied to separate the vertebrae contralateral to the side of involvement. The same respiratory correlation is adhered to as described above.

Additional treatment methods that the physician has found successful should be applied, such as acupuncture, electrotherapy, physical measures, and support for the lumbar spine. When applicable, all muscles of the pelvis and thigh should be evaluated and balanced with applied kinesiology techniques. Techniques that are specifically applicable to modular organization and gait are also important for lasting correction. These techniques include PRYT, equilibrium reflex synchronization, dural tension, coccygeal lift, and iliolumbar ligament treatment.

During treatment of an intervertebral disc protrusion, one must monitor the condition continuously. If there is progression of the condition indicated by orthopedic and neurologic tests, as well as the symptomatic condition, further testing should be done, such as CT, MRI, and nerve conduction studies. The therapeutic program outlined here is conservative and non-traumatic, with a high percentage of good to excellent results. One should take care to avoid iatrogenic progression of the condition. An antalgic position should NEVER be forcefully broken. An antalgic position is consciously or subconsciously assumed by the patient to reduce pressure on the nerve. Obviously, forcefully going against the antalgic position increases pressure on the nerve and may be responsible for causing a prolapse of the disc. Side posture rotary manipulation generally applied, such as the "million dollar roll," is potentially harmful to the disc. One must remember that the lower lumbar spine has a limited rotational factor, and shear stresses are applied to the disc by forceful rotation. If a side posture adjustment is applied to the lumbar spine, it should be controlled and very specific.

Cervical Disc Syndrome

There are numerous conditions in the cervical spine that are effectively examined and treated using applied kinesiology techniques. Kabat,[80] who uses a muscle test of the wrist flexors to diagnose cervical disc problems, states "... that compression of the cervical spinal cord by the herniated nucleus pulposus of the cervical disc is the most common cause of low back and leg pain." There may be no place else in the body where dysfunction can cause as many remote problems. Conversely, remote problems often affect the cervical spine. The answer, as usual, is to consider all the interactions within the body. Applied kinesiology examination of the cervical spine should be correlated with other AK findings, as well as orthopedic, neurologic, and general physical examination.

General Considerations

Before manipulating the cervical spine one should determine if it is necessary to examine it with x-rays, especially if there has been recent trauma or evidence that spondylosis or other disease processes may be present. Often it is valuable to view the articulations of Luschka on oblique x-rays and evaluate motion with lateral flexion and extension views.

The vertebral artery should be routinely evaluated with a modified Adson's test. The supine patient first rotates the head to one side, then extends the head and neck and holds the position for thirty to sixty seconds. The test is done both right and left, and is usually positive in only one direction.[34,57,59] If dizziness, visual disturbances, nystagmus, or syncopy develops the test should be stopped. Rotary cervical manipulation — especially of the atlas-occiput area — is contraindicated.[84] The test is especially needed with women on birth control medication, which is a contributing factor to the vertebral artery syndrome,[102] and when there has been hyperextension/hyperflexion cervical trauma. In a postmortem study of thirty-two traffic accident victims with posterior cervical trauma, 31% had trauma to the vertebral artery.[114] Generally positive findings are on the left, but when due to birth control medication the positive findings are usually on the right.[2] When the provocative test findings are positive, further arterial examination is advised.[67]

General rotary adjustments of the cervical spine are not usually productive of effective correction, as observed by applied kinesiology challenge and therapy localization; they may be traumatic, with sometimes disastrous results. Manipulation of the cervical spine — or any other portion of the spine, for that matter — should be carefully designed and implemented to give corrective movement in agreement with the physiologic movement of the joints. Although complications, such as vertebral artery injury, are exceptionally low for chiropractic manipulation (they are higher for medical and other health care practitioners' manipulation),[84] they can be reduced even further with appropriate examination to uncover contraindications and proper application of the adjustive thrust.

Many resistant cervical spine problems result from the hyperextension/hyperflexion cervical sprain, the so-called whiplash accident. When these conditions do not respond to ordinary chiropractic care, they often involve disturbance of the hyoid muscles and/or cranial faults. The cranial faults are particularly important because of cranial nerve XI's supply to the sternocleidomastoid and trapezius muscles, and the nerve's dual origin from the cervical spine and cranium.[117]

A problem that often relates with the hyperextension/hyperflexion cervical sprain is soft tissue injury, which encroaches upon the nerve root at the intervertebral foramen. The lower cervical spine is especially subject to this type of problem because there are two synovial lined joints surrounding the intervertebral foramen.[112] The canal is formed anteriorly by the articulation of Luschka and posteriorly by the articulating pedicles of adjacent vertebrae. During the repair process of recent trauma to these articulations, forceful manipulation of the vertebrae is contraindicated because it may increase swelling and encroachment on the nerve root. The respiratory adjustment is helpful when working with recent trauma or in cases where forceful adjustment is contraindicated (page 80).

Cervical Disc Syndrome

Frank herniation of the intervertebral disc in the cervical spine is less common than in the lumbar spine. There is some discrepancy in the anatomical description of the cervical disc. Burke[14] describes the annulus fibrosis as being about twice as thick anteriorly and laterally as posteriorly. He states that the normal cervical disc is extremely strong, and trauma will cause a compression fracture before rupture of the annulus fibrosis. Jackson[71] describes a "safety zone" in which the nerve is protected from the intervertebral disc. She states, "None of the nerve root fibers pass over the intervertebral disc." The safety zone is made up of the body of the vertebra lying directly anterior to the anterior nerve root fibers. Jackson further considers that the annulus fibrosis is thicker posteriorly, with the nucleus pulposus situated anteriorly. Because of this arrangement she states, "Disc material is rarely extruded posterolaterally or posteriorly in the cervical area." This is in direct conflict with Turek,[112] who recognizes herniation in the cervical spine as small, usually no larger than a pea. Because of the limited capacity of the spinal canal and nerve structure mobility at the area it can cause enormous pressure. Most take the view that the annulus fibrosis is thinner posteriorly. Montgomery[92] attributes this to some of the fibers terminating at the intervertebral foramen, making the posterior section thinner with protrusion at the weakest area.

Ninety-five percent of disc lesions occur at the 5th and 6th vertebral levels. The rest occur at C6-7 and C4-5, which are predisposed when the lower levels are rigid because of degenerative joint disease.[112] When the cervical disc ruptures, there is usually a unilateral involvement with the rupture where the annulus fibrosis is weakest and where the posterior longitudinal ligament is thin. A midline rupture is rare and may cause immediate paraplegia that simulates hemorrhage, thrombosis, or a spinal cord tumor. It is an acute surgical emergency and should be treated as such. The cervical disc problem may develop in an insidious manner. A person may attribute the pain, which may be localized or radiate, to sleeping wrong or having a "crick in the neck." Exacerbations and remissions are the rule, with subsequent attacks usually more severe than the preceding ones and lasting much longer.

Bandy[3] combines muscle tests with positioning the head and neck and applying vertebral challenge to determine levels of cervical disc involvement. Testing this way also determines how corrective effort should be applied.

The character of the associated muscle weakness in a frank intervertebral disc syndrome is different than the usual "weakness" observed in applied kinesiology muscle testing. The disc syndrome causes frank radiculopathy where there is a varying degree of weakness that does not respond to evaluation of the five factors of the IVF. One will find no strengthening by therapy localization to NL and NV reflexes and other factors of the IVF. In a disc lesion, the muscle weakness is in proportion to the degree of nerve impingement; it is not a failure of locking, as in the standard applied kinesiology manual muscle test.

The muscles associated with the various nerve levels are emphasized to varying degrees among authors. Bandy[3] has associated specific tests with various levels as having a high degree of correlation. These relationships are deltoid, C6 nerve root, C5-6 disc; triceps, C7 nerve root, C6-7 disc; and finger abductors, C8 nerve, C7-T1 disc.

Upon finding a muscle weakness that appears to be associated with an intervertebral disc involvement, the disc is challenged by pressing on the adjacent vertebra until the vector is found that strengthens the weakened muscle. It is not a rebound challenge as seen in the standard applied kinesiology challenge of a vertebral subluxation; rather, it is a direct challenge with the vertebra held to determine strengthening of the specific muscle associated with the disc level.

The next step is to determine the position of the neck for correction. Have the patient fully extend his neck. Often this position will strengthen the muscle; if not, add rotation to one side or the other to find the position negating the weakness. With the neck in this position, an adjustive thrust is applied in the vector of challenge previously determined. If optimal strength is not obtained in the specific muscle associated with the disc level, repeat the two-step procedure of challenge and neck position and re-adjust.

The cervical disc involvement may not be present unless the patient is sitting or standing. The examination of challenge and neck position is then done in the position in which the specific muscle tests weak, to find what strengthens it. The adjustment is performed in that position. Before adjusting with the neck in extension and rotation, test the vertebral artery by the modified Adson's test described earlier. A literature review done by Kleynhans[84] indicates that the most common iatrogenic cause of vertebral artery injury is adjusting with a combination of rotation and hyperextension. Referring to neck manipulation, Fisk[34] states, "The artery is at risk if there is any extension, even with traction on the neck. The combination of extension and rotation in the 'manipulative' manoeuvre is really asking for trouble.

"It should also be common knowledge that the immediate postpartum period is a potentially hazardous time, vascular-wise. Others at potential risk are the mid-thirty females on the pill, particularly if they smoke."

The intervertebral disc involvement should not recur after proper correction. According to Bandy,[3] it can take up to six months for the annular fibers to heal. To promote healing and avoid stress to the involved tissues, the patient should avoid stressing the neck, such as by lying on the stomach or propping the chin on the hand while watching television, or reading in that position. Extreme motions of the neck, especially when held for long periods, should be avoided.

Hidden Cervical Disc

The cervical disc syndrome may be gross and show motor reflex and sensation dysfunction. Applied kinesiology techniques may indicate a lesion when all of the diagnostic criteria are not present. Goodheart[41] postulates that some cervical spine pain and radicular problems result from laxity of the annulus fibrosis, causing an intervertebral disc bulge. He does not consider this a true herniation; rather, it is a "hidden cervical disc" problem. The mechanism responsible appears to be an anterior cervical subluxation.

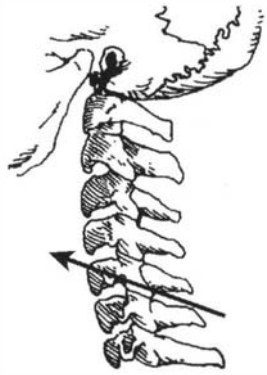
There are two types of challenge that reveal this disturbance. Goodheart uses the wrist extensors, if strong in the clear, as general indicator muscles for cervical disc problems. A general challenge is pressure on the vertex of the head in a caudal direction that applies axial compression to the disc, causing the wrist extensors to test weak when the condition is present. Often the wrist extensors will be strong when the patient is non-weight-bearing and weak when standing or sitting.

The specific challenge to locate the level of involvement is to press on the transverse process in an anterior superior direction in alignment with the facet plane. When the vertebra above the level of disc involvement is challenged this way, the indicator muscle will test weak. The

challenge may be positive bilaterally or unilaterally. There will be a considerable amount of tenderness on the inferior aspect of the spinous process.

The optimal vector of vertebral challenge usually indicates the contact and direction to apply an adustive thrust to the vertebra. In the case of a hidden cervical disc the direction of correction is opposite the positive challenge. Adjusting in the direction of positive challenge will probably make the patient's condition worse.

The hidden cervical disc will not show positive therapy localization in the usual manner. It can be used to enhance the cervical spine weight-bearing test. Simply apply therapy localization in conjunction with pressure applied to the vertex of the head, compressing the cervical spine. In some cases, the weight of the upright patient's head on the cervical spine is adequate compression to cause positive therapy localization.

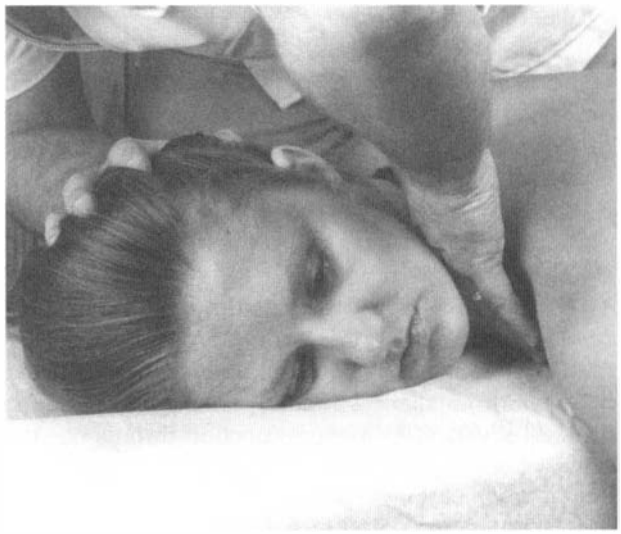


3—70. A positive challenge is in the direction the vertebra has moved to compromise the cervical disc.

There are two techniques for adjusting the vertebra responsible for the hidden cervical disc. One is with the patient supine, the other with him prone. With the patient supine, the physician contacts the spinous process below the involved disc level with the side of his index finger. The spinous process can be quickly located by going to the spinous process immediately below the one that is tender and associated with the challenge noted above. The physician cradles the patient's head with the forearm of his other arm and the hand wraps around to contact under the chin, enabling the physician to direct traction to the cervical spine while stabilizing the vertebra below the disc level. This is a controlled type of cervical imbrication adjustment.

The segment may be adjusted with the patient prone. Depending upon the challenge present, one or both sides may need to be adjusted. If the challenge is positive bilaterally, first adjust the side with maximum weakening on challenge. The position for the adjustment is with the patient's head turned toward the side to be adjusted. The physician stands on the side away from the head-turn and contacts the vertebra at the transverse process of the vertebra below the disc level (the vertebra below the one that had a positive anterior challenge). The adjustment is done rapidly, with a line of drive along the facet plane (posterior-caudal direction). If the opposite side continues to show a positive anterior superior challenge on the vertebra above the disc level, it should be adjusted in a similar manner. A little more force may be needed on the second side since the vertebra has pivoted slightly.

Re-therapy localize and challenge the disc to determine if correction was successful. There should also be a reduction of pain at the spinous process above the level of disc involvement. If pain remains, Goodheart recommends nutritional supplementation in the form of superoxide dismutase (SOD) rather than manganese, which is usually given in applied kinesiology for other disc involvements. Prior to treatment, SOD will neutralize the positive challenge and therapy localization when the patient chews the substance.



3—71. Prone adjustment of cervical disc.

3—72. Adjustment direction is on the transverse process in a posterior caudal direction to slide the vertebra down and posteriorly along the facet line.

Cervical Compaction Technique

Proper motion in the cervical spine is of extreme importance because of the very high nerve receptor concentration in the joint capsules and their role on function throughout the body.

Barry Wyke,[122,123] a noted English neurologist, describes the receptors in the cervical spine joints and their influence throughout the body. The joints of the cervical spine, like all synovial joints, have an array of mechanoreceptor (corpuscular) and nociceptor (non-corpuscular) nerve endings. This includes both the facet and Luschka articulations. The Type I receptors (low-threshold, slow adapting) have powerful reflexogenic influences on the cervical, limb, jaw, and extra-ocular muscles. These receptors are very important in postural and kinesthetic sensation. The Type II receptors (low-threshold, rapidly adapting) monitor the phasic changes of the cervical and limb muscles.

Some of the afferent branches from the Types I and II articular receptors ascend and descend within the spinal cord to synapse in the motor neuron pools of the neck and upper and lower limb muscles. Branches primarily from the upper three or four cervical spinal joints ascend in the propriospinal tracts to be distributed in the motor neuron pools of the jaw and external ocular muscles.

The role of the nerve receptors in the joints of the cervical spine emphasizes the importance of the normal function of this area. Receptor stimulation impaired by structural imbalance, trauma, pathology, or the aging process adversely influences function throughout the body.

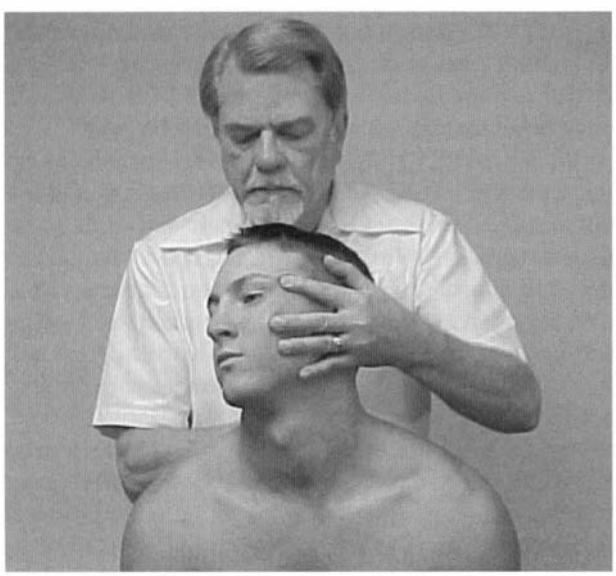
Goodheart describes a one-leg standing test developed by Freeman and Wyke that appears to monitor the cervical spine mechanoreceptors.[50] The patient is asked to stand on one leg at a time, and his deviation from centerline is compared. The deviation should be the same bilaterally. Deviation from centerline should not be more than five inches and, more important, there should be equal deviation from side to side. Unequal deviation is most often balanced with the cervical compaction technique

Cervical Motion Examination

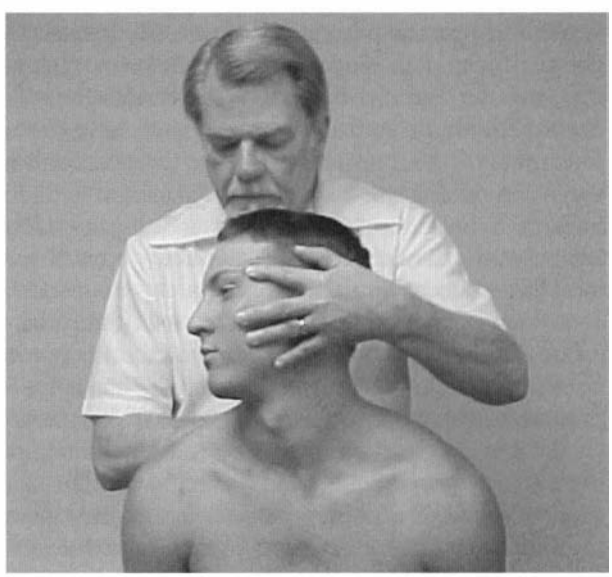
Examination for deficiencies in cervical motion that respond to cervical compaction technique is done after cervical subluxations and/or fixations are corrected, pelvic function has been evaluated and treated, and other obvious structural faults have been corrected.

Cervical range of motion is more thoroughly evaluated than in the usual orthopedic examination. First, the usual flexion, extension, rotation, and lateral bending are evaluated. Rotation is further evaluated by having the patient put his cervical spine in extension and, while maintaining the extension, turn the head to the right and left. Rotation is also evaluated with the cervical spine in flexion, then turning the head to the right and left. If there is imbalance from side-to-side of these active motions, re-evaluate the patient for subluxations and/or fixations, muscular imbalance, pelvic problems, and any other factor that restricts active cervical range of motion. When the active range of motion is balanced in these activities, evaluate the same motions on a passive basis, i.e., with the physician turning the patient's head to the right and left and comparing range of motion from side-to-side. There should be the same range of motion when the head is turned passively as was present actively.

When cervical compaction is required, passive mo-



3—73. Limited passive cervical rotation in slight extension.



3—74. Actively the patient is able to increase rotation.

tion is less and active motion is greater. When passive motion limit is felt, the patient is asked to continue turning and additional motion will be obtained. The exact reason for greater range of motion when done actively is unknown. When the movement is active, it appears that motion is improved by the patient's muscle organization recognizing where the restriction is and avoiding it. Correcting the passive range of motion by the cervical compaction technique appears to re-activate the cervical mechanoreceptors and balances the one-leg standing test deviation.

Treatment

An axial force is applied to the cervical spine while the physician moves the spine through motion. The physician applies 3 to 4 pounds of rhythmic force to the vertex of the patient's head while passively moving the cervical spine through a range of motion specific for the type of limitation. Motion and compaction are done four or five times through the corrective range of motion for the limited passive motion. Following the correction there should be full range of motion evaluated both actively and passively.

Passive limitation	Corrective motion
Lateral flexion	Ipsilateral rotation
Flexion	Extension
Extension	Flexion
Rotation in extension	Ipsilateral lateral flexion in extension
Rotation in flexion	Ipsilateral lateral flexion in flexion

Examples:

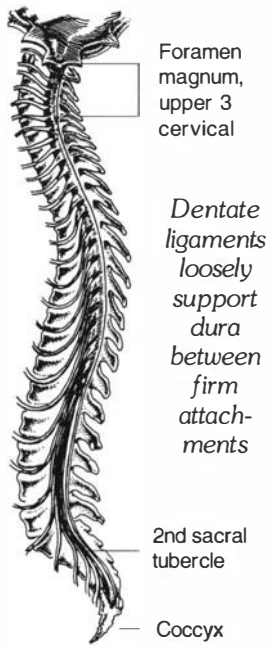
1. The patient's active cervical rotation is equal but limited to the left when done passively. Treatment is done by the physician applying pressure to the patient's vertex while rhythmically moving the cervical spine through lateral flexion. The movement through lateral flexion is done four to six times.
2. The patient first moves into cervical extension and then actively rotates his head right and left with equal ability while maintaining the cervical extension. The physician then passively compares rotation from side to side while maintaining cervical extension and finds limitation to the left. Vertex pressure is applied with the patient's cervical spine in extension, and the cervical spine is moved into lateral flexion as the physician applies rhythmic pressure to the vertex with four to six repetitions.

Pelvic Categories

Throughout applied kinesiology it is emphasized that the body functions as an integrated whole. Pelvic function is an important example of this interdependence. The pelvis is an assembly that has sub-assemblies; that is, the pelvis can move as a whole, yet there is action between the innominates and the sacrum, and between the sacrum and the coccyx. Pelvic dysfunction is divided into three categories. Categories I and II are dysfunctions of the sub-assemblies; category III is dysfunction of the intact pelvic assembly with the 5th lumbar.

The pelvic category system was developed by DeJarnette[24] and is practiced in sacro occipital technique (SOT). The original system of evaluation and correction is viable, and is the basis for additional diagnosis and therapeutic developments in applied kinesiology.

Pelvic category faults are intimately involved in creating dural tension because of the firm dural attachments at the occiput and upper cervical vertebrae, with no further firm attachment until the anterior portion of the 2nd sacral segment by the filum terminale. In between these firm attachments are the dentate ligaments, which only loosely support the dura. It appears that the wide range of dysfunction and symptomatic problems from pelvic faults is due to dural tension and pelvic ligament relations with the spine, documented by Dvorak and Dvorak.[27] If the examination for pelvic faults is negative but symptoms indicate a probable fault, cervical motion may cause a positive fault finding. Anterior or lateral cervical flexion can increase dural tension to reveal the fault.



3—75. Dural attachments

More than one type of pelvic category fault can be present at the same time. On an initial examination, usually one type of fault will be prominent. When it is corrected, another fault may be revealed. For example, after a category I fault is corrected, the pelvis may test positive for a category III fault that was not previously apparent.

There are several methods of correcting pelvic faults; some were developed in SOT and others in applied kinesiology. A recent study using SOT methods of examination and correction evaluated muscle strength change.[113] The anterior deltoid, latissimus dorsi, psoas, tensor fascia lata, adductors, and gluteus medius strength was measured pre- and post-treatment. There was significant strength increase in most of the muscles tested. The choice of corrective method can be matched to the physician's training in manipulation and treatment style.

Category I

The category I pelvic fault is torsion of the pelvis without osseous misalignment at the sacroiliac articulations; thus there are no subluxations as such in this involvement. It often relates with other distortion throughout the body, including cranial faults.

A common complaint of a category I pelvic fault is cervical spine tension. The patient complains of pain and limited motion on turning his head, making it difficult to back his car. This will usually be more marked on one side. Secondary to the pelvic torsion, there is often torsion of the shoulder girdle that may manifest as a thoracic outlet syndrome. Cranial faults are often associated with a category I as well.

Therapy Localization

A category I pelvic fault has a unique therapy localization that differentiates it from other pelvic disturbances. The patient is usually examined prone, and strong hamstrings are used as indicator muscles for the therapy localization. There will be positive therapy localization when the patient places his hands on the sacroiliac articulations, right hand on right and left hand on left. Care should be taken that the therapy localization is over the ligaments of the sacroiliac articulations. Positive therapy localization will usually be with the palmar surface of the fingertips touching the patient's skin; however, palm-up therapy localization should also be evaluated. There will be further positive therapy localization on one sacroiliac only, done with one of the patient's hands over the other. This is considered the positive side of the category I pelvic fault. Neither sacroiliac articulation will show positive therapy localization if single-handed TL is done one at a time.

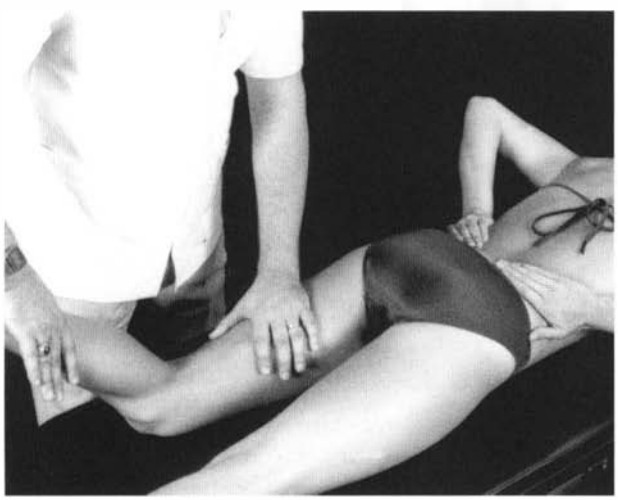
The typical pain location on digital pressure for a category I is at the inferior medial aspect of the PSIS on the positive side. This is the reason for a positive Mennell's sign[91] in standard orthopedic testing. This tenderness is increased by pulling posteriorly on the ilium, and relieved by pulling forward.

All aspects of a pelvic examination should be considered to differentiate a category I from a category II. In unusual cases, there can be a bilateral category II. In this case, there will be positive therapy localization when the right and left hands are on the right and left sacroiliac articulations, respectively. There will also be single-handed therapy localization over each sacroiliac. In this case correct the category I first, which may clear the category II. In addition, there will be differences in challenge of category I and category II pelvic faults, discussed later.

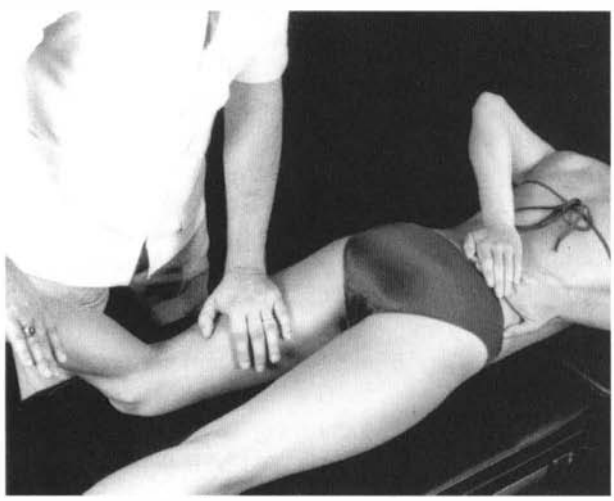
Challenge

The torsion of a category I pelvic fault consists of a posterior superior iliac spine (PSIS) on one side and a posterior ischium on the other. A positive category I challenge is simultaneous pressure applied on the PSIS and contralateral ischium in an anterior direction and released, followed by a strong indicator muscle weakening. There will be one combination of vectors that causes the maximum amount of indicator muscle weakening. Because the pelvis demonstrates a rebound-type challenge, this is the optimal vector for correction.

Positive challenge will usually be with the PSIS posterior on the side of the short leg. If this does not correlate, consider anatomical variances, such as an anatomical short leg or neurologic disorganization. If



3—76. First step in therapy localization for category I. Positive therapy localization with one hand on each sacroiliac indicates probable category I.



3—77. Positive therapy localization with two hands placed over one sacroiliac indicates positive or compromised side of category I. Does not have positive therapy localization with only one hand.

there is a discrepancy between the challenge and leg length and other factors have been ruled out, the challenge takes precedence.

A category I pelvic fault will not have positive challenge when only one sacroiliac is challenged. This is a differentiating factor between a category I and category II fault. To challenge only one sacroiliac, stabilize the sacrum with one hand and challenge the PSIS or ischium of one side. Test a strong indicator muscle for weakening.

Muscle Involvement

Several muscle dysfunction patterns are often associated with, and probably the cause of, category I pelvic faults. The piriformis is often weak on the side of two-handed therapy localization and hypertonic on the other, or bilaterally weak. If the piriformis is weak only on the opposite side of two-handed therapy localization, the patient is neurologically disorganized. Piriformis weakness is important because it crosses the sacroiliac articulation and helps provide stability. Piriformis weakness, along with that of the gluteus maximus, correlates with DeJarnette's dollar sign[24] in sacro occipital technique. Other muscles to evaluate are the gluteus medius, sacrospinalis, and quadratus lumborum. The oblique abdominals are specifically important because of the major lever factor they have on the crest of the ilium.

The muscles can be tested in the clear, but they often do not test weak until the patient is weight-bearing. The piriformis muscle can be tested in a weight-bearing position, with the patient either standing or on his hands and knees on an examination table. When muscle weakness is present only in the weight-bearing position, the muscle is treated as usual. It may be necessary to treat the muscles while the patient is weight-bearing. In addition, weight-bearing factors such as foot function should be evaluated when the test is positive.

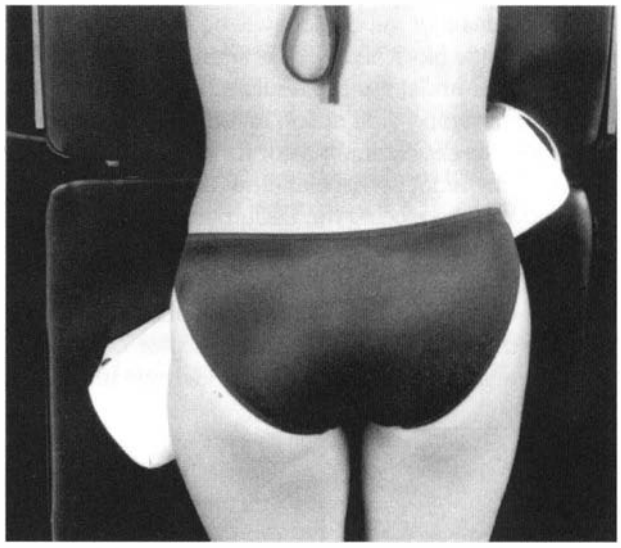
1st Rib and Thoracic Outlet

Shoulder girdle distortion is frequently secondary to a category I pelvic fault creating pain at the 1st anterior and posterior rib attachments on the side of two-handed therapy localization. The tenderness is usually exquisite but will be relieved after pelvic corrections.

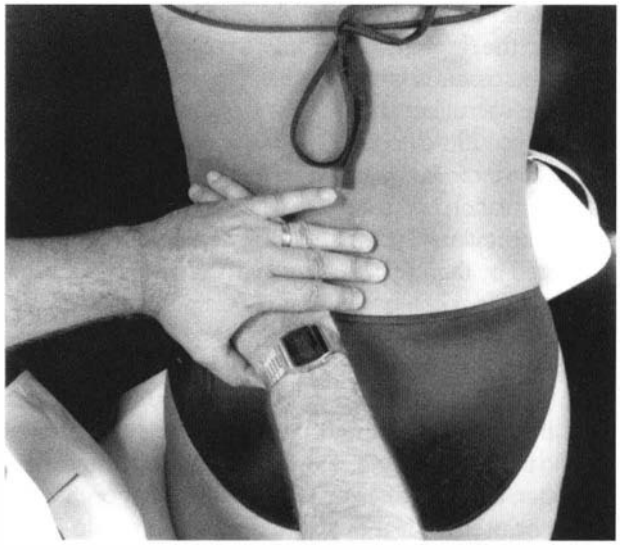
Imbalance and dysfunction of the shoulder girdle often create a thoracic outlet syndrome with compression of the neurovascular bundle. This will test positive as a costoclavicular, scalenus anticus, or pectoralis minor syndrome, among others. A positive thoracic outlet syndrome can often be corrected by making corrections only at the pelvis. In these cases the distortion and dysfunction of the shoulder girdle are secondary to the pelvic torsion of a category I pelvic fault. Elimination of positive orthopedic tests associated with a thoracic outlet syndrome by pelvic correction readily indicates the association.

Block Adjusting Technique

DeJarnette blocks are placed under the prone patient's anterior superior iliac crest and acetabulum in a manner to relieve the pelvic torsion. Block placement is determined by challenge. The posterior ilium side is the one where the PSIS was challenged from posterior to anterior and a strong muscle weakened; the contralateral side is the posterior ischium. The posterior ilium is usually on the short-leg side, but the challenge takes precedence for how to block the patient. A block is placed under the acetabulum on the posterior ilium side to bring the ischium posterior. Contralaterally, the block is placed under the ASIS to bring the ilium posterior. When the



3—78. Blocks placed for category I left PI ilium and right posterior ischium. Blocks are placed under anterior superior iliac spine and acetabulum, and point toward each other.



3—79. The manipulation is a pumping-type movement opposite the positive side. Illustration is for category I positive on right, with a posterior ilium on side of manipulation.

blocks are properly placed, there will no longer be positive bilateral sacroiliac therapy localization, and there will usually be relief of pain at the 1st rib head.

The patient's body weight lying on the blocks may adequately make a correction. A gentle thrusting-type action has been added in applied kinesiology to facilitate and speed the correction.

As mentioned earlier, one sacroiliac is the compromised or involved side; that is the one presenting the two-handed therapy localization. The uncompromised or "non-involved" side will not have positive two-handed therapy localization. The non-involved side is the side of contact for the manipulative effort. Contact is either on the PSIS or ischium in the direction of positive challenge. This indicates that the contact will be on the ischium if the DeJarnette block is under the ilium, and on the PSIS if the block is under the acetabulum. Corrective motion is a light, pumping-type action repeated approximately ten times. An excellent indicator for the number of repetitions is the reduction of tenderness at the posterior 1st rib head. Before placing the DeJarnette blocks, palpate the rib head for tenderness; compare after the blocks are in place, and after the corrective manipulation has been applied. Usually there will be great reduction of tenderness on digital pressure. A good indicator of effective correction is a minimum of 50% tenderness reduction; often it is much greater. With experience, one can readily determine the rigidity of the pelvis after the first few corrective thrusts. In a very rigid pelvis, it may take more thrusting actions than usual to obtain maximum correction. After the corrective attempt there should be no positive therapy localization or challenge to the pelvis. If a category I pelvic fault is not easily corrected or if it returns, some other factor is involved, such as muscle dysfunction, weight bearing, or gait dysfunction; this should be evaluated and corrected. A patient may test negative for a category I fault, but when tested weight bearing or immediately after gait, he will test positive. A category I pelvic fault can be evaluated for gait influence by simply having the patient therapy localize over both sacroiliac articulations while walking. When the patient stops, have him maintain the therapy localization while an indicator muscle is tested. If the category I is specifically involved with gait, an indicator muscle will test weak; it will not test weak when the patient simply walks without the sacroiliac therapy localization.

Cranial Fault Correlation

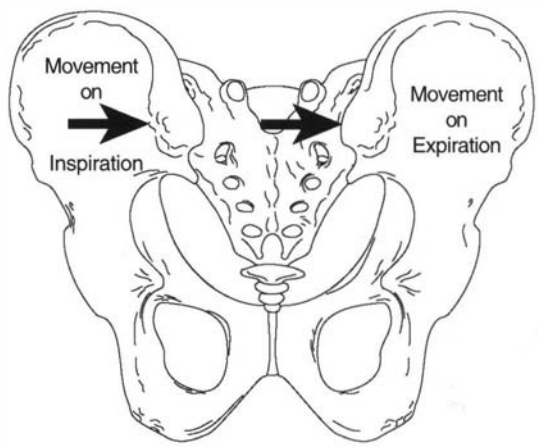
Often with a category I pelvic fault there is disturbance in cranial primary respiratory function. A routine procedure should be the evaluation of the cranium with other factors of the stomatognathic system.

Category II

There are two types of category II pelvic faults. The category II fault has been recognized as an osseous subluxation at the sacroiliac articulation. Goodheart[48] recognized an additional type of pelvic fault in which the major involvement is at the symphysis pubis. This is still considered an osseous subluxation of the pelvis as opposed to the respiratory torsion of a category I pelvic fault. Since the osseous fault is at the symphysis pubis rather than the sacroiliac articulation, this fault was originally called a pseudo-category II pelvic fault. The original category II is indicated by therapy localization at the sacroiliac articulation and is called a category II sacroiliac pelvic fault, abbreviated category IIsi. The additional type is indicated by therapy localization at the symphysis pubis and is called a category II symphysis pubis pelvic fault, abbreviated category IIsp. The change from a pseudo-category II is an effort to be more descriptive of the fault.

A typical complaint with a category II fault is leg pain that develops during the night but is not present during the day. Mid-thoracic, lumbar, or abdominal pain will often develop after heavy lifting. A category II may be associated with a dropped arch on the side of involvement.

There is movement within the pelvis with respiration. With inspiration the ilia move laterally, the symphysis pubis moves inferiorly, the sacral base moves posteriorly and the apex anteriorly. The posterior iliac spine moves medially with inspiration. The opposite of each movement takes place with expiration.



3—80. If held inspiration cancels positive PI ilium therapy localiztion, adjust the PSIS in an anterior medial direction as indicated on the left innominate. If held expiration cancels positive therapy localization, adjust in an anterior lateral direction. In both cases have the patient hold the phase of respiraton that canceled the positive therapy localization while the adjustment is being made.

Category IIsi

A category IIsi pelvic fault is an osseous subluxation between the sacrum and the innominate. It is identified by positive therapy localization over the sacroiliac articulation. Therapy localization is usually done with the patient supine, which yields a higher percentage of positive results than when done prone. Usually only one side will be positive. It is possible to have bilateral category IIsi pelvic faults, in which case a differentiation must be made between a category I and a category IIsi, as described under category I. Additional differential diagnosis is available in the characteristics of categories I and IIsi.

Pelvic respiratory movement can be used to advantage in diagnosis and treatment. When there is positive therapy localization over the sacroiliac, have the patient take and hold a deep phase of respiration. If the positive therapy localization is abolished it indicates movement in that direction is corrective. Adjust the structure in the direction of respiratory movement while the patient holds the respiration.

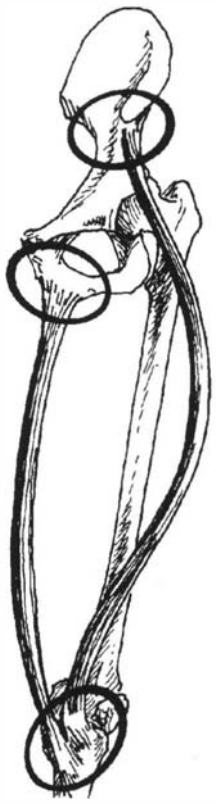
Usually the category II will be readily identified by therapy localization over the sacroiliac articulation. For a more definitive diagnosis to find a subclinical category II, add body into distortion (BID), eyes into distortion (EID), head tilt (described in Chapter 6), and held respiration to the therapy localization. In the latter case, maintain the negative therapy localization contact and have the patient take and hold a phase of respiration. If there is now weakening of an indicator muscle, the held respiration is detrimental to the sacroiliac position. Correction should be done on the opposite phase of respiration and in that innominate movement.

There are two major types of category IIsi pelvic faults: the posterior ilium and posterior ischium. They have different muscle involvements and areas of tenderness that differentiate them from each other and from a category I.

Posterior Ilium

The posterior ilium is nearly always associated with dysfunction of the sartorius and/or gracilis muscles on the side of involvement. They give anterior support to the pelvis. Sometimes the muscles will not test weak in the clear, but a subclinical weakness will be found by therapy localizing the reflex points or other factors involved with the muscles. They may also test weak only in a weight-bearing position.

The muscle relationship with the sacroiliac subluxation can be demonstrated by correcting the subluxation



3—81. The pelvis is provided anterior support by the sartorius and gracilis. Tenderness in a posterior ilium category II will be at the circled locations.

3—82. Muscles that are weak in a posterior ischium. Circles delineate tender areas.

with the usual manipulative techniques, but not strengthening the muscles. The manipulation should balance the leg length and eliminate positive therapy localization and challenge. If it does, have the patient walk a short distance and then re-evaluate for the presence of the subluxation. In many cases, when the muscles are not strengthened the subluxation will immediately return following walking. Correct the muscle dysfunction and repeat the process; the subluxation will usually not return.

There are specific correlations to the posterior ilium category IIsi pelvic fault. The leg on the side of the posterior ilium will be short in the absence of congenital variations, which are rare. A postural x-ray taken with properly aligned equipment will show the innominate longer on the posterior ilium side. There will be tenderness at the origin and insertion of the gracilis and/or sartorius and along most of the muscle's length. There will also be tenderness at the anterior and posterior 1st rib heads. Challenge on the posterior superior iliac spine for the vector that causes the greatest weakening of an indicator muscle. Adjust in the direction of positive challenge with the phase of respiration that canceled positive therapy localization.

Adjustment of the posterior ilium can be done with the patient prone or side-lying. The prone adjustment can best be done with a drop terminal point table. When done side-lying take care not to put excessive rotation into the lumbar spine that may cause disc trauma.

Posterior Ischium

The posterior ischium subluxation is not as common as the posterior ilium. It is usually secondary to weak hamstring muscles, which give posterior stabilization to the pelvis on that side. If the hamstrings are not weak in the clear, evaluate for subclinical weakness. This may be present only when weight bearing or some other factor is added to the examination, e.g., therapy localization to the neurolymphatic reflex. The leg will be long on the side of the posterior ischium in the absence of congenital anomalies. A postural x-ray will reveal a shorter innominate on the side of posterior ischium. There will be tenderness at the origin of the hamstrings on the ischial tuberosity, and there may be tenderness at any or all points of hamstring insertion. The medial hamstrings insert on the medial side of the tibia. Differentiate hamstring insertion tenderness on the tibia from that at the insertion of the sartorius and gracilis. The anterior and posterior 1st rib head attachments will usually be tender. Challenge on the posterior ischium for the vector that causes the greatest weakening of a strong indicator muscle. Adjust in that vector on the phase of respiration that canceled positive therapy localization.

Adjustment of the posterior ischium can be made with the patient in a prone or side-lying position. When done side-lying, one should take care not to put excessive rotation into the lumbar spine.

Category IIsp[48]

Associated with the pelvic torsion of a category IIsp pelvic fault is tension in the sacrospinous and sacrotuberous pelvic ligaments. These ligaments are important in pelvic balance and integrity. There are spondylogenic reflexes of the sacrospinous ligament to the occiput to C6 and of the sacrotuberous ligament from C7 to T8 that are often responsible for paraspinal pain in those areas, as described on page 129.

Therapy Localization

A category IIsp is identified by testing the sartorius for positive therapy localization over the symphysis pubis, slightly to the right and left. Prior to the therapy localization there will usually be no tenderness on digital pressure at the origin/insertion of the sartorius and gracilis muscles, but it will develop with the pubic therapy localization. Leg length will often be balanced and become imbalanced with therapy localization. The muscle weakening and tenderness are consistently present with imbalance and stress of the sacrospinous and sacrotuberous ligaments.

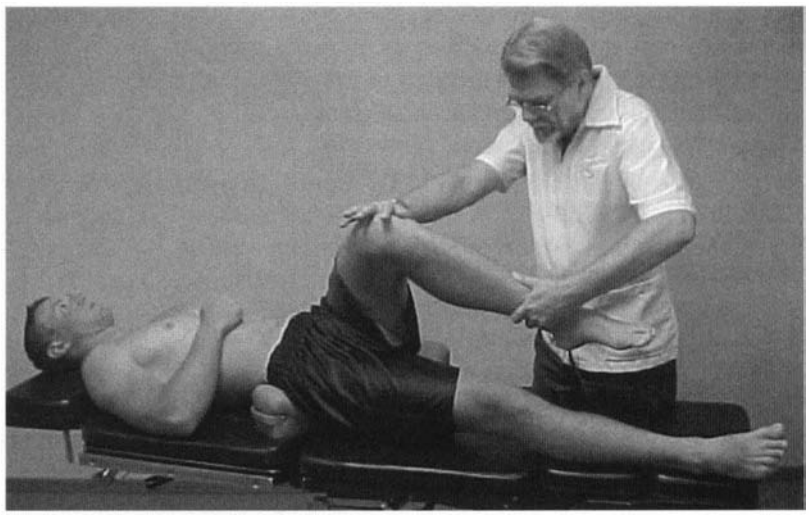
Sometimes there is symptomatic evidence that this fault is present, but there is no positive therapy localization at the pubis. Some indications are pain at the cervical and thoracic spine and/or of the shoulder and arm. Therapy localization indication may become positive when the patient flexes his neck and does a partial sit-up (curl-up). This causes increased dural tension to reveal the subclinical fault that is not evident when lying supine. Lateral flexion — and rarely extension — of the cervical spine may also reveal the fault.

Challenge

Torsion of the pelvis is posterior ilium on one side and posterior ischium on the other, with torsion mainly at the symphysis pubis where there is positive TL with none at the sacroiliac articulations. Usually the ilium will be posterior on the short-leg side, and the ischium posterior on the long-leg side. The exact torsion of the pelvis is confirmed by challenge, which takes precedence over apparent leg length. With the patient supine, place one hand under the ilium on one side and the ischium on the other side, and lift as if to lift the patient away from the table. A positive challenge is weakening of the sartorius or gracilis muscle, which is best, but any previously strong indicator muscle such as the tensor fascia lata can be used. The pelvis can then be re-challenged with a hand under the opposite ilium and ischium. There should only be one positive challenge. The challenge is of a rebound nature, as with other areas of the spine. The side on which the ilium was lifted anteriorly, causing a weak muscle, is the posterior ilium; the side on which the ischium was lifted anteriorly, causing an indicator muscle to weaken, is the posterior ischium side.

Block Adjusting Technique

A DeJarnette block is placed under the posterior iliac spine (PSIS) on the posterior ilium side, and under the ischium on the posterior ischium side. If the block placement is proper for correction, there will no longer be positive therapy localization at the symphysis pubis and pubic bone. With the patient remaining on the blocks, the physician grasps the patient's ankle and knee to move the leg on the posterior ischium side into flexion at the hip and knee. The thigh is adducted, bringing the knee across the body sufficiently to roll the patient gently onto the posterior ilium block. The knee and hip are then brought toward neutral, and the maneuver is repeated about six times in a rolling fashion. It is usually necessary for the patient to stabilize the position of the DeJarnette blocks with his hands to keep them from slipping under the pelvis. A similar motion is then done about six times with the leg of the posterior ischium side, but with thigh abduction. (Note: It is also applicable to move the legs through the motion alternately.) Following this action, the blocks are removed and the patient is re-evaluated with therapy localization, which should no longer cause weakening of the indicator muscle or tenderness at the muscle's origin/insertion.



3—83. Leg movement with supine block adjusting technique for category II.

Category III

In a category III pelvic fault the pelvis in intact. The fault is dysfunction of L5 on an intact pelvis, or an intact pelvic dysfunction on L5. Symptoms from a category III pelvic fault can be local or remote. Often there is severe sciatica that fails to respond. A category III may be in conjunction with a lumbar disc involvement, facet syndrome, or other conditions that involve the lumbosacral spine. Examination and treatment should be a combination of standard orthopedic, neurologic, and AK methods. The pain may be in the low back that eases with movement and comes back with standing. Because of the influence on the dura, there may be cranial nerve involvement, especially at the jugular foramen. There may be involvement of cranial nerves IX, X, and XI. This may cause cervical problems because of sternocleidomastoid weakness, or digestive problems as a result of vagus nerve involvement. One can observe function of the glossopharyngeal nerve (cranial IX) by observing the uvula and posterior palatal tissue as the patient says "ah." If the uvula deviates to one side, unilateral glossopharyngeal involvement is indicated.

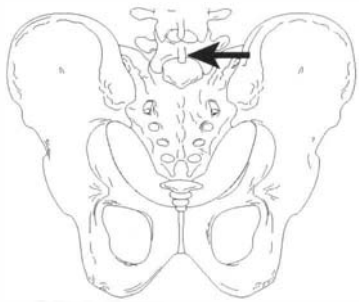
The category III pelvic fault influences hip rotation because of muscle imbalance. To evaluate hip rotation the examiner internally rotates both legs by grasping the ankles. There will be much greater internal rotation on one side.

Therapy Localization

Because the pelvis is intact, there is no positive therapy localization at the sacroiliac articulations or at the symphysis pubis.

Challenge

The category III pelvic fault is determined by challenge with the patient prone. Contact the anterior portion of the ischium and lift it posteriorly while the L5 spinous process is pressed toward the side of ischial contact. A positive challenge is indicated by weakening of a previously strong muscle, usually the hamstring group. The challenge is done bilaterally, and only one combination of ischium spinous process challenge will be positive.



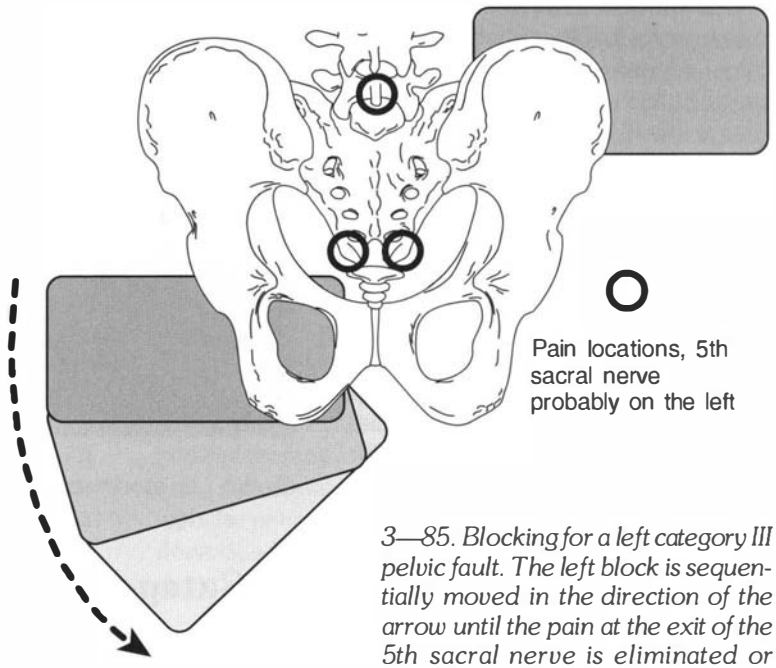
3—84. Lift ischium while simultaneously pressing on L5 spinous process to that side to challenge for a category III.

Block Adjusting Technique

Proper placement of the DeJarnette blocks is indicated by reduction of tenderness present in the area of the 5th sacral nerve and at the 5th lumbar spinous process. Locate the tenderness and observe the intensity. The tenderness at the sacral nerve is usually but not always on the positive ischial challenge side. Place one block 90° to the spine under the anterior iliac spine on the side of L5 challenge contact. Initial location of the block under the ischium is 90° to the spine. Evaluate the pain at the 5th sacral nerve and the lumbar spinous process. Progressively rotate the thick portion of the block under the ischium inferiorly until the pain is eliminated or is diminished to the greatest amount, indicating proper block placement that allows the pelvis to return to normal. Leave the patient on the blocks for a few minutes while correction takes place.

While the patient remains on the blocks, test for a sacral inspiration or expiration fault, which is a complication of a category III fault and often present. While the patient holds a deep inspiration, test the hamstring group on either side for weakening. Next, test the hamstring group while the patient holds a deep expiration. Weakening on inspiration indicates an expiration fault; weakening on expiration indicates an inspiration fault. Correct the sacral fault while the patient remains on the blocks (see Sacral Respiratory Function, page 403).

Following correction of the category III fault check for tenderness from C1 to T8; if present, apply pressure to the sacrotuberous and sacrospinous ligaments for spondylogenic reflex technique. While the pressure is applied, monitor the pain and vary the vector of force until the paraspinal pain is reduced (see page 129).



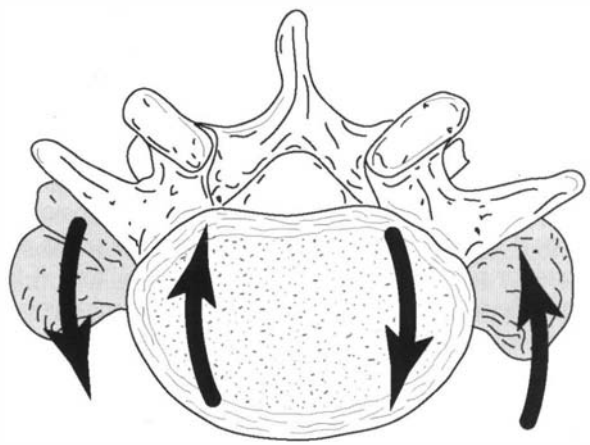
3—85. Blocking for a left category III pelvic fault. The left block is sequentially moved in the direction of the arrow until the pain at the exit of the 5th sacral nerve is eliminated or greatly reduced, which can be on either side.

PiLUS[47]

As the right leg moves forward during walking, the sacrum moves posteriorly about a vertical axis on that side. The 5th lumbar rotates anteriorly on the right leg forward side in counterrotation to the sacrum. The sacral and 5th lumbar motion is described by Illi[68] and confirmed by cadaver dissection research at the National College of Chiropractic. Illi states, "Later I discovered that if the vertebral column is bent forward, upon a fixed pelvis, the sacrum does not move, as one might expect, anteriorward, but it performs a rotation around its perpendicular axis. On each examined cadaver, the movement was asymmetrical and of variable extent. There was another surprising observation; the 5th lumbar vertebra deviated in the opposite direction from the torsion of the sacrum." He also developed special x-ray projections to view the sacral position and studied the spinal motion in gait.

Illi demonstrated that excessive tension is placed on the spinal cord with lumbar flexion if the lumbar vertebrae do not rotate in conjunction with flexion. This is depicted in his writings by illustrations showing an inflated finger cot placed under the cauda equina, with minimal pressure applied by the cauda equina. Flexion of the spine without allowing lumbar vertebrae rotation places pressure on the finger cot to separate it into two halves. The same amount of flexion with lumbar rotation places almost no traction on the cord, and minimal pressure on the finger cot.

With correction of the innominate, sacrum, and lumbar spine indicated by applied kinesiology examination, one often observes the elimination of standard or-



3—86. The sacrum rotates anteriorly as the right leg moves forward in gait. Simultaneously the 5th lumbar counterrotates posteriorly on the forward leg side.

thopedic signs such as Lindner's. In Lindner's sign for sciatica the patient sits with legs outstretched, and flexion of the head and neck causes pain in the leg(s) or lumbar region.[10] Often the sciatic radiation or other pain is eliminated when pelvic and spinal motion is corrected, as indicated by the PiLUS technique (pronounced "plus").

PiLUS Technique

Obvious pelvic and spinal problems should be corrected prior to applying the PiLUS examination technique. This method of examination is excellent for finding hidden problems and fine-tuning the system. The symptomatic pattern of PiLUS dysfunction can be almost anywhere in the body. Specifically there is most often neck pain and/or limited range of motion.

Movement of the normally functioning spine and pelvis is synchronized and predictable. As the right leg moves forward, the sacrum moves posteriorly and the 5th lumbar vertebra rotates anteriorly in counterrotation to the sacrum. In the normal spine and pelvis this action is started by facilitation of the piriformis on the left and inhibition on the right. Goodheart[47] observed this consistent facilitation and inhibition in the normally functioning person and recognized the potential of using the information to find the cause of dysfunction. In addition to the predictable function of the piriformis, there are other muscles with specific functions in conjunction with lumbar flexion. The muscles' initials are the derivation of the PiLUS acronym: Piriformis, iliacus, Latissimus dorsi, Upper trapezius, Sternocleidomastoid.

With normal function the previously strong right piriformis tests weak when the standing or sitting patient flexes the lumbar spine to approximately 35°. The exact amount of flexion is not specific. The inhibition may take place with as little as 20°, but one should make certain that the patient moves into full flexion. The same inhibition of the piriformis should be present with lumbar spine extension when the patient is sitting, standing, or prone. Furthermore, the right sternocleidomastoid and iliacus muscles also weaken, and the left latissimus dorsi and upper trapezius weaken with the flexion or extension. The iliacus is brought into this picture because of its partial origin from the sacrum ala.

The pattern of muscle weakness with lumbar flexion or extension is the same for right-handed and left-handed persons. Even in left-handed persons the left piriformis does not normally weaken with the flexion and extension.

A quick screening test indicating a need to examine further with PiLUS technique is to have the prone patient extend the lumbar spine by pushing on the examination table to lift his upper trunk from the table while the examiner tests the previously strong right piriformis for weakening.

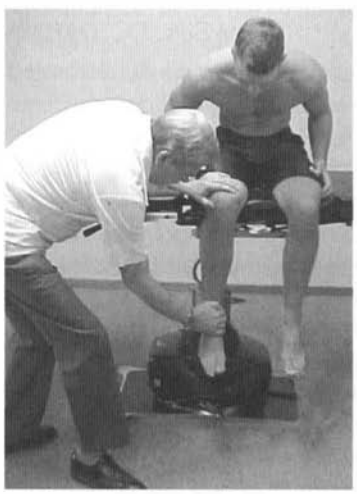
Failure of PiLUS pattern of weakness with lumbar flexion or extension indicates dysfunction of the following in a decreasing order of frequency:

All conditions should be examined non-weight bearing, sitting, and standing.

Category I
Category II
Category III
Sacral fixation/subluxation
Yaw #2 weight bearing
Iliacus malfunction, usually right sacral basic contact required
L5 fixation or subluxation
Occipitoatlantal fixation or subluxation
Upper cervical fixation or subluxation
1st rib fixation
Cervical compaction for imbalance of passive vs. active range of motion.
Foot and ankle involvement

When there is failure of proper inhibition of the PiLUS muscles, progressively therapy localize to the listed items until TL causes the muscle to weaken. Correct the factor at fault and re-test.

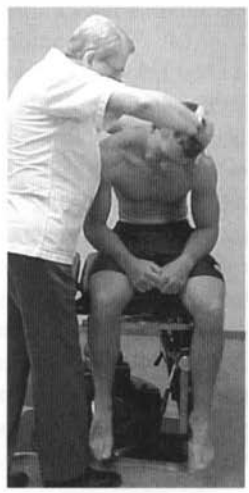
Piriformis weight-bearing tests can be done by the



3—87. Piriformis

3—88. Latissimus dorsi

3—89. Sternocleidomastoid



3—90. Upper trapezius

patient placing his hand on the examiner's shoulder for stabilization. All weight-bearing problems should be corrected in the position in which the positive test was found, if possible. A category I can be corrected with the patient standing against a hi-lo table. Position the blocks in the usual manner and have the patient hold them there.

When evaluation and correction of the items on the list fail to produce the proper muscle weakening with lumbar flexion, have the patient open his jaw with no therapy localization to the temporomandibular joint. If this activity weakens the right piriformis with lumbar flexion, the stomatognathic system is probably at fault. Usually the problem will be found in the muscles of mastication. First, evaluate for tenderness at the pterygoid pocket. Find the tenderest point of the superior, middle, or inferior portion of the pterygoid pocket and hold the tender area while tapping T2, 3, and 4 for about 30 seconds. Repeat on the other side. This treatment should reduce tenderness at the pterygoid pocket and correct the improper PiLUS function. (See page 416 for more information on this treatment.)

Using the quadriceps as an indicator muscle, have the patient open and close the jaw 10 times. Quadriceps weakening indicates positive reaction to muscles of mastication repeated muscle action. Usually the temporalis muscle is at fault. Bilaterally, treatment is origin/insertion technique to the posterior temporalis origin and insertion at the coronoid process.

The jaw or cranial treatment to correct improper PiLUS activity is typically only applicable when most PiLUS activity is correct and there is only involvement of the stemocleidomastoid or upper trapezius. If there is more involvement of PiLUS and walking gait activity, start examination and treatment at the sacrum.

In some cases PiLUS muscle weakening failure can be caused by ankle and foot dysfunction. With the patient in the flexed lumbar position, have him strongly supinate one or the other foot and test for appropriate PiLUS muscle weakening. If positive, the cause is usually the tibialis posterior, which needs percussion technique for myofascial gelosis (page 199). Examine and treat for foot and ankle dysfunction.

Lumbar Rotation with Flexion

When there is lumbar spine rotation failure with lumbar flexion, the rectus femoris or quadriceps group will test weak in the flexed lumbar position, but not when tested otherwise. This is most easily evaluated with the patient seated. Test either the rectus femoris or quadriceps group. If strong, have the patient flex forward about 30°; if the muscles weaken, evaluate thoroughly for lumbar subluxations or fixations. When corrected there will no longer be weakening on lumbar flexion. This is not part of the PiLUS technique, but it is another good method for finding hidden problems in the lumbar spine and correlates with Illi's findings, previously described.

Deep Tendon Reflex Examination

As applied kinesiology matures clinical observations are better understood, and the applicable neurology is being delineated. Standard neurologic tests can be expanded using applied kinesiology techniques, as is the case with the deep tendon reflex examination. Many applied kinesiologists in the ICAK-USA are expanding their education and becoming diplomates in neurology. The basis for AK is being expanded by them and others. The article below appeared in the *ICAK-USA News Update*, February, 1994 and is reproduced here with permission.

The Deep Tendon Reflex as a Functional Neurologic Analysis and Treatment Tool

Richard Belli, DC, DACNB

"The deep tendon reflex has been used for decades to test the integrity of afferent and efferent components of the peripheral nerves. But you will see that this simple test has much more to offer the functional neurologist than a test of a simple reflex loop. The test typically involves a fast stretch of the muscle spindle by an application of a reflex hammer to the associated tendon. This test involves both sensory and motor pathways integrated from the muscle spindle to the motor cortex and back to muscle. The standard application of this test determines the level of response of the reflex after the muscle spindle has been stretched, with the concern being whether it is either absent, hypo, normal, or hyperactive, and bilaterally even. These observations are simply noted with visual observation. However, by using manual muscle testing we can test the functional aspects of this beautifully integrated yet simple reflex.

"The mechanism behind the deep tendon reflex, which is more appropriately called the muscle stretch response (MSR), is thought by many experts to be the single most important reflex for human motion. The MSR in conjunction with the flexor reflex afferent system are imperative for normal posture, muscle tone, and smooth rhythmic movement. It is termed a monosynaptic proprioceptive reflex, however it is much more complicated than that. The reflex involves not more than one or two segments of the cord, but is modulated by descending motoneurons located in rostral portions of the central nervous system pathways such as the pyramidal, reticulospinal, and vestibulospinal tracts. The reflex is

modulated from the sensory aspect of the muscle spindle which transverses the cord as well as ascends the cord to supraspinal areas such as the cerebellum, reticular formation and cortex. When supraspinal modulation is lost, inhibition of the reflex is lost and the reflex becomes hyperactive. This is a classic sign of upper motor neuron lesion and emphasizes the importance of supraspinal modulation.

"Basically, the reflex works by depolarizing the annulospiral ending in the muscle spindle which depolarizes the Ia afferent from the muscle spindle. This neuron enters the dorsal horn and directly synapses on the alpha motor neuron of the muscle that was stretched. Collateral branches from the Ia afferent project to ascending pathways, inhibitory Ia interneurons of the antagonist muscle, and renshaw cells. Additionally, the Ia inhibitory interneuron, renshaw cells and muscle spindle are modulated from descending supraspinal pathways. When the muscle spindle is stretched there is a monosynaptic facilitation of the agonist and disynaptic post synaptic inhibition of the antagonist muscles. In addition to this there is involvement of the crossed extensor reflex. For example, if the right patellar ligament is struck to elicit the reflex, the agonist quadriceps will be facilitated, and the antagonist hamstring will be inhibited. Also the opposite quadriceps will be inhibited and the hamstring facilitated. The pectoralis contralateral to the facilitated quadriceps will be facilitated and the extensor or triceps will be inhibited. And finally, the ipsilateral pectoralis will be inhibited and the triceps facilitated.

"Keeping this in mind the doctor can elicit a reflex and use manual muscle testing to test all the muscles involved on all four limbs for appropriate facilitation and inhibition. In addition to the primary pathways, the pathways associated with inhibition of the inhibitors can be tested. An inhibited muscle in this mechanism should only be inhibited for one contraction, on the subsequent attempt at contraction the muscle initially inhibited by the test would be refacilitated. This is a function of the supraspinal pathways directing the inhibitory renshaw cells to inhibit the Ia inhibitory interneurons which will block the reciprocal inhibition of the initial muscle spindle stretch. If this does not take place, the interneurons that inhibit the inhibitory neurons are not doing their job, which can be a local dysfunction or supraspinal dysfunction.

"Correction simply entails using manipulation to restore normal motion to the segments in which therapy localization eliminates the aberrant response. This could involve any articulation in the skeletal system. The neurological implications can range from the cortex to the anterior horn and every synaptic area in between.

"There are also essential neurotransmitter components associated with this mechanism, with acetylcholine, GABA, and glycine being primary. If the aberrant reflex pattern returns it would be wise to check the cofactors for the production of these neurotransmitters, and the metabolization of norepinephrine, as it tends to inhibit acetylcholine and GABA.

"There is hardly a human function that does not involve a muscle action and associated reflexes. Manual muscle testing is therefore an invaluable tool in analyzing these responses. Our ability to test and correct these systems is limited only by our knowledge of the function of the human neuraxis."

The basic principles noted in the article can be expanded to other areas of the body and the cause of dysfunction located by AK techniques. Goodheart[49] added the inhibition of the ipsilateral sternocleidomastoid and contralateral upper trapezius to Belli's observations.

Deep Tendon Reflex

Quadriceps	Inhibits ipsilateral	Hamstrings
Quadriceps	Inhibits contralateral	Quadriceps
Quadriceps	Inhibits ipsilateral	Pectoralis sternal
Quadriceps	Facilitates ipsilateral	Quadriceps
Quadriceps	Inhibits contralateral	Sternocleidomastoid
Quadriceps	Inhibits ipsilateral	Upper trapezius

When inhibition is not correct the problem will frequently be found where the dura has attachment. When the patient's examination fails the DTR predictability test, retest the DTR while the patient therapy localizes to the following areas in descending order.

Occiput-upper cervical area
Cervical spine
Sacrum
Center of chest (rib pump)

For example, if there is predictable deep tendon reflex muscle inhibition with therapy localization to the occiput-upper cervical area, examine closely for involvement such as subluxations, fixations, and PRYT. There will be some type of dysfunction at the location of therapy localization that restores proper inhibition.

The sternum may be fixated at the manubriosternal and/or xiphisternal articulations. There will be positive therapy localization, and appropriate treatment is percussion with a percussion instrument or by tapping with fingertips. Gentle adjustment in the direction of positive challenge on the phase of respiration that abolished the positive challenge may be needed.

In the unusual case where nothing can be found with therapy localization that restores proper inhibition to the DTR, analyze the patient's health problem. There may be other problems with structure or in systems such as digestive or immune. Test with PiLUS and the walking gait configuration. Correct and re-test. If improper reaction to the DTR remains the patient still has some problem that needs correction.

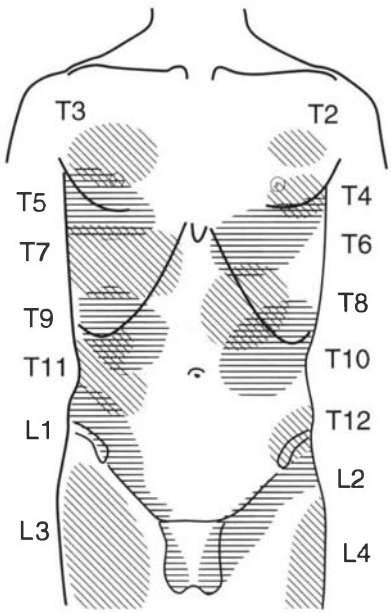
Spondylogenic Reflex

Referred pain from the vertebral complex soft tissues has been documented by many investigators.[17,33,60,69] The documentation of vertebral complex referred pain that is not due to radiculopathy, along with the neurologic work of Wyke[120,121,123] and Korr,[85] helps put the clinical reports of Dvorak and Dvorak[27] in perspective. They have organized the examination and treatment of referred dysfunction from the axial skeleton to the peripheral soft tissues. They report that Sutter, in his German writings, terms these interrelationships spondylogenic reflex syndromes (SRS). They go on to state, "Being mediated through the reflexogenic pathway of the CNS, the SRS is defined as the reproducible causative relationship between the reciprocal functionally abnormal position (segmental dysfunction) of skeletal parts of the axial skeleton and the local, anatomically determined noninflammatory rheumatic soft tissue changes. The term 'functionally abnormal position' (segmental dysfunction) is understood as a disturbance of the so-called inner function of the vertebral unit. The well-balanced interaction between the bony structures (skull, vertebrae, pelvis) and the corresponding muscle-tendon apparatus is impaired." Dvorak and Dvorak[27,28] and Schneider et al.[107] systematically describe the reflexes, their examination, and treatment. Their system is commendable because it includes in-depth palpation of the individual muscles, tender points, and structural manipulation. The discussion here deviates from their examination and treatment techniques. It will be the application of the Dvorak and Dvorak reflex associations by Goodheart,[46] using some applied kinesiology techniques of examination and treatment. First we will look at a few examples of how referred pain has been investigated.

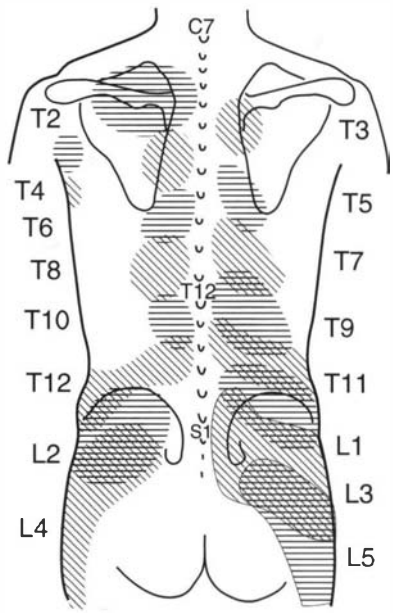
Feinstein et al.[33] irritated paravertebral muscles from the occiput to the sacrum by injecting a 60% saline solution that caused referred pain of a non-dermatomal distribution. The pain referral was consistent on an approximate segmental plan with considerable overlapping. The areas of pain referral are different from the conventional dermatomes. When the sympathetic nerves or plexus was blocked, the same referred pain was produced by the saline injection, thus suggesting a primarily spinal integrative mechanism. This indicates that peripheral nerve function played no part in this kind of referred pain. Reporting of the subjective pain was considered more reliable because the subjects were the medical doctor authors, seventy-five medical students, and three laboratory assistants who could provide better anatomical descriptions of their sensations than lay people.

Similar studies were done by Kellgren[82,83] in which interspinous ligaments were injected with 6% saline and the referred pain mapped. An overview of some of his finding is seen in figures 3—91 and 92.

Probably the most definitive study relating to cervical trauma was done by Cloward.[16,17,18] He stimulated the soft tissues, especially the intervertebral disc, while patients were alert under local anesthesia during cervical surgery, and mapped the referred pain from the patients' descriptions.



3—91. Anterior referred pain after Kellgren.[82,83]



3—92. Posterior referred pain after Kellgren.[82,83]

Examination

Evaluation for a spondylogenic reflex syndrome is done after spinal corrections, including pelvic and other conditions discussed in this chapter, are made. In addition the stomatognathic system should be evaluated and corrected if involved. Correction of these primary areas of dysfunction will often eliminate a spondylogenic reflex with greater efficiency in treatment. If a vertebral subluxation is corrected it may be associated with a spondylogenic reflex, but it will not correct the effects of the reflex. It is necessary to note the subluxation level because the information will be needed later in the correction of the spondylogenic reflex.

Indication of a probable spondylogenic reflex is soft tissue pain with altered soft tissue character. Altered tissue can be differentiated from surrounding normal tissue that blends smoothly from structure to structure. Familiarity with anatomy and skill in palpation are prerequisites. One must be familiar with the muscle patterns of the spondylogenic reflex syndrome, which comes with experience.

Dvorak and Dvorak[27] have clinically grouped muscles with spondylogenic reflex activity with a specific vertebral level illustrated in 3—94 to 3—119. In the applied kinesiology spondylogenic technique the major muscles of the spondylogenic group are the primary areas of examination. There will probably be other spondylogenic muscles or soft tissue of the vertebral level group involved, but usually not all of the muscles are involved. With experience one recognizes one or two major muscles for each vertebral level. Note that larger muscles such as the latissimus dorsi and gluteus maximus have muscle fiber portions that correlate with different vertebral level association.

Palpation of the soft tissue, usually a muscle, starts at the patient's area of pain. Often the pain is subjectively pointed out by the patient during discussion, or it may be elicited by palpation pressure during examination. The pain usually extends through the muscle from the origin to insertion.

When palpating for altered tissue character, stay at a specific tissue level. For example, the gluteus maximus is superficial to the gluteus medius, which is superficial to the gluteus minimus. Palpate one level at a time when there are overlying muscles. Test the suspected muscle, which should be strong in the clear. If not, examine and correct the cause of the weakness. When the muscle involvement is associated with a spondylogenic reflex it will weaken after a strong contraction in the manner of strain/counterstrain, which will usually be cleared by the spondylogenic reflex treatment technique. If the muscle weakens following a strong contraction, consult the Dvorak and Dvorak charts to find the vertebral association. Some of the larger muscles have strips that relate with several vertebral levels. For example, the gluteus maximus has muscle strips associated from L1 to S3. To determine the exact level, consider involvement of other muscles in the group and challenge and therapy localize for the associated vertebral subluxation.

The subluxation may be corrected now if it was not done before. Eliminating the subluxation will not clear the spondylogenic pain or the strain/counterstrain reaction. The identity of the subluxation level is important for the next step of correction.

The Lovett reactor vertebra is the key to eliminating the spondylogenic reflex. The Lovett reactor vertebra will have a hologramic or respiratory subluxation that should be examined for and corrected. There still will be no elimination of the reflex.

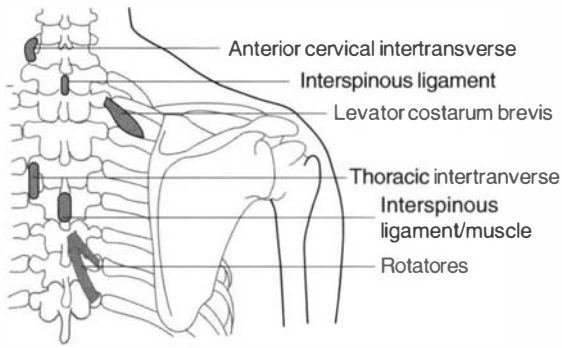
The reflex is eliminated by tapping on the Lovett reactor vertebra for about thirty seconds at a rate of one Hz. The tapping is accomplished by the physician's fingertip striking the spinous process sharply enough to bounce back, but not hard enough to cause pain. The pain and abnormal muscle characteristic will now be eliminated. If the active reflex (pain and abnormal muscle characteristic) returns on sitting, re-treat the Lovett reactor vertebra with the patient sitting.

Another method of stimulating the Lovett reactor vertebra is by a double transverse (mamillary or lamina) contact. Stimulate the contact points by light thrusting at one Hz. Do not use a high velocity thrust.

Spondylogenic Reflex Syndrome Patterns

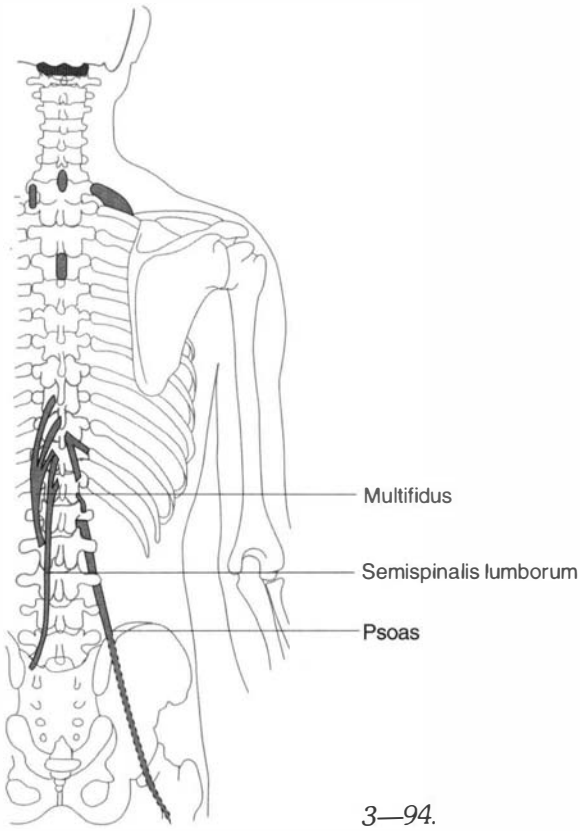
The following illustrations 3—94 to 3—119 are redrawn from *Manual Medicine, Diagnostics,* by Dvorak and Dvorak, ©Thieme-Stratton Inc. (New York, 1984) with permission.

The vertebral level for each spondylogenic reflex group is labeled, followed by the Lovett reactor vertebra in italics. Fibers of the major muscles are labeled in the illustrations. The smaller muscles are identified by their shape as identified in illustration 3—93.



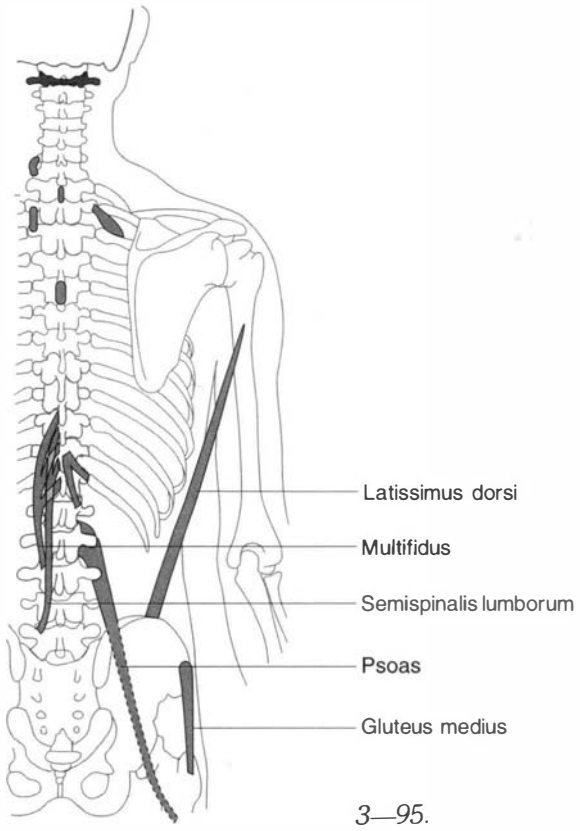
3—93.

Occiput — *Sacrum*



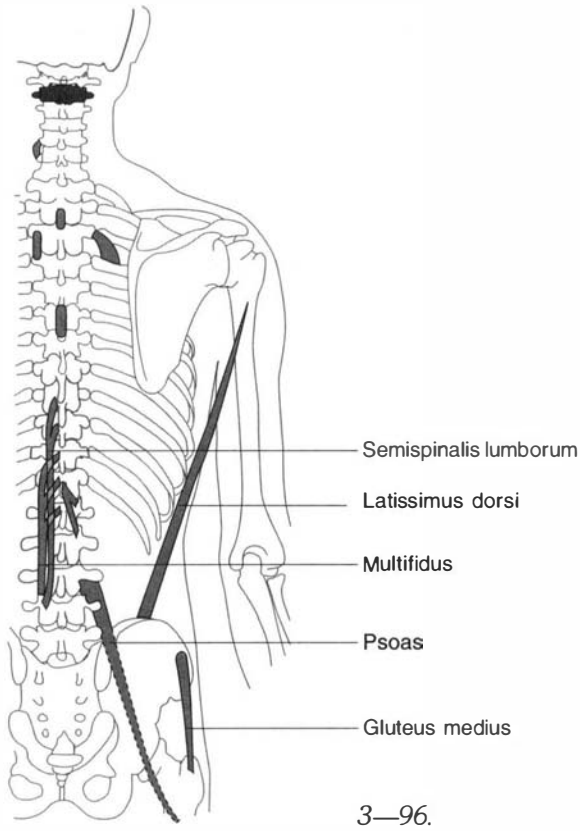
3—94.

C1 — *L5*



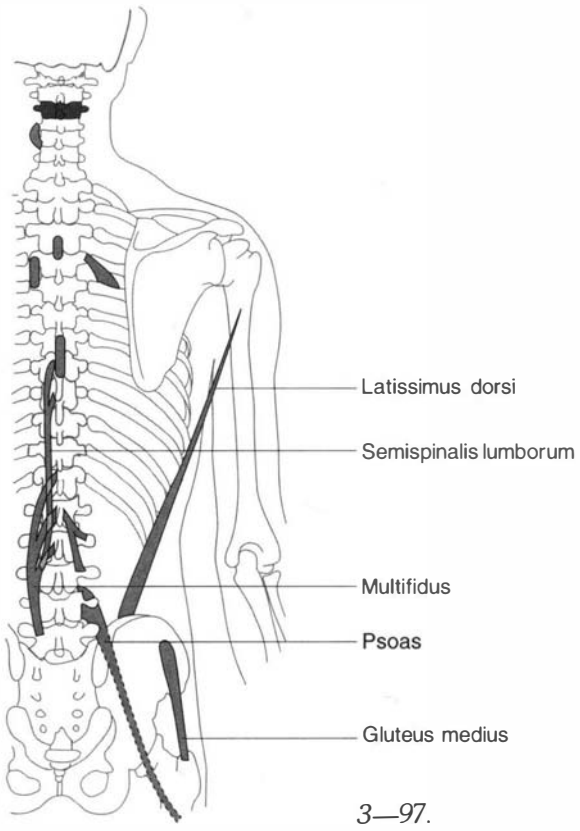
3—95.

C2 — *L4*



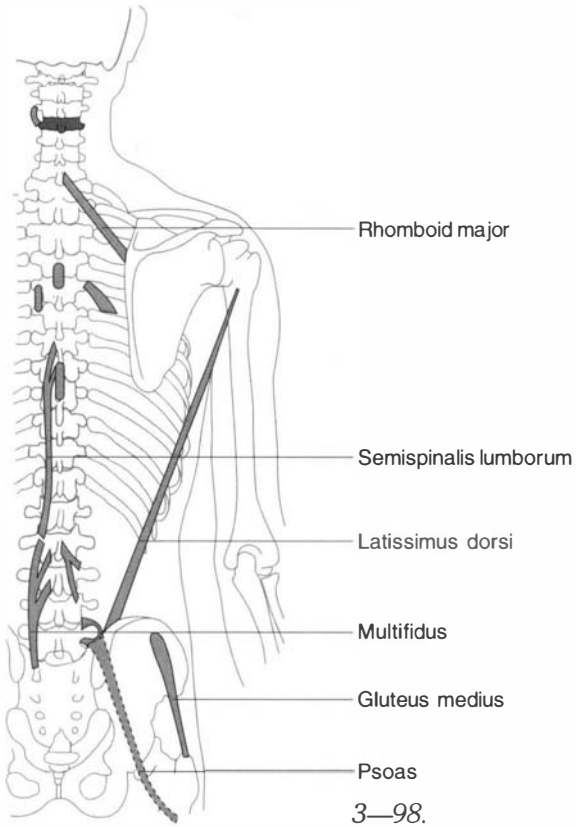
3—96.

C3 — *L3*



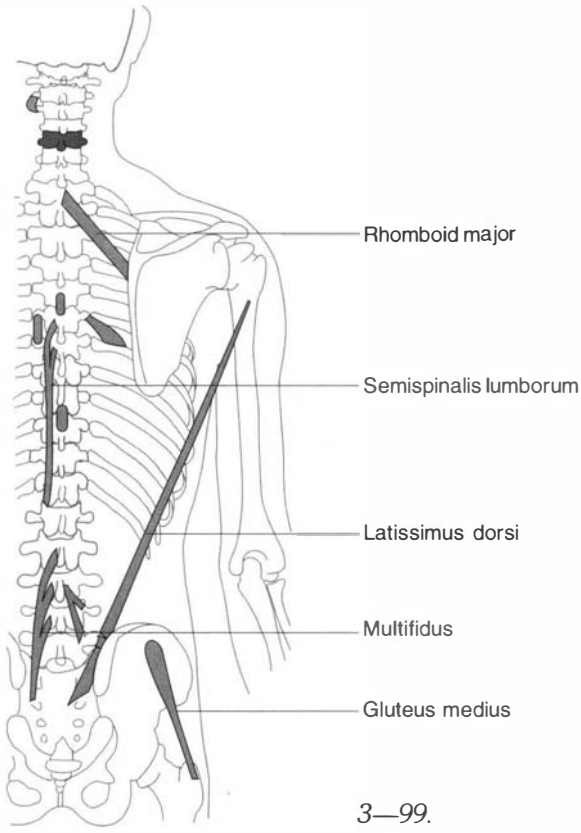
3—97.

C4 —*L2*



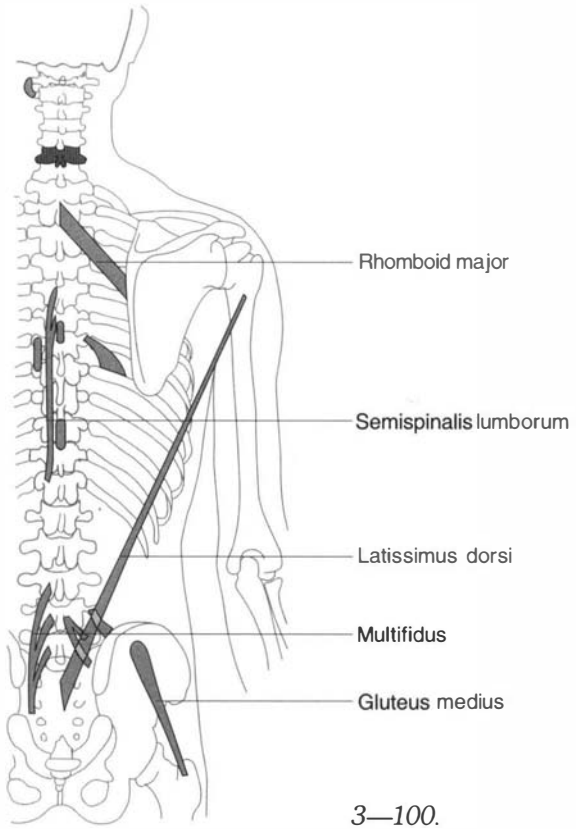
3—98.

C5 — *L1*



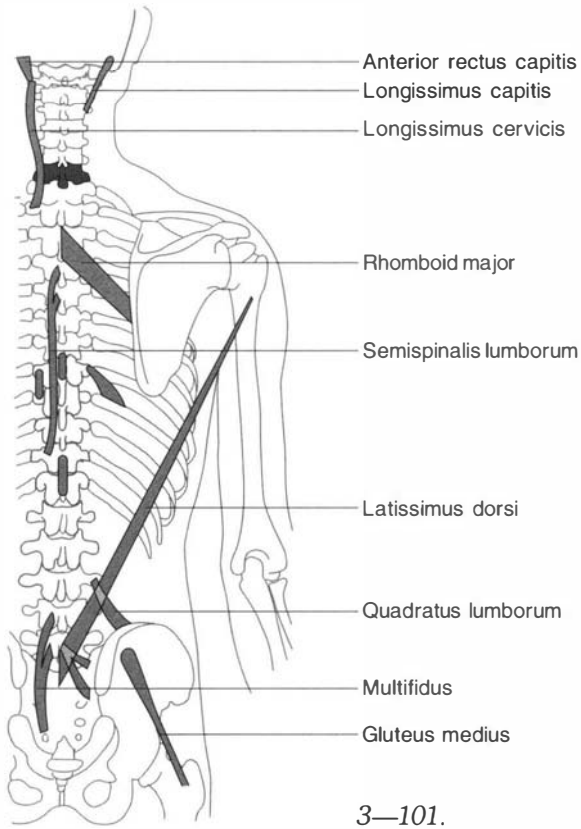
3—99.

C6 — *T12*



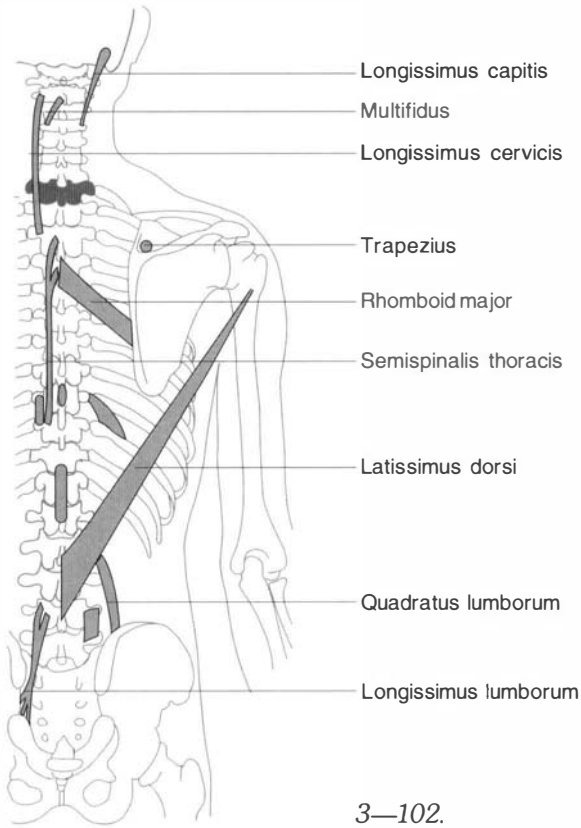
3—100.

C7 — *T11*



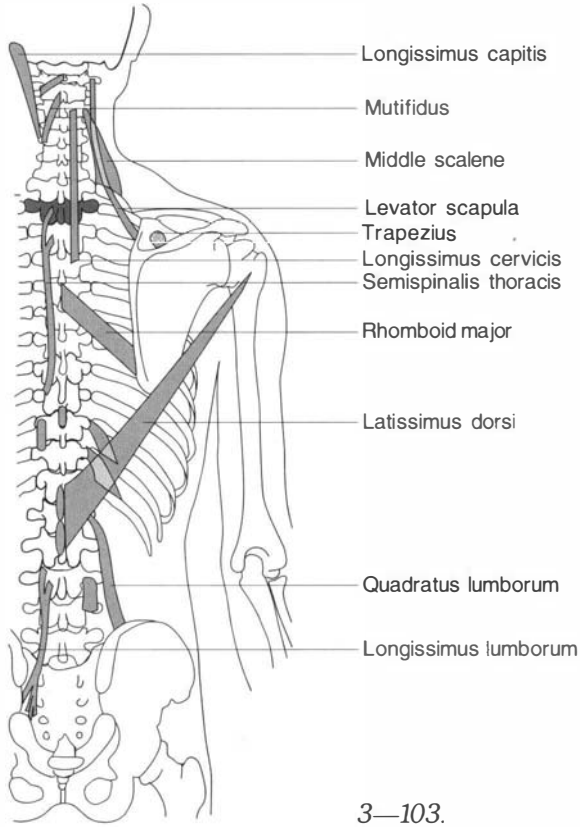
3—101.

T1 — *T10*



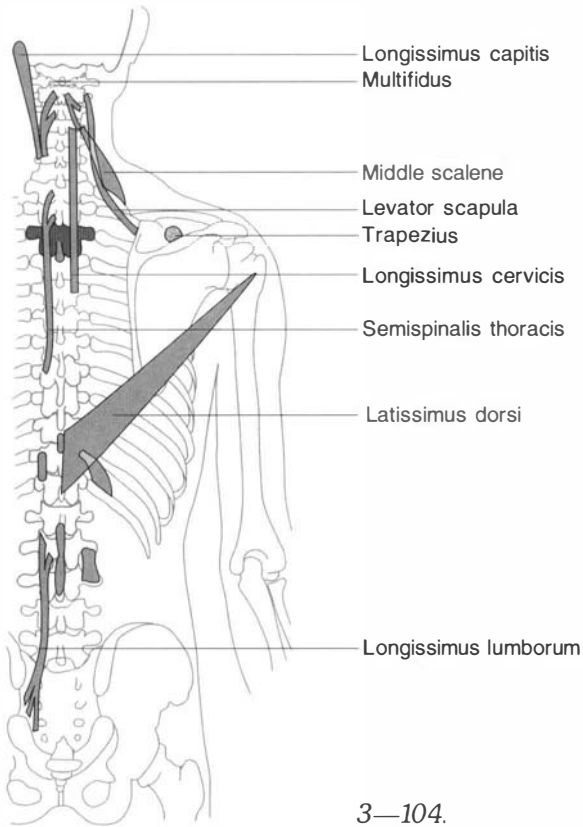
3—102.

T2 — *T9*



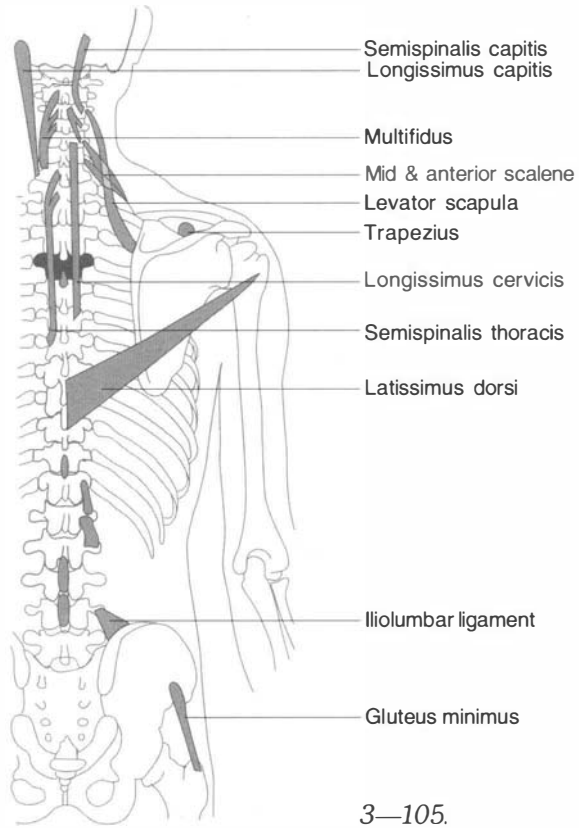
3—103.

T3 — *T8*



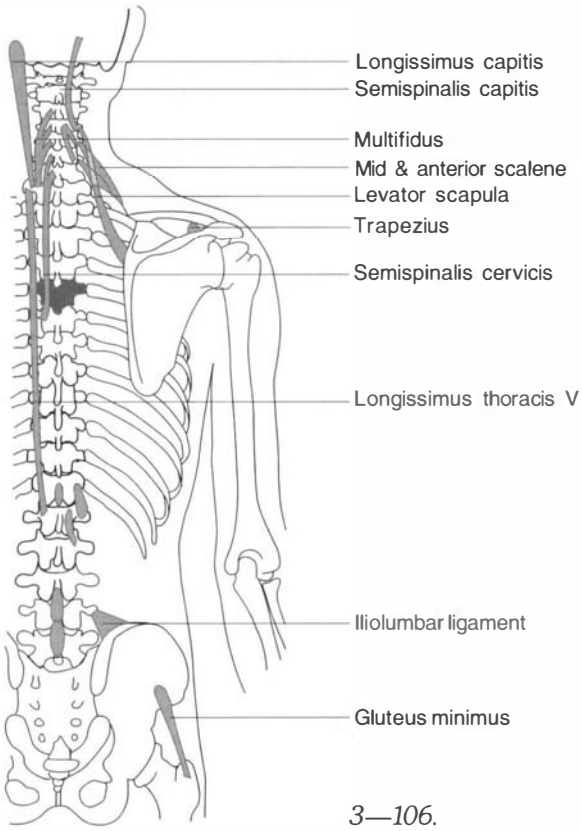
3—104.

T4 — *T7*



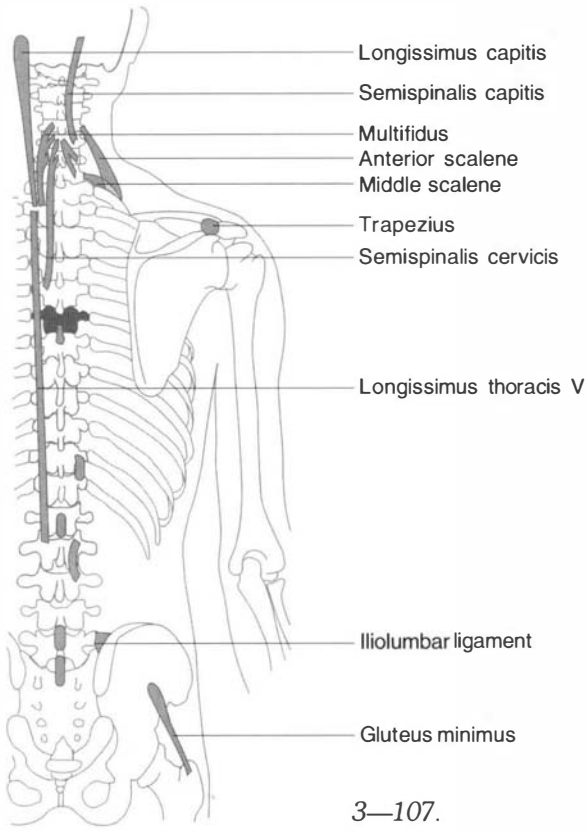
3—105.

T5 — *T6*



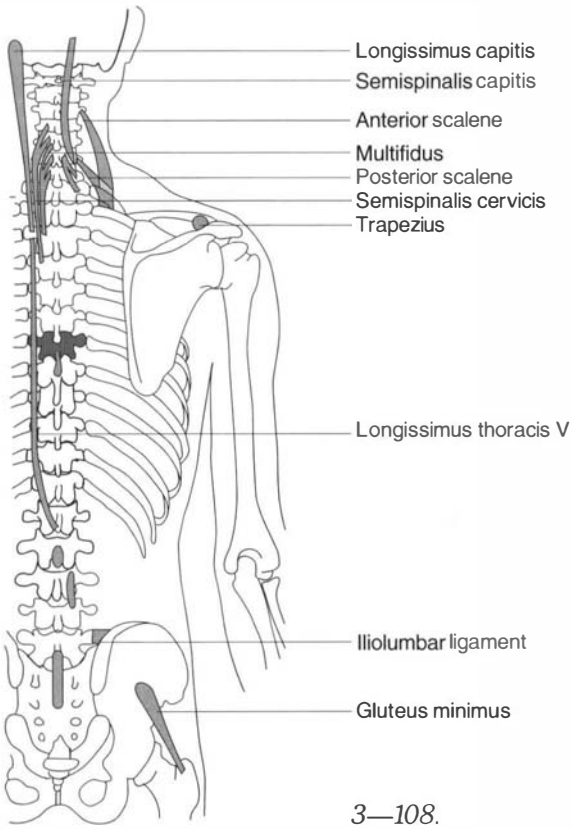
3—106.

T6 — *T5*



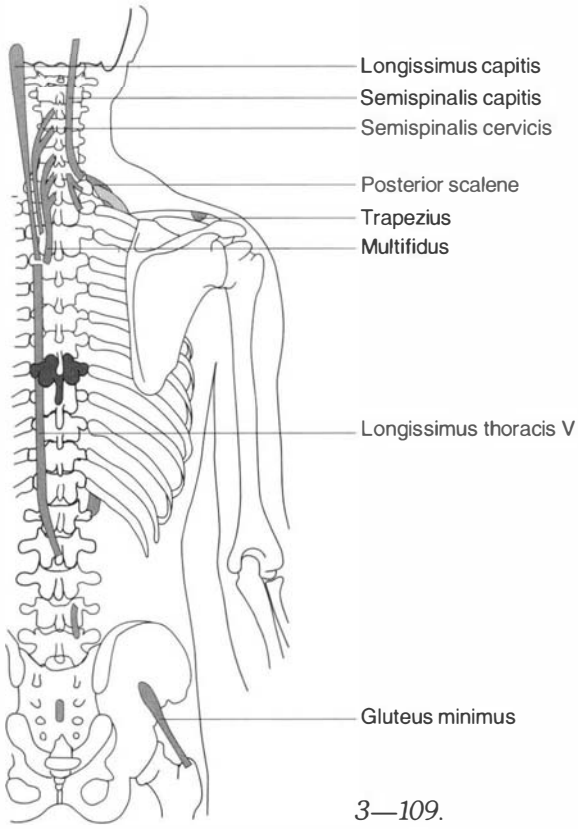
3—107.

T7 — *T4*



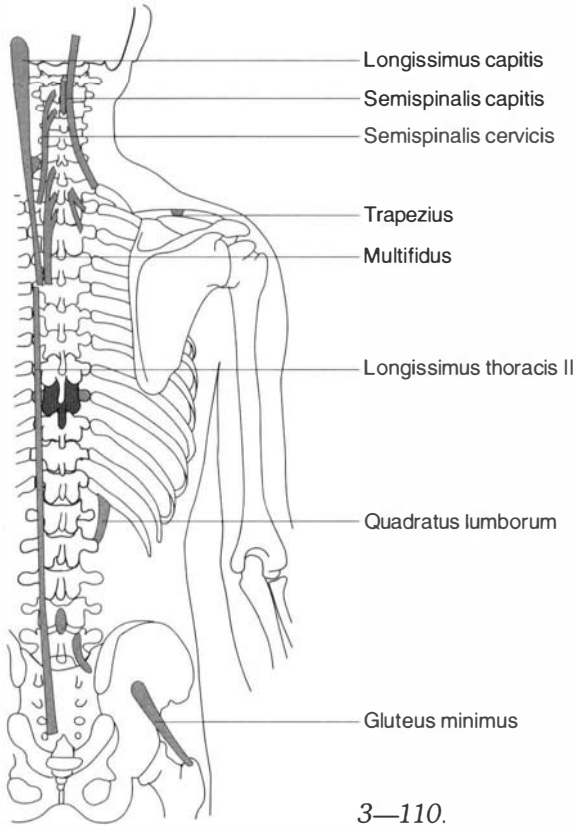
3—108.

T8 — *T3*



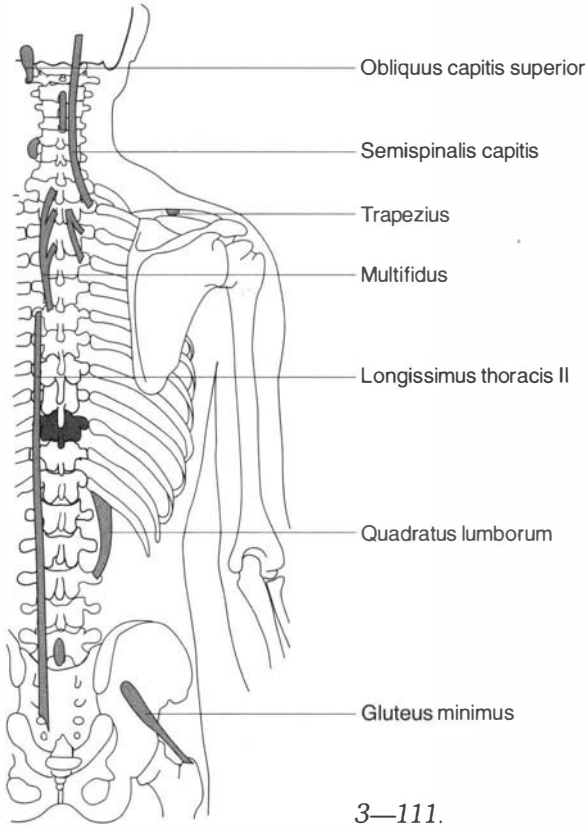
3—109.

T9 — *T2*



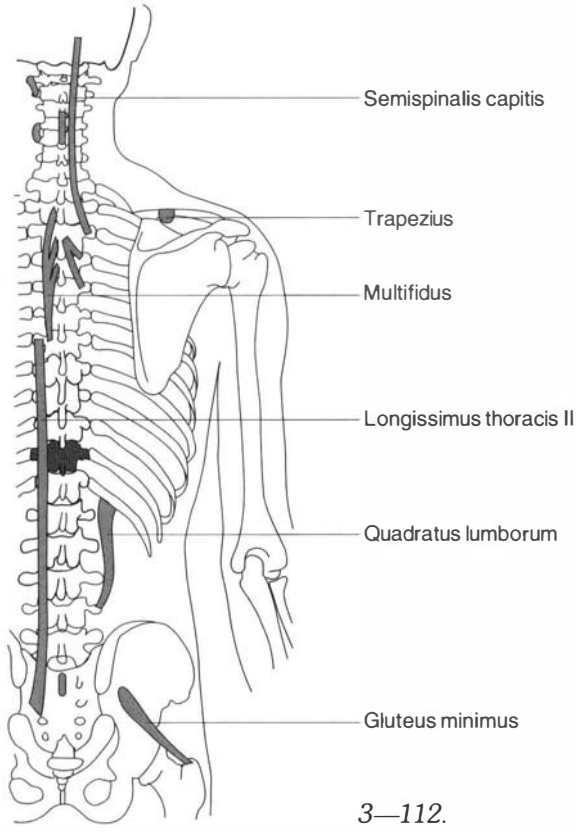
3—110.

T10 — *T1*



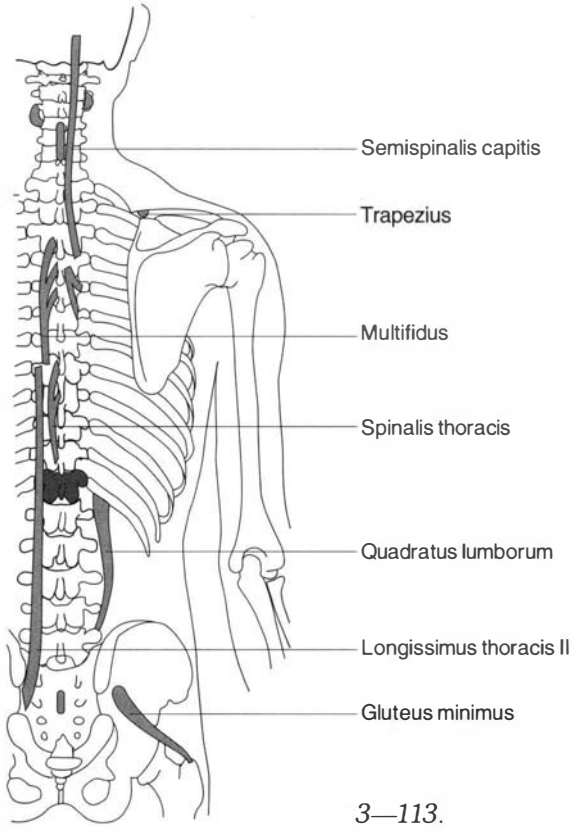
3—111.

T11 — *C7*



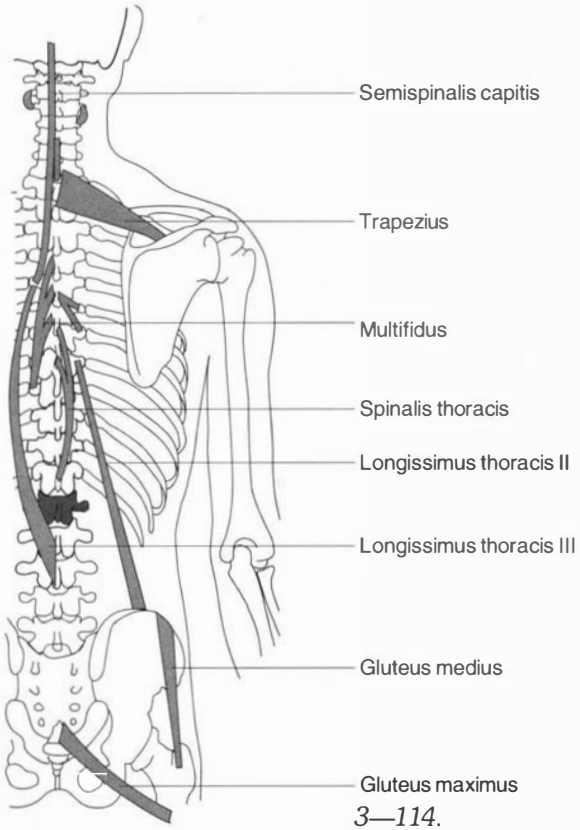
3—112.

T12 — *C6*



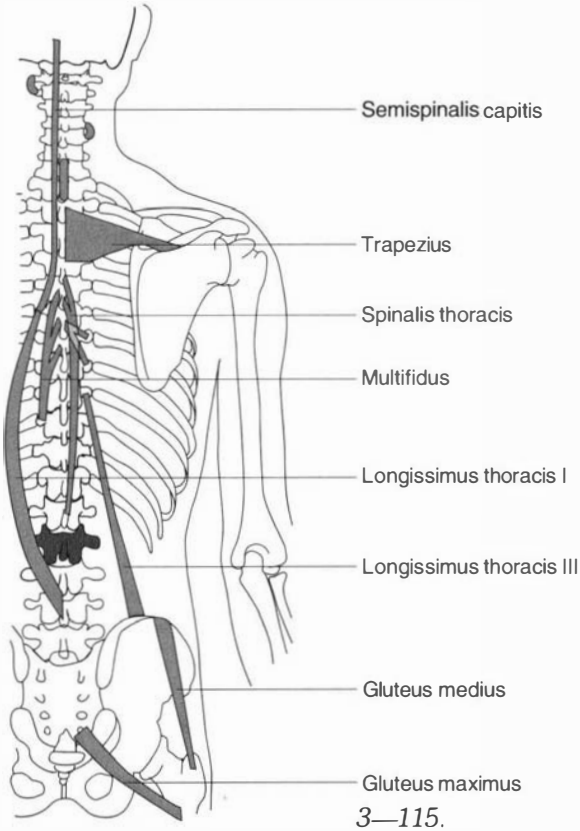
3—113.

L1 — *C5*



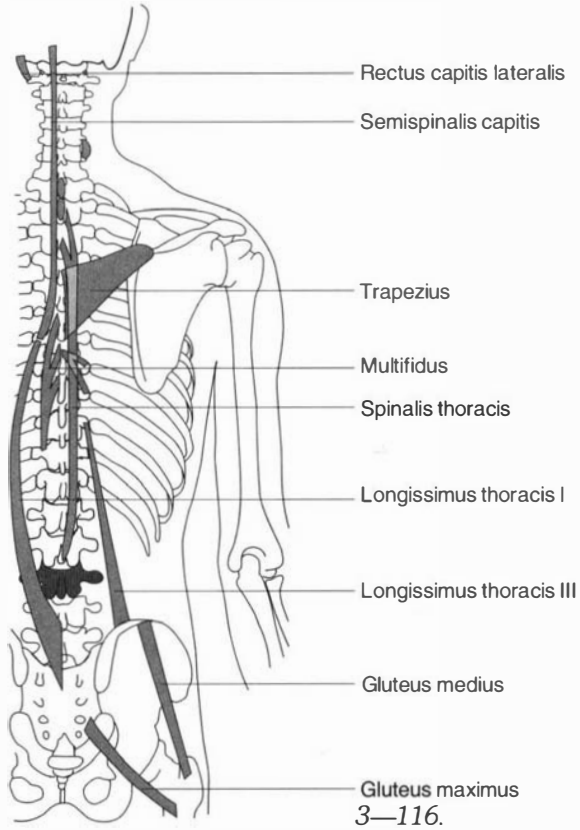
3—114.

L2 — *C4*



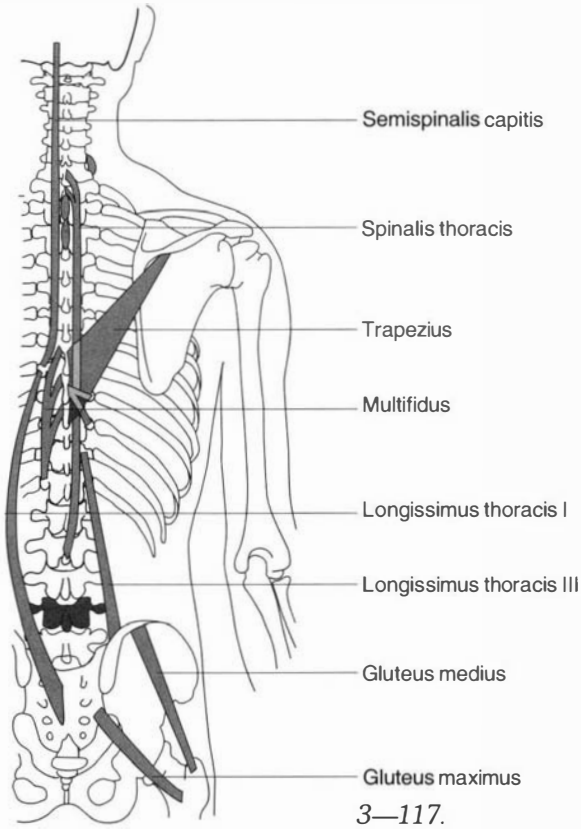
3—115.

L3 — *C3*



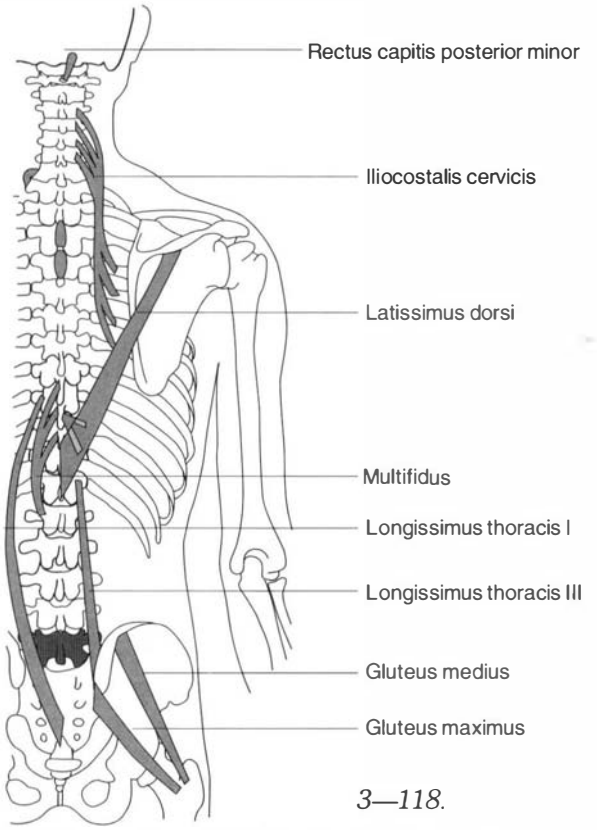
3—116.

L4 — *C2*



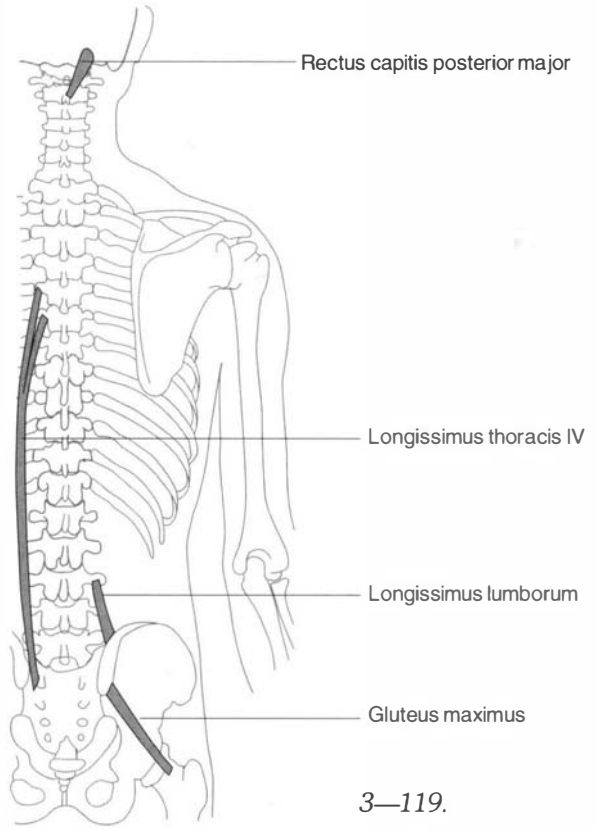
3—117.

L5 — *C1*



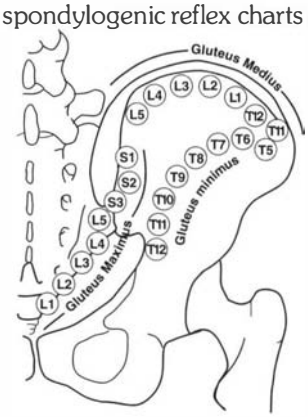
3—118.

Sacrum — *Occiput*

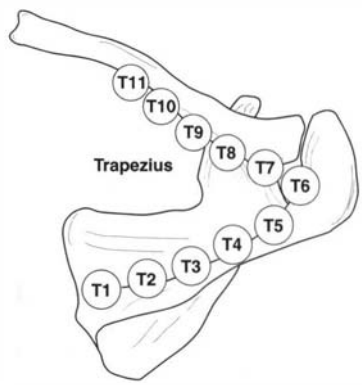


3—119.

The illustrations below give examples of a method to find active spondylogenic reflexes. Palpate along the origin and/or insertion of the muscle for tender or painful locations. Confirm the muscle's involvement by the muscle testing weak after maximum contraction in the manner of strain/counterstrain. Identify the muscle's location on the spondylogenic reflex charts and challenge to confirm a subluxation at that level. For example, if there is tenderness along the superior edge of the ilium go to illustration 3—115 and 3—116 to identify that the involvement is probably associated with L2 or L3; challenge for a regular or intraosseous subluxation. If not present, check the vertebrae above and below.



3—120. Gluteal muscles' tender or pain points.

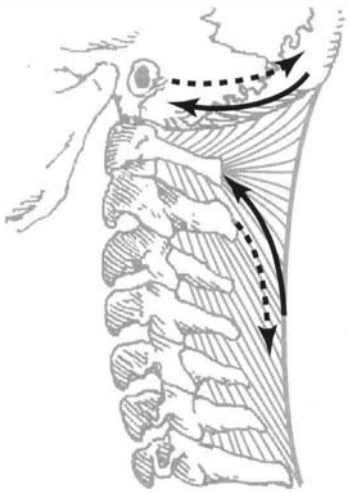


3—121. Trapezius tender or pain points.

Nuchal Ligament

The nuchal ligament is located in the midline from the external occipital protuberance down to, and between, all seven cervical vertebrae. It forms a septum between the muscles of the neck. The posterior superficial part directly connects the external occipital protuberance with C7. The fibers from the occiput to the atlas are angled anteriorly, while the fibers between the rest of the cervical vertebral spinous processes are angled posteriorly from the vertebra above to the one below. When the head is extended one would expect the spinous processes to uniformly approximate each other, and likewise with flexion to uniformly separate from each other. Surprisingly, in some areas of the cervical spine, in a high percentage of cases the motion is exactly opposite. Jirout[74,75,76,77,78,79] did

extensive x-ray analysis of the small muscles' and ligaments' influence on cervical movement. His opinion is that on head extension the base of the skull is levered anteriorly and the posterior portion of the nuchal ligament becomes relaxed, while the anterior portion is stretched and its cranial pull on the spinous processes causes ventral tilting of the lower cervical vertebrae. In head flexion the base of the skull is levered posteriorly, relaxing the anterior portion of the ligament below the axis. Owing to this relaxation the caudal traction of the ligamentous system of the upper thoracic spine is released and causes dorsal tilting, or at least a reduction of the ventral tilting of the lower cervical vertebrae.



3—122. Extension movement is in the direction of the solid arrows. After Jirout.[74]

Tension and pain in the nuchal ligament are indicators of an inferior sacrum.[42] The tension can often be seen on postural analysis. The patient may or may not complain of the tension and pain. The pain is easily elicited by palpation starting at the occiput and proceeding down to T7 close to midline. The sacrum will usually be inferior on the painful side; if it is bilateral the sacrum will probably be involved bilaterally.

Often the head is unlevel, with inferiority on the side of nuchal ligament tension and inferior sacrum. There may or may not be the usual neck flexor and extensor muscle imbalance associated with the unlevel head. If muscle imbalance is present, correcting it will usually not balance the head in the presence of sacral inferiority.

Test for an upper cervical fixation with the patient prone. If it is not present, test the bilateral gluteus maximus with the patient's head in flexion and extension. To test in flexion have the patient move up or to the side of the table so that his head hangs over the table edge. Upper cervical fixation can also be tested for by having the patient therapy localize to the upper cervical vertebrae and then turning his head from side to side; test a strong indicator muscle for weakening.

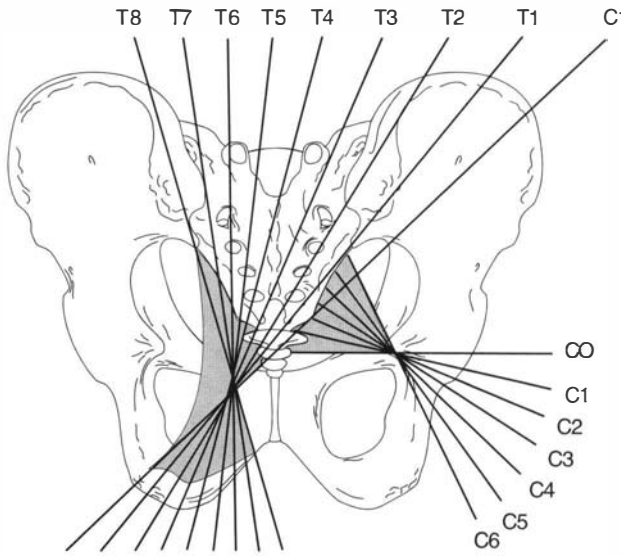
There will often be a respiratory pattern to the upper cervical fixation, i.e., the indication of fixation is removed on a deep inspiration or expiration held in. Usually the respiratory phase that eliminates the evidence of fixation is inspiration. Make the fixation correction while the patient holds that phase of respiration.

Check for and correct involvement with pitch, roll, yaw, and tilt (PRYT), and dysfunction in the stomatognathic system. Correct the inferior sacrum as discussed earlier in this chapter. The head should be level or greatly improved, and tension and pain in the nuchal ligament removed or greatly improved.

Sacrospinous and Sacrotuberous Ligaments

There are spondylogenic reflexes in the sacrospinous and sacrotuberous ligaments that reflex to the occiput to C6 and C7 to T8, respectively. Treatment may be needed following pelvic correction, such as an inferior sacrum or pelvic category. In the case of an inferior sacrum, involvement of the ligament(s) will usually be on the side of inferiority; in a category II they will be on the side of positive therapy localization.

The sacrotuberous and sacrospinous ligaments are treated by applying digital pressure. First palpate for the area of greatest paraspinal tenderness from C1 to T8. It is usually ipsilateral to the side of pelvic involvement, but may be bilateral. Apply about 10 pounds inferior to superior pressure to the sacrospinous and sacrotuberous ligaments where they cross, approximately halfway between the sacrum and innominate bones. Vary the vector of pressure while monitoring the paraspinal tenderness. There will be a dramatic reduction of tenderness when the proper vector of pressure is applied. The pressure on the ligaments stimulates the mechanoreceptors, producing the effect at C1 to T8.



3—123. Sacrotuberous and sacrospinous ligaments' spondylogenic reflexes. Redrawn from Manual Medicine, Diagnostics, *by Dvorak and Dvorak, ©Thieme-Stratton Inc. (New York, 1984) with permission.*

Iliolumbar Ligament

Illi[68] embarked upon his pelvic research to better understand the results of chiropractic manipulation of the pelvis, which were in direct conflict with the previously held belief that the sacroiliac articulation is immovable. Illi believed that there must be more to the sacroiliac than anterior and posterior ligaments, and that there must be a "...proprioceptive sensorial bed serving the sacroiliac joints designed to transmit the positional sensations from these articulations to the central nervous system; and second, the existence of an intra-articular ligament, whose major function is that of directing and limiting sacroiliac movement." At the National College of Chiropractic he dissected numerous sacroiliac articulations on cadavers and, with a lateral dissection approach that had not been done before, he discovered "Illi's ligament," an intra-articular sacroiliac ligament. Further study of the pelvis revealed "...compensatory, brief gyrating movement, obliquely up and down and concurrently anteriorly and posteriorly between the innominate bones, thus describing almost a horizontal figure eight." It is this movement and its control that allow proper compensation during walking. It has been proposed that the ligamentous structure has flexibility from its yellow elastic component and proprioceptive receptors to help organize the muscle function of gait.

There are numerous mechanisms involved in the organization of walking. Stimulation to proprioceptors in the sacroiliac articulations and their associated ligaments appears to be of great importance in this organization. Goodheart[39] applied the predictability of muscle action known to occur in gait in observing and developing what is known as the iliolumbar ligament technique. Discussed first is a brief review of expected shoulder, neck, and head muscle function in gait positions.

In normal function there is predictability of muscle facilitation and inhibition, which is readily demonstrated by manual muscle testing of gait muscles in simulated gait positions. Under normal conditions, the flexor and extensor muscles of the shoulder test strong when an individual is in a neutral standing position. Normally when a lower extremity is advanced and weight put onto it to simulate a gait position, the contralateral shoulder extensors — such as the latissimus dorsi — weaken, as do the ipsilateral shoulder flexors. The antagonists of both shoulders continue to test strong. Reversing the gait position reverses the muscle test findings of the shoulder flexors and extensors.

Disturbed function can be demonstrated by placing pencils under the 1st and 5th metatarsals of the leading foot. This simulates dropped metatarsals, with subsequent subluxations and improper stimulation of the joint and other proprioceptors. No longer will the shoulder flexors and extensors function in a predictable manner. The examiner will find that some of the muscles may continue to function as usual in the gait position, while others fail to be facilitated or inhibited. It is unknown exactly how muscles will perform when tested. What is known is that they will differ from normal gait activity. Slight movement of the pencils under the forefoot will usually change the results of shoulder muscle tests. (For a more detailed discussion of this demonstration see page 171.)

Another method of gait testing discussed on page 210 is the walking gait. It is a method of evaluating proper facilitation and inhibition of the sternocleidomastoid, upper trapezius, and deep cervical extensor muscles in gait function. Failure of proper reciprocal inhibition during walking puts excessive stress into the stomatognathic system.[117] When function of these muscles is unpredictable, the therapeutic method that usually obtains results is treatment to the muscle stress receptors.

Evaluating the predictability of muscle tests under simulated gait positions yields considerable information to the physician evaluating function of the joints, nervous system, and muscles. When muscle function lacks predictability in the simulated gait position, one will usually find the disturbance in foot subluxations or pelvic category dysfunction.

An evaluation of simulated gait position and predictability of muscle inhibition and facilitation is useful when torsion develops within the body during walking. This appears to relate with failure of the lumbo-sacro-iliac gyrating motion described by Illi.[68] Treatment is directed toward the complex on one side. It appears that the most significantly affected structure is the iliolumbar ligament. Improved function is probably the result of re-establishing normal proprioceptive function of the ligaments and joints. Because examination and treatment are directed in alignment with fibers of the iliolumbar ligament, the procedure is called the iliolumbar ligament technique.

Examination

Initial examination does not determine the side on which treatment will be necessary; rather, it indicates an involvement of the lumbo-sacro-iliac mechanism. Examination and treatment are done only after other factors — such as foot, general gait, pelvic, and other disturbances — have been examined for and corrected, if necessary.

The first portion of the examination consists of testing the individual in a neutral standing position. The latissimus dorsi is tested bilaterally to evaluate shoulder extension. The upper trapezius and deep cervical extensors are tested as a group bilaterally. The group test is most easily done by the physician placing his hand over the top of the patient's head, as indicated on page 210. All tests should be normal; if they are not, determine the reason and correct before proceeding. If

the muscles do not test strong in the clear, evaluate for the five factors of the IVF and weight-bearing problems.

Next the patient is placed in a simulated gait position by advancing one leg and putting weight on it. In this position the contralateral latissimus dorsi, ipsilateral upper trapezius, and deep cervical extensors should test weak; the ipsilateral latissimus dorsi, contralateral upper trapezius, and deep cervical extensors should test strong. This is normal gait function. Repeat the test by bringing the patient back to neutral stance and advancing the opposite leg into the simulated gait position. The test results should be reversed from the earlier gait test. If the muscles do not function in a predictable manner, there is some problem in the gait mechanism that does not relate to the iliolumbar ligament technique. Evaluate and correct, if necessary, the patient's feet, pelvis, and other gait factors discussed elsewhere.

When the above tests are normal, the patient can be further evaluated for need of the iliolumbar ligament technique. The patient is now tested in simulated gait positions similar to those mentioned; however, instead of having the patient take a step forward, he is asked to take a step backward with one leg so that the leading leg stays in the same position as it was in neutral stance. Goodheart[39] postulates that the lumbo-sacro-iliac mechanism should be flexible enough to perform the gyrating movement described by Illi,[68] whether the leading leg is advanced forward or the trailing leg moved backward. Under normal circumstances the latissimus dorsi, upper trapezius, and deep cervical extensors will test the same when a leg is advanced or moved backward to simulate gait. To repeat, then, if a leg is moved backward and weight put on the contralateral leg, the contralateral latissimus dorsi and ipsilateral upper trapezius and deep cervical extensors to the leading leg should test weak, with the opposite muscles strong.

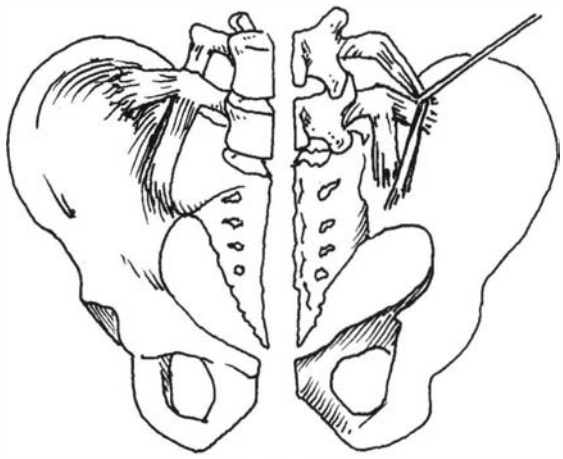
When tests are not predictable, as noted earlier, there is dysfunction, and further evaluation is done. With the patient maintaining the gait position, have him push anteriorly on the 5th lumbar transverse process on the side of the leading leg. If positive for iliolumbar ligament involvement, the latissimus dorsi contralateral to the leading leg and the ipsilateral upper trapezius and deep cervical extensors will now test weak. Reverse the procedure and test the contralateral latissimus dorsi, ipsilateral upper trapezius, and deep cervical extensors to the leading leg. If they test strong, evaluate by having the patient push anteriorly on the 5th lumbar transverse process on the side of the leading leg; the muscles should now test weak, confirming iliolumbar ligament involvement.

3—124. Gyrating counterrotation movement of the sacrum and 5th lumbar vertebra.

Apparently the fault is an inability of the lumbo-sacro-iliac mechanism to gyrate properly with gait, being limited by the ligamentous structure, primarily the iliolumbar ligament. The gait test, followed by pushing anteriorly on the 5th lumbar transverse process, is for screening only; it does not determine the side needing treatment. It is done to determine that the fault is really failure of proper gyration of the mechanism and not some other pelvic or lumbar fault. This is necessary because the test to determine side of involvement is not specific for iliolumbar ligament technique.

To test for side of involvement, the iliolumbar ligament and lumbo-sacro-iliac mechanism are challenged with the patient prone. The challenge is in the direction of iliolumbar ligament caudally directed fibers.

In major anatomy texts,[51,119] the iliolumbar ligament is described as being primarily anterior. From the 5th lumbar transverse process the more caudal bands run to the base of the sacrum, blending with the anterior sacroiliac ligament. The superior band attaches to the crest of the ilium and is continuous with the fascia. Steindler[109] shows a view of the anterior iliolumbar ligament with a large inferior division, which correlates with the more recent description of Dvorak and Dvorak.[27] They describe more attachments to the iliolumbar ligament, with an increase in magnitude of the posterior aspect. They attribute the origins to L4 and L5, which are independent of each other in their courses. Insertion is to the most medial portions of the iliac crest and the neighboring anterior and poste-



3—125. Iliolumbar ligament.

rior surfaces of the ilium.

Further testing is indicated when the step-back gait previously described is positive. With the patient prone, challenge by pressing the 5th lumbar transverse process and ilium together, contacting where the more vertical iliolumbar ligament fibers attach. A positive challenge is weakening of a previously strong indicator muscle when the contact points are pressed together to shorten the ligament. The positive challenge will be on one side only. One must recognize that challenge of this nature can be positive in the presence of a pelvic category disturbance or some other factor, which is the reason that the gait test described earlier is necessary to determine if there is an iliolumbar ligament involvement.

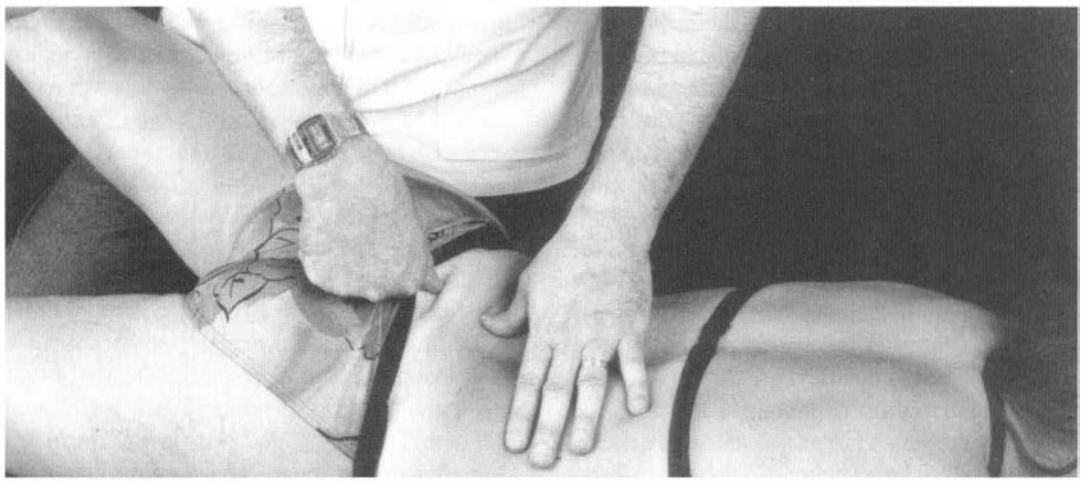
If the gait test is positive and there is no positive challenge to the iliolumbar ligament, consider the possibility of congenital difference of the iliolumbar ligament and apply different vectors of challenge. There will frequently be a positive strain/counterstrain test of the gluteus maximus on the side of positive iliolumbar ligament challenge.

The first phase of iliolumbar ligament treatment is to clear the strain/counterstrain of the gluteus maximus (page 201). This is done in the conventional manner and followed by treatment to the iliolumbar ligament. The patient remains prone, and the physician forcefully approximates the transverse process and ilium in the direction of positive challenge while maintaining hip hyperextension. It is usually easiest for the physician to place his knee upon the table, propping the patient's leg on it. Hold the leg in extension and maintain pressure to shorten the iliolumbar ligament for thirty to forty seconds. Re-challenge should be negative, as well as predictable weakness of the shoulder and neck extensors, whether the leading leg stepped forward or the trailing leg backward.

A major improvement from treating the iliolumbar ligament is that quite often the patient will no longer need to take a longer step to counteract the torsional effects of walking with an improper unilateral long stride. The patient should now test normal on blocking and other tests of dural tension (page 224).

Usually the treatment does not need to be repeated. If it does, raw bone concentrate is often appropriate nutritional therapy.

Numerous health problems benefit from correcting the torsional effects of walking, filum terminale tension, and iliolumbar ligament gait problems. One may see improvement in intermittent claudication, various joint and other structural problems, and recurrent stomatognathic system disturbances. Dysfunction of the external pterygoid muscle is a common recurring condition that may be eliminated by these techniques.



3—126. Hold approximating pressure on the ends of the iliolumbar ligament in the direction of the fibers while maintaining hip extension for thirty to forty seconds.

Sagittal Suture Tap Technique

Some of the techniques in applied kinesiology have developed as a result of serendipitous improvement in a patient's condition from the examination; such is the case with the sagittal suture tap technique. Kabat,[80] in his hidden cervical disc examination technique, attempts to simulate the forces of trauma that caused a patient's disc problem by application of force to the head in the direction trauma occurred. Goodheart[49] was using Kabat's technique of delivering a sharp tap to the top of the head to examine a patient's probable cervical spine trauma. Surprisingly, the patient remarked that following the tap he was able to straighten his leg, which he had not been

able to extend at the knee for fifteen years. This began the investigation of what effects tapping the top of the head might have throughout the body.

It is thought that there is a perfect holographic image of the body that the brain compares with the present condition. If, for example, there is a distortion of the pelvis it fails to match the perfect holographic image in the brain. It is believed that therapy localization brings these two images together for comparison, showing dysponesis that is perceived in examination by the test muscle weakening.

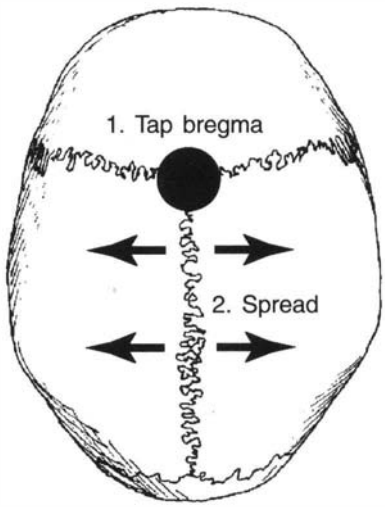
No matter how effective a correction might be, if it does not hold nothing has really been gained. Many techniques have been developed in applied kinesiology to determine the stability of corrections, such as having the patient walk after a pelvic correction is done and then retesting the patient for loss of correction. Testing the patient in supine, seated, and standing positions is another example of evaluating the stability of corrections. It is possible that there is an encoded memory of the patient's dysfunction, perhaps in the holographic pattern of the distortion (see also memory recall, page 187).

The sagittal suture tap is another method for determining the stability of a correction and, when the correction is lost, aiding in obtaining stability. The sagittal suture tap is thought to be a portal of entry into cortical and cerebellar memory and erasing encoded memory of the dysfunction.

Examination and Treatment

The sagittal suture tap technique is a simple examination and treatment procedure that is effective in eliminating recidivism. It is applicable to nearly all applied kinesiology techniques.

1. Make the usual correction indicated by positive applied kinesiology examination findings.
2. When the correction is effective, indicated by negative post-treatment examination, have the patient therapy localize the bregma and re-evaluate with the original examination technique. If the findings are again positive, sagittal suture tap technique is applicable to the patient for stabilization of this correction.
3. Tap the bregma vigorously four or five times, but not so severely that it hurts the patient. Follow by spreading the sagittal suture. After tapping the bregma and separating the suture, the positive examination findings should no longer be present.
4. Have the patient again therapy localize the bregma to determine that it no longer causes reproduction of the initial findings.
5. Each phase of correction must be accompanied by the sagittal suture tap technique.



3—127. First tap bregma four or five times and then spread the sagittal suture.

CHAPTER 3 — REFERENCES

1. Abrahams, V.C., "The physiology of neck muscles; their role in head movement and maintenance of posture," *Can J Physiol Pharmacol*, Vol 55, No 3 (Jun 1977).
2. Ask-Upmark, E., & E.R. Bickerstaff, "Vertebral artery occlusion and oral contraceptives," *Br Med J*, Vol 1 (Feb 28, 1976).
3. Bandy, J.V.N., "Cervical disc syndrome — A precise diagnostic and therapeutic procedure for correction of cervical disc lesions." Proceedings of Winter Meeting, ICAK, St Thomas, Virgin Islands, 1984.
4. Bandy, J.V.N., AK Extravaganza Seminar, Steamboat Springs, CO, Mar 1986.
5. Barlow, W., *The Alexander Technique* (New York: Alfred A. Knopf, 1977).
6. Bartelink, D.L., "The role of abdominal pressure in relieving the pressure on the lumbar intervertebral discs," *J Bone Joint Surg*, Vol 39B, No 4 (Nov 1957).
7. Basmajian, J.V., "Electromyography of iliopsoas," *Anat Rec*, Vol 132 (Oct 1958).
8. Beardall, A.G., *Clinical Kinesiology, Vol II: Pelvis and Thigh*, ed T.W. Brown (Lake Grove, OR: privately published, 1981).
9. Beardall, A.G., *Clinical Kinesiology, Vol V: Muscles of the Lower Extremities — Calf and Foot*, ed T.W. Brown (Lake Oswego, OR: privately published, 1985).
10. Blauvelt, C.T., & F.R.T. Nelson, *A Manual of Orthopaedic Terminology*, 3rd ed (St. Louis: The C.V. Mosby Co, 1985).
11. Bolton, P.S., "The somatosensory system of the neck and its effects on the central nervous system," *J Manip Physiol Ther*, Vol 21, No 8 (Oct 1998).
12. Bourdillon, J.F., *Spinal Manipulation* (New York: Appleton-Century-Crofts, 1970).
13. Breig, A., *Adverse Mechanical Tension in the Central Nervous System* (New York: John Wiley & Sons, 1978).
14. Burke, G.L., *Backache from Occiput to Coccyx* (Vancouver, B.C.: W.E.G. MacDonald, 1964).
15. Charnley, J., "Acute lumbago and sciatica," *Br Med J*, Vol 1 (Feb 5, 1955).
16. Cloward, R.B., "Cervical diskography — a contribution to the etiology and mechanism of neck, shoulder and arm pain," *Ann Surg*, Vol 150, No 6 (Dec 1959).
17. Cloward, R.B., "The clinical significance of the sinu-vertebral nerve of the cervical spine in relation to the cervical disk syndrome," *J Neurol Neurosurg Psychiatry*, Vol 23 (1960).

Chapter 3

18. Cloward, R.B., "Acute cervical spine injuries," *Clin Symp*, Vol 32, No 1 (1980).
19. Conable, K.M., "A biomechanical model of vertebral fixations," in *1984 Selected Papers of the International College of Applied Kinesiology*, ed W.H. Schmitt, Jr. (Park City, UT: ICAK, 1984).
20. Cox, J.M., *Low Back Pain — Mechanism, Diagnosis and Treatment*, 4th ed (Baltimore: Williams & Wilkins, 1985).
21. Dart, R.A., "Voluntary musculature in the human body — the double-spiral arrangement," *Brit J Phys Med*, Vol 13 (1950).
22. Dart, R.A., "The postural aspects of malocclusion (Part 2)," *Human Potential*, Vol 1, No 4 (1968).
23. Dart, R.A., "The postural aspects of malocclusion (Part 3)," *Human Potential*, Vol 2, No 1 (1969).
24. De Jarnette, M.B., *Sacro Occipital Technic*, 1981 (Nebraska City, NE: privately published, 1981).
25. Denslow, J.S., "An analysis of the variability of spinal reflex thresholds," *J Neurophysiol*, Vol 7 (Jul 1944).
26. Denslow, J.S., & G.H. Clough, "Reflex activity in the spinal extensors," *J Neurophysiol*, Vol 4 (1941).
27. Dvorak, J., & V. Dvorak, *Manual Medicine Diagnostics* (New York: Thieme-Stratton, Inc, 1984).
28. Dvorak, J., & V. Dvorak, *Manual Medicine Diagnostics*, 2nd rev ed, trans/ed by W.G. Gilliar & P.E. Greenman (New York: Thieme Med Pub, Inc, 1990).
29. Eie, N., & P. Wehn, "Measurements of the intra-abdominal pressure in relation to weight bearing of the lumbosacral spine," *J Oslo City Hosp*, Vol 12 (Oct 1962).
30. Engel, D.P., "Holographic technique: A testimonial." Proceedings of Summer Meeting, ICAK, Detroit, 1982.
31. Eyre, D.R., "Biochemistry of the intervertebral disc," in *International Review of Connective Tissue Research*, Vol 8, ed D. Hall & D.S. Jackson (New York: Academic Press, 1979).
32. Feder, K.S., "Flexion-traction technique." Proceedings of Summer Meeting, ICAK, Detroit, 1980.
33. Feinstein, B. et al., "Experiments on pain referred from deep somatic tissues," *J Bone Joint Surg*, Vol 36A, No 5 (Oct 1954).
34. Fisk, J.W., "Neck manipulation" (Letter to the Editor), *New Zealand Med J*, Vol 89 (Jan 24, 1979).
35. Gabor, D., "Twenty-five years of holography," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
36. Goodheart, G.J., Jr., *1972 Applied Kinesiology Workshop Manual* (Detroit: privately published, 1972).
37. Goodheart, G.J., Jr., "The cervical challenge," *Chiro Econ*, Vol 15, No 2 (Sep/Oct 1972).
38. Goodheart, G.J., Jr., "The fixation vertebral pattern," *Chiro Econ*, Vol 16, No 1 (Jul/Aug 1973).
39. Goodheart, G.J., Jr., *Applied Kinesiology 1975 Workshop Procedure Manual*, 11th ed (Detroit: privately published, 1975).
40. Goodheart, G.J., Jr., *Applied Kinesiology 1979 Workshop Procedure Manual*, 15th ed (Detroit: privately published, 1979).
41. Goodheart, G.J., Jr., *Applied Kinesiology 1982 Workshop Procedure Manual*, 18th ed (Detroit: privately published, 1982).
42. Goodheart, G.J., Jr., *Applied Kinesiology 1983 Workshop Procedure Manual*, 19th ed (Detroit: privately published, 1983).
43. Goodheart, G.J., Jr., *Applied Kinesiology 1984 Workshop Procedure Manual*, 20th ed (Detroit: privately published, 1984).
44. Goodheart, G.J., Jr., "The interosseous vertebral holographic subluxation, Part I," *Chiro Econ*, Vol 29, No 2 (Sep/Oct 1986).
45. Goodheart, G.J., Jr., "The interosseous vertebral holographic subluxation, Part II," *Chiro Econ*, Vol 29, No 3 (Nov/Dec 1986).
46. Goodheart, G.J., Jr., *Applied Kinesiology 1987 Workshop Procedure Manual* (Detroit: privately published, 1987).
47. Goodheart, G.J., Jr., *Applied Kinesiology 1988 Workshop Procedure Manual*, 24th ed (Detroit, MI: privately published, 1988).
48. Goodheart, G.J., Jr., *Applied Kinesiology 1992/93 Workshop Procedure Manual* (Grosse Pointe Woods: privately published, 1993).
49. Goodheart, G.J., Jr., *Applied Kinesiology 1994/95 Workshop Procedure Manual* (Grosse Pointe Woods: privately published, 1995).
50. Goodheart, G.J., Jr., *Applied Kinesiology 1997/1998 Workshop Procedural Manual* (Grosse Pointe Woods: privately published, 1998).
51. Gray, H., *Anatomy of the Human Body*, 29th Am ed, ed C.M. Goss (Philadelphia: Lea & Febiger, 1973).
52. Greguss, P., "Holographic concept in nature," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
53. Hack, G.D. et al., "Anatomic relation between the rectus capitis posterior minor muscle and the dura mater," *Spine*, Vol 20, No 23 (1995).
54. Hack, G.D. et al., "In response to Rutten et al., 'Anatomic relation....,'" *Spine*, Vol 22, No 8 (1997).
55. Hack, G.D., G. Dunn, & M.Y. Toh, "The anatomist's new tools," *1988 Medical and Health Annual*, *Encyclopaedia Britannica* (1988).
56. Haldeman, S., "Point of view," *Spine*, Vol 20, No 23 (1995).
57. Henderson, D.J., "Significance of vertebral dyskinesia in relation to the cervical syndrome," *J Manip Physiol Ther*, Vol 2, No 1 (Mar 1979).
58. Hendry, N.G.C., "The hydration of the nucleus pulposus and its relation to intervertebral disc derangement," *J Bone Joint Surg*, Vol 40B (1958).
59. Hildebrandt, R.W., "Vertebral artery syndrome resulting from manipulation of the cervical spine," *J Manip Physiol Ther*, Vol 2, No 2 (Jun 1979).
60. Hockaday, J.M., & C.W.M. Whitty, "Patterns of referred pain in the normal subject," *Brain*, Vol 90, Part 3 (Sep 1967).
61. Holmes, A., "Lumbar facet imbrication and correction," *Chiro Econ*, Vol 18, No 2 (Sep/Oct 1975).
62. Holmes, A.R., "An applied kinesiologist's look at the Cox technic and Chiromanis table." Proceedings of Summer Meeting, ICAK, Detroit, 1977.
63. Holmes, A.R., "Case history of a common problem of coexisting lumbar disc protrusion and facet imbrication producing low back pain and radicular inflammation." Proceedings of Summer Meeting, ICAK, Detroit, 1979.
64. Holmes, A.R., "Deimbrication technic update." Proceedings of Summer Meeting, ICAK, Detroit, 1981.
65. Hoppenfeld, S., *Physical Examination of the Spine and Extremities* (New York: Appleton-Century-Crofts, 1976).
66. Hoppenfeld, S., *Orthopaedic Neurology — A Diagnostic Guide to Neurologic Levels* (Philadelphia: J.B. Lippincott Co, 1977).
67. Houle, J.O.E., "Assessing hemodynamics of the vertebro-basilar complex through angiothlipsis," *JCCA* (Jun/Jul 1972).
68. Illi, F.W., *The Vertebral Column — Life-Line of the Body* (Chicago: National Coll of Chiro, 1951).
69. Inman, V.T., & J.B. Saunders, "Referred pain from skeletal structures," *J Nerv Ment Dis*, Vol 99 (1944).
70. Inman, V.T., H.J. Ralston, & F. Todd, *Human Walking* (Baltimore: Williams & Wilkins, 1981).
71. Jackson, R., *The Cervical Syndrome*, 3rd ed (Springfield, IL: Charles C. Thomas Publisher, 1966).
72. Jackson, R.T., & C.M. Epstein, "Effect of head extension on equilibrium in normal subjects," *Ann Otol Rhinol Laryngol*, Vol 100, No 1 (1991).
73. Jackson, R.T., & W.R. De l'Aune, "Head extension and age-dependent posturographic instability in normal subjects," *J Rehabil Res Develop*, Vol 33, No 1 (Feb 1996).
74. Jirout, J., "The dynamic dependence of the lower cervical vertebrae on the atlanto-occipital joints," *Neuroradiology*, Vol 7, No 4 (1974).
75. Jirout, M.J., "The dynamics of the craniocervical junction on lateral inclination of the head and neck," *Chiro Econ*, Vol 26, No 4 (Jan/Feb 1984).
76. Jirout, J., "Notes on x-ray research into joint play in the cervical spine" (Letter to the Editor), *Manual Med*, Vol 3, No 2 (1987).
77. Jirout, J., "Dynamic radiological studies of the spine and their role in prevention of spinal disorders," *Manual Med*, Vol 4, No 4 (1989).

78. Jirout, J., "Significance of the time factor in the dynamics of the cervical spine," *Manual Med*, Vol 6, No 2 (1991).
79. Jirout, J., "A new approach to testing of long-term resistance of the spine to mechanical stress," *J Manip Physiol Ther*, Vol 14, No 9 (Nov/Dec 1991).
80. Kabat, H., *Low Back and Leg Pain from Herniated Cervical Disc* (St. Louis: Warren H. Green, Inc, 1980).
81. Katz, M.M., A.R. Hargens, & S.R. Garfin, "Intervertebral disc nutrition — Diffusion versus convection," *Clin Orthop*, No 210 (Sep 1986).
82. Kellgren, J.H., "Observations on referred pain arising from muscle," *Clin Sci*, Vol 3 (1937).
83. Kellgren, J.H., "On the distribution of pain arising from deep somatic structures with charts of segmental pain areas," *Clin Sci*, Vol 4 (Jun 1939).
84. Kleynhans, A.M., "Complications of and contraindications to spinal manipulative therapy," in *Modern Developments in the Principles and Practice of Chiropractic*, ed S. Haldeman (New York: Appleton-Century-Crofts, 1980).
85. Korr, I.M., "Proprioceptors and somatic dysfunction," *JAOA*, Vol 74, No 7 (Mar 1975).
86. Macnab, I., "Disc degeneration and low back pain." Proceedings of the Royal College of Physicians and Surgeons of Canada, Report of Annual Meeting and Proceedings 3 and 4, Oct 1952. Reprinted in *Clin Orthop*, No 208 (Jul 1986).
87. Markham, B.C., "Cervical facet imbrication." Proceedings of Summer Meeting, ICAK, Detroit, 1983.
88. Martindale, R.E., "Trigger mechanisms of osteopathic spinal lesions," *Acad Applied Osteopath Yr Bk* (1956).
89. Mayo Clinic, Members of Neurology/Physiology sections, *Clinical Examinations in Neurology*, 2nd ed (Philadelphia: W.B. Saunders Co, 1963).
90. McCouch, G.P., I.D. Deering, & T.H. Ling, "Location of receptors for tonic neck reflexes," *J Neurophysiol*, Vol 14 (May 1951).
91. Mennell, J.McM., *Back Pain* (Boston: Little, Brown & Co, 1960).
92. Montgomery, R.L., *Head and Neck Anatomy with Clinical Correlations* (New York: McGraw-Hill Book Co, 1981).
93. Nachemson, A., "Electromyographic studies on the vertebral portion of the psoas muscle," *Acta Orthop Scand*, Vol 37 (1966).
94. Nachemson, A.L., "The lumbar spine — An orthopaedic challenge," *Spine*, Vol 1, No 1 (Mar 1976).
95. Nachemson, A., & J.M. Morris, "In vivo measurements of intradiscal pressure. Discometry, a method for the determination of pressure in the lower lumbar discs," *J Bone Joint Surg*, Vol 46A, No 5 (Jul 1964).
96. Nachemson, A.L., G.B.J. Andersson, & A.B. Schultz, "Valsalva maneuver biomechanics — Effects on lumbar trunk loads of elevated intraabdominal pressures," *Spine*, Vol 11, No 5 (Jun 1986).
97. Naylor, A., "Intervertebral disc prolapse and degeneration: The biomechanical and biophysical approach," *Spine*, Vol 1, No 2 (Jun 1976).
98. Obersteadt, H.L., "Study of specific muscles to determine disc lesion and correction," in *1987 Selected Papers of the International College of Applied Kinesiology*, ed D.H. Schusterman (Shawnee Mission, KS: ICAK, 1987).
99. Peterson, C.K., & T. Wei, "The use of the lateral bending sign to determine the level of disc herniation," *ACA Roentgenological Brief*, No 87/2.
100. Ravins, H.E., *Dental Orthopedics* (Beverly Hills, CA: Preventive Dental Res, 1981).
101. Rutten, H.P. et al., Letter to the Editor re: Hack et al.'s article, "Anatomic relation between rectus capitis posterior minor muscle and the dura mater," *Spine*, Vol 22, No 8 (1997).
102. Ryan, R.E., Sr., "A controlled study of the effect of oral contraceptives on migraine," *Headache*, Vol 7, No 6 (Jan 1978).
103. Schmitt, W.H., Jr., "An explanation of the muscle weakness patterns associated with vertebral fixations of the upper cervical and cervico-dorsal regions." Proceedings of Summer Meeting, ICAK, Gaylord, MI, 1975.
104. Schmitt, W.H., Jr., "The vertebral challenge," *Chiro Econ*, Vol 18, No 6 (May/Jun 1976).
105. Schmitt, W.H., Jr., "Vertebral fixations which mask other structural faults." Proceedings of Summer Meeting, ICAK, Detroit, 1976.
106. Schmitt, W.H., Jr., "A possible explanation of the mechanisms of iliac fixations and sacral fixations." Proceedings of Summer Meeting, ICAK, Detroit, 1978.
107. Schneider, W. et al., *Manual Medicine Therapy,* trans/ed W.G. Gilliar & P.E. Greenman (New York: Thieme Med Pub, Inc, 1988).
108. Shapiro, R., "The herniated intervertebral disk," in *Myelography*, ed R. Shapiro (Chicago: Year Book Med Publisher, Inc, 1975).
109. Steindler, A., *Kinesiology of the Human Body Under Normal and Pathological Conditions* (Springfield, IL: Charles C. Thomas Publishers, 1955).
110. Stroke, G.W., "Optical engineering," in *1981 Yearbook of Science and the Future*, ed D. Calhoun (Chicago: Encyclopaedia Britannica, 1980).
111. Turek, S.L., *Orthopaedics — Principles and Their Application*, Vol 1, 4th ed (Philadelphia: J.B. Lippincott Co, 1984).
112. Turek, S.L., *Orthopaedics — Principles and Their Application*, Vol 2, 4th ed (Philadelphia: J.B. Lippincott Co, 1984).
113. Unger, J.F., "The effects of a pelvic blocking procedure upon muscle strength: A pilot study," *Chiropractic Technique*, Vol 10, No 4 (Nov 1998).
114. Vanezis, P., "Vertebral artery injuries in road traffic accidents: A post-mortem study," *J Forensic Sci Soc*, Vol 26 (1986).
115. Walther, D.S., *Applied Kinesiology — The Advanced Approach in Chiropractic* (Pueblo, CO: Systems DC, 1976).
116. Walther, D.S., *Applied Kinesiology, Volume I — Basic Procedures and Muscle Testing* (Pueblo, CO: Systems DC, 1981).
117. Walther, D.S., *Applied Kinesiology, Volume II — Head, Neck, and Jaw Pain and Dysfunction — The Stomatognathic System* (Pueblo, CO: Systems DC, 1983).
118. White, A.A., III, & M. Panjabi, *Clinical Biomechanics of the Spine* (Philadelphia: J.B. Lippincott Co, 1978).
119. Williams, P.L., & R. Warwick, eds, *Gray's Anatomy*, 36th Br ed (Philadelphia: W.B. Saunders Co, 1980).
120. Wyke, B., "The neurological basis of thoracic spinal pain," *Rheumatol Phys Med*, Vol 10, No 7 (Aug 1970).
121. Wyke, B., "Morphological and functional features of the innervation of the costovertebral joints," *Folia Morphologica*, Vol XXIII, No 4 (1975).
122. Wyke, B., "Cervical articular contributions to posture and gait: Their relation to senile disequilibrium," *Age and Ageing*, Vol 8, No 4 (1979).
123. Wyke, B.D., "Neurology of the cervical spinal joints," *Physiotherapy*, Vol 65, No 3 (Mar 1979).

THIS PAGE INTENTIONALLY LEFT BLANK

Chapter 4

Nutrition

Chapter 4

Introduction

A system for evaluating nutritional effects on body function has been developed in applied kinesiology by Goodheart.[58,59] The system appears to provide additional information about how nutrition, or possibly adverse substances, may work with or affect body function. It is designed for use in conjunction with the physician's general knowledge of nutrition, and laboratory and physical diagnostic findings. It is important that the examiner using this technique be thoroughly knowledgeable about the usual methods for determining nutritional needs.

Applied kinesiology nutritional testing appears to reflect the nervous system's efferent response to the stimulation of the gustatory and olfactory nerve receptors by various substances. The nerve pathways causing change in muscle function as observed by manual testing are unclear; however, there is considerable evidence in the literature of extensive efferent function throughout the body from stimulation of the gustatory and olfactory receptors. There is also evidence of afferent modification of gustatory sensitivity and central nervous system interpretation of gustatory impulses ultimately modifying functional change as a result of oral stimulation. Applied kinesiology nutritional testing enables a physician to give individual consideration to each patient's nutritional needs. To properly apply this method of testing, one should be thoroughly familiar with the nervous system's role in nutrition and proficient in manual muscle testing.

As one begins a serious study of nutrition outside applied kinesiology, it is easy to become very confused. Consulting ten different authorities on specific questions about nutrition may provide seven, eight, or even ten diverse philosophies leading to different therapeutic approaches, or perhaps to no treatment at all. Extremes range from the belief that if a person eats balanced meals no nutritional supplements are ever needed to recommendations of so many high-potency supplements that one would hardly need eat regular food, except for fiber. Philosophies about nutrition vary from mega-dosages of numerous supplements to low-potency products of natural origin only. Some recommend that specific nutritional products routinely be given with certain medications to offset any side effects[148]; others recommend no medication, with nutrition taking its place; still others recommend minuscule dosages of homeopathic remedies for the treatment of disease. There are vitamins, minerals, isolation of natural food products (such as the essential fatty acids), herbs to treat almost any condition, and Bach flower remedies for mental and emotional conditions.[9,25,173] Why are there so many philosophies with different treatment approaches to something as essential to our life as the food we eat? The answer cannot be put into a simple statement. There are many reasons for the confusion that reigns among nutritional authorities. Paramount among these is that all nutritional factors have not even been discovered yet, and it is not known exactly how the body uses many nutritional products. As we continue with our discussion, keep in mind that the first description of treating scurvy with ascorbic acid was done by Parsons[132] only six decades ago.

The applied kinesiology method of evaluating changes in body function as the result of nutritional stimulation fills a specific void in the conundrum of nutritional diagnosis. The method consists of stimulating the gustatory nerve receptors by having the patient chew or inhale[23] the substance to be evaluated, and then manually testing a muscle for change. The muscle may appear stronger or weaker, depending upon the type of evaluation being made and the muscle's neurologic association with the substance stimulating the gustatory receptors. This system of testing is controversial. One reason for this is that there have been many modifications of Goodheart's original description. Rather than have the patient stimulate the gustatory receptors with the substance being tested, some physicians and lay people have the individual hand-hold the substance or lay it on the belly; some even have the patient hold a bottle containing the substance to be tested.[11,12,44,135,136] These modified systems are frequently taught to lay people who often do not have the anatomical knowledge necessary for accurate muscle testing, nor do they have a nutritional background or general diagnostic ability.[11,12,44,162,169]

The testing of nutrition as advocated by the International College of Applied Kinesiology is a discipline limited to the tested substance stimulating the gustatory or olfactory nerve receptors, combined with accurate and specific muscle testing. The information derived from these tests must then be correlated with a standard diagnostic work-up by a person licensed in the healing arts to be a primary health care provider. The approach discussed in this text is designed to be an adjunct to standard nutritional evaluation, not to take the place of it. Those who have the expertise to properly test nutrition, as described by the ICAK, should not use this method as a sole approach in evaluating nutrition and/or substances harmful to the body.

This writer believes that the modified testing procedures for nutrition — including hand-held, laying it on the skin, touching various areas of the skin, and teaching the material to lay people — are potentially detrimental to the health of the subject being evaluated and should be eliminated. The abuses of manual muscle testing in evaluating nutrition have sunk so low that this writer once heard a woman at a nearby table in a restaurant say, "If you don't believe me, let me show you."

She then had a man at the table stand up and hold a sugar dispenser in his hand. She proceeded to have him hold his other arm out and attempted to pull it down. He was a strong individual, and she almost lifted herself off the floor before she was able to pull his arm down. Then came the statement, "See — I told you it would make you weak."

Applied kinesiology nutritional evaluation is indeed a revolutionary method of determining nutritional needs. In my thirty-nine years of practice, I have seen many changes take place in what is considered the routine nutritional approach for health problems. Thirty-five years ago I was increasing fiber content in the diet of patients with colon disturbances. On three different occasions when I took patients off the bland, refined-sugar diet prescribed by their allopaths, I was called a stupid quack and accused of endangering the lives of my patients. Fortunately, the patients continued my therapeutic approach and had uneventful recoveries in spite of the diatribe against my procedure and me. Today, even television commercials and the National Cancer Institute[181] emphasize the need for fiber in the diet. Those same doctors who called me a quack now use the approach I used thirty-five years ago. Now the pendulum may be swinging too far to include too much fiber in some cases.

There are many reasons that nutritional needs should be evaluated on an individual basis. Applied kinesiology adds to the physician's nutritional knowledge the ability to determine, to a certain extent, the effects of various nutritional products on the specific individual being considered. Furthermore, it enables one to evaluate the difference between nutritional products that may appear to be the same according to the product's label description, but act differently from individual to individual.

People are different. Everyone does not require the same nutritional program regarding the food eaten and possible supplementation taken. Williams[178] points out that researchers must begin considering biochemical individuality in the study of nutrition. Individuality may have a genetic basis with different body composition, enzymatic patterns, and endocrine balance. Williams presents a hypothetical group of ten men (group 1), all of average height, with the same foot size. They have the same amount of hair on their heads, and an average tendency to put on body fat. They consume the same amount of alcoholic beverages, have the same sex urges, and their digestive tracts react the same to food. They all have natural teeth, without cavities or plaque buildup. Finally, they all have the same emotional reactions to the same daily stresses.

Contrast this group with another hypothetical population of ten men (group 2). In this group is one man who has lost all his hair. Another seems to gain weight just by thinking about food. Another has long, narrow feet and fingers. One supervises 100 men on a production line, with a very tight productivity schedule to meet. Another has no sex drive, and still another is a salesman with his two-martini lunch schedule.

It seems that the minimum and maximum daily requirement of nutritional complexes can be easily figured for group 1, but what are the needs for group 2? If there is considerable individuality among people, how has man survived for so long before there was any study of his nutritional needs? Is there an innate self-selection of the food needed by the body? Williams points out the body's wisdom with the illustration of an individual who has no knowledge about nutrition and little or no tendency to gain weight. If, during a ten-year period, he gains five pounds, his self-selection of food has regulated his caloric intake to a minimum error. During this period, if he were moderately active he would have consumed approximately 12,000 pounds of moist food. If there was a 1% error of caloric intake over the ten years, he would have gained or lost 120 pounds. With the five-pound gain, his body wisdom was adequate in regulating food intake to an error of less than 1/20 of 1%.

There is abundant data indicating that man and animals have innate self-selections to determine quantities and quality of food that will provide optimal health. Although this mechanism is constantly functioning to some degree and will be discussed later, it is not an answer to the nutritional question; there are many factors that interfere with proper self-selection.

In-depth study of nutrition is done to indicate the nutritional products needed for optimal health and for the treatment of various types of body dysfunction and disease processes. Why do many of the studies end with opposing conclusions? If complete data is available, one may find that one study used a natural vitamin product while the other used a synthetic one. One may have used a higher potency vitamin than the other, or the studies were biased by group selection. Conflicting conclusions may be traced to the differences in manufacturing nutritional products. One company may use heat in processing, and another cold. Keep in mind that all nutritional co-factors have not yet been discovered. Can heat or exposure to air destroy some essential co-factors present in natural food products? On the other hand, what might be missing from synthesized products? Can man really manufacture complete nutritional products? In a nutritional practice, it becomes obvious that supplements vary from manufacturer to manufacturer regardless of the labels indicating the same supplement types and concentration. Prescribing the proper supplement for a patient from the physician's knowledge of nutrition is complicated by the variation between manufacturers' products.

Choice of proper nutrition from knowledge of its effects in the body is further complicated by current labeling laws. This is aptly pointed out in a health bulletin from the University of California (Berkeley).[174]

Labels provided by the food processor often give little information about the product. Sometimes they can be frankly misleading, making it worse for the average person to read the label than to not. Many are misled by the term "natural"; it has no legal meaning. A product labeled "natural" — unless it is meat or poultry — can be highly processed, packed with additional fat and sugar, and loaded with preservatives.

It is difficult for the average person to determine the amount of sugar in a product. According to the FDA, "sugar" means sucrose (table sugar). It does not cover other forms of sugar, such as glucose, fructose, corn syrup, and products high in sugar concentration. The ingredients of a product are listed in descending order according to weight. A breakfast cereal may list its ingredients as rolled oats, brown sugar, corn syrup, sugar, and raisins, followed by other items. Note that the rolled oats are listed first; however, when the sugar products are added together, sugar may well be the major item in the product.

Buzz words, such as "enriched" and "fortified," lead one to believe that a product is higher in nutrition than the original product when, in fact, it may be lower. "Enriched" is used when foods have lost nutrients during processing and then had them replaced; it does not mean that all lost nutrients have been replaced. For example, white flour loses at least 50-80% of many nutrients. Iron, niacin, thiamine, and riboflavin may be replaced, but other nutrients lost in the milling process — such as fiber, zinc, and copper — are not.

Today there is more interest in nutrition by the general population and members of the healing arts than ever before. Most serious students recognize the difficulty in thoroughly understanding this complex subject. There is no single factor available to indicate the proper diet and/or supplementation needed for a particular patient's prophylaxis or therapy. Many factors must be considered to arrive at the optimal approach. Applied kinesiology manual muscle testing is a new and viable approach when used in combination with other methods of investigation about which the reader should be knowledgeable. When used with this discipline, it adds a new dimension to understanding nutrition and the body's needs.

Contributing to an optimal evaluation and recommendation are history, physical examination, laboratory findings, biochemical individuality, any drugs the patient may be taking,[148] interaction of vitamins and minerals, and a thorough knowledge of nutritional effects on the body. Added to this thorough consideration is the effect nutrition and food products have on body function as indicated by manual muscle testing. Each consideration in this list provides additional information for proper supplementation or diet. No single factor is adequate, since each has its limitations.

Blood tests provide information about the quantity of substances, such as protein, calcium, phosphorus, potassium, and other items in the bloodstream. When indicated, blood tests can give valuable information about the function of the body and its nutritional status.[92,175,176] Urinalysis[177] and other specialized body chemistry tests should be performed and evaluated when necessary. One has to put the results of the tests in proper perspective. When we analyze the test results the question has to be asked, "How is the body using the available material?" One can liken the distribution of food to the population to that of the bloodstream distributing food to the body. The amount of food available to a city can be calculated by combining the amount of food locally produced with that brought in by trucks, trains, and other modes of transportation. A comparison of the city's population needs and the food available may indicate that everyone in the city is obtaining optimal nutrition. This, of course, cannot be considered true unless the individuals in each household are evaluated for their use of the available nutrition. It may not be available to some because of economics, and to others because of poor food choice. When all individuals in the city are considered, there may be substantial nutritional deficiency even though it appears that food is available. The bloodstream is similar to the stream of trucks and trains coming into the city. The nutrition may be available in the bloodstream, but is it being used by the individual cells?

Hair analysis is another means of evaluating nutrition, especially minerals. There is considerable disagreement about the viability of this method. Because it is a relatively new approach, some consider that there is a lack of reference normals for the mineral levels in hair.[152] One must be certain that there is data reproducibility from particular laboratories. Early in the use of hair analysis this author had difficulty correlating changes when re-testing a patient after therapeutic efforts. The laboratory's reproducibility was evaluated by mixing a patient's hair sample and sending half of it to the laboratory under one name and the other half under another name. Correlation between the two reports was poor. This same indictment has been made by Barrett[10] but contradicted by Schoenthaler.[153] Today most laboratories have good reproducibility. Schoenthaler presents a statistical method for evaluating the reliability of laboratories. Anyone using hair analysis as a method of nutritional evaluation should take it upon himself to investigate the laboratory being used.

Applied kinesiology nutritional testing, like the other considerations, is not all-encompassing and must be correlated with other methods to determine nutritional need. In a status statement published in 1983[88,89] and updated in 1987, the International College of Applied Kinesiology states, "Nutritional evaluation [by muscle testing] should be done only with the subject tasting the substance. It is also necessary to evaluate other factors that may influence the perceived muscle

strength. Confirming diagnostic criteria for the need of any nutrition should be present from the patient's other diagnostic work-up, which may include history, type of dysfunction, laboratory tests, physical diagnosis, and dietary inadequacies...." An adequate educational background is needed in evaluating nutritional needs and manual muscle testing as discussed in a further status statement update in 1992. The use of manual muscle testing by lay salespeople has created problems due to their untrained status and enthusiasm to sell their products.

Some have described the testing of nutrition by manual muscle testing as a simple procedure,[44,135,162,169] which it certainly is not. One must be aware of the various factors that influence manual muscle testing, such as subluxations, lymphatic drainage, intrinsic neurologic dysfunction, balance of the meridian system, and function of the cranial-sacral primary respiratory system. These are only a few of the many factors that have been found to influence the manual muscle test. They must be taken into consideration when evaluating an individual for his nutritional needs.

Nutritional testing with the modified methods of holding the nutrition in the hand, laying it on the body, holding a bottle that contains the substance, and touching various "reflex points" about the body is often the main subject of a book or booklet produced for general public reading. In some instances, these procedures are taught at weekend seminars that may be sponsored by companies trying to sell their nutritional products. In fact, some nutrition companies have taught lay people to do muscle testing to convince prospective customers to buy the product. When one tries to "prove something" to another individual with manual muscle testing, errors often result. The examiner may unconsciously change the parameters of the test (or may not even know what the parameters are), and make the test come out the way he expects due to his enthusiasm for the procedure.[156] In no way should the modified procedures be confused with applied kinesiology methods. The skilled applied kinesiologist uses manual muscle testing to evaluate nutrition as an adjunct to standard laboratory and physical diagnostic methods. All factors of the examination should correlate, or something may be missed. Research sponsored by the ICAK[170] points out that manual muscle testing to evaluate nutrition, whether chewed or held in the hand, is not a viable approach in and of itself.

The primary method of testing nutrition in applied kinesiology is to have the patient chew the substance to be tested. The influence on the body appears to be the result of stimulating the gustatory and olfactory receptors. Oral absorption may also influence the body.

In simple daily observation, one can see many instances in which chewing nutrition quickly changes an individual. A hungry, crying child quiets immediately upon nursing or obtaining other food. An irritable hypoglycemic individual calms immediately upon chewing food, long before there can be any rise in the blood sugar level from the substance.

The importance of the gustatory system on health is illustrated in a report by Pangborn[131] about a Russian study by Murskii wherein dogs were "killed," then resuscitated. Early recovery of the gustatory function was always associated with successful resuscitation. In cases where recovery was difficult and cortical cells did not regain full function, the ability to distinguish food from non-foods was sometimes disturbed. The early development of taste sensation emphasizes the importance of gustatory function. The newborn human infant is able to distinguish water from sugar solutions.[113,129] Fetal sheep can taste as early as 100 days into the gestation period (term = 147 days), as measured at the chorda tympani nerve.[22]

When chewing nutrition changes muscle function as perceived by the manual muscle test, the change is almost immediate. It seems evident that the effect is due to stimulation of the gustatory and olfactory receptors. Oral absorption of some of the chewed material may stimulate remote receptors. As will be discussed later, certain substances enter the bloodstream almost immediately by oral absorption.

Most of the research done on testing nutrition by applied kinesiology methods has been clinical correlation of muscle testing results when specific nutrition is chewed, in correlation with the clinical and laboratory examinations previously mentioned. The literature has many examples of how gustatory receptors and oral absorption change body function. Research shows widespread interaction within the nervous system and the body in general from nutritional stimulation. Most of this research was done prior to the clinical knowledge of the effect chewing nutrition has on manual muscle testing. Further research must be done, taking into consideration the influence of nutritional products on the nervous system and the great amount of neuromuscular, organ, and gland interaction. Most of the basic research has been done on the control of food and water intake under normal and abnormal conditions. There have been group and isolated studies done on innate self-selection and its effect on health and diseased states. While these studies have nearly all indicated that self-selection enhances health, we will also consider how education, environment, emotions, and status satisfaction override proper innate self-selection.

Progressive research on applied kinesiology nutritional testing should not be limited to the gustatory receptors. Food and water intake is regulated by a combination of peripheral and central systems. Stevenson[166] presents an overview of this integration. Chemoreceptors, such as the glucoreceptors in the hypothalamus and liver and liporeceptors monitoring the fat depots, provide information about the body's reserves. It has been suggested that glucoreceptors provide a short-

term control of food intake relative to immediate energy needs, while the liporeceptors provide a long-term control for the maintenance of body weight. Additionally, the osmoreceptors, stretch receptors, and baroreceptors reflect blood volume and extracellular fluid volume. Even thermoreceptors play a role in regulating food intake. It is well-known that the environmental temperature influences food intake in man and animals. Hypo- or hyperactivity of endocrine glands, such as the thyroid[85] and adrenal,[46] modifies taste sensation and reaction to stimulation.

How a substance tastes to an individual does not appear to have any bearing on applied kinesiology nutritional testing. The results of a test appear to depend on how the nervous system reacts physiologically to the substance. The sensation of taste as subjectively evaluated by the subject is a hedonic one and appears to be evaluated on another level.[102] The attractiveness of food, its texture, and an individual's previous experience play a major role in what he chooses to eat. A fresh apple is identified as such by its odor. Peel and mash a raw apple and a potato to eliminate the texture characteristics. It will be difficult to determine which is which when each is tasted with the nose and eyes closed. Cold milk, beer, and soup are distinctly different in taste from hot milk, beer, and soup. One easily recognizes the optimal temperature of wine or meat. Changing the visual stimulation of the food by adding tasteless food coloring increases or decreases its attractiveness. Try serving blue-yolked eggs or black cereal! The change in desire is due to a learned response, not the dark color, since black caviar and olives rank among the most desired delicacies.

Stimulation of the gustatory receptors elicits specific preferences in drinking or eating or — on the other hand — in rejecting particular foods. Pfaffmann,[138] in discussing the "pleasures of sensation," emphasizes the hedonic aspects of sensory stimulation and suggests that sensory input to the hypothalamus and other structures of the limbic system may be involved in hedonic and reinforcing features of stimulation, as compared with cognitive and arousal functions. Some items tested with applied kinesiology methods fit into the pleasurable realm, while others are distinctly unpleasurable. Typically, sugar will cause a hypoadrenic individual to test weak on its ingestion, while a vile-tasting product for adrenal supplementation will cause a previously weak associated muscle to strengthen.

Those who use manual muscle testing to evaluate nutrition should be thoroughly familiar with the nervous system's role in nutrition. Although much of the basic research was done prior to the use of manual muscle testing to evaluate nutrition, it provides a foundation for further basic and clinical research to understand the action taking place.

The Nervous System's Role in Nutrition

Understanding the immediate effect of nutrition on the nervous system has not been thoroughly investigated. Recent studies on neuropeptides are opening new doors to eventually enable better understanding of this interaction.

Much of the neurophysiologic knowledge of nutrition's action on the nervous system comes from animal studies in which surgery was performed to establish esophageal or stomach fistulae so that food could be put directly into the system, bypassing the oropharynx, or could go through the oropharynx and not enter the stomach. In addition, information has been obtained by removing the adrenal, pancreas, thyroid, and other glands to create deficiencies and observe the change in the self-selection of foods. The nerves of taste are recorded in animals to determine their response to various types of gustatory stimulation. Considerable information has been obtained by injecting animals with insulin and other substances to determine the change in the animals' desire for food and in body function, such as nerve responses.

Much can be learned about the effect of nutrition on the nervous system from animal studies, but great care must be taken in extrapolating the animal information presented here and elsewhere to man. Regarding animal studies, Moulton[119] states, "The chemical senses show extraordinary diversity in the geometrical deposition of their organs, in their relative degrees of development, and in their structural interrelation in different species. It is clear that their relative biological significance must vary widely from group to group. This fact alone implies that extrapolation of findings concerning their interrelations and functions from laboratory animals to man must be viewed with more than the usual caution."

There are variables in taste function that are unique to various species. For example, a bird will not drink a fluid that is a few degrees above its body temperature, yet cooling the fluid down to freezing does not decrease its acceptability. Birds will rapidly notice a change in the surface texture of grain; however, they appear indifferent to viscosity. A thick viscous sucrose solution is accepted as well as pure water.[102]

A laboratory rat quickly selects sucrose or saccharin over other "non-sweet" substances. The cat and dog prefer sucrose, but they are indifferent to or reject saccharine solutions. The cat is indifferent to both solutions. There are even species differences when specific sugars are considered.[102]

When gustatory chemoreceptors are stimulated with the basic tastes of salt, sweet, sour, or bitter solutions, oscilloscope recordings of the chorda tympani and

glossopharyngeal nerves are different in goats, sheep, and calves.[14] Studies of the nervous system's role in nutrition, regardless of whether in man or animal, reveal the constant effort to maintain homeostasis. There is recall within the nervous system of how different nutritional or other substances have previously affected the body.

Learned Response

Both man and animals react to gustatory stimulation on innate and experiential bases. A better understanding of how the nervous system reacts to gustatory stimulation has developed from experiments in which animals are given a choice of foods. Often the animal is first made deficient in some nutrient and then given food choices that will or will not supply the deficiency. These are called self-selection experiments. Diet choices in self-selection experiments may be classified as one of the following: 1) simple preferences, 2) learned appetite, or 3) true hungers. Simple preferences have no relation to nutritive value; they are based on the hedonic value of flavor, odor, consistency, or some similar pleasurable quality. Learned appetites are based on an animal's experience that a certain food will give it a feeling of well-being. Hungers are based directly upon the physiologic need and require no learning process.[158] The principal difference between learned appetites and true hungers is that in the former an animal must have had a previous experience of well-being from eating the food, and in the latter there is a true nutritional need for the food for which no learning process is required.[160]

Animals develop a memory for the beneficial or detrimental effect of various foodstuffs.[102] Rats depleted of a specific nutrient such as thiamine, when offered a choice between an adequate and a deficient diet, will select much more successfully when flavor has been added to one of the choices. Apparently they can rapidly associate the physiological need with the flavor of the diet. This identification of toxicity or benefit of a foodstuff by means of taste is a serious problem in the use of poisoned baits for rodents or other animals.

In learned response, the initial proper selection of food to satisfy the animal's deficiency is innately proper, and the vitamin becomes associated with the taste. In Harris et al.'s study,[71] flavor was added to the vitamin-sufficient diet. After the rats became accustomed to it, the flavor was moved to an insufficient diet. The rats moved to the diet containing the familiar flavor and became vitamin-deficient again. A rat can be re-educated to choose whatever diet contains the vitamin, provided the diet possesses a distinctive characteristic.

A comparison between wild and domesticated animals reveals that wild animals make choices in the amount and kind of food they eat. Wild animals stop eating when their nutritional requirements are met and they are satisfied, regardless of the foodstuff's appealing flavor. Domesticated animals are self-indulgent and more concerned with self-pleasure than optimal function.[102] Maller[111] studied the differences in food selections of domesticated and wild rats. The domesticated rats' choices for sensory pleasure were evident in sweet solutions, diets high in fat content, and negatively flavored food. When the food was flavored with quinine sulfate, a domesticated rat decreased its food intake; there was no change in the wild rat's intake. Henkin,[79] in commenting on Maller's work, states, "If we can extrapolate [this] data to man, who must be the most domesticated of all animals, this kind of phenomenon certainly seems to hold to a great extent in various diseases. The alcoholic is an excellent example of this. He chooses to obtain calories from alcohol and avoid nutrients and it is only with a great deal of effort that this pattern can be changed." Pfaffmann[139] discusses the question, "Is taste necessary?" He concludes that it is for animals, especially when not looked after by man. It is becoming more evident that man needs to learn how to look after himself.

Regulation of Food Intake

Regulation of the amount of food and water intake in man and animals is an excellent example of the numerous mechanisms at work and the accuracy with which the body regulates itself through the nervous system. Jacobs and Sharma[91] describe and support "...a model in which energy balance is a critical factor in the control of food intake. When the animal is in balance or in surfeit, the metabolic properties of ingested food are critical and when it is deprived the sensory properties receive priority in regulating intake." The experiment was primarily done with dogs. Dogs that were satiated ate for calories; dogs deprived of food prior to the evaluation ate primarily for taste, to satisfy the specific nutritional needs of the body. The type of food selected depended on the animal's deficiency. Regulation of food quantity and intake was not based on caloric deficit over short periods.[93]

The interplay of neurologic factors at different levels in the regulation of water intake is demonstrated by Janowitz and Grossman's study[95] of dogs with esophageal fistulae. There are three mechanisms that regulate the amount of water a water-deficient dog will drink. When the water is sham-drunk — that is, it cannot reach the stomach because of being diverted at the esophageal fistula — a dog will drink 2.5 times as much water as the deficit. When water is allowed to reach the stomach, a dog will drink 1.2 times the deficit. If water in the amount of the deficit is put directly into the stomach without rinsing the pharynx and the dog is allowed to drink within ten minutes, it will drink the amount of the water deficit. If it is not allowed to drink for fifteen minutes after water is put into the stomach, the dog will not drink any water.[3] This study confirms that

stretching of the gastric receptors is the first mechanism that regulates water intake.[161] It also shows that moistening of the mucous membrane in the mouth gives at least temporary satisfaction,[15] causing cessation of drinking. Finally, if enough time is given for absorption of the water, the osmoreceptors, stretch receptors, and baroreceptors reflect the extracellular fluid volume[166] and cause the dog not to drink. Conflicting with this study is one by Adolph,[1] who deprived dogs prepared with esophageal fistulae of water. When the dogs were allowed to sham drink, they satisfied their thirst with a single draft of water in five minutes or less time. The amount of water sham-drunk was precisely the amount of their water deficit.

It is obvious that there are numerous factors at work in the control of how much water is drunk. It is important to consider stimulation of the receptors in the oropharynx; the same rapid change takes place in applied kinesiology nutritional evaluation. If water is administered to a rat without passing through the oropharynx, the rat is not fully satisfied.[35]

Patients with numerous muscular weaknesses are often dehydrated. Goodheart[57] observed a generalized strengthening in these patients on manual muscle testing when they drank a glass of water. The improved muscle function is immediate, and appears to relate with the same neurologic mechanism that causes a dog's thirst to be satisfied when sham-drinking. In some cases a person's muscle will test weak after multiple contractions when the muscle was strong in the clear (discussed in Chapter 6); for now consider that among other factors this has been found to be associated with dehydration. In this case drinking a swallow of water will make no change, but if water is held in the mouth while the patient does repeated muscle activation the muscle will not weaken. How important is the length of time the material is in the mouth and the gustatory receptors are stimulated? We will consider oral absorption and chewing of food later; both depend on the length of time the material is in the mouth.

Two mechanisms regulate caloric intake.[95] When there is a deficit, the taste is potentiated to increase intake, which is a short-term response. Over a longer term it becomes a learned component of intake.[90] The short-term component ensures adequate caloric intake under varying conditions of need.[96] The learned component is a wholly neural mechanism tending to maintain active ingestion, regardless of caloric need. When a learned component is present, satisfaction of the gustatory mechanism appears to be necessary regardless of caloric need. Glucose fed intravenously does not inhibit food intake in the dog.[94] This learned response accounts for the increased intake of food in the presence of obesity.

Stimulation to the oral receptors does not appear to play a major role in regulating the amount of food eaten. Additionally, the bulk of food is not the factor regulating the amount ingested. Animals will eat the correct number of calories even though the food is increased in bulk many times by water added to a liquid diet or cellulose added to a solid, powdered diet. They will also consume the right caloric intake when high concentrations of quinine are added to make the food bitter. Teitelbaum and Epstein[168] summarize their observations this way: "What we are emphasizing is that although oropharyngeal sensations are essential when the animal must find food and identify it, they are not essential when the animal's only problem is how much to eat."

Epstein[36] developed a method whereby rats can feed themselves directly into the stomach, bypassing the oropharynx. This eliminates stimulation to the oropharynx and the somesthetic sensations produced by food in the mouth and pharynx. It also reduces the proprioceptive feedback from the act of eating. The elimination of stimulation to the oropharynx does not interfere with the quantitative control of food intake; on the other hand, choice of food and motivation to eat are greatly impaired. The rat feeding intragastrically by this method will adjust its food intake to receive the same nutritive amount when the solution is diluted with water. A rat eating 30 ml of food per day will adjust its intake to approximately 60 ml per day when the diet is diluted to half its intensity with tap water.[35] At high dilutions the animal eats more meals per day to obtain the necessary amount of food.

The regulation of food and water intake provides an excellent illustration of the great amount of interplay within the nervous system for the regulation of nutrition. In applied kinesiology nutritional testing, we are concerned primarily with gustatory and olfactory receptor stimulation and oral absorption. Animal studies show that taste and smell are not necessary when the only problem is how much to eat, but they are central when an animal must select the proper food.[168]

Self-selection in Nutritional Deficiency

Numerous studies have been done in which animals have experimentally been made nutritionally deficient in various substances to determine how they would cope with the problem. The deficiency may be made by withholding an essential nutrient, or by surgically removing a gland(s) such as in an adrenalectomy. The object is generally to provide the animal with a selection of foods; some contain the needed nutrient while others are devoid of it to determine if the animal will select the proper food for its health. Successful self-selection of food has been demonstrated in many animals, including pigs, dairy cows, sheep, chickens, pigeons, monkeys, rats, and others.[142] Other studies evaluate the effect of stimulation to the gustatory receptors by sectioning the nerves of taste[137] and bypassing the oral and nasal nerve receptors to determine the animal's change in food preferences and aversions.[18]

Human studies have primarily been limited to observation of patients with various health problems.

Sodium. Sodium deficiency is usually developed in laboratory animals by adrenalectomy. Richter and Eckert[144] gave adrenalectomized rats continuous access to a solution of sodium salts (chloride, lactate, and phosphate). The rats selected these salts, gained weight, and survived. When tested with a selection of non-sodium salt solutions (chlorides of magnesium, aluminum, and potassium), the rats did not select these solutions and died. When tempted with commercially available sodium-free substitutes, adrenalectomized rats clearly avoided them in favor of the sodium solution.[124] The rats' choice to drink a sodium solution in sodium deficiency is clearly due to stimulation of the oropharyngeal receptors.

Rats select food for quantity and quality. Several studies have been made with Epstein's[36] method of rats feeding themselves intragastrically via a tube inserted through the oropharynx to the stomach. This enables the testing of self-selection without stimulating the oropharyngeal receptors. Under normal circumstances, rats will avoid high concentrations of sodium chloride; however, when feeding intragastrically, bypassing the oropharyngeal receptors, they cease to avoid the sodium chloride solution. The rats maintain quantity precision of eating but lose the choice of foods and motivation to eat.

Sodium-deficient rats choose sodium over a non-sodium solution within fifteen seconds of being given a choice.[121] This indicates that the choice is based on nerve receptor stimulation and is not a learned response. It appears that there is a specific neuroresponse for the deficient sodium ion, which may be due to a higher response of the receptors in the presence of sodium deficiency. Richter[142] found the average taste threshold for sodium chloride in normal rats was 0.055%; for adrenalectomized rats it was 0.0037%, about 15 times lower. He considers that the minute amount of salt obtained from the solutions for which the adrenalectomized rats first manifested a preference was so low it could not have had a physiologic effect. This may be true in reference to absorption of the small amount of sodium chloride, but later studies that will be discussed show physiologic change, such as glandular secretion from gustatory stimulation independent of absorption. The desire of an adrenalectomized rat to choose a sodium chloride solution over other drinking fluids is so strong that it has become a test to determine the completeness of adrenalectomy in research.[47]

Increased salt intake of sodium-deficient rats depends upon their ability to taste the solutions offered.[137] Sectioning of the chorda tympani, glossopharyngeal, or lingual nerves individually does not result in the loss of sodium appetite. When all three nerve supplies are sectioned, taste sensation is greatly reduced or eliminated. Rats made sodium-deficient under these circumstances do not increase their salt intake and consequently die.[143]

Untreated patients with Addison's disease have increased taste sensitivity, roughly 100 times more acute than that of normal subjects.[77] When they were treated with desoxycorticosterone acetate (DOCA), serum sodium and potassium returned to normal but did not alter the taste threshold. When treated with prednisolone, the taste threshold returned to normal within the first day, frequently before any change in serum electrolyte concentration or body weight. Henkin et al.[77] state, "The nature of the effect of carbohydrate-active steroids on taste is obscure. It may be related to the effect on nerve function."

The increased taste sensitivity in patients with Addison's disease is contrasted in a study by Nachman and Pfaffmann.[122] They compare the impulse recording from the chorda tympani nerve in sodium-deficient rats that increased their intake of sodium chloride with normal rats that showed a clear aversion to drinking the solution. The afferent gustatory signal revealed no difference between the two groups. They concluded that the mechanism for increased sodium intake is a central one, in which the excitability of a group of cells changes to respond differently to the unaltered afferent sodium chloride signal.

There appears to be no question that stimulation of the gustatory receptors in sodium deficiency initiates the selection of a sodium solution over others and influences the amount of solution consumed. Studies of salt-deficient sheep with esophageal fistulae reveal that sham-drinking of salt solution is partially effective in satiating sodium appetite. The amount sham-drunk depends upon the concentration of the solution offered.[20] The type of sodium solution consumed when more than one is offered also seems to be regulated by the gustatory receptors. Sheep with parotid fistulae ingest $NaHCO_3$ more readily than they do NaCl, which may be related to their loss of alkaline saliva.[32]

Dogs with esophageal fistulae begin sham-drinking immediately when hypertonic sodium chloride is administered intravenously. When Pitressin® is administered before the sodium chloride, there is an inhibition of the sham-drinking for ten to twenty minutes.[15] Pitressin® is the trade name for the antidiuretic hormone; the precise mechanism by which it acts is unknown.[64] When there is a loss of the antidiuretic hormone because of removal of the posterior lobe of the pituitary gland, animals excrete large amounts of urine, become dehydrated, and soon die. When the animal is given access to unlimited water, it consumes huge amounts — sometimes twice the body weight in water per day, keeping it alive and in good health.[142] The wisdom of the body is again demonstrated in studies of rats made hypertensive by kidney encapsulation. In general they avoid salt solutions in favor of those with no salt.[48]

The effect of gustatory stimulation is widespread throughout the body. Another example of how gustatory receptor stimulation with sodium stimulates change in function is the comparison of the results of irrigating the oral cavity of rats with tap water or a saline solution. Urinary excretion was immediately measured by chronically implanted vesico-urethral catheters. Buccal stimulation with water caused an increase of urinary flow as early as the first minute. By the sixth minute the urinary flow was two times the control level. Oral stimulation with the saline solution depressed urinary flow within the first minute.

Self-selection of sodium in sodium deficiency is probably better understood than any other self-seletion in deficiency. There are probably many factors that influence sodium selection. For example, hypothyroid rats maintain a spontaneous salt appetite when given a choice between water and a sodium chloride solution.[49] Studies indicate that regulation of sodium intake is from afferent impulses from gustatory receptors[121] and long-term absorption.[15] Afferent impulses from long-term absorption come from throughout the body and/or gut. These impulses are mediated by central control.[122] The dilemma in presently understanding the neurologic mechanism is pointed out by Denton in his study using gastric intubation and esophageal fistulae in cattle, goats, and sheep. "A restless salt-deficient animal ingests 2-3 liters of hypertonic $NaHCO_3$ in a single drinking act over 2-3 min, and this is followed by a precipitate decline of motivation and loss of interest. An increase of plasma sodium concentration follows 15-30 min later. This observation contrasts the fact that intra-carotid infusion causing a large increase in sodium concentration of the blood passing through the brain for 10-15 min has neither evident effect on motivation nor any satiating effect. The nature of the central component of the mechanism of satiation of salt appetite, including the relation, if any, to the self-stimulation areas is unknown."[33]

It appears that the self-selection of sodium in sodium deficiency and other neurologic observations made in animals are applicable to man. Richter[143] reports on a three-and-one-half-year-old boy who was taken to Johns Hopkins Hospital for study of precocious sexual development. After eating the regular hospital diet for one week, the boy died. An autopsy revealed both adrenal cortices had been almost completely destroyed by tumor growth. Questioning the mother revealed that feeding the child had been a problem. He desired large amounts of salt; he would literally eat it by the handful, and did so until the hospitalization restricted his salt intake. The loss of salt was the reason for his death.

In another case a 34-year-old man with marked Addison's disease put approximately a 1/8" layer of salt on his steak and used nearly 1/2 glass of salt for his tomato juice. He even made lemonade with salt![142]

Sugar. Proper self-selection is demonstrated in preference for a needed substance or avoidance of a substance that may be detrimental to the body. Both sides of the preference-avoidance curve are demonstrated in animals by self-selection or avoidance of sugar. Adrenalectomized rats with marked diabetes avoid carbohydrates and increase fat and protein intake on a self-selection basis. This results in a loss of their diabetic symptoms, and the blood sugar falls to a normal level.[142]

Considerable neurophysiologic action appears to take place from sugar stimulation in the oropharynx. Nicolaidis[128] points out that many diabetics carry sugar products to combat hypoglycemia and malaise as a result of too much insulin treatment. Within about twenty seconds after putting the sugar product in their mouth, they feel better. The effect is pre-absorptive. It cannot take place from absorption into the bloodstream because of the short time factor and the small amount of sugar product. Studies have shown that when rats are given labeled glucose and expired CO_2 is measured at the end of one hour, only 2% of the glucose load is entirely catabolized.[127] Nicolaidis[128] states, "Obviously the improvement is due to the reflex released endogenous glucose which provides the emergency fuel until the intentional absorption of ingestants."

In vivo blood glucose levels were measured in rats when the buccal mucosa was stimulated with sucrose dilutions or with a saccharine solution.[127] In less than a minute hyperglycemia was observed, and sometimes a secondary elevation was observed four to seven minutes later. These elevations were even observed in twelve rats that had esophageal ligatures. Stimulation of the tongue and the buccal mucosa with water most often did not modify the blood sugar level, but it did in some cases. Nicolaidis[127] points out that perhaps these findings explain "...some pathological curiosities such as the dramatic disappearance of malaise in patients with iatrogenic or idiopathic hypoglycemia immediately after they ingest a little sugar. It is quite possible that in this hyperglycemia reflex, neural or more likely, neurohumeral efferents mobilize glucose from carbohydrate reserves...." He further points out that the hyperglycemic reaction occurs more clearly in animals with esophageal ligatures, and that there may be a dual mechanism involving these reflexes since hypoglycemia or hyperinsulinemia occurs initially after gastrointestinal stimulation with sweet solutions.

Conscious dogs with esophageal or stomach fistulae were sham-fed glucose or tap water to determine insulin secretion in a study by Hommel et al.[84] When the glucose was orally administered and recovered from the stomach fistulae there was no rise in glucose; however, there was a rise in immuno-reactive insulin (IRI) in the peripheral venous blood within 2.5 minutes, which was the earliest test made for increased IRI. When glucose was administered to the esophageal fistulae,

bypassing the oropharynx, the IRI values increased only after the tenth minute. These results suggest the mobilization of insulin on the basis of nerve impulses from gustatory stimulation. An increase in insulin was also observed from sham-feeding tap water orally; however, the increase in IRI was smaller than with the glucose test. Topical anesthesia of the oral mucosa will eliminate the effect of increase in circulating insulin caused by sham-feeding of glucose or tap water.[125] "Glucose is only a weak stimulus for insulin secretion in the carnivorous dog, and further physiologic factors must be of importance."[84] When glucose was administered intravenously, the increase in IRI was smaller than after oral or stomach administration. The authors[84] point out that insulin release after electrical stimulation of the vagus nerve results in a time similar to that of oral administration.

Water stimulation of a rat's tongue or water drinking in man often results in a hypoglycemic response, which is abolished by vagotomy. The highest amount of insulin is released upon the oral administration of glucose. The next highest is with glucose given intragastrically, and lowest is when the same amount of glucose is administered IV.[128] In most cases when hypoglycemics chew refined sugar, nearly all muscles of the body will temporarily test weak. This reaction may be due to the insulin release as a result of stimulating the gustatory receptors.

The body reacts differently to equal amounts of glucose when administered orally, by IV infusion, or directly into the stomach because of stimulation to the nervous system at different levels. From the intestinal level the messages seem to be endocrin, but at the oral level they seem to consist mainly of nerve receptor stimulation to which the central nervous system responds with neuroendocrine secretion. When the oral receptors of esophageal-ligated rats are stimulated with a sweet substance (sucrose or saccharin), there is a hyperglycemic response as quickly as one minute after the oral stimulation. Post-absorptive effects cannot account for this change because of the ligature and the fact that there is a similar reaction to sugar and saccharine stimulation.[128]

Valenstein[172] comments regarding the work of Nicolaidis[127] that it is interesting that the sweet taste of non-caloric substances, such as saccharin, may produce rapid hyperglycemic responses. This enhances a study he and co-workers completed. A significantly higher percentage of animals that had been drinking a saccharine solution rather than water succumbed when given an LD50 dose of insulin. They had speculated that oropharyngeal stimuli might trigger "preparative metabolic reflexes," which in the case of a sweet taste may produce insulin secretion.

The sweet taste of saccharin sometimes elicits a response similar to sugar in studies. When it does, there is usually a quantitative difference. Smith and Capretta[163] found that rats learn to avoid saccharin when it is provided for them without food association, presumably because of its non-nutritive value. When rats are given saccharin in combination with food, they do not learn to avoid it, presumably because of the continued opportunity to associate its taste with a deficit relief. Although the sweet taste is important in early insulin response, as indicated by the saccharine-producing secretion of insulin, ingestion of unflavored bulk can also initiate the early insulin release.[128]

Animals select their food source according to its nutritive value. Adolph[2] examined the effects of flavor. He attempted to have rats ingest non-nutritive substances, such as clay and cellulose, by adding saccharin. This led to a very slight acceptance of these materials. When nutritive-flavoring materials were added, their ingestion by the rats increased. Adolph concluded that it is difficult to fool a rat into accepting a non-nutrient with artificial flavoring. We can look at many current food products and see that man is more easily fooled.

There are two insulin elevations when sugar is administered orally, one almost immediately and the second some ten minutes later. When nutrients are administered intragastrically or intravenously, only the second phase of insulin secretion appears. In animals with esophageal fistulae, insulin secretion occurs despite the absence of any food reaching the stomach.[128]

Many factors appear to be influenced by glucose stimulation of the oropharynx. Changes in metabolic rate developed as a result of oral stimulation with sucrose in fasting rats. The metabolic rate remained high for more than ten minutes, while controlled water stimulation caused no change.[128]

Protein. When animals are fed a diet very high or low in protein, food intake is depressed. When the diet is low in protein, the low food intake is usually attributed to the inability of such a diet to support normal growth. Harper[67] indicates that food intake in a low-protein diet is depressed because of the animal's limited ability to dissipate energy, with a vicious cycle developing. When the protein intake is high, the reduced food intake appears to result from a large heat increment that can inhibit food intake, according to the thermostatic hypothesis of food intake regulation. There may also be an accumulation of amino acids or some products of amino acid metabolism that decrease food intake. The decreased intake is transitory; after a few days the rats return to normal eating.

Rat studies have not shown a strong self-selection for different types of protein[159] when there is self-selection for the proper amino acid to correct a deficiency; it appears to be a sense of taste, not of smell.[67] Rats given the choice of foods where casein is provided for protein will avoid the casein because of its taste and die.[35]

Vitamin B. The self-selection of diets contain-

ing the proper component of the B complex to correct a deficiency appears to be from stimulation of the gustatory receptors.[6] Harris et al.[71] gave thiamine-deficient rats a choice of two diets, one devoid of the vitamin and the other barely adequate. The deficient rats consistently chose the adequate diet, whereas non-deficient rats ate the two diets indiscriminately. If the sufficient diet contains more than an adequate amount of thiamine, the deficient rats will eventually begin to eat from both diets. The same type of self-selection is present for riboflavin and pyridoxine. The choice is not one of simple preference, because normal animals do not choose the vitamin-rich diet except on a random basis.[158]

When a diet is chosen to correct a deficiency, a learned response develops.[147,149] Rats deficient in thiamine, riboflavin, or pyridoxine were given a diet rich in the needed vitamin and uniquely flavored. After becoming accustomed to the diet the needed vitamin was switched to the same base food, but the flavor was eliminated. Another diet was available; it did not have the added nutrient, but it did have the flavor. The rats switched to the vitamin-deficient, flavored diet. When the diet with the needed vitamin was again flavored, they returned to the flavored and enriched diet. This indicates that the learned response developed from the needed nutrient is maintained for some time. When the learned response is applied to humans, one can see why it is often difficult to change dietary habits, even when the change is toward more nutritious food.

If the learned response is not deeply established, rats will seek a diet to improve their nutritional deficiency. When thiamine-deficient rats are presented with different food selections, they repeatedly change their diet.[146] In a natural situation this would maximize the rats' likelihood of encountering a diet with thiamine. If the selection of novel diets contains no thiamine, a preference for the familiar diet develops after two to four days.

When vitamins are regulated in an animal's diet to be either sufficient or deficient, the animal chooses food that can be metabolized properly when deficient vitamins are present, or it avoids food that cannot be metabolized when the necessary vitamins are not present. A rat on a diet entirely lacking vitamin B will self-select large amounts of sucrose for the first eleven days. When the vitamin B deficiency begins to manifest, there is a decrease in sucrose selection and an increase in fat.[142]

Calcium. In the presence of calcium deficiency there is an increased self-selection on a natural basis. When parathyroidectomized rats are fed a regular diet, they develop tetany and usually die within a few days. When offered a calcium solution on a self-selection basis, they take sufficiently large amounts to keep themselves alive and free from tetany. They also have a reduced appetite for phosphorus solutions, which is in keeping with the decreased rate of phosphorus excretion in hypoparathyroidism.[142]

On a self-selection basis, pregnant rats increase calcium and sodium phosphate slightly during pregnancy and very markedly during lactation. When the litters are weaned, the sodium phosphate appetite drops immediately; calcium drops to the original level after several weeks. Fat intake also increases slightly during pregnancy, with a very large increase during lactation. Rats on a self-selection diet during pregnancy and lactation produced babies that weighed the same as control rats on a stock diet, but there was a 20% lower food intake during pregnancy; it was almost 50% lower at the height of lactation for the self-selection rats. The conclusion is that rats on a stock diet desiring a lot of calcium had to consume a large amount of the entire diet to obtain the increased calcium.[142]

The same self-selection appears to be present in the calcium-deficient human if it is not interfered with. Children with parathyroid deficiency have been reported to show a craving for chalk, plaster, and other substances with high calcium content.[142]

Influence on Enzyme Production. Stimulation of the oropharyngeal receptors is an important first step in the digestive process. Enzyme release is appropriate to the type of food the digestive system is about to receive. Optimal digestion and health are sacrificed when the oral phase is not present, such as swallowing nutritional supplements in tablet form.

Taste has an influence on the exocrine function of the pancreas. Dogs were prepared with both gastric and intestinal fistulae so food could be diverted from the body at the stomach or intestine. Previous experiments had shown that food entering the stomach had no influence on pancreatic secretion. When the dogs were fed kaolin (hydrated aluminum silicate) there was no pancreatic response, whereas sugar was effective in increasing both enzyme content and flow. A high fat diet resulted in a greater lipase flow, but a high carbohydrate diet had no effect on amylase flow. It was concluded that taste and diet are involved in modifying the exocrine function of the pancreas.[102]

Sham-feeding in dogs causes mobilization and discharge of enzymes from the pancreas within one to five minutes after feeding, which continues for approximately thirty minutes.[100,125] Although gastrointestinal hormones — such as secretin and cholecystokinin — mediate pancreatic response, the hormonal action clearly depends on the interaction with the parasympathetic nervous system. Anticholinergic drugs or surgical denervation will interrupt the hormonal effect.[125]

Sucrose, quinine, or citric acid was used to stimulate the tongues of dogs with gastric and intestinal fistulae.[13,125] Since salivary stimulation produced no pancreatic secretion, it was used as a control. Initially the stimulation by the three substances produced pancreatic secretion. The dogs, however, learned quickly that no food would be forthcoming and the response

dissipated. Naim and Kare[125] state, "It is likely that the cephalic phase of pancreatic secretion is a complex function of higher processes than simple reflex stimulation, and that taste stimulation is only a tool of these processes."

Toxic Aversion. Man and animals rapidly associate post-ingestion effects with the flavor of the diet. If there are adverse physiologic effects from eating an item, a learned response of aversion to repeating the act is rapidly associated with the flavor and odor of the foodstuff so that the act will not be repeated.[69] This identification of toxicity is of benefit for preservation of the species. It is a serious problem to man in his effort to control rodents and other animals with various forms of poisoned baits.[102] There are two methods of getting animals to select poison for pest control, in spite of their innate ability to avoid harmful substances. (1) The poisons are mixed thoroughly with their usual food to mask most of the taste. This is especially applicable when the animal is very hungry. (2) If the poison is highly insoluble in the saliva, it will not be adequately tasted.[142]

Studying rats' selection between lithium chloride, which is toxic, and sodium chloride, which may be lifesaving, shows evidence of the sophistication within the nervous system to select between substances that have a similar taste. Recordings from individual fibers of the chorda tympani nerve show that NaCl and LiCl applied to the tongue respond almost identically.[45] Since the nerve response and apparently the taste sensation are almost identical between the two substances, the self-selection of NaCl when there is a sodium deficiency shows the rat's discriminatory ability through his gustatory receptors.

Adrenalectomized rats showed a specific appetite for NaCl, and gained weight only when this salt was given. Other salts that contained sodium were also chosen by the rats, but toxic salts such as LiCl were avoided. In this experiment normal rats failed to manifest an appetite for any of the salts offered.[46] In another study the rats freely drank from solutions of NaCl and LiCl, but after a short time they developed an aversion to drinking the LiCl,[123] indicating that it is necessary for the rat to experience the toxic effects of the substance before a learned response develops.[69]

Oral Absorption

Consideration of what causes the change in muscle function, as observed by the manual muscle test, during chewing of nutrition and other substances must include oral absorption. Most of the studies regarding oral absorption have related to the effectiveness of various drugs when administered sublingually as opposed to being swallowed or given by muscle or intravenous injection. The results of these studies indicate that many substances are more effective when administered by oral absorption. In some cases, the drug must be administered by sublingual or buccal absorption to avoid passing through the liver, which all substances absorbed from the stomach, intestine, or rectum must do. The basic complex of some substances, when swallowed, is destroyed by gastric or other digestive juices. A number of drugs that show poor or erratic absorption from the gastrointestinal tract are absorbed better via the buccal or sublingual route.[54] Numerous types of drugs are administered sublingually or buccally, including cardiovascular drugs, steroids such as estradiol, progesterone and testosterone, barbiturates, and enzymes.[54] Estradiol is effectively absorbed sublingually for post-menopausal treatment.[43] Papain, given buccally, produces a modification of the inflammatory reaction and absorption of edema.[117]

One must question the term "oral absorption" because in many of the studies it is not determined whether the substance is actually absorbed into the system, or if the effects of the administered substances occur because of gustatory nerve stimulation with the nervous system mediating the change in function without oral absorption. Some studies definitely document absorption into the bloodstream. G-strophanthin can be measured in the plasma from fifteen minutes to two hours after sublingual administration.[37]

Another consideration that is not adequately understood from the literature is the speed of expected physiologic change from the administration of a substance. In most studies, the expected action was not tested for until two or more minutes after the oral administration. In any event, there is substantial data indicating physiologic change from what may or may not be oral absorption. Sometimes the effectiveness of the orally administered substance is greater than when the substance is administered by intramuscular or intravenous injection. Lorazepam (a surgical pre-medication) is more effective when administered sublingually than intramuscularly.[52]

Several cardiac medications are administered sublingually. Cardiac muscle function changes within ten minutes of the sublingual administration of Molsidomine, as indicated by ECG.[97] There is an improved absorption of tetranitrate (a medication for angina pectoris) sublingually as opposed to the buccal pouch. It is effective when administered buccally, by inunction, or injection; it is considerably less effective when swallowed.[4]

Much study has been done on the effectiveness of nitroglycerin when administered sublingually. The body initially reacts to the nitroglycerin at the first measurement of two minutes.[42] At that time there is a decrease in systolic pressure and an elevation in forearm blood flow.[114] The fall in systolic pressure is present in normal subjects performing steady exercise,[81] as well as in those needing treatment with nitroglycerin. The maximum decrease of heart rate in the seated position is between four and seven minutes after sublingual

administration of nitroglycerin.[26]

When there is lack of salivation, there is limited dissolution of the nitroglycerin tablet and delayed physiologic effects.[145] This may result from medications that have anticholinergic side effects. It is clinically evident in applied kinesiology nutritional testing that patients who have limited saliva, whether because of medication, disturbed nerve control of salivation such as treated by applied kinesiology, or dehydration, are more difficult to test for needed or adverse chemical substances, such as nutrition and toxic products.

Data currently available seem to indicate that the most reasonable explanation for the change of muscle function, as perceived by manual muscle testing during AK nutritional testing, is stimulation of the oral nerve receptors. It is possible that some of the chewed material enters the bloodstream and then stimulates remote receptors of various kinds. If this is so, the substance must be absorbed rapidly because the change in muscle function occurs almost immediately.

The speed of absorption through the oral mucous membrane is indicated in a study by Miles and Dellow.[115] They devised a system by which the oral cavity of a rabbit could be isolated from the remaining gastrointestinal system and the nasal cavity. Tritiated (radioactive) water was recovered from the external jugular vein within twenty to forty seconds of the commencement of the oral profusion. Blood samples were taken at twenty-second intervals for 200 seconds. The recovery rate increased linearly up to 200 seconds. The investigators state, "Under normal circumstances the level of absorption can be assumed to be greater than the experimental because of the influences of muscular activity, blood flows, and increased areas of mucosal exposure."

Direct Pathway to the Brain. An intriguing area that requires further investigation is a direct pathway from the oropharynx to the brain, described by Kare[101] and Maller et al.[112] Isotopically labeled glucose and sodium chloride were introduced into the oropharyngeal cavity of a rat. The esophagus and trachea were ligated proximal to the submaxillary gland. The experimental rat was able to breathe through a slit in the trachea below the ligature. The labeled glucose and sodium chloride were left in the oropharynx four minutes, then the material was rinsed out with distilled water. The animal was then quick-frozen in liquid nitrogen. Radio tracer studies found the isotopically labeled glucose and sodium concentrated in the tissues of the face region, and clearly in the intracranial cavity and in the brain. None of the active material was found below the ligation, including blood samples taken from the heart. This indicates that there was no absorption into the bloodstream; therefore, it was not due to sublingual absorption. Kare[99] states, "...if the isotopes are introduced into the gut, they are not detectable in the brain but are demonstrable in the liver and blood." In some trials the isotope was applied to the oral cavity for less than thirty seconds, and detectable activity was found in the brain.[100] In addition to the studies of Kare[99,100,101] and Maller et al.,[112] only one other study was found investigating the possibility of a direct pathway; it failed to confirm the earlier findings.[141] Further investigation is needed to determine the possible influence of this type of pathway on applied kinesiology nutritional testing.

Olfactory Response

Stimulation of the olfactory receptors is part of the gustatory response. People with a cold often say, "I've lost my sense of taste," revealing the importance of olfactory function in taste. Much of what is perceived as taste is actually smell. Probably part of the applied kinesiology nutritional test when an individual chews a substance is also stimulating the olfactory receptors. There are also procedures to directly use the olfactory function in testing substances, as well as in administering certain types of nutrition and therapy[23]; these will be discussed later.

The olfactory receptors are unique. It is estimated that there are 100,000,000 olfactory cells in the olfactory epithelium, interspersed between sustentacular cells.[64] The olfactory nerves are constantly replaced. They are the only nerve endings out in the open, protected only by a thin film of mucus.[118] The fibers from the olfactory cells pass to both the medial and lateral olfactory areas of the brain. This may be important in a holographic hypothesis of the gustatory system.

There are fewer studies of the effect of stimulating the olfactory receptors on physiology than for the oropharynx receptors. It is clear, however, that medication administered this way sometimes affects the body more dramatically than when injected. When Pitressin® is administered intranasally there is a transitory, sharp rise in blood pressure and associated pallor of the stomach mucosa. When Pitressin® is administered by hypodermic injection the effect is similar to nasal administration, but it is less marked.[180]

Some of the effects of nasally administered medication may be derived from absorption through the mucous membrane and in the lungs. Calcitonin is used in the treatment of Paget's disease. It is absorbed by nasal spray with no side effects, producing an effective treatment.[31]

When substances are tested by sniffing the vapor, the effect on the body as measured by manual muscle testing is immediate. Immediate action is also seen in the application of some drugs. When inhaled, amyl nitrite immediately causes a decline in arterial pressure.[114]

The sense of smell varies with different disease states. Patients with adrenocortical insufficiency were asked to select by olfaction the one solution of three that differed from the others. Two bottles contained water and one contained a solution of salt, sucrose, urea, or HCl. When compared with normal volunteers, the patients with adrenocortical insufficiency had an increase in sensitivity up to 10^7 times. Treatment with steroids returned smell sensitivity to its normal range in each patient.[78] Increased sensitivity in certain diseases may be a factor in the self-selection of appropriate nutritional needs. It may also contribute to the sensitivity of applied kinesiology nutritional testing.

Human Food Selection

The study of self-selection of food by humans is basically limited to young children who have not yet developed a learned response for food preference. In early and follow-up classic studies of fifteen children by Davis,[28,29,30] there is strong evidence that self-selection — if not interfered with — provides the proper foods for the body's needs.[30] The children were brought into the six year program as infants age six to eleven months at the time they were weaned. Prior to entering the study, they had not received any food other than milk from nursing. The children were brought into the study at this young age to eliminate any outside influence on their selection of food. All children were in the program for at least six months; all but two remained for one to four-and-one-half years.

The children were allowed to choose from a large selection of natural foods. Most of the food was cooked to avoid loss of soluble substances; no salt or seasoning was added. Some items were served both raw and cooked to allow a choice.

When the study began, four children were poorly nourished and underweight, and five had rickets. During the first weeks of the experiment, the children selected many types of food but finally narrowed their choices down to groups of likes and dislikes. Each child's diet varied from the others. There were no failures to regulate the diet by self-selection, and no influence from adults. Throughout the program, the children's health was excellent except for colds lasting approximately three days. There were no serious illnesses throughout the six-year period. At one time the entire group contracted acute Pfeiffer's glandular fever on the same day. During the convalescent period unusually large amounts of raw beef, carrots, and beets were eaten.

Two occasions indicate that the children selected the type of food needed by their bodies. One child with the highest gastric pH chose a diet of much higher alkaline food than other children. Another child began the experiment with rickets. In addition to his food selection, his tray contained cod liver oil and milk containing cod liver oil. Throughout the period the disease was active, the child's food selection included the cod liver oil. About the time the blood calcium and phosphorus reached normal and x-rays showed the rickets had healed, the child ceased taking any pure cod liver oil.

It appears that the general calcification of bones in this group was above average. The roentgenologist who studied all the children wrote in his report to Davis, "The beautifully calcified bones in roentgenograms of your group of children stand out so well that I have no trouble in picking them out when seen at a distance." The excellent bone formation was present whether or not the child had rickets when he entered the study.

The children's dietary selection varied from time to time and did not follow any standard conventions. The unorthodox diets of orange juice and liver for breakfast and several eggs, bananas, and milk for supper was typical of the varying patterns. Can you imagine a mother of four children accepting individual requests of this type for meals?

The psychological evaluation of the children by an individual outside the study states, "I saw them on a number of occasions and they were the finest group of specimens from the physical and behavioral standpoint that I have ever seen in children of that age."

Almost all children five years of age like the taste of cod liver oil. As they grow older they develop a dislike for it. In a group of 5-year-old children, nearly 100% liked cod liver oil; in a 14-year-old group, only 30% liked it. Some of the 14-year-olds had an almost insatiable appetite for cod liver oil. When allowed to satisfy their craving, they took as much as 16 tablespoons in one day and continued to do so for five to ten days. After that time, they desired only a small amount, finally stating that they no longer liked it. The children's desire for the cod liver oil correlates with rats deficient in vitamin A or D self-selecting cod liver oil until the deficiency was satisfied.[142]

One must consider the psychology of eating as well as the physiology. Children's innate ability to properly self-select foods needed can often be used to advantage in handling young children's eating problems. Adult influence may interfere with proper self-selection. Often parents think their children are not eating adequately and try to coax them into greater food indulgence. The

attitude of many mothers is colorfully stated by Sweet[167] when he says that he is certain that many modern mothers pray, "Oh! Dear Lord, I beseech Thee. Wilt Thou not make my children fat and me thin?" Sweet recommends allowing very young children with eating problems to self-select food for three weeks. During this period the parent keeps track of the food eaten and the physician reviews it weekly. Frequently the child will indulge in certain foods in the beginning and eat a more balanced proper diet toward the end of the three weeks. Children allowed to omit a food at will usually do not develop a lasting dislike for it, as they often do when it is forced on them against their will. Sometimes children will voluntarily omit a food to which they are hypersensitive, eliminating allergic reactions.

Boyd,[21] in discussing Sweet's presentation, points out that when children are given a three-week period of self-selection, the food available must be proper. Sometimes the diet selection on the family table is so poor that the child's innate abilities are given no opportunity to function properly. He states, "We shouldn't direct our attention to the child when it is the parent that is the chief offender."

Under certain circumstances adults do appear to recognize food that is needed when deficiencies are present. During World War II American soldiers in a German prison camp were given well below the minimum amount of food to sustain life. A high point was the arrival of Red Cross food parcels. Points were assigned to each type of food for trading purposes among the soldiers. The highest listed item was a can of powdered milk. The reason given for its popularity was that "...it satisfied — even more than the chocolate — the prisoners' craving for something rich to eat." This is especially significant since powdered milk was so unpopular among American soldiers outside the prison camps.[109]

Unfortunately, most humans do not have the ability to make proper self-selection for their nutritional needs. In addition, it is impractical to eat whenever one wants to and almost impossible to have available whatever is desired.

Learned Response of Humans

There are many factors that influence the type of food eaten by man. The unhealthy diet of most hypoglycemics may have started with the innocent remark of a parent, "Be good at Uncle John's and I'll get you an ice cream cone." When adverse foods are used as rewards for a young child, it is only reasonable for him to develop a strong desire for such items. The food customs with which a child grows up will influence his food choice in adulthood. Richter[142] helps put this in perspective: "Most children are brought up by their parents to distrust their own appetites. Often when they like a food they are told not to eat it, and when they dislike it they are equally often told that it is nourishing and good for them. In later life such persons are much more apt to depend on food faddists than on their own taste sensations."

In some countries various foods have prestige or status value, causing certain individuals to avoid more wholesome foods for those with less value but providing an emotional satisfaction. Orientals prefer white polished rice. It has a status level much higher than the "dirty" rice pounded by the native women with primitive utensils. The white polished rice is far inferior to the whole rice; it contains no vitamin B_1 to protect the population against beriberi. "In China, for instance, there is a pronounced preference for white polished rice, and the man who eats brown, so-called dirty rice loses considerable face."[164] Only the Chinese overseers who could afford the nutritionally inferior white rice suffered from beriberi. Snapper[164] goes on to state, "Aborigines complete their qualitatively insufficient food intake by consuming insects, reptiles, and small game living in the jungle forest. Their health status is immediately endangered if they are persuaded to abandon their original way of living."

Advertising techniques used by food processors may lead to erroneous nutritional concepts and a completely unbalanced diet. Sugar products have long been advertised as "quick energy" food. No wonder a weak, shaky hypoglycemic automatically reaches for another candy bar or soda pop.

The appearance and odor of food and the surroundings in which it is served contribute to the appeal or rejection of the food. The presentation of the food itself may be highly desirable, but if there are strong adverse odors in a dirty room, most people will find that their appetite diminishes. When the visual appearance of food is disturbed by unappealing mixtures of otherwise desirable food, it is less likely to be accepted. Sometimes the environment and appearance of food is such that it may disturb some people to the point of regurgitation.[110]

Again, we can learn from animals how difficult it is to change a learned response. As a physician you face this difficulty every time you prescribe dietary changes. The learned response is so strong that an animal will maintain an improper diet to the point of death when a lifesaving diet is present. If a rat develops a sugar preference before an adrenalectomy, it will maintain that diet after surgery, avoiding the choice of the sodium chloride solution that would preserve its health. On the other hand, if the choice of sugar or sodium chloride solutions is not given until after the adrenalectomy, the rat will choose the sodium chloride solution and survive with no weight loss.[68]

Food selection in man is made by education through advertising, parental guidance, prestige, and status. The Western way of life has limited instinctive, correct choice.

Importance of Chewing Food

It is obvious from the earlier discussion that secretion of enzymes and other digestive products results from food stimulating the gustatory receptors. There are numerous isolated case studies reported in the literature that indicate chewing and stimulation of the gustatory receptors are important first steps in digestion. Wolf and Wolff[180] report on a nine-year-old individual who drank extremely hot soup, causing an esophageal stricture. Efforts to regain esophageal patency were futile, and a gastrostomy was performed. For six years the patient was fed directly into the stoma. He then began feeding himself, and developed a method of chewing the food before putting it into his stomach. It was not until then that he really began to gain weight and become robust. When food was introduced directly into his stomach, it failed to satisfy his appetite. Wolf and Wolff did an extensive study of this individual when he was fifty-seven years old. They observed his stomach function through the stoma during different stages of emotions, dietary influence, medication, and other factors. It is an interesting and unusual study.

Hollander et al.[83] report on an eighteen-year-old man with a completely obstructed esophagus. A jejunostomy was done for feeding. His weight dropped to a low of 70 pounds. The young man wanted to chew food, even though it would soon be regurgitated from his esophageal sac. Using this procedure, he reached a high of 114 pounds after six months. Later, after esophageal reconstructive surgery was done, he reached a high weight of 120 pounds.

Nicolaidis[128] reports studies in which rats suffered as a result of lack of oral food stimulation. When there was a suppression of oral feeding supplemented with IV continuous nutritional infusion, there was loss of body weight. When there was complete IV feeding, there was a greater weight loss; however, when a minute amount of powdered food was given to the rat, there was a beneficial effect on body weight. It is interesting to note that body weight increased when saccharin was used for oral stimulation, but it did not continue like the minimum powdered food. This seems to indicate that the powdered food provides a complex, multifactorial stimulation to the olfactory, gustatory, and gastrointestinal receptors.

Rats deprived of food for four days were divided into control and experimental groups. The experimental group was provided with food. Within the first twenty minutes following ingestion of the food, there were only three instances of urination among the experimental group; there were twenty-six instances among the control group (n13). The rapidity of the response raises the possibility of signaling factors separate from postingestional influences. The experimenters conclude, "This effect, which appears at maximum strength almost immediately after the introduction of food, cannot be adequately accounted for either in terms of postingestional absorption of the food or on the basis of the dehydrating properties of food in the stomach." They go on to state, "It would appear that the introduction of food into the mouth or stomach, or both, may provide sensory information that makes it possible for the organism to prepare for the forthcoming absorption."[98]

Neurologic Aspects of Gustatory Control

Gustatory control as discussed here relates to the interplay between stimulation to gustatory receptors, remote chemoreceptors, liporeceptors, glucoreceptors, osmoreceptors, stretch receptors, and baroreceptors, among others. Little is known about the afferent pathways and interpretation of these impulses for the final efferent impulses to control the action the body will take. The influence of learned response will mediate the activity, as will nutritional deficiencies and other bodily needs. The control of food and water intake involves many peripheral and central systems.[166] There are many levels of mediation within the systems. We will consider the sensitivity of the gustatory receptors, the numerous nerves that transmit gustatory impulses, and some central mechanisms. Can this function that is so widespread be explained by holographic comparison of the transmitted nerve wavelengths from the body's remote environment, with the wavelengths of the taste sensation of the gustatory mechanism? Is there a match of wavelengths to an innately known and needed body chemistry?

Gustatory Receptors

It is obvious that stimulation of the gustatory receptors has a widespread influence on the nervous system. All the ramifications are yet to be discovered. In general, it is considered that taste originates from stimulation of the taste buds in the oral cavity, but even this understanding may be limited. Henkin[80] discusses studies in which ethanol or bile salts are injected intravenously into the arms of subjects. After seven to twelve seconds the odor is smelled, and within ten to fourteen seconds it is tasted.

The first consideration of neurologic aspects of gustatory control is the specificity and sensitivity of taste receptors. Are the taste buds sensitive to only one of the four primary tastes? Do the taste buds always have the same sensitivity to a specific type of stimulation?

Halpern[66] discusses numerous experiments indicating neurophysiological, pharmacological, anatomical, and behavioral control of gustatory afferent responses. Following is a summary of some of the studies Halpern cites in his convincing presentation entitled "Some Relationships Between Electrophysiology and Behavior in Taste." In most cases the altered gustatory apparatus tends to contribute to normal homeostatic conditions.

A shift in balance of the autonomic nervous system changes the sensitivity of the gustatory receptors. General enhancement of gustatory responses is mediated through the sympathetic nervous system. Sympathetic stimulation increases the firing rate and number of units recorded in the glossopharyngeal nerve when the tongue is stimulated in a frog preparation.[66] A change in balance of the neurotransmitters may cause an individual to react differently to an applied kinesiology nutritional test from time to time. There is evidence that the contents of the digestive system will influence the sensitivity of the gustatory receptors.

There is rapid modification of taste receptor sensitivity following gastric stimulation. Gastric distention with water produces a 50% increase in gustatory response to sodium chloride. In contrast, quinine irrigation produces a 17% reduction in the impulse rate, as measured at the glossopharyngeal nerve.[66]

Further evidence that the contents of the digestive system can influence the sensitivity of the gustatory receptors is provided by the change in an animal's avoidance-acceptance curve. Halpern[66] reports on a study in which a dog's stomach was loaded with a hypertonic NaCl solution through a fistula; NaCl in milk solutions that the dog previously accepted were refused within three minutes. The stomach was then emptied and washed with warm water and normal NaCl in milk, and acceptance returned within one to three minutes. This indicates that the gastric or duodenal chemoreceptors supply the afferent neural inflow for the observed preference changes.

The concentration and constituents of the blood also influence the nervous system to change the avoidance-acceptance curve for various substances. When there is a decrease in plasma Na^+ concentration, there is an increased oral intake of NaCl. The internal afferent input might be through a CNS interoceptor in the blood vessel. Support for chemoreception in the blood vessel is seen by neural response in the distal portion of the chorda tympani nerve following intravenous injection of saccharin, nicotinic acid, and the bile acid desoxycholic acid.[66]

Halpern states, "Any disruption of normal metabolized levels that alters the environment or substrates of the gustatory receptors or transfer nuclei is likely to change gustatory responses, though probably not in a taste-specific fashion. One of the more potent and easily disrupted classes of metabolites is that of the vitamins."

The differential recognition of stimuli by the gustatory apparatus can be observed by the topical application of gymnemic acid and its potassium salt to the human tongue. Gymnemic acid applied to the mouth temporarily abolishes the sense of taste for sweet and bitter substances, but not for those that are pungent, sour, or astringent.[51] Another substance — gymnema sylvestre — abolishes only the sense of taste for sweet substances when applied to the tongue.[186] Halpern[66] proposes, "The primary sites of action of the gymnemic acid and potassium salt could be on the gustatory receptor membrane, within the receptor cells, or on the primary afferent neurons or their synapses with the receptors. Alternatively, the differential suppression of human recognition thresholds by gymnemic acid and potassium salt might be a central effect caused by complex change in the afferent code, or possibly brought about by lingual absorption of gymnemic acid and its subsequent action on the CNS." Measurement of the incised human chorda tympani nerve response when gymnemic acid was topically applied to the tongue indicated the action was primarily on the peripheral portion of the gustatory system.

Halpern summarizes his review of the gustatory afferent system by saying, "The responses of the gustatory afferent system to adequate stimulation can be altered through salivary, neuroendocrine, direct neural, and topical pathways...."

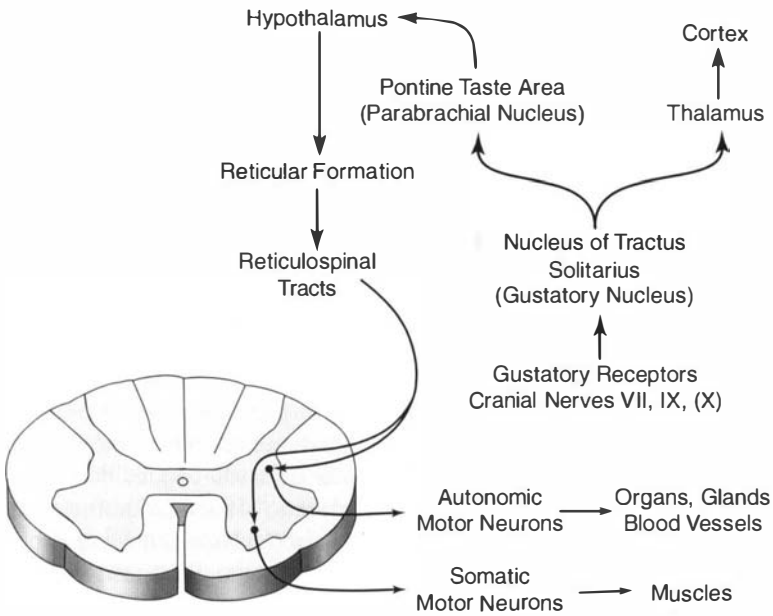
A dramatic example of topical application of a substance changing the sensitivity of the gustatory receptors is miracle fruit (synsepalum dulcificum), which is from a shrub indigenous to tropical West Africa.[87] When the tongue is subjected to the pulp of its berry, sour items such as lemons taste sweet for approximately two hours afterward. The West African natives often use these fruits to render their stale and acidulated maize bread (kankies) more palatable, and to give sweetness to sour palm wine and beer (pitto). Generally any sour material eaten or drunk will taste pleasantly sweet for several hours after exposure. Salty and bitter taste responses do not appear to be influenced.

The sensitivity change of the gustatory receptors appears to be an important consideration in applied kinesiology nutritional testing. If the body is in homeostasis regarding a specific vitamin or mineral, the gustatory receptors may not be sensitive to it, causing less afferent supply for the central nervous system to consider. On the other hand, in deficient states the gustatory receptors may be more sensitive, alerting the central nervous system to the need.

Gustatory Nerves

Sensation from taste buds on the anterior two-thirds of the tongue passes through cranial nerve V and then through the chorda tympani into cranial nerve VII. Impulses from the circumvallate papillae on the back third of the tongue and other posterior regions of the

mouth are transmitted by cranial nerve IX. From the base of the tongue and other parts of the pharyngeal region, impulses are transmitted by cranial nerve X. Taste impulses of the three cranial nerves — VII, IX, and X — are transmitted into the tractus solitarius. There are also impulses from cranial nerve XII with taste stimuli to the tongues of rats.[183]



4—1. Proposed pathways of motor response to gustatory receptor stimulation.

Certain taste buds are characteristically classified as sensitive to one of the four primary tastes, with a tendency for grouping into special areas. The sweet taste is located principally on the anterior surface and tip of the tongue, the salty and sour taste on the two lateral sides of the tongue, and the bitter taste from the circumvallate papillae on the posterior of the tongue.[64]

Evaluation of taste at various localities is a neurologic examination of the cranial nerves. When solutions of the four basic tastes are applied to different areas of the tongue, there is great chance of error and difficulty in determining the deficient area.[19,105] An improved method of evaluating taste is by anodal galvanic stimulation.[106]

There is an overlap of the different taste sensations throughout the areas of the oropharynx. Taste cannot be completely abolished unless all three nerves are severed,[130,143] and even then it is difficult. In considering the four types of taste, Moulton[119] asks, "Is there any evidence that interaction among these systems does in fact occur? If so, at what level? Does it enhance capacity of the organism to make chemosensory quality discrimination...?"

Erickson[38] points out that it may be misleading to label a taste fiber as a "salt" fiber because it is maximally sensitive to salts, since it is probably responsible for signaling a number of stimuli.[133]

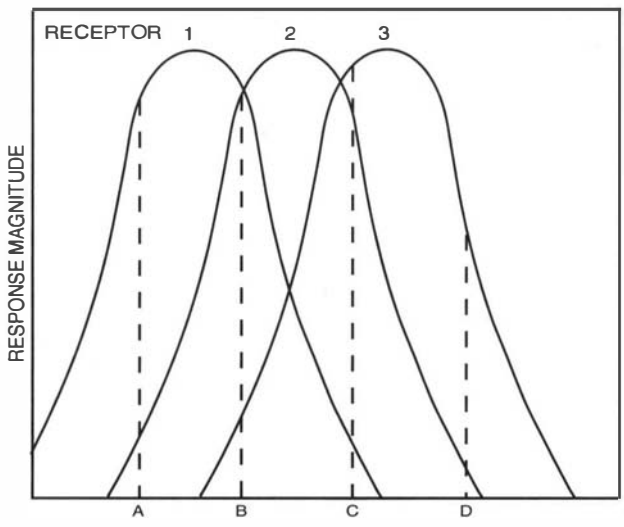
The lack of specificity of taste receptors is one reason that it is difficult to abolish taste and problematic to localize lesions in the nervous system by testing for lack of taste. Microelectrode recording from individual taste receptor cells shows no, all, or nothing spikes characteristic of neural tissue. Different size spikes are seen in recording strands of the chorda tympani nerve when there is stimulation to the gustatory receptors. The recorded potentials appear to be graded responses to the taste stimuli. Each receptor cell responds to a number of taste stimuli. Discrimination of the basic taste qualities of sweet, sour, salty, and bitter is not receptor-specific. Yamamoto et al.[183] classified four types of cranial nerve XII responses to stimulation of the tongue in lightly anesthetized, non-decerebrate and acute decerebrate rats. Two distinctive groups developed — a sucrose-sodium (NaCl) group and a hydrochloric (HCl)-quinine one. In addition, there are extraneous factors that influence the afferent impulse from the receptor. Patton[133] states, "The complex sensations aroused by mixed gustatory stimuli are a fusion of the four primary modalities along with various somatosensory and olfactory components."

It appears that the first "analysis" of the substance being chewed is by the sensitivity of the oral receptors, depending upon the homeostasis of the body. The afferent information that reaches the central nervous system from a substance(s) stimulating the gustatory receptors depends upon the summation of impulses. Erickson[38] tested the hypothesis of a "cross-fiber pattern" of response for a number of taste solutions. This was done by recording from thirteen fibers of the chorda tympani nerve to establish a pattern for different taste quality to determine if the pattern was similar for substances with similar taste. This portion of the hypothesis was substantiated. It is further supported by Zotterman,[186] who found that different biological sugars stimulating the gustatory receptors produced different responses in the recording of the chorda tympani nerve. Erickson[38] correlated this physiologic information with behavioral tests of the rats based on shock-based avoidance of drinking from one salt to others. He concluded that "...the neural message for gustatory quality is a pattern made up of the amount of neural activity across many neural elements."

Individual fiber recording at the chorda tympani nerve indicates very similar nerve transmission from stimulating the gustatory receptors with lithium chloride or sodium chloride,[45] yet the rat learns to avoid toxic lithium chloride,[123] apparently by different neural

taste codes.[70] Since an animal's behavior pattern varies with different taste receptor stimulation and there is little or no difference in the nerve impulse as recorded at the chorda tympani nerve, there must be some method of higher taste discrimination. Zotterman[186] puts electronic nerve recording in perspective when he says, "It is not known whether the central nervous system 'sees' the responses from the chorda tympani in the same way as the electronic apparatus."

Coding of taste is an integration of the responses of the population of gustatory neurons, rather than an activity in particular neuron types related to the four basic tastes. Stimulus intensity is coded by the total amount of activity elicited in a population of responding neurons.[179] Erickson[38,39] analyzed the function of the much better understood color vision. Can the combination of different types of fibers and impulses account for the many tastes as they do for varied color sensations received? Assume that there are only three receptor-fiber types to perceive color. Now suppose that four colors are individually used as stimuli and recordings are obtained from the three receptor-fiber types. The receptor-fiber types are indicated by 1, 2, and 3 on the accompanying graph. The color stimuli are indicated by A, B, C, and D. When the stimulus is from color A, the receptor-fiber types are stimulated to different degrees or not at all. The summation of activity is at the ordinance of A with the receptor type. The same is true for colors B, C, and D.



4—2. See text for discussion.

Central Control

There is widespread distribution of taste impulses in the brain. The central system is focused in the hypothalamus, limbic lobe, and midbrain. Certain areas have been designated for specific function. For example, the lateral hypothalamus has been regarded as the "center" for feeding and drinking.[166] Regardless of these designations, it seems obvious that the integration of efferent and afferent activity of the gustatory system is widespread. Grossman[63] proposes the hypothesis "...that the hypothalamus, far from integrating and interpreting all of the complex regulatory influences that appear to act on the nutritive process, may, in effect, serve primarily as the sensory end organ of a much more complex system which involves much, if not all, of the old cortex and related subcortical relay stations." Yamamoto[182] states, "In comparison to the relatively simple and stereotyped reflexes mediated in the brainstem, more complicated or well-organized responses relating to motivational, effective, reinforcing, arousal, and ingestive behaviors are involved in taste functions of the limbic and hypothalamic areas." He continues, "Neurons in the parabrachial nuclei project to the cortical taste area via the thalamic taste relay nucleus. This thalamocortical pathway may be concerned with taste discriminatory function." "Taste input projects to different locations in the rat cortex. Direct thalamocortical inputs are received at the ventral taste area and are important in taste discrimination. The second area may receive direct thalamocortical input, but its functional importance is still vague."[184]

To help understand some of the rapid homeostatic adjustments made by the body, Nicolaidis[127] recorded two areas of the cat's brain — at the supraoptic nucleus, and above it in the central zone of the medial prosencephalic fasciculus. When an intercarotid injection of NaCl was given, response in the brain was almost immediate. Buccal stimulation with NaCl solution produced a similar activation in the brain. The application of the NaCl solution to the tongue caused a reduction in urinary flow. Bathing the buccal area with water did not produce the increase in brain responses, and often the basal activity was progressively inhibited. A surprising finding was a more ventrally located area to the prosencephalic fasciculus where the response was exactly opposite that described for the supraoptic nucleus. It seems possible that these centers in the brain are the recording area for the object and reference beams of holographic neurologic function, with the impulses reporting concentrations in the blood being one beam and the stimulation to the gustatory receptor impulses the other.

There are numerous areas in which there are antagonistic centers in the brain. For example, the appetite centers located in the hypothalamus are the lateral nucleus for "hunger or feeding," and the ventral medial nucleus is the "satiety center." These areas may be the reference and object beams of holographic function. The analogy of taste with color vision by Erickson[38,39] was made prior to the current holographic model of the nervous system; however, it fits well into this pattern.[64] Dolgoff[34] reports that von Bekesy of the University of Hawaii has shown that the mathematical impulses of the holographic theory applied to the neuroreactions to stimuli in the gustatory system. Pres-

ently the exact neurologic mechanisms responsible for the clinical observation of applied kinesiology nutritional testing are unclear; however, there is ample evidence of a widespread neurologic function throughout the body from stimulation to the gustatory receptors. There is also considerable evidence that integration of remote receptors and those of the gustatory system is important in maintaining the body's nutritional homeostasis.

Gland and Other Tissue Support

Glandular and other tissue products taken orally for health problems have received varying degrees of acceptance. Much of the initial research on tissue products was done in Europe. There are currently health spas, primarily in Europe, that provide cellular injection for tissue support. This, too, has proponents and opponents.

A strong proponent of oral ingestion of various animal tissues in the early twentieth century was Harrower.[72] In one of his books[73] he quotes extensively from medical literature, such as the *Journal of the American Medical Association, British Medical Journal, New York Medical Journal*, and many others, as to the efficacy of oral tissue ingestion, which he calls "organotherapy." It seems that many of the early glandular products actually contained hormones and were part of the reason for the success of the treatment.

As hormones were isolated into crystalline substances and synthesized, the potency increased and organotherapy became less prevalent in allopathic medicine.

Currently organ, gland, and tissue substances are primarily used by natural health practitioners. Clinical evidence indicates effective treatment when properly prescribed, but there is not the dramatic and immediate change seen with synthetic concentrated hormone therapy.

Greater knowledge is developing regarding tissue therapy. Burns[24] reviews some of the literature in his article, "Accumulating Scientific Evidence Supports Glandular Therapy." A review of this literature helps put the action of tissue therapy in perspective and gives direction for further research. Briefly discussed here will be breakdown of the products in the gut, transfer across the gut wall, neuroactive peptides, effects in the body, and some examples of known action from the oral ingestion of peptides.

Breakdown in the Gut. Criticism frequently directed toward oral administration of tissue concentrates is that they are thoroughly digested in the gut to basic amino acids prior to assimilation. That this does happen to a certain degree is an undeniable fact; there is considerable peptide hydrolase activity in the intestinal mucosa providing for the breakdown of peptides. Some peptide sequences, however, are relatively stable to enzymatic hydrolase. Phosphopeptides, those containing B-amino acids, and one containing pyroglutamate at the N terminus, are prominent examples of such stability. In light of this Gardner[53] states, "However, since all the amino acids (rather than just serine and a few other amino acids) appeared to be represented in peptide fraction crossing the intestine, hydrolysis-resistant phosphopeptides are unlikely to be the sole reason for this phenomenon."

Gardner[53] strongly supports the absorption of intact peptides and protein in an extensive review of the literature. In his introduction on protein metabolism he states, "However, one special aspect that has been largely neglected hitherto is the possibility that peptides produced during digestion *in vivo* of a protein meal may enter the circulation in intact form, and that they may thus reach peripheral tissues where they could exert biological activities." He goes on to state, "...there is now a substantial body of evidence, albeit not widely known, that significant quantities of larger molecules, including peptides and even intact proteins, can cross the intestine." Most still regard the intestinal epithelium as an absolute physical barrier preventing ingress of micromolecules; however, there is overwhelming evidence that this concept has become untenable.

Hemmings and Williams[75] demonstrated by electron microscopy that large protein molecules are absorbed intact across the gut wall. These intact tissues are found throughout the body where they are degraded, which "...might be termed distributed digestion, occurring in all body cells rather than in the gut lumen." They go on to state, "Tentatively it is put forward that there is a universal necessity for body cells to be 'permeable' to proteins of many types and that there is a constant traffic of protein molecules into all body cells."

An example of absorption through the gut wall is orally administered chymotrypsin and trypsin.[5] Miller's[116] specific interest was in studying trypsin, chymotrypsin, and bromelain transfer into the bloodstream. He used sophisticated radioactive and fluorescent dye-tagging methods that conclusively revealed these large protein molecules to be directly absorbed into the bloodstream. The percentage of absorption varied with the substance and with the health of the individual. He concluded, "The opinion that such enzymes are not absorbed should be abandoned."

When considering the permeability of absorption across the gut wall, one must remember that there should be a selectivity to act as a protective mechanism. This should minimize the entry of potentially noxious molecules into the body. In some states of illness, such as celiac disease, the intestine fails in this function.[75] This may be the reason for some food sensitization, such as bovine milk, enteropathy — espe-

cially of infants — and the reason individuals with celiac disease are so sensitive to gluten.

Inert solvent particles have been shown to cross into the system from the intestines, including latex spheres, carbon fragments in India ink, metallic iron particles, lycopodium spores, viruses, and others. These are several microns in diameter, illustrating the size and non-specific nature of material that can cross the gut in trace amounts. Even large molecules such as insulin, which has a molecular weight of 6,000, can pass into the system across the intestine in quantities sufficient to produce hypoglycemia under some circumstances, including the presence of protease inhibitors or hypertonic luminal solutions.[53]

Absorption from the gut is enhanced at an area of specialized cell types, known as "M" cells, which overlie Peyer's patches. They permit subepithelial lymphocytes to come very close to the intestinal lumen, and they contain many vesicles. It has been suggested that their function is to facilitate access of luminal antigens to lymphocytes, thus stimulating an immune response. It is here that macromolecules and solid particles, including viruses, pass into the milieu from the gut.

Gardner's review[53] of the literature reveals that there are many routes and mechanisms that appear to be involved in the passage of some peptides and macromolecules into the blood, including (1) trans-cellular passage through obvious "pores," (2) trans-cellular passage through lipid regions, (3) area-mediated transport by specific trans-cellular mechanisms, (4) pinocytosis through normal epithelial cells, (5) pinocytosis through specialized "M" cells, and (6) para-cellular passage through tight junctions and extrusion zones.

Effect in the Body. It is only with relatively recent research that the effect of neuroactive peptides has become known. Within a short time, over sixty neuropeptides have been identified; undoubtedly there are many more. Gardner[53] states, "It is important to stress that small peptides can have very diverse biological activities, and also that many are intensively potent substances. Hence the amount of peptides that may have significant biological activities are liable to be below the limits of detection by chemical and physical methods; also, one is hindered from applying bio-asays because one does not necessarily know what the nature is of the biological activity that may be present in the plasma of postprandial subjects."

Candace Pert,[134] Chief of Brain Biochemistry, Clinical Neuroscience Branch, National Institute of Mental Health, has made a strong case for the unifying of mind-body, with neuropeptides being the unifying factor. She and her co-workers propose that receptor molecules for neuropeptides "...do not become more complex as an organism becomes more complex. The identical molecular components for information flow are conserved throughout evolution. The whole system is simple, elegant and it may very well be complete." These important breakthroughs seem to shed light on why animal tissue products have specific effects on human health.

Although many hormones have been found to have neuropeptide activity, tissue therapy cannot be classified as hormone therapy. It does not conform to the laws of dosage, intensity, and duration.[126]

Animal studies have been done with cellular injection. In such cases, as with oral administration, effects are not immediate. Neumann[126] observes that, "Numerous observations indicate that the effects typical for cellular therapy do not occur immediately, i.e., within hours or days but rather after a latent period which is believed to be 12 to 18 days. Usually, pharmacological studies are concerned with an immediate effect, the intensity of which increases until the resorption at the site of the injection reaches its peak, and decreases as soon as the drug is catabolized or excretion causes a decrease in the blood-level or in the depots in storing organs."

One study showed a much more rapid change.[165] Liver extracts were infused into dogs that induced liver growth in the test animals. Tissue was evaluated for regeneration at 24, 48, and 72 hours after infusion. Regeneration was present at 48 hours. The reason for the increased liver growth is unknown. Starzl et al.[165] observe, "The identity of the growth factor(s) in regenerating liver is speculative. The fact that it cannot be found in liver remnants until regeneration is far advanced reduces the possibility that it is an uncomplicated initiator. Yet, its probable importance as a biological determinant is underscored by the fact that its effects far outlast a short-lived administration."

The administration of cellular therapy has different reactions in healthy and diseased states. Injections of radioactive liver into rats concentrated twice as much in rats' damaged livers as in healthy livers.[126]

Neuropeptides, present throughout the body, have a wide range of function. Among other things, they are involved in the autoimmune system. The peptide glutaurine from the parathyroid influences the natural killer activity of lymphocytes in cancer, under some conditions.[107] Peptides may not even be specific for a particular action in order to influence it. Small exogenous peptides may inhibit the degradation of endogenous peptides. This is accomplished by whole or even partially degraded peptide hormones (fragments) competing with endogenous peptides for sites on peptidases. In this manner the peptidase is inactivated, sparing the degradation of the endogenous peptides and thus prolonging their activity. The cells of the immune system have receptors for various neuropeptides, and they also make neuropeptides themselves.[134] Presented here are only a few examples of how neuropeptides affect body function.

Thymus. The thymus is an endocrine gland that produces a family of hormonal-like peptides. They control the development of the thymic-dependent lymphoid system and participate in the process of immune regulation. The active polypeptides from the thymus are given the prefix "thymosin," followed by a subscript number α_1, α_2, β_1, β_2, and so on. Thymosin α_1 is highly active in amplifying T-cell immunity. Some of the thymosin fraction has been chemically synthesized in the laboratory and has been shown to have similar effects. Some of the effects of thymosin are to induce bone marrow cells to develop into intrathymic lymphocytes. Thymosin fraction V induces T-cell differentiation and enhances immunological function in animal models and in humans. Other types reduce contact sensitivity, stimulate interferon production, enhance antibody production, elevate serum calcium concentration, and decrease serum inorganic phosphate. Thymosin peptides are effective in inducing the differentiation of specific sub-classes of T-lymphocytes (killer, helper, and suppressor cells).[56] Thymosin V treatment in HTLV-III (positive pre-AIDS) may prevent progression to frank AIDS. It restores some aspects of depressed immunity.[155]

Thyrotrophin-releasing Hormone (TRH). Thyrotrophin-releasing hormone is a neuroactive peptide that can be given orally. As a drug it is known as protirelin. It is used as an emergency treatment in shock, spinal cord injury, and as a CNS depressant antagonist.[82] The effect in treating spinal cord injury is as an antagonist to endorphins.[40,41] When TRH is administered orally, the increase in serum thyrotrophin is slower than when administered IV, but it lasts longer.[65] There have been some effective results in administering TRH to depressed patients. This improvement is through its effect as a CNS depressant antagonist.[55] TRH is absorbed in the upper part of the small intestine. It is stable against digestive enzymes and first pass through the liver.[185]

Applied Kinesiology Nutritional Testing

As previously discussed, applied kinesiology nutritional testing should always be correlated with standard clinical and laboratory methods of determining nutritional deficiencies to make a final determination of a patient's needs. In general, nutritional testing is simple. The complex part is relating the nutritional muscle test results with the rest of the examination findings. Nutritional testing requires a specific protocol. The most frequently abused portion of the protocol is accurate muscle testing. First, the test should be of a specific muscle, isolating it to the maximum so there is little opportunity for recruitment of synergistic muscles. Since muscle testing is done before and after administration of the tested substance, one must be very careful to reproduce the test in exactly the same manner.

The procedure for testing a patient's response to nutrition is to have him chew or suck on the substance until the gustatory receptors are stimulated. A muscle is then tested for change. A weak muscle may become strong, or a previously strong muscle may test weak.

When attempting to find the proper nutrition to support an organ or gland, it is best to test the muscle(s) associated with that organ or gland. If a patient is diagnosed as having a liver disturbance and the associated pectoralis major (sternal division) muscle tests weak, have the patient chew a substance that may help the liver, such as vitamin A. If the weak muscle is associated with the liver disturbance and the vitamin A is appropriate treatment, the muscle will test strong. The vitamin A is placed in the patient's mouth, and he chews or sucks on it to stimulate the gustatory receptors. Chewing the substance is the most effective means of testing because it simulates the oral movements of eating. There is an increased response from the oral chemoreceptors if the papillae are moved in conjunction with the substance tested.[14]

The substance chosen to test in various health conditions is determined by the examiner's general knowledge of nutrition. Substances found to frequently improve function of various muscles are listed in the muscle testing chapter of this text. The list is not all-inclusive, nor does it indicate that the substance will actually improve muscle function in all circumstances.

Sometimes there will be numerous nutritional factors that will cause a weak muscle to test strong. A method to determine the best product has been described by David Leaf as reported by Goodheart.[62] First ascertain the tenderness at the associated neurolymphatic reflex by palpation. Individually test each of the nutritional products that improved muscle function by having the patient chew the substance and then re-evaluate the neurolymphatic reflex tenderness. The product that will provide the best improvement for the patient will dramatically relieve the tenderness on digital pressure. It is usually not necessary to supplement the patient with all of the factors that improve the muscle test.

Adverse Compounds

Adverse effects of compounds on the body can be determined in a manner similar to that for nutritional needs. If a food product, nutritional item, or environmental chemical is detrimental to the body, it will cause associated muscles to weaken. In some adverse cases, all muscles of the body will temporarily test weak after administration of the detrimental substance. Again, it is best to test an associated muscle. For example, many items are detoxified by the liver. The pectoralis major

(sternal division) is an excellent muscle to test for weakening caused by detrimental substances.

Some have said that the administration of refined sugar will weaken everyone. This is not correct, and if one consistently finds it to happen, he should re-evaluate the quality of his muscle testing and mind-set.[156] Truly healthy individuals usually do not weaken with the ingestion of refined sugar. In some cases, refined sugar will make a previously weak muscle test strong because of the body's physiologic needs at the time. Certain patterns are frequently — but not always — present. Chemicals toxic to the body, such as carbon tetrachloride, will especially weaken the pectoralis major (sternal division). Alcohol will often weaken the sartorius and gracilis muscles in an individual with relative hypoadrenia. Although there are many such patterns, 100% applicability to all individuals is rarely present. If one consistently obtains the expected results from muscle testing to evaluate nutrition and other compounds, he should evaluate the quality of his muscle testing. Unexpected results are good evidence of quality muscle testing.

Olfactory Stimulation

Therapeutic products, such as vitamins, herbs, homeopathic preparations, and Bach flower remedies, can be tested for need and administered therapeutically by inhalation. Brimhall[23] introduced this concept to applied kinesiology in 1979, and it has been a useful procedure under many conditions.

Absorption of heavy toxic metals by respiration is commonly recognized.[154] There has been a major effort to reduce lead from automobile emissions because of absorption into the body by respiration. Great care must be taken in a dental office to prevent mercury from going into the vapor state because of its high absorption into the system by inhalation.

Testing therapeutic and harmful products for applicability to a patient's condition via inhalation is very similar to chewing the substance. Simply hold a container of the product to be evaluated under the patient's nose and have him sniff the odor. If it is appropriate therapy, a weak associated muscle will strengthen. For example, if the infraspinatus muscle — associated with the thymus — strengthens upon inhalation of the vapors of echinacea, an herb that supports the immune system, there is evidence it will be of value in the immune-deficient patient. Harmful substances can be tested by sniffing and testing a strong muscle for weakening.

A therapeutic substance can be given to a patient over a protracted time by inhalation absorption. The method of application is usually by a vaporizer. Approximately one dropper of the substance can be put in two quarts of water to vaporize in the bedroom while the patient sleeps.

Interaction

There is considerable nutritional interaction within the body. For example, a patient may have hypothyroidism, but the usual nutritional products that improve the condition are ineffective. Applied kinesiology testing methods may provide information about an interaction causing the endocrine system to malfunction. Another gland may be involved with the hypothyroidism. If it is nutritionally supported, the hypothyroidism may improve. The examination for interaction is done with double-handed therapy localization. Prior to testing for interaction, determine if direct nutritional therapy to the gland in question is applicable. In the case of hypothyroidism, have the patient place his hand on a thyroid reflex area, such as the neurolymphatic reflex, and test a strong indicator muscle for weakening. If it weakens, have the patient chew thyroid substance and/or iodine. Re-therapy localize and test the indicator muscle. In many cases this will neutralize the positive therapy localization, indicating the substance is appropriate for the thyroid. If the positive therapy localization is not abolished, have the patient place his other hand on another gland or organ reflex point, such as the adrenals, while continuing to therapy localize the thyroid. Re-test the indicator muscle; if there is no change, continue with other organs or glands until one is found that neutralizes the positive thyroid therapy localization. If therapy localizing the spleen cancels the positive therapy localization, there is indication that the spleen is also involved. Have the patient chew spleen substance or vitamin C and re-test the thyroid reflex area with single-handed therapy localization. There will be no positive therapy localization to it if the spleen substance helped the problem.

The interaction of nutritional support is indicated in a study by Leaf.[108] The study was done to determine the effect on a weak teres minor muscle of putting nutritional substances on the skin or in the mouth. Subjects participating in the study answered a survey form containing thirty symptoms relating with either hypo- or hyperthyroidism. Four nutritional substances — niacinamide, vitamin E, inositol, and thyroid extract — were placed on the skin, and the teres minor muscle was tested for strengthening. The substances were then put in the mouth, and the teres minor was again tested for strengthening. Placement of the nutrition on the skin produced a random response. When the nutrition was put in the mouth, individuals who marked over twenty symptoms on the survey showed strengthening of the teres minor muscle with all four nutrients. Individuals who marked seventeen to twenty responses showed strengthening of the muscle as follows: niacinamide (28%), vitamin E (22%), inositol (24%), and thyroid extract (100%). When there were thirteen to sixteen positive responses on the questionnaire, muscles strengthened to niacinamide (13%), vitamin E (15%), inositol (11%), and thyroid extract (91%). There was

no response in 1%. The patients marking four to twelve symptoms proved most positive in selectively strengthening the teres minor muscle by thyroid extract. Results were as follows: niacinamide (3%), vitamin E (5%), inositol (4%), and thyroid (92%). There was no response in 3%. When there were three or fewer positive responses on the questionnaire, there was random response of the teres minor strengthening, similar to when the nutrition was placed on the skin. This study indicates a selectivity of chewed nutrition affecting an apparently associated muscle. When there is a very high symptomatic pattern relating to the muscle-gland association, more types of nutrition strengthen the muscle than the expected individual association. When there is a lower number of positive responses on the symptom questionnaire, the expected nutrition-muscle-gland association shows a higher percentage.

Allopathic or Nutritional?

There are two approaches in the use of nutrition. The megavitamin approach, advocated by many, is basically an allopathic approach to nutrition. A nutritional product is given to create a specific response in the body, frequently to counteract some health problem or potential health problem. Often the nutrition used in megavitamin dosages must be synthetic to obtain the high dosage level. The labeling of nutrition as "natural" does not necessarily mean it is 100% so. Vitamin C labeled as "natural" from rose hips can contain some rose hips, but it may be brought up to the label dosage with ascorbic acid.

Megavitamin dosages can sometimes create side effects by depleting the body of the co-factors necessary to process the high dosage of the administered nutritional product. Megavitamin dosages are often of value in treating specific conditions. One should take care that the megavitamin dosage is not creating imbalance within the body, ultimately causing a new problem.

Some nutritional products are processed to maintain the product in as natural a state as possible. These products are usually low in dosage, and an attempt is made to maintain the natural synergistic factors as nature developed them. Use of this type of nutritional supplementation is a true nutritional approach, rather than an allopathic one. Many of these products can be used on a continuing basis without concern for creating an imbalance within the body.

Sometimes nutritional products will cause a previously strong indicator muscle to weaken, indicating that some factor is detrimental to body function. There are many constituents of various nutritional products that can cause this to happen. A patient may be sensitive to artificial coloring, the carrier, or to the nutrition itself. An example of a patient being sensitive to an item is when too much of a substance is provided in the patient's diet. Sometimes patients who are hypoglycemic and hypoadrenic are given a diet high in whole fruit and fruit juices, which provide considerable potassium. In the hypoadrenic, potassium is retained in the body. Under these circumstances, having the patient chew or suck on a potassium tablet or consume some of the fruit may cause general indicator muscles of the body to weaken — evidence to reduce fruit in the diet.

Nutritional Dosage and Administration. There is no satisfactory method for determining dosage requirements with applied kinesiology. The best method to date is to determine initial dosage empirically, then adjust it according to the amount needed to maintain normal function of the associated muscle.

There is considerable evidence that it is important to chew nutritional supplements to stimulate the gustatory receptors, alerting the body to the type of food it is receiving. This activates the enzyme system and other processes necessary to properly use the nutritional product. Swallowing a tablet is similar to putting food into the stomach through a stoma.

Certain nutritional products are more effective when administered throughout the day. In severe conditions, one may have a patient chew a low-potency product every fifteen minutes for the first day or two. In some cases, it appears that stimulation of the gustatory receptors is more important than the quantity of nutrition taken. Equally effective treatment may be obtained by cutting the tablets into quarters so that the patient chews a quarter of a tablet every fifteen minutes.

Parotid and Thymus Role. Until recently the thymus gland was very much overlooked in the adult. Its role in the autoimmune system is being recognized, but that subject is outside the scope of this text.

Goodheart[61] proposed that the thymus, being an autoimmune gland that processes ribonucleic acid, codes the RNA released from dead cells by antigen-antibody reaction as a result of tissue degeneration. After this coding, the RNA is secreted by the parotid gland during the chewing process. The coded RNA from the saliva combines with the food, tagging it for specialized use in the body. This provides a selective use of the available nutrition to regenerate the areas of degeneration.

This hypothesis resulted from the observation that when thymus nutritional support was tested, it would not influence any muscle other than that associated with the thymus gland (infraspinatus). Parotid glandular substance likewise caused no major change. When thymus and parotid substances were placed in the mouth together, weak muscles associated with the endocrine system strengthened. Use of nutrition for specialized areas, such as the adrenal, ovaries, and others, was enhanced when thymus and parotid substances were administered with a specialized tissue. This activation and correlation of nutritional products in the chewing mechanism seem reasonable because of the

necessity of chewing nutritional products to obtain optimal benefit.

Dehydration

Many individuals are dehydrated. This may interfere with adequate nutritional testing. As previously indicated, nitroglycerin is less effective for the treatment of angina pectoris when the mouth is dehydrated, primarily because the nitroglycerin fails to dissolve and be absorbed.[145] In nutritional testing there is lack of response in the presence of dehydration, even if the tablet is chewed, perhaps because mixing saliva with food for processing changes the gustatory receptors' sensitivity to stimulation. No studies of this have been found.

Dehydrated patients may not show effective therapy localization. When dehydration is present, positive therapy localization may be present after the patient wets his fingertips when it was not present before.

Sometimes a patient's general dehydration will cause nearly all muscles to test weak. When this occurs, the patient will often regain normal function in some or most of the muscles after drinking a glass of water. A greater effect is obtained if the patient holds water in his mouth while the test is performed. The effect is immediate, without adequate time for water absorption into the system. Human studies were done to determine the amount of perspiration in dehydrated subjects when water or saline solution was administered orally.[127] In the dehydrated subjects, there was an initial discharge of sweat 2.7 seconds after beginning to drink. In rehydrated subjects, the same sweating response was not observed. The minimum time required for sweating to begin in the dehydrated subjects suggests a reflex within an orogastric origin. When there is poor therapy localization because of dehydration, the patient may not need to wet the fingertips if a glass of water is drunk.

Ribonucleic Acid

Research into where and how memory is stored has produced many hypotheses and theories. Pietsch[140] summarizes this nicely: "Name the molecule, cell, or lobe, or stipulate the physiological, chemical, or physical mechanism, and somebody, someplace, has found memory on, in, around, or associated with it and, in spite of the generally good to splendid quality of such research, there is probably someone else, somewhere, whose experiments categorically deny a given result." Numerous types of testing with applied kinesiology seem to indicate that memory is located throughout the body. Researchers tend to think in terms of their own pet theories to the exclusion of all others. It seems that there are many types of memory, perhaps used for different purposes in the various functions that are necessary for preservation of the species.

Chemical memory is one type that has been proposed, and primarily associated with it is ribonucleic acid (RNA). The RNA molecule is an extremely large one, consisting of a string of hundreds or even thousands of sub-units of four different kinds. It has been proposed that new information entering the nervous system introduces a change in an RNA molecule of specific neurons reserved for the purpose. This unique protein is a memory of the occurrence. New information is compared with the established RNA/protein combination already present. If the match succeeds, memory is recognized.[7] Studies have been done in which rats or worms were trained, then an attempt was made to chemically transfer the memory of the training to naive subjects.

Both the chemical and neurologic natures of memory were demonstrated in a unique planarian experiment at the University of Michigan.[27] The worms were split into two groups for feeding. Group A was trained to associate an electrical shock and light with feeding time; Group B received the electrical shock and light at random times, not with feeding. Group A developed a conditioned response to go to the feeding place. During one stage of their existence, planarian earthworms are cannibalistic. Groups A and B were fed to two naive groups. The group (AA) that ate Group A appeared to have a chemical memory transfer from that group. A significant number of them went to the feeding site when the electrical shock and light were turned on. The naive group (BB) that ate Group B showed no effects from the electrical stimulation and light. Another study[74] indicates that planaria that cannibalized either trained or untrained planaria did better than naive planaria that did not cannibalize other planaria. This indicates that something in the cannibalistic process is involved in these findings.

In a blind, controlled study by Babich et al.,[8] rats were trained to approach a food cup when a distinct click was sounded. Ribonucleic acid was extracted from the brains of these rats and injected into untrained rats. Without any training, the injected rats manifested significant tendencies to approach the food cup when the click was sounded.

It is obvious that much is to be learned about the memory process. The exact role of RNA in memory is unclear, but it has been associated with function change in applied kinesiology testing in such a way that it appears to relate with memory.

It is not unusual for a patient to have difficulty passing the Romberg test, yet no spinal cord posterior column disease is present. Goodheart[60] postulates that short-term memory is required to maintain orientation in space with the eyes closed. To determine if RNA improves orientation in space, have the patient stand on one foot with his eyes closed. Note the ability to maintain this position, and possibly record the time the patient can stand on one leg without veering to the side. Have the patient chew an RNA tablet and repeat the

process. If there is no improvement, have the patient chew another tablet and re-test. Continue this procedure until definite improvement is observed, or numerous tablets have been chewed without improvement. The number of tablets needed for therapy is somewhat indicated by the number chewed to obtain results. The most important factor is to re-check the patient's balancing time on subsequent visits, regulating the number of tablets accordingly.

Occasionally a patient's orientation in space will be worse after chewing the RNA. When this happens, have the patient chew an extremely small amount of the substance, similar to a homeopathic dosage. Some people are very sensitive to this substance when chewed. In this case, very small and frequent dosage will usually re-establish normal orientation in space.

Of course, it is necessary to rule out any central nervous system disease.[76] In addition, disturbance of the equilibrium proprioceptors from cranial faults or an upper cervical subluxation or fixation can cause poor orientation in space, as can foot dysfunction and other factors.

Ribonucleic acid may be of value in testing a patient. Sometimes there are obvious health problems even though manual muscle testing does not reveal any dysfunction in the nervous system. The body remembers everything that has ever happened to it. Although the record is present at all times, it cannot always be drawn out at will. To help uncover hidden problems, have the patient chew RNA. RNA appears to enhance the memory pattern, and dysfunction that was previously not apparent appears as indicated by manual muscle testing. Often RNA is supplied in a yeast form. Be certain that the patient is not sensitive to yeast, as in candida albicans.[120,171]

Medication can often cause a confusing pattern as observed by applied kinesiology examination. Some of the medications causing the greatest problems are tranquilizers, mood elevators, birth control pills, and diuretics. When the symptomatic pattern and other aspects of physical examination are not correlating with applied kinesiology tests, it may be due to medication the patient is taking. Often chewing ribonucleic acid will change the results of manual muscle testing so that all aspects of the examination correlate.

Research in Nutritional Testing

Much more research is needed before there will be a thorough understanding of the relationship between chewing nutrition and the performance of a muscle when tested manually. One of the major problems in designing a research study is the number of factors that influence the manual muscle test, such as the cranial-sacral primary respiratory system, reflexes, subluxations, and many others. As Goodheart[59] points out, "... the nutritional factor is only one component of the composite whole of the particular problem posed by the particular patient." The study by Leaf,[108] previously discussed, dramatically shows that there is no one nutritional factor that correlates with a specific muscle; the patient's general health level influences the number and kind of nutrition that will change muscle function. Each individual or group who has done a study is to be congratulated. There have been negative and positive studies indicating that chewing nutrition has an effect on the manual muscle test. From each study something has been learned, ranging from the value of the AK procedures to how to design improved studies.

Rybeck and Swenson[150] evaluated the effect of chewing sugar on the latissimus dorsi muscle, which is associated with the pancreas in applied kinesiology. The population of the study was seventy-three healthy students who were unfamiliar with applied kinesiology. In random questioning, as many believed that sugar might increase strength as decrease it; most had no opinion. The subjects were first evaluated for a normally functioning latissimus dorsi with manual muscle testing. Only those judged to have solid muscle function were used in the study. Two types of muscle tests were performed: 1) the standard manual muscle test used in applied kinesiology, and 2) one against a force transducer.[86] The tester was blind as to whether the subject received a sugar cube or nothing as a control. The hypothesis being tested was whether sugar in the mouth would cause the latissimus dorsi to weaken. The results were insignificant for the mechanical test. For the manual muscle test there was a significant difference between the control and experimental groups. The Wilcoxin Rank Sum Test showed the experimental group to be significantly different from the controlled one ($p=0.0062$).

There are two interesting factors about this study. First, it is not expected that everyone will weaken when sugar is placed in the mouth. As noted previously, depending on the physiologic needs of the body at the time of the test, it is expected that some individuals will strengthen when sugar is placed in the mouth. On a clinical basis it is observed that individuals who have sugar-handling stress more frequently weaken when sugar is placed in the mouth than does the random population. The significantly positive outcome of this study may relate with the population study of students under stress. The second interesting factor is the difference between the manual muscle test results and the muscle test results against a mechanical transducer. The

failure to correlate with the manual muscle test is supported by Blaich's[16] and Blaich and Mendenhall's[17] studies in comparing the manual muscle test with Cybex II testing.

Scopp[157] evaluated the nutrition-muscle association described in applied kinesiology by giving individuals with unilaterally weak muscles either a placebo or the indicated nutrient. Muscle strength was measured by a JayMar dynamometer, with a 21% gain in strength for the nutrient group. This was statistically significant ($p < .05$) as compared with the placebo group. The placebo group showed a small non-significant pre/post decrease in muscle strength. The muscles were tested according to methods described by Kendall and Kendall.[103]

In a double-blind study, Sandweiss[151] tested the pectoralis major (sternal division) muscle on twenty-nine individuals with a normal manual muscle test in the clear, and another group of twenty-six individuals with a pectoralis major (sternal division) muscle that was weak in the clear. The individuals were tested with vitamin A and a placebo. The placebo was manufactured by the same company as the vitamin, so that the taste and appearance of the tablets would be similar. The tablets were administered so that neither the subject nor tester knew which was being tested. The tablet tested first was on a random basis. The placebo effect was definitely operative in this test; however, vitamin A outperformed the placebo in strengthening the pectoralis major (sternal division) by six to one. An interesting aspect of the study is that the placebo effect was more operative when the placebo was the first tablet tested.

It is difficult to design an effective study to evaluate applied kinesiology nutritional testing. Foremost, the designer must be familiar with all aspects of applied kinesiology. A major effort is required to eliminate as many variables as possible. A study by Friedman and Weisberg[50] is an example of a poorly designed study that does not adhere to proper muscle testing principles. Possibly the most important factor in obtaining accurate test results is the examiner's knowledge of ways the subject may change the test parameters to appear strong in the presence of weakness. Subjects will often shift body position, change direction of force, and otherwise modify the test to recruit synergistic muscles. These factors must be observed by the examiner so they can be placed into the equation that finally evaluates whether there is muscle strength or weakness on manual muscle testing. The study by Friedman and Weisberg was designed to evaluate dental vertical dimension, Golgi tendon organ manipulation, and the effect of chewing sugar. In order to make the examiner blind, the subject was placed behind a screen with his arm held out so that the examiner on the other side of the screen could contact the subject's wrist for an arm pull-down type of test. This gives the examiner no ability to control the patient's body shift when he makes an effort to recruit synergistic muscles or otherwise change the test parameters.

A study by Kenney et al.[104] purports to investigate applied kinesiology testing of nutritional supplementation but studies nothing that the ICAK supports or teaches. In addition the study is poorly designed. Nutritional "deficiencies" were evaluated with "Ridler" points or by the use of acupuncture meridians. Ridler points are an arbitrarily designated set of body surface points of unknown value whose use has nothing to do with applied kinesiology. While some aspects of meridian therapy are used in applied kinesiology, they are not used to determine nutritional needs. The diagnostic method used in this study bears no relation to anything supported by the ICAK or in the literature of diplomates of ICAK. This in itself places the study outside of the realm of applied kinesiology. Thorough examination, considering all the alternatives, is necessary to arrive at a final conclusion and diagnosis. This is the main reason that applied kinesiology seminars taught by diplomates of the organization are limited to those licensed to be primary health care professionals. This study was done using two lay persons and a chiropractor whose education in applied kinesiology was not identified. The study implies that the "...chiropractor and two lay-persons ... are recognized and experienced in applied kinesiology techniques." They are not recognized by the ICAK because two participants are lay people, and they used techniques not recognized by the ICAK. The lack of qualification of these practitioners and the fact that what they were doing was not applied kinesiology makes their findings essentially irrelevant.

A study by Triano[170] found that there is no one-to-one association between certain muscle weakness and a specific nutrient that always strengthens the muscle. Research on AK nutritional testing requires a much more complex design. There are many factors that can cause a muscle to test weak that need to be taken into consideration. As noted previously in this chapter, interaction within the body may be the reason that nutrition will strengthen a muscle where no association is apparent.

Continued research is necessary to put in perspective the applied kinesiology evaluation of nutritional effects on body function. It is important to reiterate that the results from AK testing must be correlated with all other standard procedures for determining the patient's needs for nutritional supplementation. AK testing adds a functional dimension to the final prescription.

CHAPTER 4 — REFERENCES

1. Adolph, E.F., "Measurements of water drinking in dogs," *Am J Physiol*, Vol 125 (1938).
2. Adolph, E.F., "Urges to eat and drink in rats," *Am J Physiol*, Vol 151 (1947).
3. Adolph, E.F., "Thirst and its inhibition in the stomach," *Am J Physiol*, Vol 161 (1960).
4. Altman, G.E., & J.E.F. Riseman, "Sublingual erythrol tetranitrate in the treatment of angina pectoris. Effect of varying the dose and rate of administration," *Am J Med Sci*, Vol 240 (Jul 1960).
5. Ambrus, J.L., H.B. Lassman, & J.J. De Marchi, "Absorption of exogenous and endogenous proteolytic enzymes," *Clin Pharmacol Thera*, Vol 8, No 3 (1966).
6. Appledorf, H., & S.R. Tannenbaum, "Specific hunger for thiamine in the rat: Selection of low concentrations of thiamine in solution," *J Nutr*, Vol 92 (1967).
7. Asimov, I., *The Human Brain — Its Capacities and Functions* (New York: New American Library, 1963).
8. Babich, F.R. et al., "Behavioral modifications in naive rats produced by injections of ribonucleic acid extracted from trained rats," *Worm Runner's Digest* (1965).
9. Bach, E., *The Bach Flower Remedies* (New Canaan, CT: Keats Publishing, Inc, 1979).
10. Barrett, S., "Commercial hair analysis — science or scam?" *JAMA*, Vol 254, No 8 (Aug 1985).
11. Barton, J.E., *How to Take Care of Yourselves — Naturally* (Medford, OR: Harman Press, 1977).
12. Barton, J., & M. Barton, *Which Vitamin? Nutritional Care through Muscle Testing* (E. Longmeadow, MA: Celecom Corporation, 1979).
13. Behrman, H.R., & M.R. Kare, "Canine pancreatic secretion in response to acceptable and aversive taste stimuli," *Proc Soc Exp Biol Med*, Vol 129 (1968).
14. Bell, F.R., & R.L. Kitchell, "Taste reception in the goat, sheep and calf," *J Physiol*, Vol 183 (1966).
15. Bellows, R.T., "Time factors in water drinking in dogs," *Am J Physiol*, Vol 128 (1938).
16. Blaich, R.M., "Manual and machine muscle testing before and after correction of 'respiratory faults,'" in *Selected Papers of the International College of Applied Kinesiology*, ed B.C. Markham (Detroit, MI: ICAK, 1981).
17. Blaich, R.M., & E.I. Mendenhall, "Manual muscle testing and Cybex machine muscle testing, a search for a correlation," in *1984 Selected Papers of the International College of Applied Kinesiology*, ed W.H. Schmitt, Jr. (Park City, UT: ICAK, 1984).
18. Borer, K., "Disappearance of preferences and aversions for sapid solutions in rats ingesting untasted fluids," *J Comp Physiol Psychol*, Vol 65, No 2 (1968).
19. Bornstein, W.S., "Cortical representation of taste in man and monkey," *Yale J Biol Med*, Vol 13 (1940).
20. Bott, E., D.A. Denton, & S. Weller, "The innate appetite for salt exhibited by sodium-deficient sheep," in *Olfaction and Taste II*, ed T. Hayashi (New York: Pergamon Press, 1967).
21. Boyd, J.D., in discussion of article by C. Sweet, "Voluntary food habits of normal children," *JAMA*, Vol 107, No 10 (Sep 5, 1936).
22. Bradley, R.M., & C.M. Mistretta, "Investigations of taste function and swallowing in fetal sheep," in *Oral Sensation and Perception — Development in the Fetus and Infant*, ed J.F. Bosma (Bethesda, MD: U.S. DHEW, 1973).
23. Brimhall, J.W., "Inhalation therapy." Proceedings of Summer Meeting, ICAK, Detroit, 1979.
24. Burns, D., "Accumulating scientific evidence supports glandular therapy," *Chiro Econ*, Vol 30, No 3 (Nov/Dec 1987).
25. Chancellor, P.M., ed, *Handbook of the Bach Flower Remedies* (London: The C.W. Daniel Co Ltd, 1971).
26. Christensson, B. et al., "Haemodynamic effects of nitroglycerin in normal subjects during supine and sitting exercise," *Br Heart J*, Vol 31 (1969).
27. Cornwell, P., & G. Cornwell, "The effects of vibration as the UCS in the conditioning of planaria," *Worm Runner's Digest*, Vol 1 (1959).
28. Davis, C.M., "Self selection of diet by newly weaned infants — an experimental study," *Am J Dis Child*, Vol 36, No 4 (Oct 1928).
29. Davis, C.M., "Studies in the self-selection of diet by young children," *JADA*, Vol 21 (Apr 1934).
30. Davis, C.M., "Results of the self-selection of diets by young children," *Can Med Assoc J*, Vol 41 (Sep 1939).
31. De Deuxchaisnes, C. et al., "New modes of administration of salmon calcitonin in Paget's disease," *Clin Orthop*, No 217 (Apr 1987).
32. Denton, D.A., "Evolutionary aspects of the emergence of aldosterone secretion and salt appetite," *Physiol Rev*, Vol 45 (1965).
33. Denton, D.A., "Salt appetite," in *Handbook of Physiology, Sec 6: Alimentary Canal. Vol I. Control of Food and Water Intake*, ed C.F. Code (Washington, DC: American Physiol Soc, 1967).
34. Dolgoff, E.J., "A holographic brain model," in *Holography in Medicine — Proceedings of the International Symposium on Holography*, New York, 1973, ed P. Greguss (Surrey, England: IPC Business Press Ltd, 1975).
35. Epstein, A.N., "Oropharyngeal factors in feeding and drinking," in *Handbook of Physiology, Sec 6: Alimentary Canal. Vol I. Control of Food and Water Intake*, ed C.F. Code, (Washington, DC: American Physiol Soc, 1967).
36. Epstein, A.N., "Feeding without pharyngeal sensations," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
37. Erdie, H. et al., "Absorption and excretion of g-strophanthin after intravenous or sublingual adminstration," *Deutch Med Wochenschc*, Vol 104, No 27 (1979).
38. Erickson, R.P., "Sensory neural patterns and gustation," in *Olfaction and Taste — Proceedings of the First International Symposium*, Stockholm, Sep 1962, ed Y. Zotterman (New York: The Macmillan Co, 1963).
39. Erickson, R.P., "Neural coding of taste quality," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
40. Faden, A.I., "Opiate antagonists and thyrotropin-releasing hormone. I. Potential role in the treatment of shock," *JAMA*, Vol 252, No 9 (Sep 7, 1984).
41. Faden, A.I., "Opiate antagonists and thyrotropin-releasing hormone. II. Potential role in the treatment of central nervous system injury," *JAMA*, Vol 252, No 11 (Sep 21, 1984).
42. Fallen, E. et al., "Relief of coronary artery spasm by nitroglycerin: Time-dependent variability in drug action," *Cathet Cardiovasc Diagn*, Vol 4, No 3 (1978).
43. Fiet, J. et al., "Post-menopausal concentrations of plasma oestradiol, oestrone, FSH and LH and of total urinary oestradiol and oestrone after a single sublingual dose of oestradiol 17B," *Acta Endocrinol*, Vol 101 (Sep 1982).
44. Fischman, W., & M. Grinims, *MRT* (New York: Richard Marek Publishers, 1979).
45. Fishman, I.Y., "Single fiber gustatory impulses in rat and hamster," *J Cell Comp Physiol*, Vol 49 (1957).
46. Fregly, M.J., "Specificity of the sodium chloride appetite of adrenalectomized rats; substitution of lithium chloride for sodium chloride," *Am J Physiol*, Vol 195, No 3 (Dec 1958).
47. Fregly, M.J., "NaCl appetite of adrenalectomized rats," *Soc Exper Biol Med*, Vol 97 (1958).
48. Fregly, M.J., "Specificity of sodium chloride aversion of hypertensive rats," *Am J Physiol*, Vol 196, No 6 (1959).
49. Fregly, M.J., "The role of hormones in the regulation of salt intake in rats," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
50. Friedman, M.H., & J. Weisberg, "Applied kinesiology — double-blind pilot study," *J Prosthet Dent*, Vol 45, No 3 (Mar 1981).

51. Friel, J.P., ed, *Dorland's Illustrated Medical Dictionary*, 25th ed (Philadelphia: W.B. Saunders Co, 1974).
52. Gale, G.D., S. Gallon, & W.R. Porter, "Sublingual Lorazepam: A better premedication?" *Br J Anesthesiol*, Vol 55, No 8 (1983).
53. Gardner, M.L.G., "Intestinal assimilation of intact peptides and proteins from the diet — a neglected field?" *Biol Rev*, Vol 59 (1984).
54. Gibaldi, M., & J.L. Kanig, "Absorption of drugs through the oral mucosa," *J Oral Ther Pharmacol*, Vol 1, No 4 (Jan 1965).
55. Gold, P.W. et al., "Pituitary thyrotropin response to thyrotropin-releasing hormone in affective illness: Relationship to spinal fluid amino metabolites," *Am J Psych*, Vol 134, No 9 (Sep 1977).
56. Goldstein, A.L. et al., "Current status of thymosin and other hormones of the thymus gland," *Recent Progress Hormone Res*, Vol 37 (1981).
57. Goodheart, G.J., Jr., 20567 Mack Avenue, Grosse Pointe Woods, MI 48236. Personal communication.
58. Goodheart, G.J., Jr., *The Cranial Sacral and Nutritional Reflexes and Their Relationship to Muscle Balancing* (Detroit: privately published, 1968).
59. Goodheart, G.J., Jr., "Structural imbalance and nutritional absorption (a new route to the brain)," *Chiro Econ*, Vol 13, No 3 (Nov/Dec 1970).
60. Goodheart, G.J., Jr., "The R.N.A. meridian relationship to applied kinesiology," *Chiro Econ*, Vol 15, No 5 (Mar/Apr 1973).
61. Goodheart, G.J., Jr., *Applied Kinesiology 1979 Workshop Procedure Manual*, 15th ed (Detroit: privately published, 1979).
62. Goodheart, G.J., Jr., *Applied Kinesiology 1987 Workshop Procedure Manual* (Detroit: privately published, 1987).
63. Grossman, S.P., "The central regulation of food and water intake," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
64. Guyton, A.C., & J.E. Hall, *Textbook of Medical Physiology*, 9th ed (Philadelphia: W.B. Saunders Co, 1996).
65. Haigler, E.D., Jr., et al., "Response to orally administered synthetic thyrotropin-releasing hormone in man," *J Clin Endocrinol Metabol*, Vol 35 (Nov 1972).
66. Halpern, B.P., "Some relationships between electrophysiology and behavior in taste," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
67. Harper, A.E., "Dietary protein and amino acids in food intake regulation," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
68. Harriman, A.E., "The effect of a preoperative preference for sugar over salt upon compensatory salt selection by adrenalectomized rats," *J Nutr*, Vol 57 (Oct 1955).
69. Harriman, A.E., & M.R. Kare, "Preference for sodium chloride over lithium chloride by adrenalectomized rats," *Am J Physiol*, Vol 207 (Oct 1964).
70. Harriman, A.E., D.M. Nance, & J.S. Milner, "Discrimination between equimolar NaCl and LiCl solutions by anosmic, adrenalectomized rats," *Physiol Behav*, Vol 3 (1968).
71. Harris, L.J. et al, "Appetite and choice of diet. The ability of the vitamin B deficient rat to discriminate between diets containing and lacking the vitamin," *Proc Roy Soc Lond*, Series B, Vol 113 (1933).
72. Harrower, H.R., *Practical Organotherapy — The Internal Secretions in General Practice,* 3rd ed (Glendale, CA: The Harrower Laboratory, 1922).
73. Harrower, H.R., *An Endocrine Handbook* (Glendale, CA: The Harrower Laboratory, 1939).
74. Hartry, A.L., P. Keith-Lee, & W.D. Morton, "Planaria: Memory transfer through cannibalism reexamined," *Science*, Vol 146 (Oct 1964).
75. Hemmings, W.A., & E.W. Williams, "Transport of large breakdown products of dietary protein through the gut wall," *Gut*, Vol 19 (1978).
76. Henderson, D.J., "Significance of vertebral dyskinesia in relation to the cervical syndrome," *J Manip Physiol Ther*, Vol 2, No 1 (Mar 1979).
77. Henkin, R.I., J.R. Gill, Jr., & F.C. Bartter, "Studies on taste thresholds in normal man and in patients with adrenal cortical insufficiency: The role of adrenal cortical steroids," *J Clin Invest*, Vol 42, No 5 (1963).
78. Henkin, R.I., "Abnormalities of taste and olfaction in various disease states," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
79. Henkin, R.I., comment in chapter on "Specific appetite" by O. Maller, in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
80. Henkin, R.I., comment in chapter on "The interrelations of the chemical senses" by D.G. Moulton, in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
81. Hoeschen, R.J. et al., "Haemodynamic effects of angina pectoris, and of nitroglycerin in normal and anginal subjects," *Br Heart J*, Vol 28 (1966).
82. Holaday, J.W., & E.W. Bernton, "Protirelin (TRH) — a potent neuromodulator with therapeutic potential," *Arch Intern Med*, Vol 144 (Jun 1984).
83. Hollander, F., H.A. Sober, & J. Bandes, "A study of hunger and appetite in a young man with esophageal obstruction and jejunostomy," *Ann NY Acad Sci*, Vol 63 (1955).
84. Hommel, H. et al., "The mechanism of insulin secretion after oral glucose administration," *Diabetologia*, Vol 8 (Apr 1972).
85. Hoshisima, K., "Endocrines and taste," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
86. Human Performance Recorder, produced by Human Performance Systems, P.O. Drawer 1324, Fayetteville, AR 72701.
87. Inglett, G.E. et al., "Taste-modifying properties of miracle fruit (Synsepalum dulcificum)," *J Agr Food Chem*, Vol 13, No 3 (May/Jun 1965).
88. International College of Applied Kinesiology, "Applied Kinesiology — Adopted Status Statement of the ICAK," *Chiro Econ*, Vol 26, No 3 (Nov/Dec 1983).
89. International College of Applied Kinesiology, "Status Statement— International College of Applied Kinesiology," *Am Chiro* (Jan/Feb 1984).
90. Jacobs, H.L., "Taste and the role of experience in the regulation of food intake," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
91. Jacobs, H.L., & K.N. Sharma, "Taste versus calories: Sensory and metabolic signals in control of food intake," *Ann NY Acad Sci*, Vol 157 (1969).
92. Jamieson, T.K., & D. Duffy, *A Manual for Metabolic/Nutritional Evaluation of the SMA/25, CBC w/Differential and Related Tests* (Cannonsburg, MI: Balancing Body Chemistry with Nutrition, 1984).
93. Janowitz, H., & M.I. Grossman, "Effect of parenteral administration of glucose and protein hydrolysate on food intake in the rat," *Am J Physiol*, Vol 155 (Oct 1948).
94. Janowitz, H.D., M.E. Hanson, & M.I. Grossman, "Effect of intravenously administered glucose on food intake in the dog," *Am J Physiol*, Vol 156 (Jan 1949).
95. Janowitz, H.D., & M.I. Grossman, "Some factors affecting the food intake of normal dogs and dogs with esophagostomy and gastric fistula," *Am J Physiol*, Vol 159 (Oct 1949).
96. Janowitz, H.D., & F. Hollander, "The time factor in the adjustment of food intake to varied caloric requirement in the dog: A study of precision of appetite regulation," *Ann NY Acad Sci* (1968).
97. Jansen, E., & H. Klepzig, "Studies on the influence of Molsidomin on coronary heart disease," *Med Klin*, Vol 71, No 47 (1979).
98. Kakolewski, J.W., V.C. Cox, & E.S. Valenstein, "Short-latency antidiuresis following the initiation of food ingestion," *Science*, Vol 162 (Oct 25, 1968).
99. Kare, M.R., comment in chapter on "Feeding without oropharyngeal sensations" by A.N. Epstein, in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).

100. Kare, M.R., "Digestive functions of taste stimuli," in *Olfaction and Taste*, ed C. Pfaffmann (New York: The Rockefeller U Press, 1969).
101. Kare, M.R. et al., "Direct pathway to the brain," *Science*, Vol 163 (Feb 1969).
102. Kare, M.R., "Some functions of the sense of taste," *J Agr Food Chem*, Vol 17, No 4 (Jul/Aug 1969).
103. Kendall, H., F. Kendall, & G. Wadsworth, *Muscles — Testing and Function*, 2nd ed (Baltimore: Williams & Wilkins, 1971).
104. Kenney, J.J., R. Clemens, & K.D. Forsythe, "Applied kinesiology unreliable for assessing nutrient status," *J Am Diet Assoc*, Vol 88, No 6 (Jun 1988).
105. Krarup, B., "On the technique of gustatory examinations," *Acta Otolaryngol*, Supp 140 (1958).
106. Krarup, B., "Electro-gustometry: A method for clinical taste examinations," *Acta Otolaryngol*, Vol 49, No 4 (Jul/Aug 1958).
107. Lang, I. et al., "Glutaurine enhances the depressed NK cell activity of tumor patients," *Immunolog Communications*, Vol 12, No 5 (1983).
108. Leaf, D.W., "Nutrient testing evaluation." Proceedings of Summer Meeting, ICAK, Santa Monica, CA, 1985.
109. Lepkovsky, S., "The physiological basis of voluntary food intake (Appetite?)," in *Advances in Food Research*, Volume I, ed E.M. Mrak & G.F. Stewart (New York: Academic Press, Inc, 1948).
110. Lepkovsky, S., "The role of the chemical senses in nutrition," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (New York: Academic Press, 1977).
111. Maller, O., "Specific appetite," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
112. Maller, O. et al., "Movements of glucose and sodium chloride from the oropharyngeal cavity to the brain," *Nature* (Feb 18, 1967).
113. Maller, O., & J.A. Desor, "Effect of taste on ingestion by human newborns," in *Oral Sensation and Perception — Development in the Fetus and Infant*, ed J.F. Bosma (Bethesda, MD: U.S. DHEW, 1973).
114. Mason, D.T., & E. Braunwald, "The effects of nitroglycerin and amyl nitrite on arteriolar and venous tone in the human forearm," *Circulation*, Vol 32 (Nov 1965).
115. Miles, T.S., & P.G. Dellow, "The oral absorption of water," *Can J Physiol Pharmacol*, Vol 49 (1971).
116. Miller, J.M., "The absorption of proteolytic enzymes from the gastrointestinal tract," *Clin Med*, Vol 10 (Oct 1968).
117. Miller, J.M., C.J. Papastrat, & M. Ginsberg, "The administration of papain buccally in the treatment of inflammation and edema," *Antibiot Med Clin Ther*, Vol 5 (1958).
118. Monmaney, T., "Are we led by the nose?" *Discover*, Vol 8, No 9 (Sep 1987).
119. Moulton, D.G., "The interrelations of the chemical senses," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
120. Mowles, R., & W.H. Schmitt, Jr., "An applied kinesiological approach to candida albicans allergy," *Chiro Econ*, Vol 28, No 4 (Jan/Feb 1986).
121. Nachman, M., "Taste preferences for sodium salts by adrenalectomized rats," *J Comp Physiol Psychol*, Vol 55, No 6 (1962).
122. Nachman, M., & C. Pfaffmann, "Gustatory nerve discharge in normal and sodium-deficient rats," *J Comp Physiol Psychol*, Vol 56, No 6 (1963).
123. Nachman, M., "Learned aversion to the taste of lithium chloride and generalization to other salts," *J Comp Physiol Psychol*, Vol 56, No 2 (1963).
124. Nachman, M., & L.P. Cole, "Role of taste in specific hungers," in *Taste*, ed L.M. Beidler (New York: Springer-Verlag, 1971).
125. Naim, M., & M.R. Kare, "Taste stimuli and pancreatic functions," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (New York: Academic Press, 1977).
126. Neumann, K., "Basic pharmacological studies concerning cellular therapy," in *Cell Research and Cellular Therapy*, ed F. Schmid & J. Stein (Thoune, Switzerland: Ott Publishers, 1967).
127. Nicolaidis, S., "Early systemic responses to orogastric stimulation in the regulation of food and water balance: Functional and electrophysiological data," *Ann NY Acad Sci*, Vol 157 (1969).
128. Nicolaidis, S., "Sensory-neuroendocrine reflexes and their anticipatory and optimizing role on metabolism," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (New York: Academic Press, 1977).
129. Nisbett, R.E., & S.B. Gurwitz, "Weight, sex, and the eating behavior of human newborns," *J Comp Physiol Psychol*, Vol 73, No 2 (1970).
130. Oakley, B., & C. Pfaffmann, "Electrophysiologically monitored lesions in the gustatory thalamic relay of the albino rat," *J Comp Physiol Psychol*, Vol 55, No 2 (1962).
131. Pangborn, R.M., "Some aspects of chemoreception in human nutrition," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
132. Parsons, L.G., "Scurvy treated with ascorbic acid," *Clin Orthop*, No 222 (Sep 1987).
133. Patton, H.D., "Taste and Olfaction," in *Physiology and Biophysics, I: The Brain and Neural Function*, 20th ed, ed T. Ruch & H.D. Patton (Philadelphia: W.B. Saunders Co, 1979).
134. Pert, C.B., "The wisdom of the receptors: Neuropeptides, the emotions, and bodymind," *Advances*, Vol 3, No 3 (Summer 1986).
135. Peshek, R.J., *Balancing Body Chemistry with Nutrition* (Riverside, CA: Color Coded Systems, 1977).
136. Peshek, R.J., ed, *Nutrition for a Healthy Heart — for the Treatment of Pain* (Riverside, CA: Color Coded Charting, 1979).
137. Pfaffmann, C., "Taste preference and aversion following lingual denervation," *J Comp Physiol Psychol*, Vol 45 (1952).
138. Pfaffmann, C., "The pleasures of sensation," *Psychol Rev*, Vol 67, No 4 (1960).
139. Pfaffmann, C., "Critic's comments," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
140. Pietsch, P., *Shufflebrain* (Boston: Houghton Mifflin Co, 1981).
141. Pilcher, C.W.T., S.P. Jarman, & D.A. Booth, "Route of glucose to the brain from food in the mouth of the rat," *J Comp Physiol Psychol*, Vol 87, No 1 (1974).
142. Richter, C.P., "Total self regulatory functions in animals and human beings," in *The Harvey Lectures, Series XXXVIII*, ed Harvey Society of New York (Lancaster, PA: Science Press Printing Co, 1943).
143. Richter, C.P., "Salt appetite of mammals: Its dependence on instinct and metabolism," in *L'Instinct dans Le Comportement des Animaux et de L'Homme*, ed M. Autuori et al. (Paris, France: Masson Et Cie Editeurs, 1956).
144. Richter, C.P., & J. F. Eckert, "Mineral metabolism of adrenalectomized rats studied by the appetite method," *Endocrinology*, Vol 22 (1938).
145. Robbins, L.J., "Dry mouth and delayed dissolution of sublingual nitroglycerin," *N Engl J Med*, Vol 309, No 16 (Oct 20, 1983).
146. Rodgers, W., & P. Rozin, "Novel food preferences in thiamine-deficient rats," *J Comp Physiol Psychol*, Vol 61, No 1 (1966).
147. Rodgers, W.L., "Specificity of specific hungers," *J Comp Physiol Psychol*, Vol 64, No 1 (1967).
148. Roe, D.A., *Drug-Induced Nutritional Deficiencies* (Westport, CT: Avi Publishing Co, Inc, 1976).
149. Rozin, P., "Adaptive food sampling patterns in vitamin deficient rats," *J Comp Physiol Psychol*, Vol 69, No 1 (1969).
150. Rybeck, C.H., & R. Swenson, "The effect of oral administration of refined sugar on muscle strength," *J Manip Physiol Ther*, Vol 3, No 3 (Sep 1980).
151. Sandweiss, Y.J., "An effect of vitamin A on pectoralis major sternal muscle dysfunction." Unpublished paper, Aug 7, 1984.
152. Schoenthaler, S.J., "Commercial hair analysis: Lack of reference norms and high reliability within and between seven selected laboratories for seventeen trace minerals," *International J Biosocial Res*, Vol 8, No 1 (1986).
153. Schoenthaler, S., "An alternate view of the accuracy of hair analysis," *Comp Med Magazine*, Vol 2, No 2 (Nov/Dec 1986).
154. Schroeder, H.A., *The Poisons Around Us* (New Canaan, CT: Keats

Publishing, Inc, 1978).
155. Schulof, R.S. et al., "Phase I/II trial of thymosin fraction 5 and thymosin alpha one in HTLV-III seropositive subjects," *J Biol Response Modifiers*, Vol 5 (1986).
156. Schwartz, J.P., "Some dangers in the development of new techniques in applied kinesiology." Proceedings of Winter Meeting, ICAK, St Maarten, 1980.
157. Scopp, A.L., "An experimental evaluation of kinesiology in allergy and deficiency disease diagnosis," *J Orthomolec Psychiatry*, Vol 7, No 2 (1978).
158. Scott, E.M., & E. Quint, "Self-selection of diet. III. Appetites for B vitamins," *J Nutr*, Vol 32 (Sep 1946).
159. Scott, E.M., & E. Quint, "Self-selection of diet. IV. Appetite for protein," *J Nutr*, Vol 32 (1946).
160. Scott, E.M., & E.L. Verney, "Self-selection of diet. VI. The nature of appetites for B vitamins," *J Nutr*, Vol 34 (Nov 1947).
161. Share, I., E. Martyniuk, & M.I. Grossman, "Effect of prolonged intragastric feeding on oral food intake in dogs," *Am J Physiol*, Vol 169 (Apr 1952).
162. Sheinkin, D., M. Schacter, & R. Hutton, *The Food Connection* (Indianapolis: The Bobbs-Merrill Co, 1979).
163. Smith, M.P., & P.J. Capretta, "Effective drive level and experience on the reward value of saccharine solutions," *J Comp Physiol Psychol*, Vol 49 (1956).
164. Snapper, I., "The etiology of different forms of taste behavior," in *The Chemical Senses and Nutrition*, ed M.R. Kare & O. Maller (Baltimore: The Johns Hopkins Press, 1967).
165. Starzl, T.E. et al., "Growth-stimulating factor in regenerating canine liver," *Lancet* (Jan 20, 1979).
166. Stevenson, J.A.F., "Sensory mechanisms and multi-factor regulation of food and water intake," *Ann NY Acad Sci* (May 15, 1969).
167. Sweet, C., "Voluntary food habits of normal children," *JAMA*, Vol 107, No 10 (Sep 5, 1936).
168. Teitelbaum, P., & A.N. Epstein, "The role of taste and smell in the regulation of food and water intake," in *Olfaction and Taste — Proceedings of the First International Symposium*, Stockholm, Sep 1962, ed Y. Zotterman (New York: The Macmillan Co, 1963).
169. Thie, J.F., *Touch for Health*, revised ed (Marina del Rey, CA: DeVorss & Co, 1979).
170. Triano, J.J., "Muscle strength testing as a diagnostic screen for supplemental nutrition therapy: A blind study," *J Manip Physiol Ther*, Vol 5, No 4 (Dec 1982).
171. Truss, C.O., *The Missing Diagnosis* (Birmingham, AL: privately published, 1983).
172. Valenstein, E.S., comment in article on "Early systemic responses to orogastric stimulation in the regulation of food and water balance: Functional and electrophysiological data" by S. Nicolaidis, *Ann NY Acad Sci*, Vol 157 (1969).
173. Weeks, N., *The Medical Discoveries of Edward Bach, Physician* (New Canaan, CT: Keats Publishing, Inc, 1979).
174. Wellness Letter Staff, "A food label lexicon," *Wellness Letter* (U of CA, Berkeley), Vol 3, No 12 (Sep 1987).
175. Whitman, M.E., "The complete blood count and its clinical nutritional implications," *Chiro Econ*, Vol 29, No 5 (Mar/Apr 1987).
176. Whitman, M.E., "Blood chemistry (SMAC) and its clinical nutritional implications," *Chiro Econ*, Vol 29, No 6 (May/Jun 1987).
177. Whitman, M.E., "Urinalysis and its clinical nutritional implications — Part 4," *Chiro Econ*, Vol 30, No 1 (Jul/Aug 1987).
178. Williams, R.J., *Biochemical Individuality — The Basis for the Genetotrophic Concept* (Austin, TX: Univ of Texas Press, 1956).
179. Willis, W.D., Jr., & R.G. Grossman, *Medical Neurobiology*, 3rd ed (St. Louis: The C.V. Mosby Co, 1981).
180. Wolf, S., & H.G. Wolff, *Human Gastric Function* (New York: Oxford University Press, 1943).
181. Wynder, E.L., "Cancer prevention," in *1981 Medical and Health Annual*, ed E. Bernstein (Chicago: Encyclopaedia Britannica, 1980).
182. Yamamoto, T., "Neural mechanisms of taste function," in *Frontiers of Oral Physiology, Vol 4 — Oral Sensory Mechanisms*, ed Y. Kawamura (New York: S. Karger, 1983).
183. Yamamoto, T. et al., "Hypoglossal motor nerve activity elicited by taste and thermal stimuli applied to the tongue in rats," *Brain Res*, Vol 238 (1982).
184. Yamamoto, T. et al., "Gustatory reaction time under variable stimulus parameters in human adults," *Physiol Behav*, Vol 29 (1982).
185. Yokohama, S. et al., "Intestinal absorption mechanisms of thyrotropin-releasing hormone," *J Pharm Dyn*, Vol 7 (1984).
186. Zotterman, Y., "The recording of the electrical response from human taste nerves," *Handbook Sensory Physiol*, Vol 4 (1971).

Chapter 5

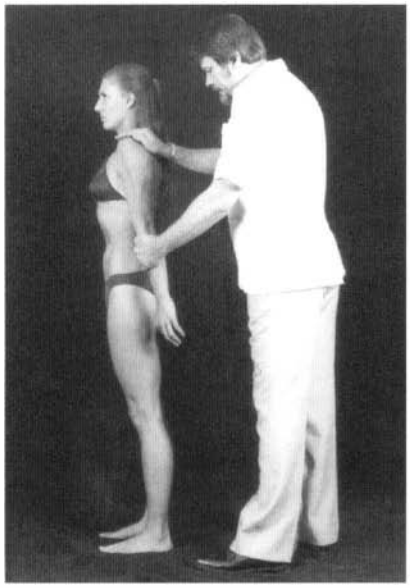
Neurologic Disorganization

Neurologic Disorganization

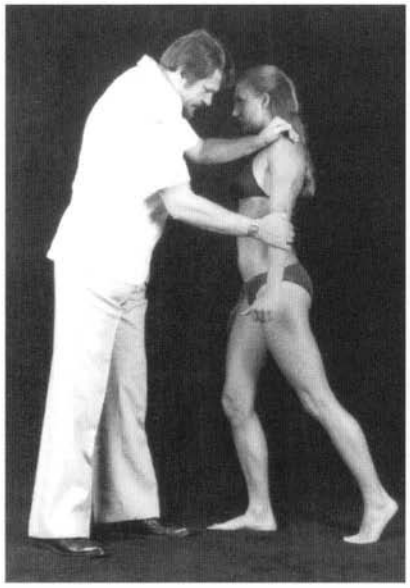
When a person is healthy, muscles function in a predictable manner. Under certain conditions, muscles should test strong with manual muscle testing; under other conditions, they normally test weak. An example of this is shoulder flexor and extensor facilitation and inhibition during gait. This can easily be demonstrated in a normal individual by first testing the general shoulder flexors and extensors with the person standing; normally the muscles will test strong. When the subject is put in a simulated gait position, there will be inhibition of one of the groups. The simulated gait position is static, with the majority of weight on the leading leg. In this position, the shoulder flexors on the leading leg side and the extensors on the trailing leg side test weak, which is the normal function coinciding with the arm swing of gait.

When one performs the simulated gait test, care should be taken in putting the subject into as proper a gait position as possible, paying attention to the usual knee, ankle, and forefoot positions. When the general shoulder flexors and extensors are tested, the subject should not be allowed to rotate the trunk and change the parameters of the test. It may be easier to test an individual shoulder flexor or extensor, such as the latissimus dorsi, for shoulder extension.

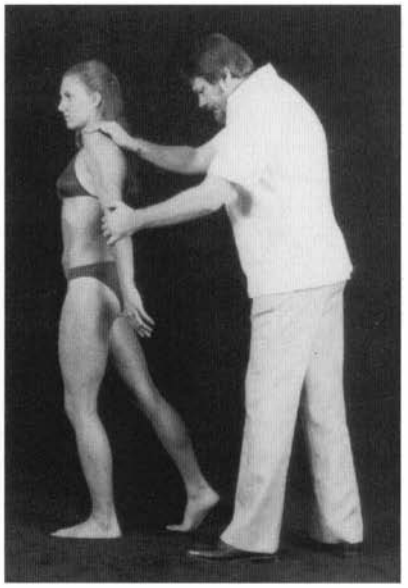
Inhibition of the shoulder flexors or extensors with gait position results from the change of stimulation to the proprioceptors of the joints, muscles, and skin. This afferent supply is then mediated in the central nervous system to cause facilitation and inhibition of not only the shoulder flexors and extensors but also all other muscles alternately active during gait. This normal action can be disturbed if there is improper transmission from the afferent receptors. For one reason or another, it appears that any of the receptors can be stimulated or malfunction to create inappropriate afferent impulses. The central nervous system, acting on the erroneous afferent information, causes inappropriate facilitation and inhibition of muscles. This can readily be demonstrated in an individual who tests normal on the above gait demonstration. Place pencils under the 1st and 5th metatarsals of the leading leg in the simulated gait position. This creates artificial subluxations in the metatarsal arch, somewhat similar to dropped metatarsals. The proprioceptors of the joints and muscles are then inappropriately stimulated, sending afferent impulses not in keeping with normal gait activity. In nearly all cases, facilitation and inhibition of the shoulder flexors and extensors will no longer be predictable. Some or all of the muscles will not test either strong or weak to correlate with proper facilitation and inhibition. In rare cases, the artificially created subluxations in the foot will not change the predictability. It appears that these individuals have highly organized nervous systems that are capable of rapidly adapting to the inappropriate stimulation from the forefoot receptors. One must recognize that there are receptors in other areas of the foot, leg, knee, hips, and pelvis reporting the simulated gait position.



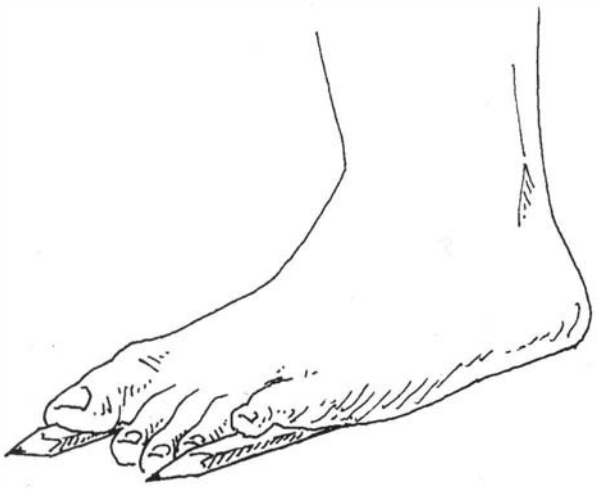
5—1. Shoulder flexors being tested in normal, balanced position.



5—2. With the contralateral leg forward, shoulder extensors will test weak in a normal subject.



5—3. With ipsilateral leg forward, the shoulder flexors will test weak.



5—4. Pencils placed under the 1st and 5th metatarsals create artificial subluxations, altering the afferent receptor response.

Neurologic disorganization appears to result from afferent receptors sending conflicting information for interpretation by the central nervous system. The pencils placed under the 1st and 5th metatarsals stimulate the forefoot in a manner different from the normal gait position. The receptors in the rest of the foot, ankle, leg, knee, hips, and pelvis continue to send information of a normal gait position. Since the central nervous system can only act on the information it receives, the resulting inhibition and facilitation of muscles is not in keeping with the gait position. Subluxations of the foot appear to improperly stimulate the joint receptors in a manner similar to the simulated subluxations caused by the pencils.

Improper afferent stimulation is not limited to joint subluxations. The many types of nerve receptors in the body can be inappropriately stimulated in a manner similar to the demonstration with pencils under the forefoot. Trauma to ligaments, muscles, fascia, skin, and many other structures can parallel the improper stimulation to nerve receptors in the "pencil under the forefoot" demonstration. Disorganization from these factors relates to the structural side of the triad of health. Inappropriate stimulation to the chemical and mental sides of the triad can also be responsible for unpredictable muscle function in a manual test.

Early in applied kinesiology Goodheart recognized unpredictable muscle function. A high shoulder without upper trapezius involvement is usually caused by a weak latissimus dorsi muscle. In some cases, the latissimus dorsi fails to test weak on the high shoulder side; rather, it tests weak on the low shoulder side. Another example is muscle weakness associated with a deficient meridian. If the circulation sex meridian is deficient on the right and normal on the left, weakness of the gluteus maximus and medius — if present — should be on the right. An example of disorganization is when the gluteus maximus is weak on the left and the gluteus medius weak on the right. Since disorganization is often related to right and left switching of function, the term "switching" was coined to describe the disorganization. Because the disturbance always appears to relate in one way or another with the nervous system, a more descriptive term is "neurologic disorganization."

Sensory Nerve Receptors

The basic types of sensory receptors[8] are as follows:

1. **Mechanoreceptors** are stimulated by mechanical deformation of the receptor, or of cells adjacent to the receptor. They include — among others — joint, muscle, and skin receptors, baroreceptors for detection of fluid pressure changes, and the equilibrium receptors.
2. **Thermoreceptors** detect changes in cold or heat.
3. **Nociceptors** detect pain and are primarily responsible for detecting damage in the tissues by physical or chemical means.
4. **Electromagnetic receptors** are recognized in standard physiology as being those of the rods and cones of the eye. Additionally, in applied kinesiology we recognize meridian and other acupuncture points as electromagnetic receptors.
5. **Chemoreceptors** detect various chemical changes in the body. Important in applied kinesiology are the receptors in the hypothalamus and its relation to taste, as well as the general chemical evaluation of body function.
6. To this standard list of sensory receptors we add **mental receptors**, including how mental processes affect the nervous system and in turn affect health, both positively and negatively. It appears that stimulation to any of the sensory receptors not in keeping with the body's needs causes neurologic disorganization, with resulting disturbed function.

When one understands the cause of neurologic disorganization, such as improper function of the shoulder flexors and extensors when pencils are placed under the foot, it is predictable disorganization. In other words, when subluxations are created in the foot from standing on the pencils, it is expected that the nerve endings are improperly stimulated, with resultant shoulder muscle dysfunction. On the other hand, when the latissimus dorsi is strong in the presence of a high shoulder and there is no other muscle dysfunction or structural distortion, one may not be able to understand the presence of this discrepancy; thus the neurologic disorganization is unpredictable, i.e., the cause is unknown.

Goodheart[6] found that simultaneously stimulating acupuncture point KI 27 (located at the junction of the clavicle, 1st rib, and sternum) and the umbilicus causes the latissimus dorsi on the high shoulder side to test weak, as would be expected. The stimulation also eliminates other disorganization, at least temporarily.

When using manual muscle testing as an indicator

for therapeutic approach, it is necessary that the nervous system be organized to provide correct information; otherwise, therapy might be directed to the wrong area. Disorganization may also result in failure to find dysfunction, or may indicate problems that are not actually present.

The standard AK method for determining if a person is neurologically disorganized is testing KI 27 with therapy localization. Positive findings indicate probable neurologic disorganization. A routine procedure has been to stimulate KI 27-umbilicus when positive therapy localization at KI 27 is present.

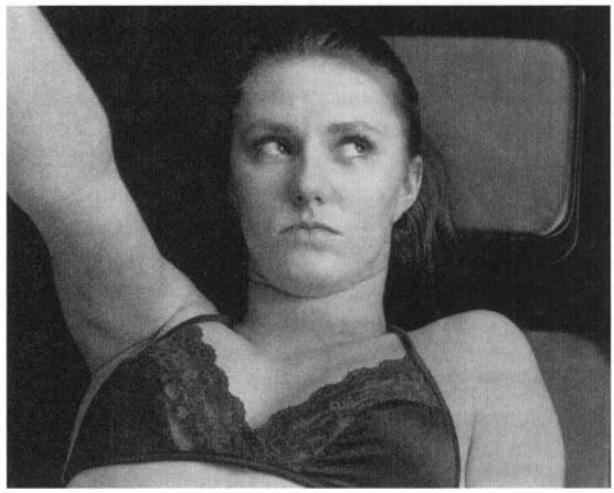
It has been readily observed that some patients (those who responded well) had a positive KI 27 only on the initial or first few visits; thereafter, therapy localization to KI 27 was negative. Patients who fail to respond adequately often continue to show a positive KI 27 with therapy localization on repeated examinations. Whenever there is neurologic disorganization, there is always a basic underlying cause. In most instances it is possible to find that cause and correct it, eliminating the neurologic disorganization. (This is accomplished with techniques discussed later.) To routinely find the cause requires considerable knowledge about applied kinesiology examination techniques and the application of treatment used for correction.

When an individual is first learning applied kinesiology, there will often be times when the cause of neurologic disorganization cannot be determined. During this stage of the learning process, it is valuable to use the KI 27-umbilicus stimulation technique and others developed early in applied kinesiology to "unswitch" the patient and avoid inappropriate treatment. Additional methods of unswitching a patient are auxiliary KI 27, GV-CV connection, and nasal tap.

Ocular Lock

An example of and common test for neurologic disorganization is "ocular lock," which is a failure of the eyes to work together effectively.

Eye motion and position are intricately involved with the equilibrium proprioceptors, which include the visual righting, labyrinthine, and head-on-neck reflexes. When the eyes are turned in a specific direction and a previously strong muscle weakens, it is a positive ocular lock. Frequently when ocular lock is present, there will also be positive therapy localization to KI 27. Ocular lock can also be observed when the examiner moves his finger in a circle for the patient's eyes to follow. This is first done clockwise or counterclockwise, and a previously strong indicator muscle is tested for weakening. Then it is done in the opposite direction, and again a strong indicator muscle is tested. Usually the patient with positive ocular lock will weaken when the eyes go in only one circular direction; occasionally, weakening can develop in both directions. When ocular lock is present with the eyes following the circling finger, there is usually a saccadic movement — a quick, jerky motion — of the eyes at a particular portion of the circle. It is at the point of the saccade that the individual usually weakens when the eyes are held in that direction. Ocular lock can usually be temporarily eliminated by treatment of KI 27-umbilicus. The usual basic cause of ocular lock is a cranial fault.

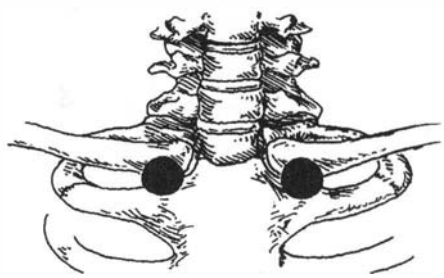


5—5. When movement of the eyes is not organized, there is lack of organization within the equilibrium proprioceptors.

Organization

KI 27-Umbilicus. KI 27-umbilicus stimulation is indicated when there is positive therapy localization at KI 27 and lack of predictable results with manual muscle testing. One must remember that therapy localization tells that something is dysfunctioning at the area being therapy localized, but it does not tell **what**. In the general area of KI 27 there could also be a positive neurolymphatic reflex for the intrinsic spinal muscles, subluxation or strain of the sternoclavicular articulation, or 1st rib subluxation.

The KI 27-umbilicus method of treating neurologic disorganization is to first vigorously stimulate one KI 27 and the umbilicus for about twenty seconds; then the other KI 27 point is vigorously stimulated along with the umbilicus for twenty seconds. This should result in negative therapy localization at KI 27 and predictable results on manual muscle testing. If ocular lock is present prior to KI 27-umbilicus stimulation, re-evaluate by having the



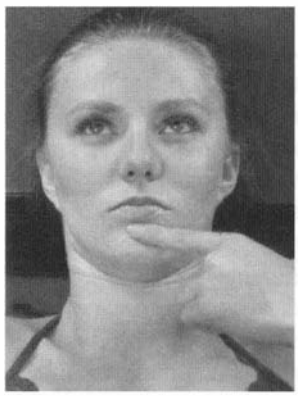
5—6. Location of Kidney 27 (KI 27).

patient put his eyes in the position that previously caused an indicator muscle to weaken. In most cases the muscle will no longer weaken with the eye position, indicating a positive effect from the stimulation.

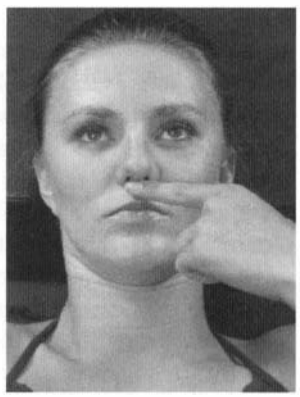
Nasal Tap.[7] If there is a positive ocular lock and KI 27 and the umbilicus have been treated, one can test further by having the patient maintain the positive eye position and quickly take two deep nasal sniffs. If the indicator muscle weakens, rapidly tap the bridge of the patient's nose on both sides for sixty seconds. Following the tapping, ocular lock should not be present after two deep nasal sniffs. It is unknown exactly what is affected by the tapping. It does help to temporarily organize the patient for examination and treatment. It seems likely that the tapping affects the cranial primary respiratory mechanism to temporarily eliminate the ocular lock.

Auxiliary KI 27. If muscle function fails to organize after stimulating KI 27-umbilicus, one should consider other applied kinesiology methods to unswitch an individual. Additional stimulation points are dubbed the auxiliary KI 27 points, located adjacent to the transverse processes of the 11th thoracic vertebra bilaterally. If these points therapy localize, stimulate them one at a time while simultaneously stimulating the umbilicus. Re-evaluate the individual for positive therapy localization adjacent to T11. Again, one must remember that other factors can therapy localize at this point, such as vertebral subluxations. If stimulation of auxiliary KI 27 and the umbilicus is successful in unswitching the individual, there will be predictable muscle function on manual muscle testing.

Conception Vessel-Governing Vessel. Switching can sometimes be temporarily eliminated by connecting the energy patterns of the governing vessel and conception vessel, two midline meridians of energy in the meridian system. This is evaluated by therapy localizing CV 24 or GV 27. CV 24 is the last point on the conception vessel meridian, located in the centerline just below the lower lip. GV 27 is the next-to-last point on the governing vessel meridian, just above the upper lip. When therapy localization to either of these points is positive, contact CV 24 and CV 2 (upper symphysis pubis) simultaneously with a solid pressure for twenty to thirty seconds. Next, contact the luo point (connector point) of GV 1 (located at the tip of the coccyx) and CV 2, and hold simultaneously for twenty to thirty seconds. There will often be a vertebral subluxation in the vicinity of the associated point for the governing vessel, which is BL 16. BL 16 is located close to T6 and 7. Challenge these vertebrae for a subluxation and correct in the usual manner, if present. Re-therapy localize GV 27 and CV 24 to determine if correction of switching has been obtained.



5—7. CV 24.



5—8. GV 27.

Therapy localization.

Finding the Cause of Neurologic Disorganization

When neurologic disorganization is treated by stimulating KI 27-umbilicus, umbilicus-auxiliary KI 27, or GV-CV treatment, the correction is only temporary unless the cause of the neurologic disorganization is also found and corrected. The return of disorganization without other treatment can nearly always be demonstrated after eliminating the positive therapy localization or ocular lock by the methods previously described. Have the patient perform normal daily functions that may include walking, moving the mandible and clenching the teeth, eating a meal, or any other regular activity. If the factor that is the basic cause of the neurologic disorganization is stressed, such as use of the feet when there are foot subluxations, evidence of the neurologic disorganization will return.

The stimulation methods discussed to temporarily eliminate neurologic disorganization are valuable for organizing an individual so that examination and treatment can proceed when the basic cause of the neurologic disorganization cannot be found. If the disorganization does not return, it is because treatment has eliminated the basic cause. This is fine when it works, but it is making the correction by accident rather than on purpose. If one is familiar with most of the examination and treatment techniques in applied kinesiology, the basic cause of neurologic disorganization can nearly always be determined and corrected. This, then, is correction by design, not by accident.[16]

Discussed first is switching in the clear, which simply means that positive therapy localization to KI 27 is present with the patient supine. Hidden switching will be discussed. This is present when an individual shows evidence of neurologic disorganization only under certain conditions; there is no positive therapy localization to KI 27 with the patient supine. It appears that all problem patients have neurologic disorganization, but it may not be present in the clear.

To find the cause of neurologic disorganization,

positive therapy localization at KI 27 is used as a tool for further examination. Stimulation is not applied to KI 27 and umbilicus to organize the individual. The basic concept is to use applied kinesiology examination tools to find what eliminates the positive therapy localization to KI 27. For example, the patient therapy localizes to bilateral KI 27 points and a strong indicator muscle is tested. When the indicator muscle tests weak with the therapy localization, there is evidence of switching. The examiner proceeds to evaluate various areas and functions of the body, as indicated by body language. For example, the examiner may observe calluses under the mid-distal metatarsals, indicating a dropped metatarsal arch that would be similar to the example of placing pencils under the 1st and 5th metatarsals. Using this clue, he has the patient continue to therapy localize the KI 27 points while he challenges the metatarsal bones in a direction for probable correction. If the dropped metatarsal arch is the cause of the neurologic disorganization, the positive KI 27 therapy localization will be eliminated when the proper vector of correction is obtained.

Challenge, therapy localization, nutritional oral stimulation, and body movement can be used to find the cause of neurologic disorganization in a similar manner. When the factor is found that eliminates the positive therapy localization to KI 27, it is treated; this should eliminate any further positive therapy localization to KI 27 without stimulating it. If the correction holds and there is no other factor causing neurologic disorganization, evidence of it will not recur.

Any factor in the triad of health — structural, chemical, or mental — can cause neurologic disorganization. Since most doctors tend to be oriented to one side of the triad, it is valuable to make a concerted effort to consider all of its aspects as possible causes of neurologic disorganization.

Structural. The most common cause of neurologic disorganization is dysfunction of the cranial-sacral primary respiratory mechanism. When ocular lock is present, one will nearly always find the cause of neurologic disorganization in this system. It may require treatment to the stomatognathic system in general, which includes jaw function, dental occlusion, cranial faults, and cervical spine function. Probably the reason so much neurologic disorganization is caused by dysfunction of the stomatognathic system is the intricate relationship between the system and the equilibrium proprioceptors. (The stomatognathic system is discussed in Chapter 9.)

To evaluate the stomatognathic system as a cause of neurologic disorganization, one uses the tools of applied kinesiology to determine what will eliminate the positive therapy localization to KI 27. It may be eliminated by a phase of respiration, challenge to an area of the skull, having the patient stretch the jaw wide open, or moving the jaw into a certain position. Movement of the jaw pulls on the bones of the skull by way of the masticatory muscles and, in effect, is a type of challenge to the skull.

Unless the basic underlying cause of switching is found, it is mandatory to use the unswitching techniques previously described before treatment so that improper treatment is not applied as a result of erroneous examination findings. Recognize that the results of many muscle tests are different after an individual is unswitched with the techniques previously discussed.

When a patient has positive therapy localization to KI 27, the only factors that should be treated are those which, when challenged, therapy localized, or otherwise evaluated, eliminate the positive KI 27. One will find that the challenge and other examination information in applied kinesiology are the same for the factors that eliminate the positive KI 27 before or after it and the umbilicus are digitally stimulated. This does not imply that one should vigorously stimulate KI 27 and the umbilicus when treating the cause of neurologic disorganization. When the proper cranial fault or other causative factor is corrected, there will no longer be positive therapy localization to KI 27.

Whenever the stomatognathic system is treated, the pelvis should be evaluated for category I, II, and III faults. The sacrum should also be routinely evaluated when cranial faults have been corrected.

The second most common cause of neurologic disorganization on a structural basis is foot dysfunction, which may be excessive pronation, tarsal tunnel syndrome, individual subluxations, and/or muscle dysfunction.

Almost any malfunction examined and treated by applied kinesiology methods can be the cause of neurologic disorganization. After the cranial-sacral primary respiratory system and foot dysfunction, the more common causes of neurologic disorganization are equilibrium reflex synchronization, PRYT, gait organization, and dural tension. On a less frequent basis, one should consider everything else treated in applied kinesiology as being a potential cause of neurologic disorganization, including local muscle dysfunction, spinal subluxations, and active reflexes, among others.

At one time, cross-pattern exercise (page 178) was routinely prescribed when an individual was switched. With more effective current methods for finding the basic underlying cause of the neurologic disorganization, cross patterning is not usually necessary for lasting corrections. It is appropriate when a child fails to go through the developmental stages, especially the bilateral phase, prior to developing lateral dominance. This may happen as a result of trauma or parents restricting normal development.

Chemical. Chemical causes of in-the-clear neurologic disorganization usually relate to some form of nutrition, which in one way or another influences the neurotransmitters. Having the patient chew an adrenal substance or choline may eliminate the positive therapy localization to KI 27. Goodheart[7] relates ribonucleic acid (RNA) to the foundation upon which memory builds. It has been used in applied kinesiology as a method to bring out hidden faults during examination. Chewing RNA may

eliminate positive therapy localization to KI 27. In this instance it appears that the reason RNA brings out hidden faults is that they were hidden by neurologic disorganization.

Mental. Mental causes of neurologic disorganization may be intrinsic to the patient's physiology, or extrinsic in his environment and interaction with people. Sometimes positive TL to KI 27 can be eliminated by having the patient therapy localize to the bilateral frontal bone eminences. This is the location of the neurovascular points for the pectoralis major (clavicular division). Successful treatment to the emotional neurovascular points will eliminate the positive therapy localization to KI 27.

Another type of emotional neurologic disorganization is evidenced by cross-therapy localization to KI 27, in which the right hand therapy localizes the left KI 27 and vice versa. Care should be taken that the two hands do not contact each other, and that the therapy localization is actually over the KI 27 point. This type of therapy localization is present in an individual who has a homolateral crawl pattern, discussed under "Cross Patterning." It is often associated with aberrant sensory disturbances and may correlate with schizophrenia. There is additional discussion of this type of disturbance on page 438 and elsewhere.[5.15]

Hidden Switching

Many problem patients fail to respond because they are not examined in the way in which they live. Most examination is done with the patient in a prone, supine, or standing static position. Under these circumstances, the dysfunction may not reveal itself. As mentioned before, most problem patients have neurologic disorganization but it may not be present during the usual static examination. When switching is hidden, therapy localization to KI 27 is negative; it becomes positive under different circumstances. Again, evaluate for all sides of the triad of health.

Structural. Hidden switching may be brought out by simply having the patient stand and therapy localize KI 27. Hidden switching is present when therapy localization is positive to KI 27 with the patient standing, but not when he is recumbent. This indicates that something in the weight-bearing mechanism is at fault, usually the feet.

There may be no positive therapy localization when standing, but when the patient continues to therapy localize KI 27 and walks, a previously strong indicator muscle weakens. It is not necessary for the patient to continue walking during the muscle test; the positive therapy localization will remain long enough to test an indicator muscle. The indicator muscle will again test strong as soon as the patient quits therapy localizing KI 27. This differentiation is necessary to determine that, in fact, the weakening is due to neurologic disorganization. Patients who develop dural tension while walking will continue to have a weak indicator muscle for a considerable time after they discontinue the therapy localization to KI 27.

When there is positive evidence of neurologic disorganization with walking, one can then examine the supine or prone patient with gait, dural tension, and PRYT tests. The only difference from the usual testing procedure is testing the patient as he therapy localizes to KI 27. Treatment is performed in the usual manner.

Dynamic testing for hidden switching can include evaluating the stomatognathic system by having the patient chew some substance that does not cause weakening when there is no therapy localization to KI 27. The test is positive when weakening develops as the patient chews the substance while therapy localizing to KI 27.

Chemical. Hidden switching resulting from chemical influences on the body relates with any element that may be toxic or out of balance. One should consider the toxins an individual may encounter in his home or place of work. When the patient is in your office he may test normal because he is away from his usual environment.

Patients are sometimes on an imbalanced nutritional supplement regimen that throws body chemistry off-balance. Under these circumstances, chewing the offending nutrition will cause positive therapy localization to KI 27 when it was not present in the clear.

Mental. A patient's indicator muscle may weaken when he activates a disturbing thought process. The best muscle to test is the pectoralis major (clavicular division) when it is strong in the clear. This may or may not relate to hidden neurologic disorganization. If present, there will be weakening when the patient activates the thought process with therapy localization to KI 27, but not without.

The use of KI 27 as an indicator for switching, both in the clear and hidden, is simply an investigative tool. When evaluating for hidden neurologic disorganization one must consider the life-style, physical activity, mental processes, and chemical environment in which the patient lives. Failure to correct the patient's health problem may result from not testing him in the manner in which he lives.

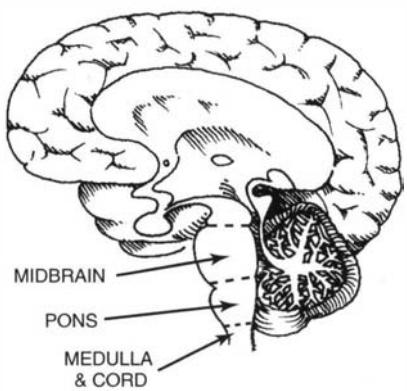
Correction of neurologic disorganization is important in obtaining optimal health. It also improves one's human performance[1] and is a key factor in many cases of learning disabilities.

Cross-Pattern Activity

Normally an infant is born with a genetically determined specialty for each hemisphere of the brain. This assigns specific dominance to each side of the brain. The dominance must be organized throughout the body for proper function. Doman and Delacato[3] present a working hypothesis on the development of the nervous system through bilateral function and final dominance. They describe the development of the nervous system from infancy where there is reflex action only, to homolateral activity where the two sides of the body function independently, to use of the two sides of the body together, and finally to cortical hemispheric dominance. Their hypothesis indicates that some individuals may not go through the normal stages of development. As a therapeutic approach, they developed a system of taking an individual back through the stages of crawling and creeping to develop homolateral, bilateral, and finally neurologic dominance. Their application is to individuals with speech and reading problems, as well as other types of learning disabilities and functional neurologic problems.[9,11,12] In many instances the evaluation and treatment procedures extend to children diagnosed as having minimum brain damage.

The concepts presented by Doman and Delacato were actively pursued in the fields of education and rehabilitation in the 1960s, with many excellent results. There are those in the educational field who now downgrade the importance of their work. Certainly this approach is not a panacea for all reading, speech, and learning difficulties. Their concepts, however, provide many correlative findings with those in applied kinesiology. The key is to have a method of diagnosing the need for cross patterning, which applied kinesiology appears to supply.

Diagnostic methods, as described by Doman and Delacato, may sometimes indicate a need for a program of crawling and creeping. Applied kinesiology examination and findings may point to some other mechanism interfering with the normal expression of properly developed neurologic function. In these cases, treatment to other factors may restore normal body organization without cross patterning. This may shed light on why some individuals who seem to need Doman and Delacato's therapeutic approach do not respond adequately to the procedures; there may be some other aspect interfering with normal function that has not been found and corrected.



5—9.

Delacato hypothesizes that a child goes through five stages of development, beginning with the lower spinal cord and medulla oblongata reflexes that are present at birth to approximately sixteen weeks. Next, homolateral function of the visual and auditory mechanisms develops during the pons level at sixteen weeks to six months. From six months to one year the midbrain develops, providing the cross-pattern mechanism and using both sides of the body together. This is an important area of development that prepares the child for function in an upright position. Early cortical function develops within the age range of one to five years. During this stage there is continued bilateral development, and walking begins. Finally, from three to eight years cortical and hemispheric dominance develops, giving right- or left-handedness and continued neurologic organization.

Physical activities from infancy through the development of bilaterality and cortical hemispheric dominance are important in developing the nervous system's organization. At the early spinal cord and medulla oblongata stage, there is no purposeful mobility; it is undulating and fish-like in character. As the infant develops into the pons level, sight and sound become important but the eyes and ears are not organized to function together; they function homolaterally, unable to locate sounds or have efficient depth perception. At about six months of age the midbrain stage develops. Here the child develops the ability to use both sides of the body together and coordinate body function, such as the hand with the eye. During this period, the child learns to use his arms and legs together and crawl in a cross-pattern motion. "Cross pattern" means that he moves his arm and leg into flexion on opposite sides, while the contralateral arm and leg move into extension. This is an important phase of development to complete before moving on to the development of cortical hemispheric dominance.

Early cortical function begins at about one year of age. Bilateral use becomes increasingly efficient. At this stage the child begins to pull himself up on furniture and takes his first few steps in becoming a biped. His arms do not function in a cross pattern; rather, they act as balancing staffs. According to the hypothesis, early cortical function should not begin until there is fairly efficient bilateral function. Entering this stage too soon seems to retard the efficient development of bilateral function.

Cortical hemispheric dominance begins to develop at about three years of age. Dominance usually begins

with hand choice, followed by eye, foot, and ear dominance. One-sided dominance is unique in man. It is usually fully developed by five to eight years of age.

The same dominance should be developed throughout the body; that is, an individual should be either right- or left-footed, -handed, -eared, and -eyed. A high percentage of individuals have neurologic disorganization. A military study of 38,430 personnel found 15% to have mixed dominance. This group required a disproportionate amount of orientation and training to achieve the desired results in marksmanship and combat training.[2]

Intrauterine — 16 weaks
Spinal cord and medulla oblongata, reflex actions only.

16 weeks — 6 months
Pons. Homolateral activity of visual and auditory functions.

6 months — 1 year
Midbrain. Cross pattern, quadruped crawling, development of using both sides of the body together, important area of development to prepare child for upright position.

1 year — 5 years
Early cortical function, walking, and continued bilateral development.

3 years — 8 years
Cortical hemispheric dominance; develops right or left dominance and continued neurologic organization.

5—10.

An individual's hand dominance is most easily recognized. Sometimes an individual will have mixed dominance of hand function, writing with the right hand and performing sports activities with the left. Observation of an individual's writing should be supplemented by observation of physical activities such as hammering, catching a ball, and other ordinary activities. When asked to cross the arms, the typical reaction is to place the dominant one on top.[2]

The most common clinical observance of crossed dominance is failure of hand and eye dominance to be the same. There are several methods for determining near- and far-point eye dominance. To determine far-point binocular dominance, give the individual a tube to hold in both hands at arm's length and have him look at a distant target. The examiner observes which eye the individual uses in lining up the tube to observe the target. A monocular far-sighted test is done by giving the individual a sheet of paper with a half-inch hole cut in it. The subject is asked to line up the distant target in the hole and bring the paper to his face, maintaining visual contact with the target. The opening in the paper will be brought to the dominant eye.

Evaluating near-point sight is similar to evaluating far-point. The individual being tested is seated at a desk and given a tube 3" to 5" in length, with an opening of about 3/4". On the desk is a sheet of paper with an "x" marked on it. He is asked to look at the x through the tube and bring the tube slowly back to the eye, without losing sight of the x. The eye to which the tube is brought is the dominant eye.

Another method of near-point sighting is for the examiner to hold his finger approximately 36" from the patient's face. The patient is then asked to align his index finger with the examiner's finger. The patient's finger should be approximately halfway between the examiner's face and his own. The patient's index finger will line up with the dominant eye.

Each of these sighting tests should be done three times. It is best to alternate them so that a practice or habit pattern does not develop.

Foot dominance is determined by observing which foot the individual uses in kicking and doing intricate activities, such as picking up marbles. Pedal dominance can be observed by having the individual step up on a chair or a step. Since approaching the object and then stepping on it may predetermine the foot used, the individual should be placed directly in front of the object and the activity begun from a standing position. The individual will typically use his dominant foot to step on the object.[2]

To determine ear dominance, give the individual a watch or something else with a low sound, but not a musical one. It will usually be held to the dominant ear. The clicking sound is best interpreted by the logical mathematical left brain in the average individual, while the musical sound is best interpreted by the tonal right brain. Each side of the brain receives input from both ears. The crossed connections are stronger than the uncrossed ones.[10]

5—11. Normal cross crawling of child develops bilaterality.

Disturbed Development

The child's development from early cord and medulla stages through pons, midbrain, early cortex and, finally, through cortical hemispheric dominance can be interfered with in many ways. During development the child can have a brain injury, severe febrile disease, or any other disease that interferes with normal nerve function. Since freedom of activity is very important in developing through the different stages, restriction can cause lack of normal development. Adults often cause the restriction by confining children in plastic carrying baskets, playpens, and walkers. Heavy clothing, if worn frequently, is also restrictive.

Normally, when a child breast-feeds, one eye and arm are alternately restricted as the mother uses the opposite breast on subsequent feedings. The bottle-fed baby is usually held with the mother's left arm while she holds the bottle with her right hand. Consistently feeding the baby in the same direction restricts one arm and often an eye, retarding bilateral development. The bottle-fed baby should be held in alternating arms from feeding to feeding.

5—12. Constantly holding child in same position restricts the use of the limbs and eyes bilaterally. The child should be held alternately in the right and left arms from feeding to feeding.

When the baby begins eating solid food, there is a tendency for an adult to place the child in a restrictive high chair and encourage the use of a spoon. During the development of the midbrain and early cortical stage, bilateral function is enhanced by eating with both hands. Using a spoon at this stage forces the child into unilateral dominance prior to readiness.

Adults tend to force a child to stand and walk too rapidly. Parents take pride in a child's early walking and early self-feeding. Actually, this often forces the child into advanced development for which he is not ready. Proficient development of cortical hemispheric dominance may be interfered with or delayed. Children develop at different rates and should be allowed to go through the stages of development at their own speed.

5—13. Use of eating utensils begins development of unilaterality and dominance. Observe that this child's eyes are not functioning together, an indication that bilaterality has not yet been developed. The child is not ready for unilateral dominance development.

Adults should not attempt to change a normally developing left-dominant child to a right-dominant one. If the genetic code is present for left dominance it should

5—14.

5—15.

Children should not be forced into early walking.

5—16. When the child is ready to start walking he will first pull himself up into a biped position, then begin taking steps on his own.

develop normally, even though the left-dominant individual will have more problems than the right-dominant one. It is recognized that it is very difficult for a left-dominant person to live in a right-dominant world, but it is worse to have poor neurologic organization.

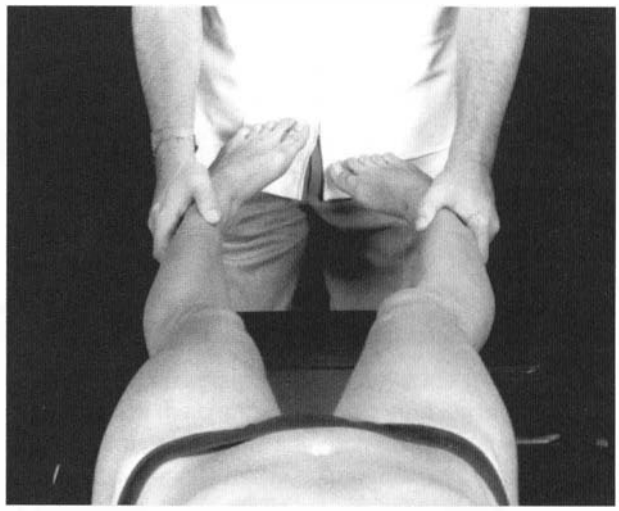
Cross-Pattern Treatment

Cross-crawl patterning was introduced into applied kinesiology by Goodheart.[4] It is a modification of Doman and Delacato's creeping pattern.[3] Creeping, in Doman and Delacato's work, is the cross-pattern hands and knees crawl in quadruped fashion. This is called crawling in applied kinesiology. Early in applied kinesiology nearly everyone who was neurologically disorganized was put on a cross-patterning exercise. With the methods of finding the cause and correcting the neurologic disorganization described previously, cross patterning is not needed as often. It is prescribed when there is evidence of improper development. This may be the result of restrictive childhood actions, described previously. The child may have had a prolonged restriction, such as a leg or an arm cast as a result of bone fracture. Severe health problems during the neurologic developmental stages may have interfered with normal progression.

Crossed dominance and general disorganization are indications for cross patterning. One should first evaluate for neurologic disorganization and correct its cause. When there is recurrent neurologic disorganization or difficulty in obtaining correction, cross patterning often helps obtain correction and better organization.

Patterning is usually done with the patient supine. His opposite arm and leg are flexed to the maximum and then returned to the table. To complete one cycle, the other arm and leg are then fully flexed. The therapeutic effect of the cross patterning is enhanced by turning the head to one side during half the cycle. The patient is considered patterned to a particular side when the head is turned to the side of shoulder flexion; the head is maintained in a neutral position when the opposite shoulder is flexed. The direction in which the patient is patterned does not necessarily correlate with the dominant hemisphere or any aspect of bilateral brain function. The patient may require patterning to either side, but not to both. The side to which the patient is to be patterned can generally be determined by testing internal thigh rotation. The examiner grasps the patient's ankles and internally rotates both legs. The side of greatest internal thigh rotation is usually the side toward which the patient should turn his head when that arm flexes. The greater internal thigh rotation usually relates to relative weakness of the psoas and piriformis. Greater weakness of muscles on one side of the body also indicates the need for cross patterning and the side toward which to pattern.

Although the thigh rotation test generally shows the appropriate side for patterning the patient, it is *absolutely necessary* to use a therapeutic trial to determine if this



5—17. Leg internal rotation test showing right leg with more available medial rotation, indicating a probable right cross-crawl pattern.

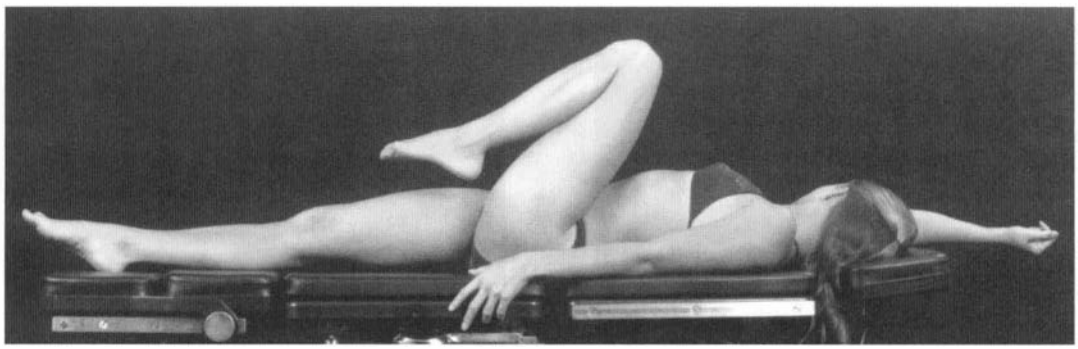
direction enhances muscle function. This is accomplished by having the patient perform the procedure for approximately six cycles, then testing previously weak muscles to determine if function has improved. An important sequential test that should always follow is to have the patient pattern in the opposite direction; the muscles should again test weak. This is extremely important because improper application of cross patterning not only fails to help the patient, it can potentially harm him.

There is a type of patterning that differs significantly from the cross pattern described here. It is called a "homolateral" crawl pattern, which Goodheart[5] associates with schizophrenia. A homolateral pattern refers to flexion of the ipsilateral arm and leg, first on one side and then on the other. An individual organized in a homolateral pattern will test weak throughout the body for a short time after doing cross-pattern activity. Cross-pattern activity administered to an individual who is in homolateral organization can cause significant exacerbation of symptoms.

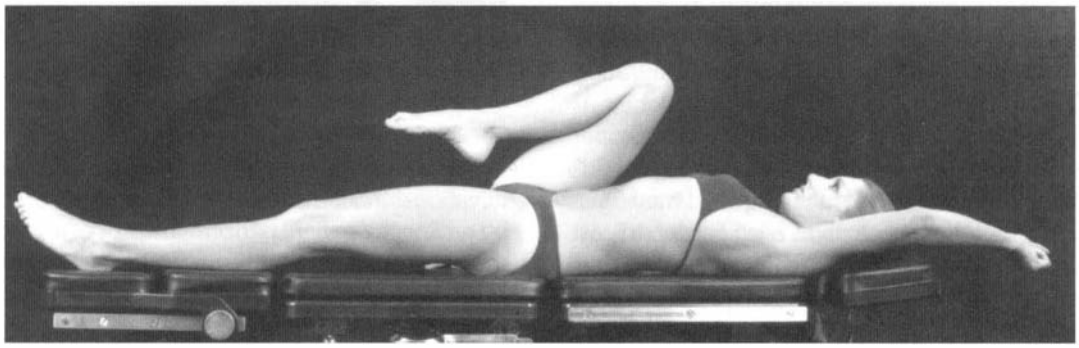
Homolateral organization requires a specific therapeutic approach. Cross patterning should not be administered to an individual with this type of organization. (This is further discussed on page 438.) This is one reason it is absolutely necessary to do a therapeutic trial whenever cross patterning is prescribed for a patient.

Some types of exercise apply an improper neurologic pattern and cause recurrent neurologic disorganization. An example is a rope and pulley arrangement where the individual pulls the leg into hip flexion with homolateral arm flexion.[14] The same type of exercise, but done on a cross pattern, will probably cause no harm.

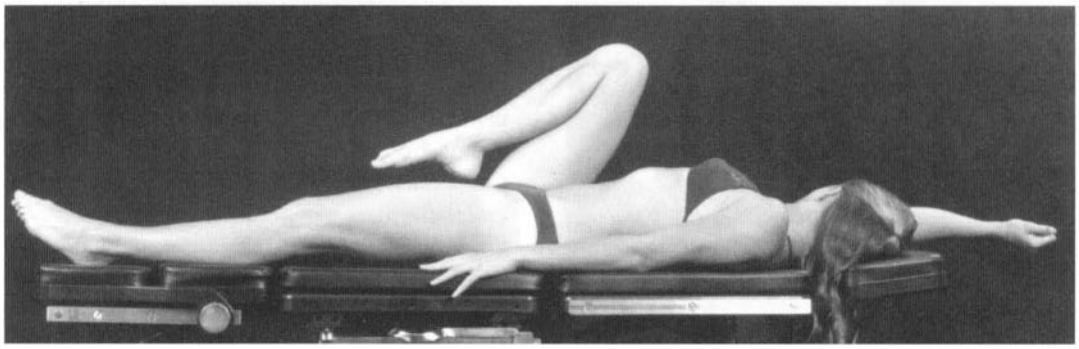
Many individuals who require cross patterning are neurologically disorganized. They often fail to understand instructions properly, and it is quite possible that the procedure may be done improperly at home. It is advisable, especially with children, to have another individual



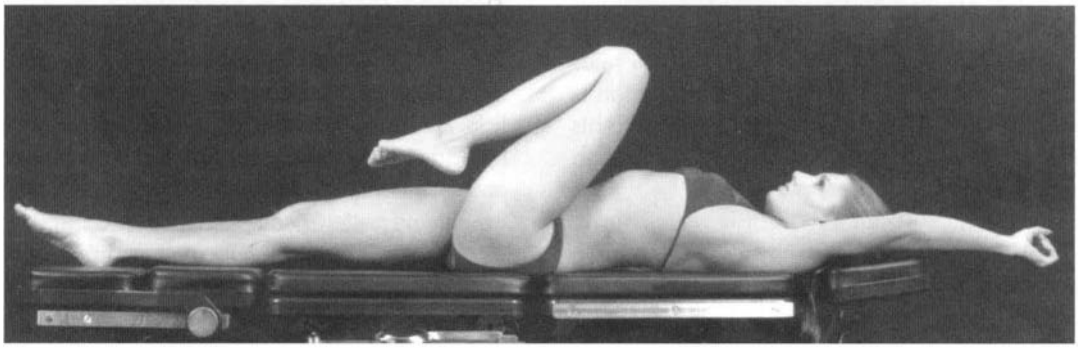
5—18. Right cross pattern. Head turns to the right as right arm and left leg flex.



5—19. Right cross pattern. Head stays straight as left arm and right leg flex.



5—20. Right homolateral pattern. Head turns right as right arm and leg flex.



5—21. Right homolateral pattern. Head stays straight as left arm and leg flex.

observe the procedure and note the instructions for doing it. One should also write down the instructions so the patient has the correct information to refer to at home.

It is important to re-evaluate the patterning as treatment proceeds. Neurologic organization may change. The individual may need patterning to the opposite side, or he may no longer need patterning.

Thirty cycles a day are usually sufficient for establishing neurologic organization. In some severe cases it may be necessary to do thirty repetitions three times a day. If the individual engages in some activity, such as swimming or running, it may be advisable to do the procedure immediately before and after the activity. The length of time the procedure must be performed varies from several weeks to many months, depending on the condition's severity. If the activity is still needed several months after diligent patterning, some other factor is probably causing the neurologic disorganization. The patient should be thoroughly re-evaluated for its cause, as indicated previously in this chapter.

There should be mind-body integration with the cross-patterning activity. Sometimes when one has done the cross patterning for a few weeks it becomes almost automatic, needing little or no thought. In this case weak muscles will fail to test strong after the patterning. Simply have the patient think through the process, repeating to himself, "My right arm and left leg are going up; now they're going down. My left arm and right leg are going up; now they're going down." When the procedure is done this way, the weak muscles will test strong. As one becomes proficient in cross patterning, additional procedures can be added. The eyes can be integrated into the procedure. Have the patient follow the hand as the arm flexes above the head. This eye movement can be done to both sides. The importance of eye position and motor re-education as a result of disease or trauma is stressed by Stejskal.[13] Deviation of the eyes influences the muscles of the neck, trunk, and limbs. This movement principle, called "eyes-hands," is a naturally associated movement. Stejskal recommends that it be widely used in neuromuscular re-education.

Cross patterning can also be done in a standing position, with the contralateral arm and leg in extension or abduction. Doing the standing procedure is not routine practice, but it can be helpful in cases where an individual does the procedure too rapidly, or it is determined that additional patterning activity is needed for optimal function. The head is turned in one direction to the side of arm movement. A therapeutic trial is used to determine that the pattern is correct.

There is clinical evidence that in some cases nutritional support may be needed to enhance neurologic organization. The nutrition required is usually brain concentrate or nucleoprotein extract, or ribonucleic acid, as determined by applied kinesiology testing. Although any indicator muscle associated with the disorganization will usually improve when the appropriate substance is chewed, the supraspinatus is generally the most effective muscle to test, if it tests weak in the clear.

CHAPTER 5 — REFERENCES

1. Blaich, R.M., "Applied kinesiology and human performance." Proceedings of Winter Meeting, ICAK, Palm Desert, CA, 1987.
2. Carrick, R., & M. Watson, "Application of the neurological organization theory to nonpathological subjects," in *Neurological Organization and Reading*, ed C.H. Delacato (Springfield, IL: Charles C. Thomas Publisher, 1966).
3. Delacato, C.H., *The Diagnosis and Treatment of Speech and Reading Problems* (Springfield, IL: Charles C. Thomas Publisher, 1963).
4. Goodheart, G.J., Jr., "Cross-pattern crawling and muscle spasm," *Chiro Econ*, Vol 11, No 5 (Mar/Apr 1969).
5. Goodheart, G.J., Jr., "The schizophrenic pattern," *Chiro Econ*, Vol 13, No 1 (Jul/Aug 1970).
6. Goodheart, G.J., Jr., *Applied Kinesiology 1975 Workshop Procedure Manual*, 11th ed (Detroit: privately published, 1975).
7. Goodheart, G.J., Jr., *Applied Kinesiology 1977 Workshop Procedure Manual*, 13th ed (Detroit: privately published, 1977).
8. Guyton, A.C., & J.E. Hall, *Textbook of Medical Physiology*, 9th ed (Philadelphia: W.B. Saunders Co, 1996).
9. Kabot, R.R., "A study of improvement in reading through improvement of neurological organization," in *Neurological Organization and Reading*, ed C.H. Delacato (Springfield, IL: Charles C. Thomas Publisher, 1966).
10. Kimura, D., "The asymmetry of the human brain," *Sci Am*, Vol 228 (1973).
11. McGrath, Father F., "The St. Philip summer remedial reading course," in *Neurological Organization and Reading*, ed C.H. Delacato (Springfield, IL: Charles C. Thomas Publisher, 1966).
12. Sister Mariam O.P., "A comparative study of the reading disability in neurologically organized and neurologically disorganized fifth grade children," in *Neurological Organization and Reading*, ed C.H. Delacato (Springfield, IL: Charles C. Thomas Publisher, 1966).
13. Stejskal, L., "Postural reflexes in man," *Am J Phys Med*, Vol 58, No 1 (1979).
14. Stevenson, R., "Cross crawl: An adjunct to other exercise programs, *Chiro Econ*, Vol 19, No 4 (Jan/Feb 1977).
15. Walther, D.S., "An additional approach in the treatment of schizophrenia." Proceedings of Summer Meeting, ICAK, Detroit, 1980.
16. Walther, D.S., "Locating the cause of neurologic disorganization," in *1983 Selected Papers of the International College of Applied Kinesiology*, ed W.H. Schmitt, Jr. (Detroit, MI: ICAK, 1983).

THIS PAGE INTENTIONALLY LEFT BLANK

Chapter 6

Basic AK Testing and Treatment Procedures

Injury Recall

The nervous system records and maintains memory of the events that happen in one's lifetime. There may be an encoded memory association maintaining a health problem that needs to be disassociated to regain normal function.

There are two approaches to bring out the encoded memory of trauma that may be causing a patient to fail to recover, recover only partially, or lose corrections. Injury Recall Technique is an examination and treatment system to locate and eliminate the memory association, and Memory Recall deals with the immediate loss of corrections.

Injury recall is presented at the beginning of this chapter on Basic AK Examination and Treatment Techniques because if historical trauma is affecting the patient's condition, it is paramount to eliminate this factor early in the treatment. Failure to eliminate the influences of historical trauma on the nervous system does not mean the patient will not respond to treatment, but the effects of treatment may not be long-lasting or the condition brought to the highest plateau.

Injury Recall Technique (IRT)

Schmitt[70] became aware of a podiatric method from the work of Robert P. Crotty, D.P.M., and described by Bronston[8] that is used by some to locate a remote area of the body creating stress that might interfere with the successful outcome of podiatric surgery. The examiner palpates the patient's foot for a painful area, and when found the patient touches a remote area of trauma that has occurred anytime in his history. If the trauma is associated with the foot, the palpation pain will greatly diminish while the patient touches the remote trauma area. Bronston relates this to a "muscle chain reaction," and acknowledges that specific anatomical relationships have not as yet been defined.

When remote trauma is associated with the foot in this manner, the stress is reduced during post-surgical recovery by disassociating the remote area of trauma. This is done by stimulating the remote injured area as in the initial test while podiatric support is applied to the foot. Within seconds after the support application, the foot tenderness exacerbated by the test will diminish or disappear.

Stimulated by the success of Bronston et al., Schmitt[70] used AK techniques to investigate the role prior trauma might have on the recovery of patients regardless of the length of time since the trauma. This led to the injury recall technique (IRT).

Initial investigation was directed toward the foot and ankle because of the podiatric experience. The podiatric view is that almost every injury of significance in a patient's history is reflected in the patient's foot or feet, usually ipsilateral to the injury. Neurologically the foot and ankle afferents are important in the organization of the neuraxis. There are several neurologic reflexes in the foot and ankle including the positive support reaction, magnet reaction, and the role foot and ankle position has in facilitation and inhibition of gait muscles.

Important in maintaining homeostasis are postural balance and organization within the equilibrium proprioceptors. The foot and ankle mechanoreceptors, along with the labyrinthine and visual righting reflexes, provide the most important afferent supply for postural balance. Among the most important proprioceptive information needed for maintenance of equilibrium is that which comes from the mechanoreceptors of the neck.[40] Equilibrium is internal balance as well as postural balance, a state of homeostasis. As we deal with the triad of health with its structural base, it is not surprising that the two areas found to remove encoded memory of historical trauma from the nervous system are at the ankle mortise and upper cervical spine.

Ankle Mortise

The relationship of the foot and ankle to trauma is directed primarily to the talus relationship with the distal tibia and fibula to make the ankle mortise. It is thought that the ankle is related to trauma by the withdrawal reflex mediated through the flexor reflex afferents and the associated crossed extensor reflex. Depending on the position of the person at the time of trauma there may be an extensor withdrawal, so the term withdrawal reflex is adopted to cover both flexion and extension. One can often see the effect of the withdrawal reflex when there is a painful reaction to treating a supine patient; the foot will consistently move into dorsiflexion.

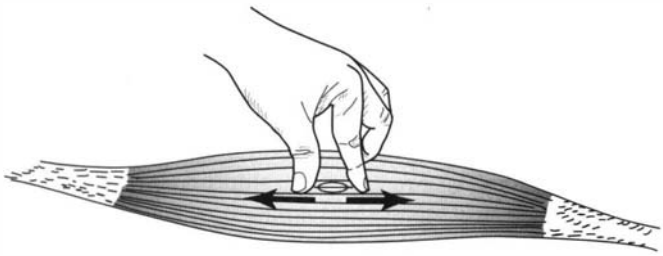
There are two methods to determine if historical trauma encoded in the patient's memory is still adversely influencing the neuraxis. 1) Test a strong indicator muscle for weakening in combination with stimulation to the injured area while approximating the talus into the ankle mortise. 2) Test a type 3 weak muscle for strengthening to neuromuscular spindle cell manipulation. (See pages 63 and 306.)

Examination — Strong Muscle Weakens. Schmitt found that simultaneous compression of the ankle mortise and stimulation to a previously injured area by therapy localization, rubbing, pinching, or applying cold causes a strong indicator muscle to weaken.

The ankle mortise is challenged by cephalad pressure on the talus to compress the ankle mortise. When positive a previously strong indicator muscle will weaken. Applying the same stimulus to either the trauma area or talus individually fails to produce the same weakening of an indicator muscle.

Most often, the talus challenge is applied ipsilaterally to the side of trauma. In some cases of midline trauma, the challenge must be done to both tali.

Examination — Weak Muscle Strengthens. A muscle weak with both types 1 and 3 (G2 submax) should strengthen with neuromuscular spindle cell spread apart manipulation (autogenic facilitation). This is done by simply using the thumb and forefinger to



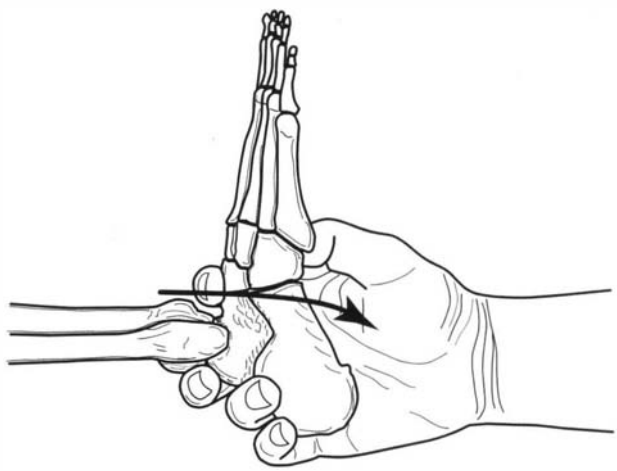
6—1. A weak type 3 muscle should strengthen with neuromuscular spindle cell apart technique. If not, IRT technique is needed.

spread apart in the muscle belly. Failure of the muscle to strengthen indicates the need for injury recall technique.

Suspected areas of recent or long-term historical trauma from the patient's history or other cause for suspicion are evaluated. This is done by stimulation over the suspected area of trauma by rubbing or gently scratching the skin. When there is trauma associated with the weakness, the muscle will test strong to both types 1 and 3 tests. Stimulation over the trauma area stimulates mechanoreceptors to block a pathway, such as the flexor withdrawal or the crossed extensor reflex that is causing the muscle to test weak. The muscle strengthening will last for ten or fifteen seconds and then return to weakness. The same type of skin stimulation over any other area will not strengthen the weak muscle.

Strengthening a weak type 3 muscle with neuromuscular spindle cell apart stimulation is used more often because of its efficiency. There are times when the strong muscle weakening method is preferred. There may be several areas of historical trauma affecting a single muscle, but this is infrequent. In this case, it is easier to use the strong muscle weakening method of testing. The strong muscle weakening method is also best when the suspected area of trauma is intraoral, close to the genitals, or in some area where it is advisable to not stimulate the skin over the suspected area of trauma.

Treatment. Treatment to eliminate the encoded trauma connection in the nervous system is simply to connect the area of trauma with the locking mechanism, which in this case is the ankle mortise. Stimulate the skin over the area of trauma by the same method used in the examination phase while separating the ankle mortise by micromanipulation. The inferior manipula-



6—2. The talus maneuver is a gentle inferior pull to separate the talus in the ankle mortise.

tion is done by grasping the talus' anterior superior surface while gaining leverage with the examiner's thumb under the metatarsals. The maneuver is not a high-velocity forceful manipulation; rather it is a gentle, flipping action with most of the force applied to the anterior superior surface of the talus to bring it inferior.

Head and Neck

Encoded memory association of trauma to the head and neck appears to usually reside in a cervical extensor reflex pattern. The correction can be made at the ankle mortise or cervical spine; the latter is usually more effective.

Examination. The same type of skin stimulation over the trauma area is done while the patient's neck is placed in extension. A positive test is for a strong indicator muscle to weaken with the stimulation and cervical extension. The most important portion of extension is the upper cervical area, including the occiput. Cervical extension or skin stimulation over the trauma done individually will not be positive. The test is usually done with the patient supine, but it can be done sitting or standing; sometimes it will only be positive in these positions.

If cervical extension without the accompanying skin stimulation is positive, refer to atlas-on-occiput

extension (page 96) and treat accordingly. Following successful treatment of atlas-on-occiput extension, re-evaluate for injury recall.

There are many common types of encoded trauma found with the head and neck technique. There may be intraoral involvement from dental trauma, both accidental and from dental procedures, scars from surgery such as tonsillectomy, and/or sutures from cranial trauma. Cervical trauma of whiplash dynamics can cause trauma anywhere in the stomatognathic system. The skull and neck should be thoroughly covered with therapy localization and tested in combination with cervical extension for encoded memory problems. Therapy localize to the neck systematically at segmental levels anteriorly, posteriorly, and laterally. When there is dysfunction in structures innervated from cervical spine levels, it is valuable to test for encoded memory with the head and neck method in addition to the talus method.

Temporomandibular joint problems are often associated with past trauma, many of them with whiplash trauma. If the patient is wearing a night guard (bite plane, splint), do the testing with the patient clenching his teeth with the appliance in and out.

Treatment. Treatment is opposite the diagnostic test, i.e., move the upper cervical-occiput area into maximum flexion. Grasp the sides of the patient's head with a full hand contact to avoid pain or excessive force anywhere on the skull. With the patient maintaining TL to the positive area move his head and upper cervical spine into flexion, with concentrated movement at the occiput-atlas level. At the end of motion, apply about three gentle but firm flexion movements to the limit of motion range. The maneuver does not go past the range of motion and should not be painful to the patient.

Cerebellum Role

Schmitt[71] presents a neurologic suprasegmental, primarily cerebellar adaptation model for the effects of injury recall technique. The cerebellum monitors and makes corrective adjustments to motor activity. The most important mechanoreceptor afferent supply to the cerebellum is from the neuromuscular spindle cells. An example of adaptation is the afferent supply from the gastrocnemius and soleus for control of postural sway. When one leans forward the stretch reflex contracts the muscles to push the forefoot against the floor, returning postural balance. If the floor moves up under the toes, causing the same dorsiflexion as leaning forward, the muscle stretch reflex tends to make the person fall backward, thus requiring further modification. If the floor continues this movement there is an adaptive response to decrease the stretch reflex response to meet the new environmental input. This adaptation has been felt by many when on a rolling boat. At first one has to consciously make adjustments to the swaying. After a short time there is adaptation to the reflexes, referred to as "now I have my sea legs." When going back to shore there is again a need for adaptation. When first stepping on solid ground, most persons feel like they are still on the boat.

This adaptation is from projections to the cerebellum from the cortex that receive afferents from the neuromuscular spindle cells. At the time of and during repair of trauma, there are various and numerous alterations in stretch reflexes as they adapt to flexor reflex afferent pathways arising from nociceptors. This, then, is the new norm that must be adapted to much the same as one adapts to the rolling boat. Schmitt[71] proposes "...if the trauma is significant enough, an adaptation of cerebellar modulation of stretch reflexes, which must take place, becomes the new norm as nociceptors continue to fire and change stretch reflex responses while the injury heals."

If adaptation to the trauma becomes stable an encoded memory develops that may cause a deviation in posture, changed temporal firing of muscle, change in gait pattern, or anything else that was part of the adaptation to the trauma and during the healing phase. Postural changes can be reflected in proprioceptive changes in the ankle mortise or suboccipital area.

The model indicating that IRT is at least partially associated with cerebellar adaptation is applying functional neurological assessment of cerebellar function in combination with IRT. The cerebellum is constantly receiving input from the semicircular canals of the vestibular complex. Movement of the head causes activation of at least one of the three bilateral canals. Semicircular canal stimulation causes characteristic reflex changes in postural muscle activity.

Six head positions to activate specific canals when supine are as follows:

1. Head rotated to right — right lateral canal
2. Head rotated to left — left lateral canal
3. Head tilted anteriorly and left — left anterior canal
4. Head tilted posteriorly and right — right posterior canal
5. Head tilted anteriorly and right — right anterior canal
6. Head tilted posteriorly and left — left posterior canal

To observe the role of the cerebellum in IRT, simply activate the area of injury by patient therapy localization or doctor pinching, and instead of approximating the ankle mortise place the head in one of the six head positions to activate one of the semicircular canals. This results in a strong indicator muscle weakening in the same way as approximating the talus in the ankle mortise. Treat with the usual IRT, and the positive reaction of head turn will no longer be present. This suggests that the encoded memory problem is associated with plastic adaptation in the cerebellum that is no longer present following IRT correction.[71]

Summary of Injury Recall Technique

All areas being screened for IRT must be negative to therapy localization before proceeding further for IRT. If positive to therapy localization, clear the cause before continuing with IRT examination.

Ankle Mortise

Strong muscle diagnosis:

- Test any strong muscle while gently challenging the mortise joint, i.e., push talus cephalad during one of the following:
- Patient TLs area of previous trauma.
- Doctor pinches area of previous trauma.
- Cold shock area of previous trauma (e.g., ethyl chloride).

Weak muscle diagnosis:

- Apply neuromuscular spindle cell spread apart technique to a Type 3 muscle weakness. If the muscle does not strengthen, injury recall technique treatment is needed.

Treatment:

- Gently pull (not thrust) talus inferiorly (in direction of opening mortise joint) while simultaneously performing one of the following:
- Patient TLs to the area of previous trauma.
- Pinch area of previous trauma.
- Cold shock area of previous trauma.

Head and Neck Technique

Diagnosis:

- Positive test is when therapy localization to areas of previous trauma on head or neck in combination with head and neck extension weakens a strong indicator muscle.

Treatment:

- Patient maintains TL to area of previous injury while doctor firmly but gently flexes atlanto-occipital area to limit of motion three or four times.

Memory Recall

The encoded memory of physical or emotional trauma may have a lasting effect on structural dysfunction. The mental recall of the incident may cause the correction of a structural fault to be lost. The cause of the correction being lost as a result of this type of memory recall is especially applicable when a patient identifies physical or emotional trauma with the onset of his condition. After corrections have been successfully made, have the patient accurately think about the incident he associates with the beginning of the health problem. If the corrections are immediately lost, there is evidence that memory recall is continuing to interfere with lasting corrections needed to return normal health. The trauma associated with the onset of a health problem is most applicable, but any physical or emotional trauma can be associated.

After successfully correcting a structural fault, have the patient concentrate on the trauma. The association of the trauma with the recurrence of dysfunction can be seen by the fault(s) immediately returning. When the recall of trauma is specifically associated with the patient's loss of correction, only the muscle weakness associated with the successfully corrected dysfunction will be present. If there is general weakening of all muscles, the triad of health emotional side needs examination and treatment.

The encoded memory can be a combination of physical and emotional, such as an auto accident in which someone died or other emotional complexity. In the case of emotional trauma it is not necessary for the physician to be made aware of the specifics. If there is no definitive memory, have the patient hold the emotional neurovascular reflex during correction.

The encoded memory association with the structural fault can be erased by having the patient accurately concentrate on the trauma while correction(s) are made. There may be more than one associated trauma; if so, correct the fault again with the patient concentrating on the additional trauma. If successful there will no longer be a loss of correction when the patient recalls the trauma.

Repeated Muscle Action

Aerobic/Anerobic Muscle Function

Muscles have a mixture of aerobic and anerobic fibers, with dominance of one type. These are the slow and fast twitch fibers, respectively. The aerobic fibers dominate in the endurance muscles as for postural control. Anerobic fibers dominate in muscles that are used more rapidly. In the chicken and turkey this is apparent in the postural muscles, such as the thigh and leg, with the heavy dark color indicating a predomi-

nance of aerobic fibers. In the pectoral muscles used for the quick action of flying, there is a predominance of anerobic fibers, thus the white color of the muscle. In the human, the dominance of one fiber over the other is not as apparent.

Muscle Endurance

Muscles sometimes fail to function when required to do so under sustained use. A method of checking for this in applied kinesiology is called aerobic/anerobic muscle testing. Goodheart[31] first observed this in a competitive downhill skier who could not maintain a tight tuck at the end of a downhill run. Although all of the skier's leg and pelvic muscles tested extraordinarily strong, it was obvious that there was muscle failure toward the end of the run. A basic principle in applied kinesiology is to test the patient the way he lives, works, and plays. In order to do this, Goodheart tested the leg muscles on a repeated basis, that is, one test after another after another until he was confident that the muscle could continue to function under sustained use. When the skier's hamstrings were tested in this manner, the muscles became extraordinarily weak after a few tests. When the neurolymphatic or neurovascular reflex was therapy localized, the muscles no longer weakened with the series of tests. Because therapy localization to the neurolymphatic or neurovascular reflex eliminated the positive test, it was hypothesized that the weakening was caused by failure to adequately clear the interstitial space of blood and lymph to carry away waste products and bring in a replenishment of nutrition.

With continued use of the repeated muscle testing, it was found that some muscles weakened from fast repeated testing and others from slower, more sustained testing. This appears to relate with the concentration of fast or slow fibers in the muscles. Muscles with a higher concentration of slow fibers are tested about ten times in ten seconds; those with a higher concentration of fast fibers are tested about twenty times in ten seconds. When positive, the muscle will usually weaken somewhere in the middle of the test series.

The testing procedure of the slow muscles is aerobic function. They require both myoglobin for oxygen and fat for conversion to adenosine triphosphate (ATP).

Aerobic/anerobic muscle testing is done when a muscle is strong in the clear and does not weaken with therapy localization to the five factors of the IVF. If the muscle does weaken, treat the indicated factor before progressing to repeated muscle testing. Positive aerobic/anerobic findings may be responsible for individuals becoming fatigued throughout the day. Athletes who begin a sport with good function but progressively slow down and lose efficiency may have a positive repeated muscle test. A tennis player may have no problem playing the first two sets, but he develops fatigue and poor coordination in the third set. If an individual has a problem with the duration of an exercise as opposed to the strength necessary for it, one should think of aerobic/anerobic testing. For example, a weight lifter who effectively lifts heavy weights once but cannot repeat the activity probably has aerobic/anerobic muscle dysfunction. Cramping at the end of a day of snow or water skiing indicates this type of dysfunction. Recurrent foot problems in athletes can be from aerobic/anerobic weakness of the tibialis anterior or posterior.

Within the framework of applied kinesiology there are many routinely observed postural and movement deformities that have predictable muscle imbalance as their cause. When a standing individual flexes at the hips into Adam's position and the lumbar spine is higher on one side, it is often because of weakness of a psoas or gluteus maximus muscle. If this predictable weakness is not found with the usual manual muscle testing, including therapy localization to the five factors of the IVF, test the patient for aerobic/anerobic muscle dysfunction. Quite often the muscle will test weak on the aerobic muscle test since these are both postural muscles and, in most cases, have a higher concentration of slow fibers. Correcting the aerobic muscle dysfunction, if found, usually balances the patient in Adam's position. If there is failure to gain balance, evaluate the muscles — especially the gluteus maximus and hamstrings — for muscle stretch reaction or shortness.

Treatment is applied to the neurolymphatic or neurovascular reflex that eliminated the positive aerobic or anerobic muscle test when therapy localized. The neurolymphatic reflex is more often involved; occasionally both may be. Prolonged treatment to the reflex may be necessary.

Nutritional support helps prevent the condition from returning. When the involvement is with aerobic muscles, indicated by the slower testing procedure, iron is indicated.

Klepper[48] evaluated 20 patients for aerobic muscle fatigue by testing the pectoralis major, lumbar division of the psoas major, and general hip abductors at the rate of one test per second. Those who showed deterioration of strength in six repetitions or less were classified as positive for aerobic fatigue. Those who sustained contraction for 20 repetitions were classified as negative for aerobic fatigue. The fatigue group was then further tested by chewing a tablet of iron (Nutri-West Core Level Iron), and in each case the patient no longer tested positive for aerobic fatigue. A statistical analysis of the blood was made, revealing low serum iron and ferritin in the group positive for aerobic fatigue. There was no statistical correlation between the two groups for hemoglobin, RBC count, and hematocrit.[48]

Anerobic muscles that cannot sustain proper function, indicated by the fast testing procedure, are helped by pantothenic acid. Nutrition is indicated when the repeated muscle test is negative after insalivation of the

appropriate nutrition without any other treatment administered.

Since aerobic muscles burn fat, the condition may be helped by a source or sources of the essential fatty acids, in which many individuals are deficient. The testing method is to have a patient insalivate the oil being tested and re-evaluate for the muscle weakening on repeated muscle testing. Sources of fatty acids frequently found effective in eliminating the condition are Linum B_6, sesame seed oil, evening primrose oil, fish oils, and linseed oil fractions. These essential fatty acids also help raise the pH level of the saliva when it is too acid. Essential fatty acids are indicated more when there are numerous muscles with a positive aerobic muscle test. A common symptom of essential fatty acid deficiency is a sensation of coldness when the environmental temperature is normal. When only one muscle weakens to aerobic activity, a source of iron is usually needed as nutritional supplementation.

Aerobic or Anerobic Exercise

The group of people who benefit most from aerobic activity are generally those who have low metabolism and wish to lose weight or inches in the form of fat. This type of exercise increases the use of oxygen and improves oxidation of fats. Anerobic activity will probably not raise metabolism. It gets its energy from sugar in the form of glucose. It must be determined whether the aerobic or the anerobic system is to be enhanced by exercise. When this is known, the exercise level is maintained to stay within that type of exercise. This can be accomplished in an exercise physiology laboratory by measuring the oxygen uptake (VO_2 max.) and the CO_2 produced in relation to the O_2 consumed. Also, the production of lactic acid is measured. This is not done on a routine basis except in research and the training of elite athletes.

Maffetone[50,51] recognized improvement in muscle dysfunction, as observed by applied kinesiology muscle tests, when athletes would run aerobically. As the muscle function improved, their running gait also improved. When the athletes' gait improved, it was primarily with aerobic running but not anerobic running. Consequently, Maffetone surmised that it was not the running per se that caused the improvement; rather, it was the metabolic phase of the running. From this observation, he developed applied kinesiology challenge tests to categorize individuals as aerobic deficient or excess, and anerobic deficient or excess. Aerobic/anerobic imbalance can be attributed to imbalance of physical activity and/or nutritional or digestive deficiency. From the physical standpoint, too much or not enough of either aerobic or anerobic exercise is detrimental to optimal health. From the chemical aspect there may be nutritional deficiency prohibiting proper fat or sugar metabolism, or failure to provide co-factors necessary for proper waste product elimination.

The challenge mechanism for evaluating aerobic/anerobic balance is simply to have the patient perform some of each activity and observe the improvement or detriment to muscle function. These tests should be done in the initial stage of examination, before the patient is treated.

1. Determine several muscle weaknesses in the clear. These can be found by analysis of posture and the TS line. The muscles that test weak should be associated with different systems of the body, e.g., the pectoral muscles, quadriceps, latissimus dorsi, and piriformis, being upper and lower body muscles and associated with different organs or glands in applied kinesiology.

2. Determine one or two muscles that test strong in the clear and can be made to weaken by stimulation to the meridian sedation point or by the origin/insertion reciprocal inhibition technique. This is done to determine that the muscle is capable of weakening and is not in an abnormal hypertonic state.

Aerobic Challenge. Have the supine patient slowly alternately flex at the hips to raise and lower his left and right leg for eight repetitions, taking approximately 20 seconds to do so. Test the weak muscles for strengthening and the strong muscles for weakening. The muscle tests should be done quickly, since the effects of the challenge only last for a short period. In normal aerobic/anerobic balance the muscle activity will neither strengthen the weak muscles nor weaken the strong muscles.

Aerobic Deficiency. The usual type of aerobic deficiency is a lack of aerobic exercise. In this case, most — if not all — of the weak muscles will strengthen for a short period after the aerobic activity and aerobic activity is needed. If the muscles do not strengthen it indicates that aerobic system deficiency is because the body is incapable of properly metabolizing fat to supply energy during what would normally be aerobic activity. The problem is a metabolic one. Aerobic activity in this patient will not be beneficial until the structural, chemical and/or mental problems are corrected. The most common deficiency is nutritional or small intestine absorption.

Aerobic Excess. In rare cases there may be no strengthening of the previously tested weak muscles, and there is a weakening of the strong indicator muscle(s). This is usually seen in those who have continued an effective aerobic exercise program for a long period. These are usually the individuals who benefit from their exercise program and then plateau, and may even develop over-training injuries. To bring back balance between aerobic and anerobic activity, the individual simply includes anerobic training in his exercise program. This can be accomplished by doing anerobic activity for 30 minutes twice a week, such as moderately fast running, light weights with high repetitions, racquetball, or other hard, higher heart rate activities.

Maffetone recommends that the person warm up and cool down aerobically. The program is continued for a minimum of about six to a maximum of about twelve weeks. The exact length of the activity can be monitored by the aerobic/anerobic challenge, as described here.

In aerobic system deficiency, the problem is usually a requirement of essential fatty acids. There may be a need for co-factors such as B-complex and the minerals zinc, manganese, and iron, as well as carnitine. Other nutritional factors may be required, such as niacin, vitamins B_{12}, E and C, or the minerals magnesium, selenium, phosphorus, and molybdenum. A very important factor is to evaluate the small intestine and direct treatment to it if it is dysfunctioning. The problem may be a congested lymphatic system, or a malabsorption condition in which the villi are filled with mucus. A common condition in aerobic system deficiency is a congested liver.

Anerobic Challenge. The anerobic challenge is similar to the aerobic challenge in that you determine muscles that test weak and are unassociated, and determine a muscle or two that test strong and can be weakened by stimulation to the meridian sedation point and/or the origin/insertion reciprocal inhibition technique. The supine patient is asked to make a fist with both hands and alternately flex and extend the forearm as rapidly as possible for ten seconds. The upper extremity muscles are dominated more by anerobic fibers, and the rapid activity challenges the anerobic system. With normal balance of the aerobic/anerobic systems, this activity will neither strengthen the weak muscles nor weaken the strong muscles.

Anerobic Excess. In anerobic excess the strong indicator muscles will weaken for a short period, while there is no effect on the weak muscles. From an exercise standpoint this is most commonly seen in an individual who has overdone anerobic exercise. It could eventually occur in the individual who is doing anerobic exercise to balance an aerobic excess, and would be the indication to return to more aerobic activity. From a nutritional standpoint, it is usually caused by failure to clear lactic acid or lactate from the system. It must be converted to pyruvic acid and then to oxyaloacetic acid in the citric acid cycle. The common co-factors found include pantothenic acid, niacin, biotin, and vitamins B_1, B_2 and B_6. Common minerals include magnesium and molybdenum. Phosphorus should also be considered.

The anerobic system can also be challenged by the chemical approach. In this case the patient tastes a source of lactate, such as calcium, magnesium, or sodium lactate. With an anerobic excess, generalized muscle weakening will be observed.

Anerobic Deficiency. This is similar to aerobic excess and is uncommon. The anerobic challenge causes strengthening of the weak muscles, with no change in the strong muscle. This is found in an individual who has over-trained aerobically over a long period. Anerobic activity, which is fast and moderately hard for relatively short distances, should be the same as that described for aerobic excess.

Computing Heart Rate

It is important to determine the maximum heart rate at which a person can exercise, yet still be aerobic. Maffetone[51] recommends the following method that is simple, quite accurate, and safe.

1. Subtract one's age from 180 (180 – age).
2. Modify this number by selecting one of the following categories:
   a. If there is a major illness or recovering from one (such as heart disease, any operation, or hospital stay) or if on medication, subtract 10.
   b. If just beginning to exercise, recently injured, going downhill in a training program or competition, or if there are frequent colds, flu, or allergies, subtract 5.
   c. If exercising up to two years without any real problems, and have not had colds or flu more than once or twice per year, subtract 0.
   d. If exercising more than two years without any problems, while making progress in competition without injury, add 5.
3. Example: Age 40 years and fits into category b: 180 – 40 = 140, then 140 – 5 = 135 beats per minute, which is the maximum pulse at which the body can maintain aerobic activity before becoming anerobic.

Pulse Monitoring

A common method of maintaining oneself at an aerobic or anerobic exercise level is to monitor the pulse rate. It is important that an instrument be used for this. Attempting to monitor the pulse rate by counting the pulse for a specific number of seconds is not a good practice and will probably produce an inaccurate result. For example, monitoring for a short period such as 6 seconds and then multiplying by 10 can miss the actual pulse rate by 10 or 20 beats per minute by only missing the count by 1 or 2. This prevents determining whether one is in an aerobic or anerobic state. Most often when the pulse rate is monitored in this manner, exercise is stopped for a brief period to count the pulse; the heart rate rapidly reduces when exercise is stopped, even for short periods to take a 6-second count. If the pulse is monitored for a longer period for greater accuracy, there is even greater reduction of pulse rate.

Another method used by some is to feel the pulse at the carotid artery while continuing exercise. Even a light pressure here may stimulate the carotid sinus and lower heart rate, giving an incorrect count. Even more significant is the danger of possibly fainting. The ideal heart monitor straps to the chest and monitors the

pulsation from the heart within the thoracic cavity. Pulse rate is presented on a digital read-out on the instrument. An improved model connects the instrument to a digital read-out that is worn on the wrist so it can be more easily read during exercise.

Other pulse monitors clip on the finger or earlobe. A light shines into the capillary bed and is reflected back to a photocell. The pulsing of the blood in the capillary bed is then evaluated by an electronic circuit, and a digital read-out is presented. This type of monitor is not good for running and other mobile sports, but it can be used on a treadmill, stationary bicycle, or skiing machines.

Exercise is slowly brought up to the maximum aerobic pace and maintained below that level within 10 beats per minute. Before discontinuing the exercise, the pulse rate is slowly brought down over about a ten-minute period. Keeping at an aerobic pace is usually a relatively slow activity in comparison to how most individuals exercise when they think they are aerobically exercising. Moving above the maximum heart rate for an aerobic pace takes one into the anerobic stage of exercising, which is sugar-burning and fat-storing. This is often the reason for exercise ineffectiveness, lack of improvement, injury and ill health. In the competitive athlete, this is referred to as over-training.

Maximum Aerobic Pace. Measuring the length of time it takes to do a specific amount of physical activity while maintaining an aerobic pulse rate can be used to evaluate the progression of health from continued exercise. This is called the maximum aerobic pace (MAP). For example, one can measure the time it takes to cover a mile while walking, running, or cycling while maintaining an aerobic pace. As one improves, it takes less time to perform the same activity while in an aerobic pace.

Repeated Muscle Activation Patient Induced

Another type of muscle failure occurs when a person repeatedly uses a muscle and it becomes weak. This has been termed "Repeated muscle activation patient induced" (RMAPI) by Goodheart.[37] This type of repeated muscle action weakness differs from the aerobic/anerobic type in the following ways: (1) The contraction is not against resistance, it is simply the patient contracting a muscle repeatedly. (2) The weakness is not eliminated when the patient therapy localizes the neurolymphatic reflex while doing the contraction. (3) The repeated action tends to weaken the muscles generally, not just the one being activated. (4) Most often there is adaptation by compensatory contraction of the involved muscle. (5) Treatment for correction is different than for aerobic/anerobic type of dysfunction.

There is no indication of the muscle dysfunction on the TS line until the patient does the repeated muscle action. Immediately thereafter the associated TS line point will become active. It can be palpated and will have positive therapy localization. In about sixty seconds it will go away.

A hypertonic muscle caused by a weak antagonist muscle was an early finding in applied kinesiology. Often the weakness is not observed by muscle testing until a subclinical weakness is found, i.e., the muscle becomes weak when one of the 5 factors of the IVF is therapy localized. In this case the body recognizes the subclinically dysfunctioning muscle and contracts the antagonist, even though the weakness is not observed by manual muscle testing in the clear.

In the case of structural imbalance due to RMAPI, it appears that the body recognizes the muscle dysfunction and reacts by contracting the muscle. Thus the muscle that weakens due to RMAPI is in a shortened state in the clear and tests strong.

An example of RMAPI is when a patient continues to have an unlevel head after all other potential causes have been investigated and dysfunction corrected. In the presence of bilaterally strong sternocleidomastoid muscles in the clear, have the supine patient actively turn his head to the high occiput side ten times, thus activating the sternocleidomastoid muscle on the low side. The previously strong sternocleidomastoid muscle on the low side will now test weak if the muscle is involved with RMAPI. If the head is repeatedly turned to the low occiput side, activating the sternocleidomastoid on the high occiput side, there will be no weakening. It is paradoxical that the muscle with RMAPI is on the low occiput side; it is thought that the body contracts the muscle in an effort to compensate for its dysfunction. Following RMAPI correction there will be no weakening with repeated muscle action to either side. Correcting this cause of an unlevel head is very important because of the neurologic ramifications disorganization in the upper cervical area can bring about.

Muscles throughout the body can be involved with RMAPI. The abdominal muscle may weaken with repeated muscle action with the lumbar extensor muscle hypertonic, compounding the increased lordosis and anteriorly tipped pelvis. A patient may initially pass the Trendelenburg test but fail it after gluteus medius repeated muscle action. Testing with repeated muscle action will uncover many reasons for the problem patient.

Treatment. Origin/insertion treatment is applied to the muscle that weakens with repeated action. It usually requires a rather hard application of pressure at the muscle's origin and insertion.

If there is still weakness on repeated muscle action, have the patient activate a remote muscle group such as the wrist extensors ten times. If the original muscle being evaluated weakens without additional

activity, it indicates a systemic problem in addition to the origin/insertion problem. Goodheart[37] hypothesizes a deficiency in synthesizing acetylcholine. He discusses the work of Copeland published in the *Journal of Nutrition* (July 1954), indicating that vitamin E is necessary to bring about the synthesis of acetylcholine. Goodheart has found that vitamin E eliminates the weakening on repeated muscle action. Vitamin E can be supplied in dry tablet form or by wheat germ oil. In some cases vitamin B is also needed. As with other nutritional testing, have the patient taste the substance while actively contracting the muscle ten times and test for its weakening. The muscle will remain strong if the nutrition is effective in treating the condition.

Muscle Stretch Reaction

Initial treatment in applied kinesiology is usually directed toward muscles that test weak in the clear. It is possible that a muscle strong in the clear has some type of dysfunction that may be found by numerous applied kinesiology techniques. One such technique is the muscle stretch reaction.

Normally, when a muscle that tests strong in the clear is stretched and then re-tested, it tests equally strong or stronger because of facilitation by the myotatic reflex. A positive muscle stretch reaction is found when a previously strong muscle is stretched and then tests weaker.

The muscle stretch reaction is associated with the muscle not functioning harmoniously with its fascia, or with trigger points within the muscle. Evidence to examine for muscle stretch reaction is a shortened muscle, local or referred pain, or poor function in the associated organ or gland.

To test for muscle stretch reaction, a muscle must be strong in the clear. It is possible to have a muscle stretch reaction in a muscle that tests weak; however, one must use the five factors of the IVF to strengthen the muscle before it can be tested for muscle stretch reaction.

With the muscle strong in the clear, stretch it to its full range of motion and apply a slight additional stretch at the end; immediately re-test for weakening, which indicates positive stretch reaction. Take care not to stretch the muscle too vigorously, because the normal reaction to a vigorous stretch is temporary inhibition.

Muscles highly dominant in fast or slow fibers may require stretching at speeds appropriate for the fiber dominancy. Postural muscles heavily dominated by slow fibers require a slower stretch than those dominated by fast fibers, which require a faster stretch. If there is a question about the dominancy of slow or fast fibers in a muscle, test both ways.

When a muscle is tested before and after a procedure, such as with the muscle stretch, one must take care that the starting position and follow-through of the test are exactly the same. In general, if the test is started with the muscle longer than its resting length, it contracts more forcefully than when it is shortened at the time of activation. The optimal length for strong contraction varies with different muscle fiber arrangements. The reason for the varying strength is the filament relationships in the sarcomere, explained by the sliding filament theory.[40]

In order to stretch a muscle it may be necessary to modify the usual test procedure. For example, the sternocleidomastoid muscle is usually tested with the patient supine. To evaluate for muscle stretch reaction, it is usually best to test the muscle with the patient seated, taking care to stabilize the shoulders adequately. The psoas can be tested in the usual position; then have the patient turn on his side for the muscle stretch and immediately return to the test position. The psoas can also be stretched by having the supine patient drop his leg off the side of the table.

When testing for muscle stretch reaction, it is necessary that all factors be considered. In addition to stretching the muscle, joints are taken through range of motion. If there is a subluxation of a joint that is moving during the muscle stretch, there may be a challenge to the joint proprioceptors that causes the following test to be weak. Cutaneous receptors may be involved, as well as ligaments (discussed in the ligament stretch reaction). Differential diagnosis must be done to determine that, in reality, there is a muscle stretch reaction when the muscle tests weak after stretching.

The muscle stretch reaction is relatively common. It has given an additional diagnostic component to three common methods of treatment: trigger points,[82] fascial massage,[67] and myofascial gelosis.

Fascial Release

The muscle and its surrounding fascia should be the same length and function together. When the muscle contracts, the fascia should be able to move smoothly with the muscle. As long as the muscle and fascia operate as a unit, the body interprets their activity as integrated and consistent. Rolf[67] describes fascia as fibrous connective tissue forming a network of support that connects and communicates throughout the body. It supports each visceral organ and muscle with elastic membranous sheaths, and allows muscles to

glide smoothly over one another. Trauma causes fascia to become denser and thicker as it heals, and it may also cause layers to adhere to one another.

Fascial release technique is based on the premise that the muscle and its fascia are not functioning in harmony. Stretching the muscle causes a neurologic feedback of the disparagement between the two structures, causing the muscle to temporarily test weak. The muscle will be shortened in its resting length, causing a limited range of motion.

Muscle-Organ Correlation

Goodheart[29] likens muscles to pumps for the lymphatic and circulatory systems. A muscle that needs fascial release has an active relationship with its neurovascular and neurolymphatic reflexes. Positive therapy localization in the clear is usually not present; however, when the reflex is therapy localized while the muscle is stretched, the positive stretch reaction is abolished. When a muscle and its fascia are treated and the NL and NV reflexes are stimulated, there is often improved function in the associated organ or gland. The response may be due to the muscle/fascia dysfunction causing poor lymphatic and vascular flow by reacting with the neurolymphatic and neurovascular reflexes. In addition, treatment to the reflexes helps obtain better response from the fascial release with reduction of recurrence. An example of the application of fascial release to improve organ/gland function is presented with thyroid treatment on page 549.

Fascial release treatment is simple to apply. It is the deep massage of a muscle designed to break any adhesions between the fascial layers so the muscle and fascia can function harmoniously. The deep massage of the muscle is not direction-specific; it can be with or against the fibers. The only directional consideration is that any movement must be with the flow of vessels, the usual consideration in massage. This is to avoid rupturing the one-way valves of the veins and lymphatic system by forcing fluid against the direction of their flow. After treatment with fascial release, re-test the muscle for muscle stretch reaction. If it is still present, the fascial release was not effectively applied, or the muscle may have trigger points in addition to or in place of fascial disharmony.

Neurolymphatic and neurovascular reflexes for the muscles should be routinely treated when fascial release is applied. Vitamin $B_{12,}$ in combination with stomach and liver concentrate or nucleoprotein extract, helps prevent recidivism on a clinical basis. It is routinely recommended three times a day for at least two weeks. A low dosage of B_{12} — 5 micrograms — appears to work better than higher dosages. Vitamin B_{12} without the stomach and liver co-factors does not appear to have any value on a clinical basis.

Trigger Points

Numerous methods of examining for and treating trigger points have been used for over thirty-five years. The most common method is Travell's,[80] who popularized the idea of trigger points. Travell and Simons have exhaustively covered the subject.[82,83] Applied kinesiology muscle stretch reaction adds another aspect to the diagnosis of trigger points and, more important, immediately indicates whether the treatment was effective.

Travell describes a trigger area as "...a small, hypersensitive region from which impulses bombard the central nervous system and give rise to referred pain."[81] Although there is consistency of the referred pain, it does not necessarily follow the distribution of sclerotomes, dermatomes, or peripheral nerves. The location of pain is known as the reference zone. The trigger point may lie within the reference zone or be remote from it.

There are active and latent trigger points. When digital pressure is applied to an active trigger point, pain in the reference zone will increase or be produced if none is present. Latent trigger points are locally tender but they will not activate a referred pain. The trigger point will have a hypertonic (shortened) muscle associated with it that will have a positive muscle stretch reaction. The patient's symptomatic pattern may relate directly to the trigger point or to the hypertonic muscle with stretch reaction. The hypertonic muscle can cause structural imbalance with resulting joint strain, or the muscle may fail to support a joint after the stretch during normal physical activities.

Many trigger points may be associated with a particular condition. Usually there is one that is most active and clinically evident. The additional points are called satellite trigger points; they must be found and treated for lasting correction. Webber[89] states that there may be as many as five or six trigger points directly affecting the shoulder, with more in the arm or hand. He also states that there may be as many as fifty trigger points in the body affecting headaches; however, this author has never found that many.

Trigger points may simulate many conditions and/or cause them. Travell[78,79] specifically refers to trigger points in the pectoralis major creating breast pain and soreness, and associates trigger points in the temporalis, pterygoid, upper trapezius, sternocleidomastoid, and other muscles as causes of headaches, dizziness, and other symptoms. Cohen,[9] in a study of the neck proprioceptive mechanisms, established their important role in body balance as specific physiological mechanisms giving support to mechanical dysfunction of the cervical region, causing vertigo and disorientation.

Goodheart consistently emphasizes the necessity of having a level head in postural orientation. (The subject is discussed in depth in this author's Volume II applied kinesiology text.[86])

Etiology

Trauma is a common cause of trigger points. The trauma appears to be to the muscle involved with the complex in the form of direct injury, excessive stretching, or contraction. Once a trigger point develops, repeated muscular stress of a lesser degree can activate pain in the reference zone, especially when the muscle becomes fatigued.

Diagnosis

The primary method used to diagnose trigger points and their relationship to referred pain has been Travell's charts. The trigger point frequently is not in the area of the patient's major complaint. It can generally be palpated as a fibrous, band-like area. As one palpates over the band, the muscle — and often the patient — will jump, giving rise to the "jump sign" described by Simons.[73] In addition to the palpatory indicators and jump sign, the involved muscle has a limited range of motion and a positive muscle stretch reaction.

To elicit the jump sign, the muscle being evaluated is placed under moderate tension and the examiner briskly pulls his finger across the firm band of muscle. A positive jump sign is contraction of the muscle band. This response is found consistently in muscles that contain trigger points, but not in normal muscles.

Trigger points are also found in ligaments, tendons, and fascia. When the tendon and fascia are involved, there will almost always be a positive muscle stretch reaction. Trigger points in ligaments exhibit the radiation of pain to the reference area. Often there is a joint subluxation associated with trigger points in ligaments that must be corrected for lasting results.[89] Structural distortion often serves as a nucleus for the myofascial trigger area. Webber[89] points out the importance of manipulation to regain structural balance.

Correction

Many treatment approaches have been described for the elimination of trigger points. Mentioned here are the intermittent cold with stretch and digital pressure methods.

The method of intermittently chilling the muscle with stretch has gone through revisions. Initially ethyl chloride was used for stretch and spray. Because ethyl chloride excessively chilled the muscle and was highly flammable and toxic, it was replaced by Fluori-Methane®, which had the advantage of being non-toxic, nonflammable, and did not provide excessive cold that is to be avoided. Because of the probable detrimental effect on the ozone layer of the upper atmosphere, Fluori-Methane® containing chlorofluorocarbons has been discontinued in the treatment of trigger points.[83]

Chilling the skin to elicit the reflex effects can be done by intermittently stroking the skin with ice. Ice can be prepared by filling a small paper cup with water and inserting a stirring stick as a handle before freezing. The paper is torn back to expose the ice and then covered with thin plastic sheeting so the ice does not make direct contact with the patient's skin. The skin should remain dry because dampness reduces the rate of skin temperature change and prolongs and diffuses the cooling effect.[83] Adequate stimulus for thermoreceptors is a change in temperature, not the absolute temperature.[7]

There are two techniques for chilling the area, depending upon the location of the trigger point and the reference zone of pain. In some cases, the only involvement is within the muscle containing the trigger point. In this instance, the muscle is covered from origin to insertion while a gentle stretch is placed upon the muscle. As the process continues, a slow increase in muscle length will be observed.

Usually the trigger point is outside the reference zone of pain. Begin at the trigger point and cover the muscle from origin to insertion, continuing the application into the reference zone. Use the edge of the ice to repeat applications at about four inches of movement per second and about one inch apart until the entire trigger point, muscle, and reference zone are covered. During the application the muscle is gently stretched, as in the muscle-only involvement. There will generally be an increase in muscle length. Care must be taken with either approach not to overstretch and strain the muscle. Also, it must not be overcooled, causing increased hypertonicity.

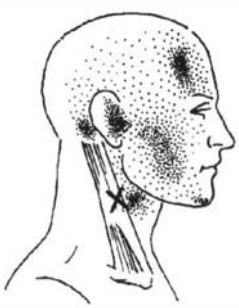
Mennell[56,57] offers a hypothesis of the mechanism involved in intermittent cold with stretch. A hypertonic shortened muscle excessively stimulates afferent nerve fibers, possibly from the neuromuscular spindle cell, Golgi tendon organ, or joint receptors. The noxious impulses appear to reflex at the spinal cord level, or they are processed at higher centers to reflex back to the reference zone of pain. It appears that stimulating the thermoreceptors produces impulses that interfere somewhere in the cycle of noxious impulses to break the reflex. The exact mechanism is unknown, but as Mennell states, "... we shouldn't wait years for development of scientific evidence before giving relief to suffering patients."

Another method of effectively treating trigger points is to hold prolonged, heavy digital pressure over the trigger point. This usually causes a strong pain to radiate into the reference zone. A common area effectively treated this way is the intrinsic muscles of the upper cervical vertebrae. The trigger point will often be found in the obliquus capitis inferior or superior, or in the rectus capitis posterior major or minor. The physician's thumb pressure into the trigger point radiates pain into the vertex, frontal, or behind-the-eye

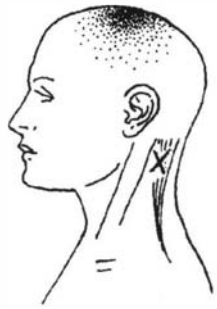
areas of the head. It may be necessary for the physician to move his thumb slightly and change the vector of force to obtain the maximum amount of radiation to the reference area. A solid, heavy pressure is held on the trigger point until the pain in the reference zone subsides. This may take four or five minutes in severe cases. Sometimes the reference pain is so severe that only a light pressure can be applied at the start, increasing slowly as the patient's tolerance level increases. Although the trigger points are found in the suboccipital area, the patient may not have pain there until the digital pressure is applied.

The reader is encouraged to study the texts of Travell and Simons on trigger points.[82,83] They have done an exhaustive study and report.

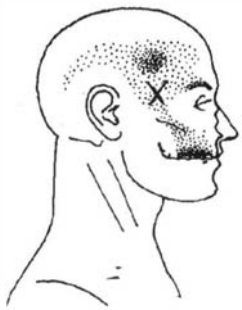
Teaching the patient to apply the intermittent cold with stretch technique is discouraged. If repeated use of the technique is necessary to control pain, the basic underlying cause has not been corrected. When there is effective relief from pain but the condition returns, one should first consider the possibility of an organic disease or some other condition that causes referred pain, such as cardiac, gallbladder, and hiatal hernia conditions. After eliminating these, consider structural strain anywhere in the body. Satellite trigger points are often produced by structural strain and contribute to the recurrence of a primary trigger point.



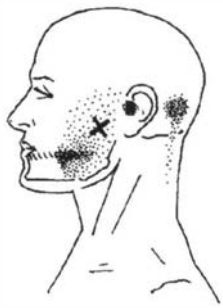
6—3. Sternocleidomastoid



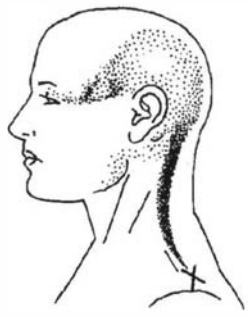
6—4. Splenius capitis



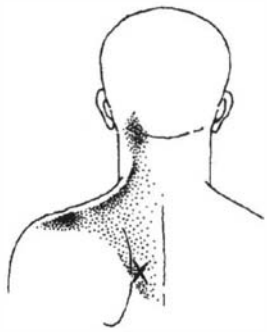
6—5. Temporalis



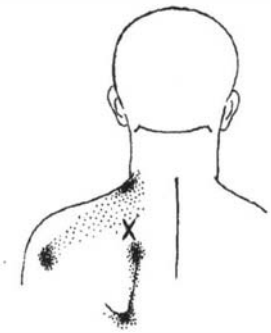
6—6. Masseter



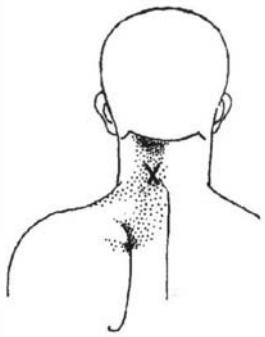
6—7. Trapezius



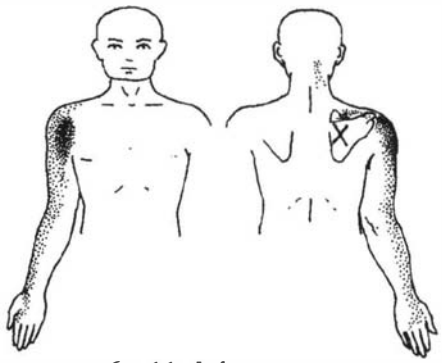
6—8. Trapezius



6—9. Levator scapula

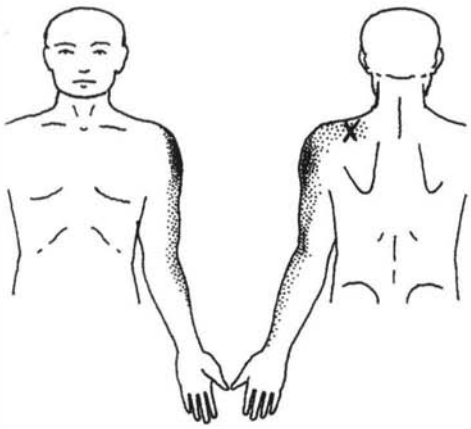


6—10. Posterior cervical

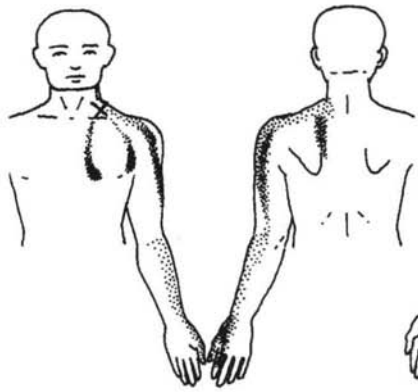


6—11. Infraspinatus

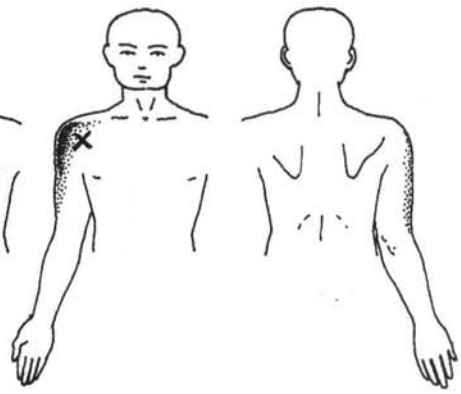
6—3 through 6—40 (above and on following pages). Predictable patterns of referred pain from trigger points, with captions indicating muscles in which offending trigger points are situated. The location of each trigger point is designated by "X." The shaded areas are the constant areas, the heavily stippled areas are commonly observed areas, and the lightly stippled areas are associated areas of pain referred from the trigger points. The referred pain pattern may be reproduced by palpation of the trigger point. By observing the pattern of pain the location of the trigger point may be predicted. (From Travell and Rinzler;[81] with permission of Dr. Travell and Postgraduate Medicine.*)*



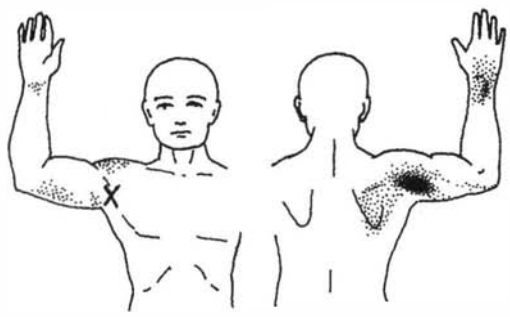
6—12. Supraspinatus



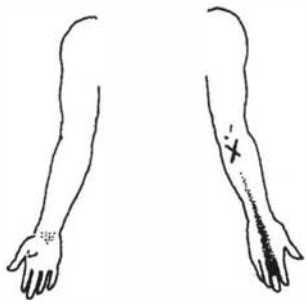
6—13. Scaleni



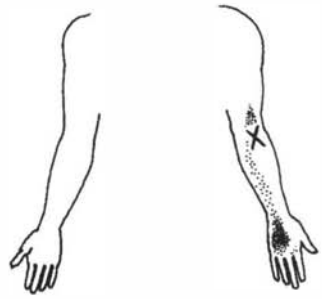
6—14. Deltoid



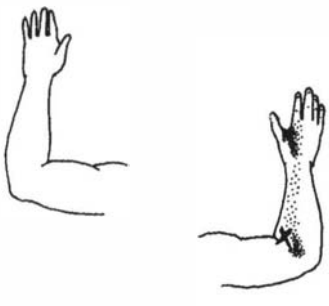
6—15. Subscapularis



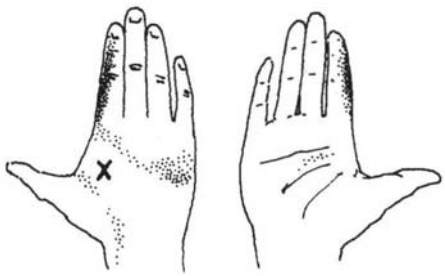
6—16. Middle finger extensor



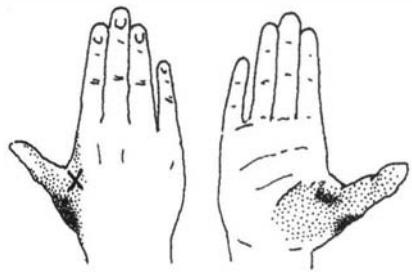
6—17. Extensor carpi radialis



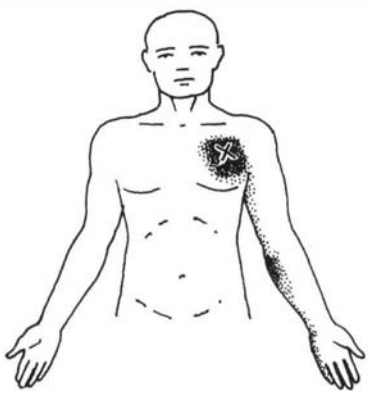
6—18. Supinators



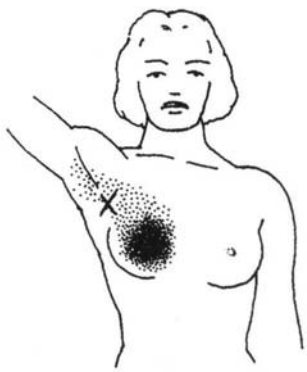
6—19. First interosseous



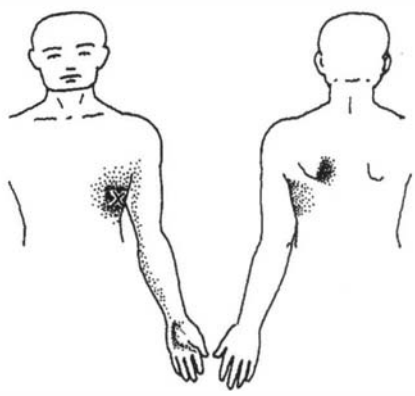
6—20. Adductor pollicis



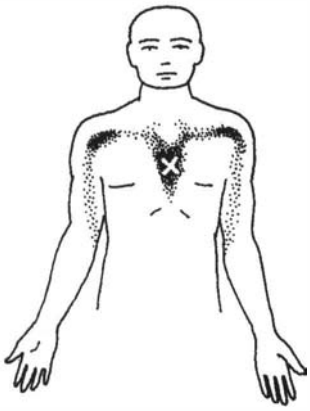
6—21. Pectorals



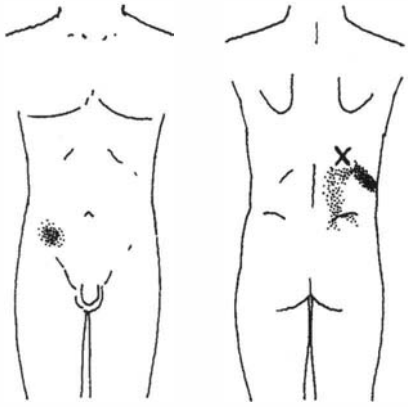
6—22. Pectoralis major



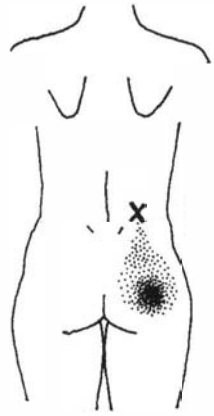
6—23. Serratus anterior



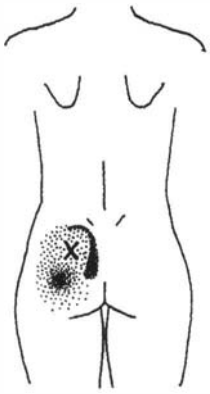
6—24. Sternalis



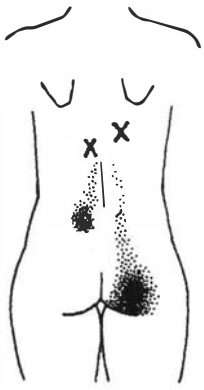
6—25. Iliocostalis



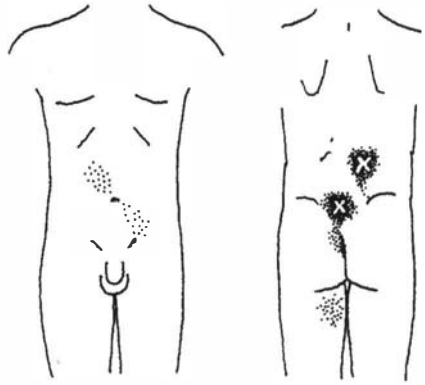
6—26. Iliocostalis



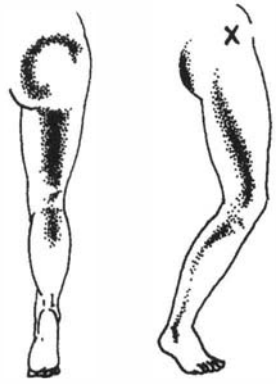
6—27. Gluteus medius



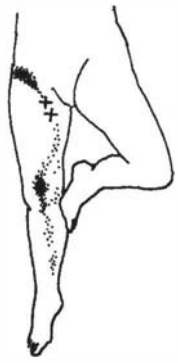
6—28. Longissimus



6—29. Multifidus

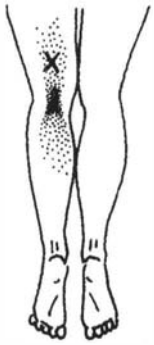


6—30. Gluteus minimus

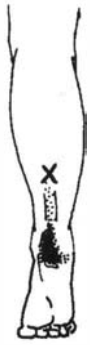


6—31. Adductor longus

6—32. Vastus medialis

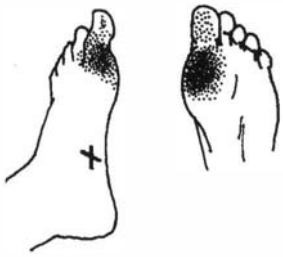


6—33. Biceps femoris

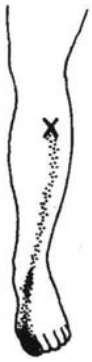


6—34. Soleus

6—35. Gastrocnemius



6—36. Abductor hallucis



6—37. Tibialis anterior

6—38. Long extensors

6—39. Short extensors

6—40. Peroneus longus

Myofascial Gelosis

Connective tissue is pervasive throughout the body. If the possibility existed to remove all of the tissue from the body except connective tissue, there would be little change in the body outline. The skeleton would be maintained by ligaments. The muscle form would be preserved by the connective tissue sheaths of the muscle fibers and the intermuscular septa. Blood vessels and the nervous system would maintain their shapes as well as the abdominal organs that would be held in position by the peritoneal attachments.[65,84]

Aside from the bony skeleton most of the connective tissue is arranged in the form of fascia. The soft tissues are supported in the body by the connective tissue, with a continuity from the toes to the head forming muscular attachments, supporting membranes, intermuscular septa, visceral ligamentous attachments, and investing sheaths for the blood vessels and nerves.[90] This connective tissue in turn is supported by its connections to the bony skeleton. Primary distortions in the skeleton place secondary tension in the fascia; on the other hand, fascial tension distorts the skeleton. Muscles will react to these distortions, or muscle imbalance in the first place may be the cause of the distortion. Pain results in addition to general health problems caused by the distortions, which is probably the most common reason patients seek a doctor's assistance.

The most important point is the continuity of fascia. One can travel from any portion of the body to another and never leave the fascia. Structural imbalance in the foot may start disorganization that results in fascial tension. The foot strain can be the remote cause of tension in the cervical spine fascia. In the cervical area the fascia is continuous with the dura, stressing structures of the skull foramina.[64] Rolf[67] notes that fascial tension is often observed by pain at the crown of the head from sagging postural drag.

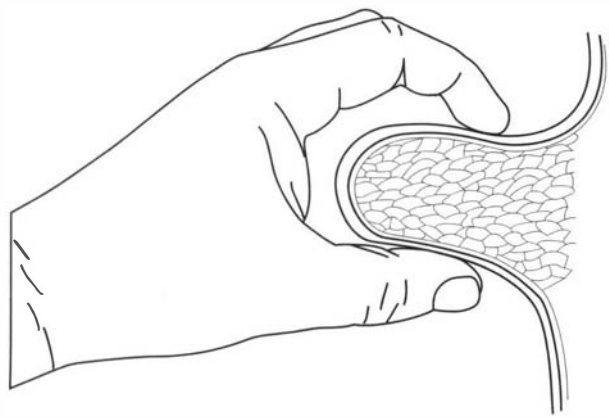
Fascia is characteristically collagenous fibers embedded in a ground substance of amorphous semifluid gel. According to Travell and Simons,[82,83] myogelosis (an older term) is "Circumscribed firmness and tenderness to palpation in a muscle or muscles. *The name is derived from the concept that the regions of circumscribed firmness were due to localized gelling of muscle proteins. Focal tenderness and palpable taut muscle fibers are also characteristic of myofascial trigger points. Most patients diagnosed as having myogelosis also would be diagnosed as having myofascial trigger points."* Rolf[67] refers to inflammatory illness or trauma causing layers of fascia to adhere to each other "... — they seem to be 'glued' together." A similar consideration of collagenous fibers losing elasticity and becoming sticky is the gel-sol description in which gel is in a more soluble form when there is freedom of motion.

Goodheart[38] has described a diagnostic method in applied kinesiology that appears to be specific for

distinguishing myogelosis from other similar types of muscular dysfunction that require treatment, such as intermittent cold with stretch, and fascial release. All three of these show the muscle stretch reaction requiring differentiation to determine the best method of treatment.

First one must know when to suspect myofascial gelosis. It is suspected when there is reduced range of joint motion. It may be gross limitation, but more frequently it is observed as a subtle restriction as one moves the structure through its range. The latter feels like a restrictive barrier that can be moved through to a full range of motion. As Goodheart[38] states, "The 'art' of diagnosis is the gently feeling or sensing for the slight restriction of 'bind' or 'barrier' when R.O.M. is carefully evaluated."

The muscle involved with myofascial gelosis is identified by pinching the muscle tissue and observing the muscle or an indicator muscle weakening. The pinching is similar to the "pincer palpation" of Travell and Simons[82] in trigger point diagnosis. The force of the pinch is solid, but not strong enough to cause pain to the patient. One must take care that the muscle tissue is being pinched. There is no effect from pinching the skin.

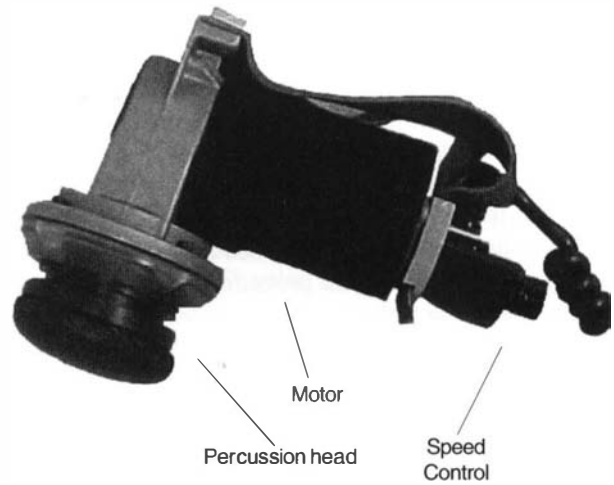


6—41. A solid, but not painful pinch of muscle tissue causes the muscle or an indicator muscle to weaken if percussion is needed for myofascial gelosis. Take care that actual muscle tissue is being pinched. (*After* Myofascial Pain and Dysfunction, The Trigger Point Manual, Vol. 2, *Janet G. Travell and David G. Simons*.)

Usually the muscle being investigated can be tested for weakening after the pinch test. In the case of muscles that can't be easily tested, such as the rectus capitis posterior minor and masseter, the weakening reaction to the pinch can be observed by testing a remote indicator muscle.

Percussion over the involved muscle provides rapidly effective treatment for myofascial gelosis. Percussion can be done by simply tapping on the muscle with one's fingertips. When done in this manner the fingers should be struck over the muscle sharply enough that they bounce back and the force is not painful to the patient. The tapping is done at about one Hz. Travell[83] found that striking the area of tenderness over the quadratus lumborum with a percussion hammer at one Hz is effective in eliminating trigger points or myofascial gelosis. Eight to ten taps are applied with about the force one would use to elicit a deep tendon reflex from the quadriceps muscle group.

An effective treatment for myofascial gelosis is application of percussion with a percussive device as popularized by Robert Fulford, D.O.[23] At this time there are two versions of this instrument. The original instrument[20] is manufactured in Germany. The percussion head is powered by a flexible shaft connected to a remote motor. The more recent version[44] manufactured



6—42. The American version of the percussion device is self-contained and easier to manipulate when treating a patient.

in the United States is a self-contained unit. Percussion application with an instrument may feel like a vibrator to the patient but there is a significant difference. The percussor action is an in-and-out action like a jackhammer. Vibrators as used in massage usually have circular motions and are ineffective in correcting myofascial gelosis.

When using the percussive device for myofascial gelosis, the speed setting should be set to very slow for best results. Percussor application for myofascial gelosis is done by applying a light force with the percussor and monitoring the application with the physician's other hand. The monitoring hand is often referred to as the "listening" or "smart" hand. It is the one that evaluates the application of percussion. The monitoring hand is placed opposite the area being percussed to sense the percussion. Reduce the percussion rate or force to just below what the monitoring hand can feel. Maintain the percussion for 30 to 60 seconds. Range of motion should be increased, and pinching the muscle should no longer cause muscle weakening, nor should it weaken following stretching.

Muscle Stretch Reaction Differential Diagnosis

When a strong in the clear muscle weakens following stretching the cause must be differentiated between trigger points, fascial adhesion, and myofascial gelosis. There are many common factors among the three types of dysfunction. All have a reduced range of motion which should improve following effective treatment. As noted previously, Travell and Simons[81] list myogelosis as an older term that today would probably be diagnosed as trigger points. Illustration 6—41 of the pincer diagnosis for myofascial gelosis is after an illustration in their text that originally had a circle in the muscle mass depicting a trigger point. Fascial release and myogelosis techniques are involved with the continuity of the fascia. Because of this either can cause remote problems in addition to the local involvement.

In AK it is important to determine the cause of the muscle stretch reaction to determine the best treatment method for lasting results.

Trigger points are identified by a localized fibrous area often in a band-like area. They have localized tenderness, and specific identification is activation of referred pain from digital pressure; however, referred pain is not mandatory for classification as a trigger point.

Muscles needing fascial release are more involved with the applied kinesiology muscle-organ/gland association. When either or both of the muscle's neurolymphatic or neurovascular reflexes are therapy localized while the muscle is stretched, it will no longer weaken. Treatment is directed to the neurolymphatic and neurovascular reflex(es) that eliminated the positive stretch reaction, in addition to the deep heavy massage of fascial release. Nutritional support may also be needed, especially if there is an apparent muscle-organ/gland association as previously discussed.

The first clue that myofascial gelosis may be involved is subtle restriction as the limb is moved through its range of motion. There is a "stickiness" to the motion. When myofascial gelosis is present the muscle will test weak following a solid pinch of the muscle mass. General indicator muscles will also test weak following pinching of the muscle mass. There is no such global effect when pinching a muscle with trigger points or in need of fascial release.

Ligament Stretch Reaction

A positive ligament stretch reaction is present when muscles that previously tested strong test weak after the ligaments of an associated articulation are stretched. The cause of positive ligament stretch reaction appears to be generalized stress of the adrenal glands. If the individual is not hypoadrenic, he will be a type A individual who is constantly pushing himself and is usually very successful in whatever endeavors he attempts. The relationship of the adrenal gland and the ligaments was first observed by Goodheart[27] with regard to low back and cervical pain associated with overachievers. This is called emotional backache and is discussed on page 437. Deutsch[16] associated Goodheart's findings with ligament stretch reaction throughout the body when there is adrenal stress. The generalized ligament stretch reaction usually has an active adrenal neurolymphatic reflex and needs adrenal concentrate; to a lesser extent, the neurovascular reflex, meridian system, and cranial stress receptor are involved.[69]

A specific indication to evaluate for ligament stretch reaction is generalized pain around a joint or in several joints of the body. There are numerous reasons to routinely evaluate all patients for ligament stretch reaction. Durlacher[18] points out that individuals with ligament stretch reaction are more vulnerable to injury. Both muscle and ligament support are important in the articulations when they are at their extremes of motion. An individual running and quickly turning puts strain into the knees and other articulations. Normally the muscles should contract at extremes of motion to support the joint. Korr[49] points out that a muscle is the only active somatic tissue capable of rapidly changing forces. The other tissues are passively moved, immobilized, pushed, pulled, compressed, and altered by forces external to themselves, i.e., those forces of muscular origin and those external to the body, such as gravity, and forces of locomotion, especially when turning and twisting.

The role of adrenal stress on the ligaments and supporting muscles of the joints is important in nearly all daily activities. Of special interest are the factory worker and athlete. An athlete is often under considerable stress during competition, even though this may be eustress.[72] If there is an underlying adrenal stress disorder, the ligament stretch may be present and cause the muscle function so necessary to perform optimal joint motion to fail. A factory worker may be under production stress or in conflict with fellow workers or his employer, creating constant stress day after day and making him more susceptible to structural problems as a result of the ligament stretch reaction. The emotional strain on the adrenal gland can be compounded by other factors of stress, such as excessive refined carbohydrate intake, overheating or overchilling, and long physical workouts leading to exhaustion.

Evaluating an individual for ligament stretch reaction is a good preventive approach for on-the-job injuries, and it helps an athlete perform to his maximum ability without injury. Another reason to routinely evaluate for ligament stretch reaction is the prevention of iatrogenic problems from joint manipulation. When a routine adjustment is made and the patient reacts severely to it, it is often due to ligament stretch reaction. The ligaments appear to be more susceptible to injury under these conditions.

Examination

Examination for ligament stretch reaction is relatively simple; however, a few precautions must be taken. The optimal muscles to test are those directly associated with the joint ligaments to be tested. For example, when testing the knee ligaments, evaluate the quadriceps, hamstrings, sartorius, or gracilis. The muscle tested must be strong in the clear, and the test after stretching the ligament should duplicate the original one as closely as possible.

Stretch the ligaments, then immediately test a previously strong muscle for weakening. In most cases it is best to attempt to move the joint in a direction that is not normal. For example, when testing the knee ligaments try to abduct or adduct the tibia on the femur. Since there is limited motion in this direction, the associated muscles are not stretched, causing the muscle to weaken from a muscle stretch reaction. In addition, one should stretch the ligaments in several directions. If there is a knee subluxation, the muscle will weaken when the joint is moved opposite the direction to correct the subluxation. When there is ligament stretch reaction there will be weakening no matter which direction the joint is moved. Of course, it is possible to have a subluxation and a ligament stretch reaction in the same articulation.

Usually the ligament stretch reaction appears to be systemic, affecting all the ligaments of the body. Blaich[5] reports specificity of the ligament stretch reaction to the knee in acute and chronic conditions. Evaluation and correction are the same, whether the condition is systemic or limited to only one articulation — often the knee.

Correction

Therapeutic effort is directed toward support of the adrenal gland. This is done nutritionally and with the five factors of the IVF as described with adrenal stress disorder in Chapter 11. Often the patient has relative hypoglycemia or some other imbalance of the endocrine system.

Strain/Counterstrain

For normal pain-free function there must be organization between the muscles, their proprioceptors, and the nervous system in general. Disorganization often comes from improper afferent stimulation. When proprioceptors send conflicting information, there may be simultaneous contraction of the antagonists,[59] which may result in extensor muscle contraction during flexion. Without antagonist muscle inhibition, joint and other strain results. A reflex pattern can develop in the neuromuscular system that causes muscle or other tissue to maintain this type of continuing strain. It often relates to the inappropriate signaling from muscle proprioceptors that have been strained from rapid change that does not allow proper adaptation.[49]

When an individual slips or otherwise rapidly changes position, there is a quick stretching of a muscle or a group of muscles with shortening of the antagonists. The neuromuscular spindle cells from the rapidly lengthened muscle immediately send afferent impulses over the Ia fibers, signaling the central nervous system for adaptation to take place. Meanwhile, the neuromuscular spindle cells of the hypershortened antagonistic muscle are silent.[39] Korr[49] offers the hypothesis that the central nervous system, seeking a response from the hypershortened and silent primary ending, begins an extraordinary outflow followed by unusually fast stretching, resulting in high gamma gain that the body is unable to reduce to normal. The recipient muscle of the imbalanced nerve supply obviously does not function in keeping with optimal body needs.

Jones[46] has developed a technique to treat this type of muscle dysfunction, which is often related with painful somatic conditions. He named the technique strain and counterstrain to describe the cause and treatment. The etiology is strain described by Jones as "...overstretching of muscles, tendons, ligaments, and fascia with the attendant neuromuscular strain reflexes. The focus of attention is directed especially at the neuromuscular reflexes rather than the tissue stresses." It is hypothesized that the condition develops as a result of overstretching (strain) of structure, with a rapid return to neutral. It is further hypothesized that if the same overstretching is not returned rapidly but slowly, no neurologic deficit occurs. Jones' treatment is to apply counterstrain to the neuromuscular reflexes originating the condition. Counterstrain, then, is "...a mild strain (overstretching) applied in a direction opposite that false and continuing message of strain from which the body is suffering."

Jones' initial observation of the efficacy of counterstrain was with a patient who was unresponsive to treatment. The patient had been unable to sleep because of pain. Jones attempted to find a comfortable position for the patient to aid him in sleeping. After

twenty minutes of trial and error, a position was finally achieved in which the patient's pain was relieved. Leaving the patient in this position for a short time, Jones was astonished when he came out of the position and was able to stand comfortably erect. The relief from pain was lasting, and the patient from that point had an uneventful recovery. Positioning the patient for comfort had succeeded where four months of traditional chiropractic and osteopathic manipulations had failed.

The technique — strain/counterstrain —presented here is a modification by Goodheart[34] of Jones' original work. Positions for counterstrain treatment developed by Jones are presented in his text, *Strain and Counterstrain*,[46] the perusal of which is highly recommended. Goodheart has contributed by adding an applied kinesiology diagnostic approach and placing the technique in perspective with other AK therapeutic measures.

Etiology and Body Language

The treatment devised by Jones is primarily directed toward painful conditions. The diagnostic approach added by Goodheart has revealed that neuromuscular function may also be improved by strain/counterstrain technique in the absence of a painful condition. There will usually be localized pain during examination, but it may not be the patient's primary complaint. The key to using strain/counterstrain technique is to direct treatment to the correct muscle. Once the approach to painful conditions is learned, diagnosis and treatment with strain/counterstrain technique in nonpainful conditions will be obvious.

Neuromuscular dysfunction that responds to the strain/counterstrain technique may be from a recent trauma or buried in the patient's history. The condition may be caused by an individual slipping, resulting in a severe overstretching of a muscle that is immediately counteracted by the muscle's contraction. The neuromuscular problem that develops in this pattern is not in the muscle that is stretched; rather, it is in the antagonistic muscle that is hypershortened and then immediately lengthened by the protective reaction of the initially stretched and strained muscle. Korr[49] proposes the hypothesis that under certain conditions the gain of the gamma system may be set too high, keeping the intrafusal fibers in a chronically shortened state. It appears that when the length of the antagonistic muscle rapidly changes without the central nervous system ordering it, the neuromuscular spindle cell fails to reset properly, leaving the intrafusal fibers contracted. Although the hypershortened muscle is not the area of initial strain, the abnormal neuromuscular pattern is set up within it and treatment must be directed there. Pain in the posterior of the body requires examination and treatment in the anterior or antagonistic area. One can think in terms of extension pain needing treatment in flexor muscles.

Under normal conditions the initially strained muscle is sore, but it returns to normal within a reasonable time. When the strain/counterstrain pattern is present, the antagonistic muscle fails to come back into neurologic equilibrium, maintaining neuromuscular imbalance. The initially strained muscle or joint remains painful past the time the trauma should be healed.

Although the patient does not usually complain of pain in the secondarily shortened muscle where the continuing disturbance is present, there will be tenderness in it that is located by palpation. The patient easily recognizes the pain when it is pressed by the examining physician's fingers. When the involved muscle is put in the position of original strain, the tenderness is relieved and relaxation begins. When away from the position of original strain, the gamma system is out of balance. Balance, although not proper, is obtained in the position of original strain. If the original movement out of this position had been slower, the condition would not have developed because the system would have had time to adapt. Treatment is directed toward allowing the body to reset the system to proper equilibrium.

Jones' approach to treating this condition is to passively find the position of relief that is close to or at the point of initial strain. This is the point Jones refers to as counterstrain, or the point of treatment. When the proper counterstrain is obtained, the patient is held there for ninety seconds; then slowly and passively the structures are returned to neutral. This appears to allow the gamma system time to reset normally.

The strain causing the neurologic dysfunction may be relatively mild and not even recognized by the patient as the cause. Jones illustrates this type of strain by a generally sedentary individual squatting to pull weeds in a garden. In this position the psoas muscle is shortened for a prolonged period, while the back extensors are strained by overstretching. If the person rapidly stands, low back pain may be felt that persistently continues. Although the pain is felt in the back, the dysfunction is probably in the psoas muscle that was quickly lengthened upon standing. When rapidly coming out of the strained position, the neuromuscular spindle cell(s) in the psoas are not reset for proper relationship between the intra- and extrafusal fibers. Apparently this is because the gamma system remains set too high, which keeps the intrafusal fibers shortened. The intrafusal fibers are erroneously in balance with the extrafusal fibers when the muscle is shorter than its normal resting length. Under these circumstances, when an individual is in a neutral position there is disparagement between the psoas intra- and extrafusal fibers. They are only in equilibrium when in the position of original strain. Although the cause of the disturbance is in the psoas muscle, the patient rarely complains of pain at that location; however, when the physician palpates the psoas the patient easily recognizes a small

localized area that is very painful. Hip flexion, which shortens the psoas muscle, reduces the disparagement between the intra- and extrafusal fibers and relieves the localized pain in the psoas. When the exact original position causing the involvement is reached, there will be little or no localized pain in the psoas on digital pressure. This is where the neuromuscular system is erroneously in balance.

Examination and Diagnosis

Understanding that the cause of the continued pain one suffers in a strain/counterstrain condition is usually not at the location of pain but in an antagonistic muscle is the most important step in solving this problem. Because the perpetuating cause is located away from the pain, many of these cases are treated by various techniques directed at the pain with no success. The location of pain may be in a muscle, tendon, or ligament. The perpetuating problem is in the neuromuscular spindle cells of a muscle that was shortened during the strain and then rapidly lengthened, causing failure of the neuromuscular spindle cell(s) to adapt through the gamma system to the new muscle position. Pain will increase in certain directions and decrease in others. The position that decreases pain is the one that shortens the muscle whose neuromuscular dysfunction is perpetuating the condition. The muscle needing treatment can usually be determined by analysis of the pain location, postural analysis, muscle testing, and movement that accentuates and relieves the pain. Imbalance of the neuromuscular system is increased with certain movements and decreased with opposing movements. Increase of neuromuscular imbalance causes pain; decrease relieves it. The patient can easily tell you which movements increase pain, but he is rarely aware of movements that decrease it. After the patient has explained the movements that increase pain, consider which muscles are antagonists. They are the ones shortened in the original strain, and are where the disturbance will usually be found.

Postural balance will often be affected because of the neuromuscular imbalance. The muscle that was shortened during the strain will have its gamma system set to maintain a shortened position. This causes a structural imbalance toward the muscle needing treatment. Evaluating A to P and bilateral balance often provides a clue about the location of the disturbance.

If the trauma that caused the strain is known, it can be analyzed for the muscle(s) that would be hypershortened during the strain.

The most specific diagnostic factor indicating that a muscle needs strain/counterstrain technique was developed by Goodheart.[34] The muscle needing treatment, when strong in the clear, will weaken after it is maximally contracted for three seconds. Following successful treatment, the test will be negative. This enables the physician to determine the exact muscle requiring treatment, as well as whether treatment is successful or if there may be secondary areas in the muscle needing treatment.

Jones delineates sixty-four locations he frequently finds involved. With the principles of examination delineated previously, one can usually determine where treatment is needed. Discussed here will be various types of conditions, possible etiology, and their analysis and treatment. Throughout applied kinesiology material are examples of specific strain/counterstrain techniques applicable to different conditions.

Psoas

A common condition requiring strain/counterstrain technique is low back pain relating with the psoas muscle, as discussed in the gardening example. Differential diagnosis of this condition is typical of all conditions that may or may not relate with strain/counterstrain.

Initially examine the lower back for evidence of local involvement, such as subluxations, fixations, sprain, intervertebral disc involvement, and other conditions of the lumbar area.

If there is a history of pain when rapidly rising from a squatting position, one can easily recognize possible involvement of the psoas from having been shortened for a prolonged period and then rapidly lengthened. In this case, simply palpate the psoas for a localized tender point, as described below.

When there is no history indicating strain setting up a secondary muscle imbalance, such as that treated by strain/counterstrain technique, one can analyze the condition to find the probable secondary muscle perpetuating the condition. As mentioned, patients will usually not complain of pain in the secondary muscle, but they can readily tell you what motion increases the pain. Analysis, then, is of what muscles would shorten with the strain.

The gardening example provides us with a condition to analyze. In this case the patient comes in with low back pain but no history of etiology. Local examination of the lower back is negative, or any findings have been corrected and the pain continues. There is an antalgic position of right anterior flexion of the lumbar spine. The patient points out increased pain on extension, indicating the initial strain was probably flexion because motion that lengthens the involved muscle further stresses the dysfunctioning neuromuscular system. One then evaluates the muscles antagonistic to the motion that increases pain. The primary muscle shortened in lumbar and hip flexion is the psoas. The rectus femoris and abdominal muscles could possibly also be involved, but they would be secondary in consideration. The rectus femoris is only minimally shortened, because in the squatting position the knee flexion lengthens the rectus femoris while the hip flexion shortens it. Likewise, the abdominals are generally ruled out

because there would need to be considerable spinal flexion for the rectus abdominis to be significantly shortened. The direction of the fibers of the oblique abdominal muscles is not in alignment for much shortening in a squatting position.

The muscle considered as the probable secondarily involved muscle to the original strain will have a localized spot tender to digital pressure. In addition to being acutely tender, it will show positive therapy localization. The most common place for the tenderness in the psoas is where the muscle crosses the pubic bone.

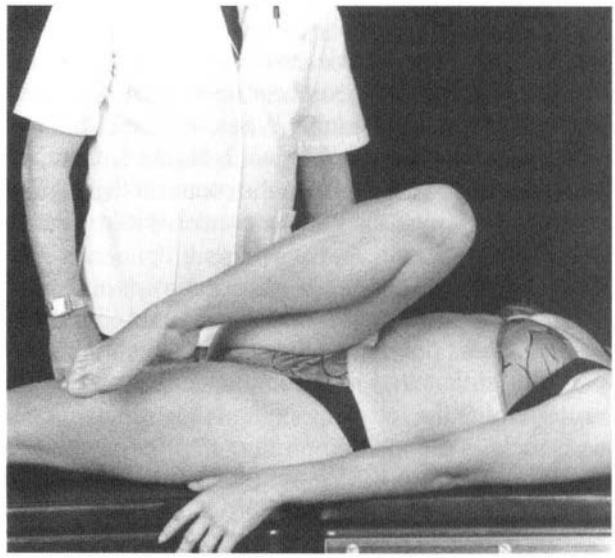
To use Goodheart's muscle test indicating the need for strain/counterstrain technique, one first rules out other factors that might be involved with the psoas. Usually the muscle will test strong in the clear. If it does not, treat with the usual applied kinesiology techniques for a weak muscle. Determine that the muscle does not have a positive muscle stretch reaction or the repeated muscle action weakness of aerobic/anerobic testing. Treat those factors if positive.

With the patient supine, have him maximally contract the psoas by hip flexion, adduction, and internal rotation. This is held for a minimum of three seconds, after which the psoas muscle is re-tested. Weakness indicates the need for strain/counterstrain technique.

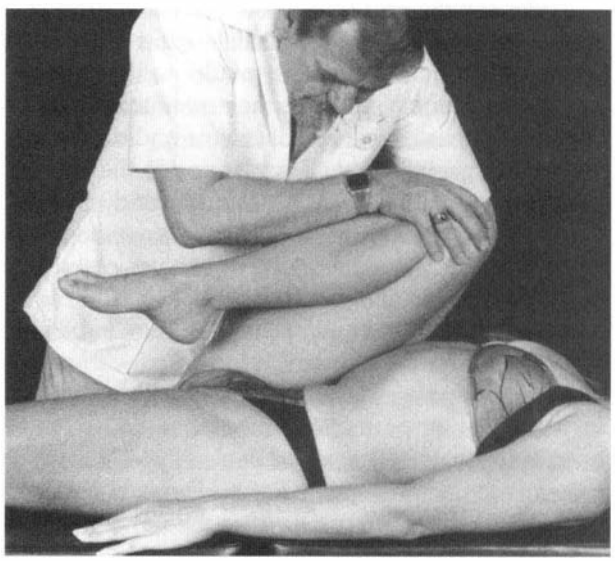
To treat the psoas, locate the tender spot in the belly of the psoas, usually where the muscle crosses the pubis. The patient is then positioned to shorten the psoas while the physician continues to monitor the tender point. When the muscle shortening equals the position in which the original neuromuscular strain developed, the tenderness at the point will be greatly diminished or gone.

Maneuvering for psoas shortening can be done with the patient supine, semi-seated — that is, leaning against a partially upright examination table backrest — or in a seated position. The thigh is brought into flexion by the examiner's passive action. Make certain that the patient does not assist the movement, as there is often an attempt to "help." While passively moving the thigh to shorten the psoas, continue to monitor the tender point. The position to relieve the tender point in nearly all cases is close to maximum shortening of the muscle. As relief of the tender point is obtained, make very small movements to obtain maximum relief. A common mistake is moving past the point of relief. There is a very specific and limited position that obtains maximum relief. Jones calls this last movement to optimal position "fine tuning," where he maintains the position for ninety seconds and then slowly returns the patient to a neutral position. It is extremely important that the return to neutral position be made slowly and passively; rapid return is what caused the condition in the first place. The slow return allows the neuromuscular system to adapt to normal.

Goodheart[34] has added a therapeutic approach



6—43. Have patient maximally contract psoas and test for weakening.



6—44. Flex hip and/or spine to shorten psoas until tenderness in point is improved.

that reduces the time in which the patient must be maintained in the non-painful position. It includes manipulation of the tender point while inspiration or expiration is held. The phase of respiration depends on whether the muscle being treated is a flexor or extensor. Dart[12] states: "All muscles are either flexors or extensors; all torsions of the body as a whole or of its constituent parts whether they be rotation of the spinal column, pronation or supination, inversion or eversion, and adduction, abduction or circumduction of the extremities — are based upon tonically maintained differential pulls between adjacent groups of flexor or extensor segments." In general, the anterior body muscles are flexors and the posterior ones are extensors.

The tender point is assumed to be a neuromuscular spindle cell. When the painless position is located,

the physician spreads his fingers over the previously tender point and has the patient inspire or expire deeply, depending on whether the muscle being treated is a flexor or extensor, respectively. For the psoas muscle, the patient takes a deep breath and holds it while the physician spreads his fingers over the previously tender point. The patient is held in the fine-tuned position with the physician's finger spreading the point and respiration assist for thirty seconds, as opposed to ninety seconds without the assisting factors. Again, on completion, the patient is slowly and passively returned to a neutral position.

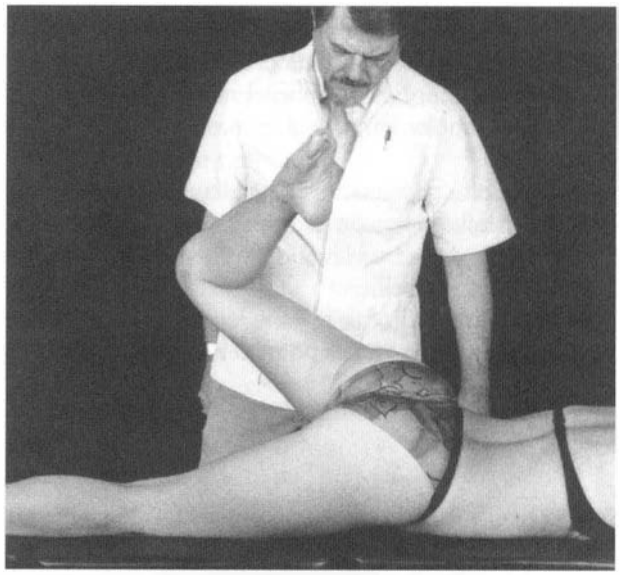
After treatment with the patient in the neutral position, the tender point in the psoas should be gone, and any associated pain — such as low back pain — should be gone or greatly reduced. Re-test the psoas after the patient has maximally contracted it for three seconds. There should now be no weakening if treatment was successful.

Usually only one point needs treatment with this technique. By re-testing the muscle, one may find an additional point or points that need treatment. Repeat the procedure for the additional point(s) as indicated previously until there is no weakening of the muscle after a maximum contraction for three seconds.

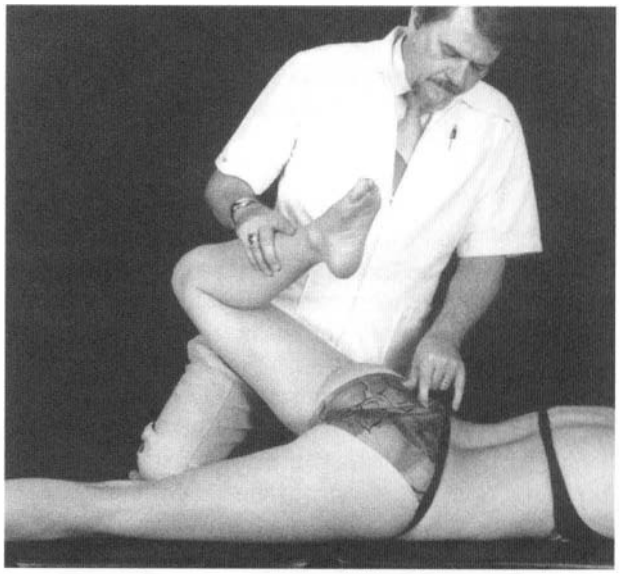
Gluteus Maximus

Another muscle commonly treated with strain/counterstrain is the gluteus maximus. It is often involved when there is body and dural tension from poor pelvic and walking patterns. Evaluating the gluteus maximus for need of strain/counterstrain technique should be routine when the dural tension pattern (described later) is found. There is often no body language of a quick strain related by the patient, and usually no specific type of pain to indicate the need of treatment.

With the muscle strong in the clear, having been evaluated for muscle stretch reaction and weakening on repeated testing, have the prone patient flex the knee and lift it off the table as far as possible to maximally contract the gluteus maximus for three seconds. Re-test the muscle; if it weakens, strain/counterstrain technique is indicated. Palpate to locate the tender point, which is usually in the heavy belly portion of the muscle over the ilium. Extend the thigh while continuing to monitor the tender point for pain reduction. The easiest way to position the patient is for the physician to stand on his leg that is toward the patient's head and flex the other one at the knee and hip, sliding his thigh under the patient's thigh after first lifting it by hand contact on the patient's flexed knee. In this manner the weight of the patient's lower extremity rests on the physician's thigh, which greatly reduces his fatigue during the thirty-second treatment period. When there is optimal relief of the tender point, the patient should take a deep breath and then hold exhalation as the physician spreads his thumb and forefinger over the previously



6—45. Patient maximally contracts gluteus maximus for three seconds. If the muscle then tests weak, treat with strain/counterstrain technique.



6—46. Position the patient's leg to relieve the tender point.

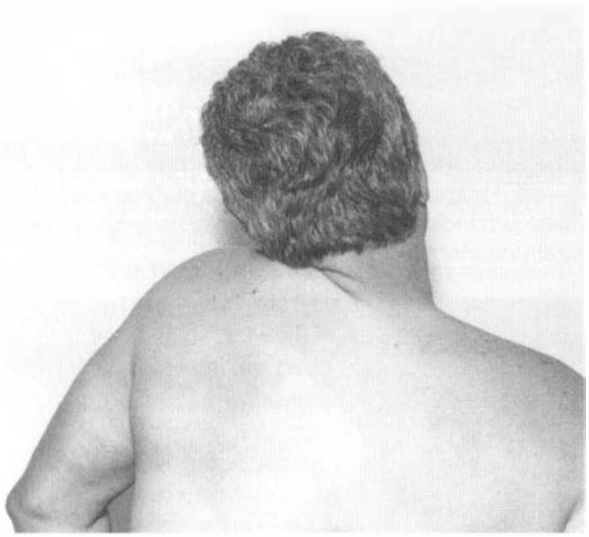
tender point. This is held for thirty seconds; then the patient's lower extremity is slowly returned to neutral. If the patient cannot hold the exhalation for the thirty seconds, continue to hold the pressure for the full length of time while he breaths naturally. Re-test the muscle after a maximum three-second contraction to determine if treatment was successful.

Levator Scapula

A common muscle requiring strain/counterstrain treatment is the levator scapula. It may be the result of hyperextension of the cervical spine, especially when there is slight rotation associated with the strain. This often happens in an auto accident with whiplash dynamics. Prior to evaluating for strain/counterstrain tech-

nique, evaluate the levator scapula for any other fault. In some instances, dysfunction of the levator scapula may be secondary to peripheral nerve entrapment of the dorsal scapular nerve as it passes through the scalenus medius.[85]

With other dysfunction of the levator scapula ruled out, test it after a maximum contraction of three seconds. This is done by elevating the shoulder and laterally flexing the neck, with slight extension and head rotation to the side of test. If the levator scapula weakens, palpate for the tender point. It is usually in the belly



6—47. Maximum lateral flexion and slight extension of levator scapula to test for need of strain/counterstrain treatment.

of the muscle at approximately the level of the 1st rib; however, it could be above or below. While monitoring the tender point, extend the cervical spine with slight lateral flexion and rotation to the side of involvement. Considerable extension of the cervical spine may be necessary to relieve the tender point. It may be uncomfortable to the patient's cervical spine. When the proper position is located, have the patient take a deep breath and exhale. With the patient holding the exhalation, the physician spreads his fingers over the tender point and holds for thirty seconds. Slowly and passively, the patient is moved back to neutral. There is usually excellent relief of pain and increased range of motion following treatment. Re-test the muscle after maximum three-second contraction to determine that the treatment was effective, and that there are no more areas requiring treatment.

If the trauma is recent and there has been ligament injury, treatment by strain/counterstrain may be contraindicated. The range of motion necessary to relieve the tender point may cause additional soft tissue injury, aggravating the condition. Wait until the soft tissue has adequately healed to apply strain/counterstrain technique.

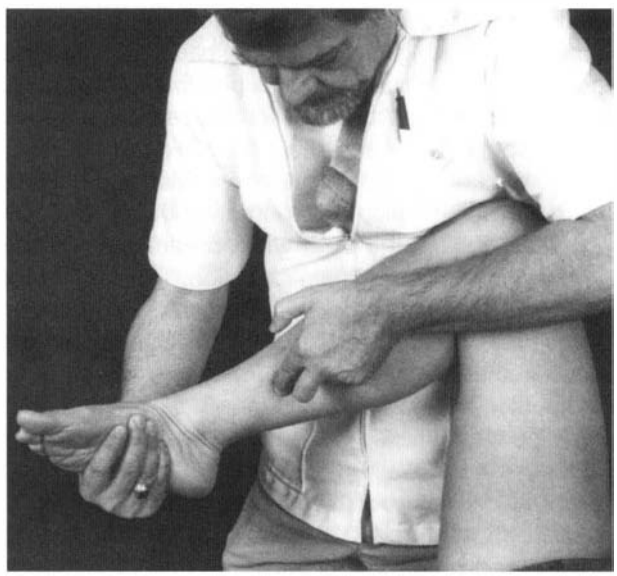
Ankle and Foot

There is often a requirement for strain/counterstrain treatment relating to the ankle and foot. This may follow a strain or sprain to the area. Body language consists of an inordinate length of time for healing and relief of pain. Under normal circumstances one should become asymptomatic when the tissues heal from a strain or sprain. The neuromuscular dysfunction relating with strain/counterstrain causes the pain to remain past the tissue healing.

The most common type of ankle strain is inversion.[24] A muscle quite frequently needing strain/counterstrain treatment is the medial head of the gastrocnemius. With inversion sprain, the medial head of the gastrocnemius shortens. In athletic injuries the inversion sprain is often associated with plantar flexion, which shortens the medial head of the gastrocnemius even more.

In the presence of strong medial and lateral heads of the gastrocnemius, have the patient maximally contract the muscle for three seconds by increasing the test position of knee and plantar flexion. Weakening after the contraction indicates need for strain/counterstrain technique.

The tender point is frequently located near the musculotendinous junction. The physician's passive maneuvering of the limb includes both the ankle and the knee. Position the foot in inversion and plantar flexion, and flex the knee to shorten the gastrocnemius. When the position is obtained that maximally relieves the tender point, have the patient take a deep breath and exhale. The physician's fingertips are spread over the tender point and held for thirty seconds, while the patient maintains the exhalation. Return slowly to the neutral position. Following treatment there should be no weakening after maximum contraction, and the pain should be greatly reduced or removed.



6—48. Position that often relieves the tender point in the gastrocnemius following an ankle strain.

General Considerations

Occasionally after strain/counterstrain technique a patient will complain of pain in the general area the following day. This appears to be related to the release of excessive amounts of lactic acid accumulated from prolonged muscle dysfunction. A cold pack applied to the area will usually provide relief. An ice pack is not recommended; rather, use cold tap water in a plastic bag laid over the area and allow it to come to room temperature.

Nutritional support for strain/counterstrain is that which usually applies to the muscle involved. For example, the gastrocnemius is related in applied kinesiology to the adrenal gland. Adrenal concentrate is usually the choice nutrition.

In small muscles, such as those in the hand, it may not be possible to spread the tender points with two fingers. In this case use one finger to stretch the tissue over the area. Similar shortened treatment time will result as if two fingers had been used.

Gait Testing

Walking and running are complex neurologic activities that have received much attention in applied kinesiology. It is important to test a patient in the way in which he lives. Often examination, including manual muscle testing, is done with the patient prone or supine; this fails to consider the aspects of weight bearing, walking, running, and other movements. There are many techniques that enable the physician to evaluate the effect of these daily activities on a patient's health. For example, an individual may have a negative category I or II therapy localization and challenge to the pelvis; however, when he therapy localizes to the sacroiliac while walking, a positive test is seen.

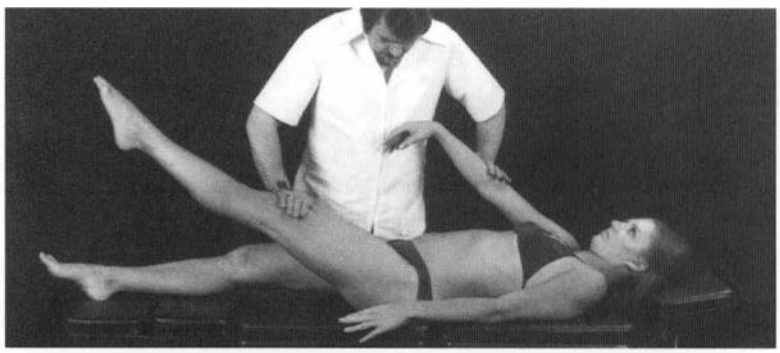
As a leg advances in gait activity, the contralateral arm swings forward. The trailing leg is organized with the contralateral arm as both the hip and shoulder move in extension.

Further organization in gait is present in simultaneous activity of the leg adductors with the contralateral arm adductors, and the leg abductors with the contralateral arm abductors. Additional organization is present in the gluteus medius with the contralateral abdominal muscles. The gluteus medius must contract to support the pelvis during stance at the same time the contralateral abdominals lift the pelvis on the opposite side.[17]

Organization of muscle groups in gait can be evaluated by applied kinesiology gait testing.[3,26] Although all types of gait problems are not identified by this testing procedure, it is an effective clinical tool for finding the cause of many gait disturbances. The system is to simultaneously test groups of muscles that are organized in gait. A positive test is when one or both groups test weak when tested simultaneously, but are strong when tested individually. The most efficient method of gait testing is to simultaneously test both muscle groups first. If they are strong, proceed to the next pair. If one or both muscle groups test weak, then test each group individually. Again, for a positive gait test, both groups must test strong when tested individually, but one or both groups test weak when tested simultaneously. If one or both groups test weak individually, the muscles must be strengthened by treatment with the five factors of the IVF before a satisfactory gait test can be performed.

Contralateral Shoulder and Hip Flexors

The supine patient flexes his hip and shoulder, keeping the knee and elbow extended. The arm and leg are raised approximately the length of a normal step. The examiner directs force against the arm and leg in the direction of extension, testing the general muscles of flexion.

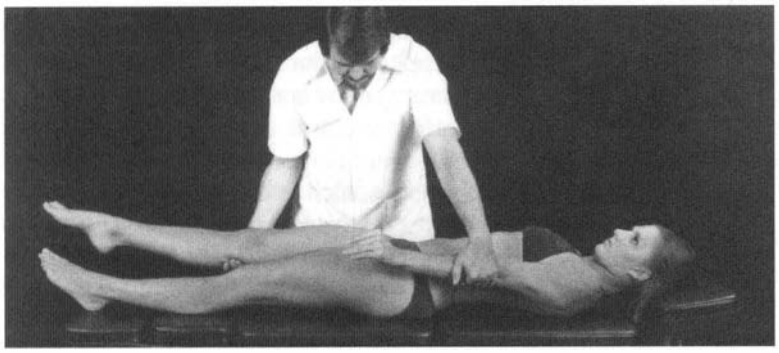


6—49. Contralateral shoulder and hip flexors.

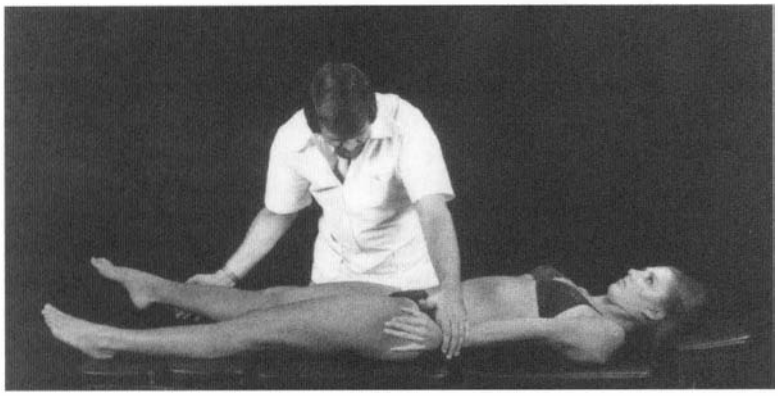
Contralateral Shoulder and Hip Extensors

This test can be done with the patient supine or prone. If done prone, the patient extends his hip and contralateral shoulder, keeping the knee and elbow in extension. The examiner directs force on the distal thigh and humerus in a direction of flexion.

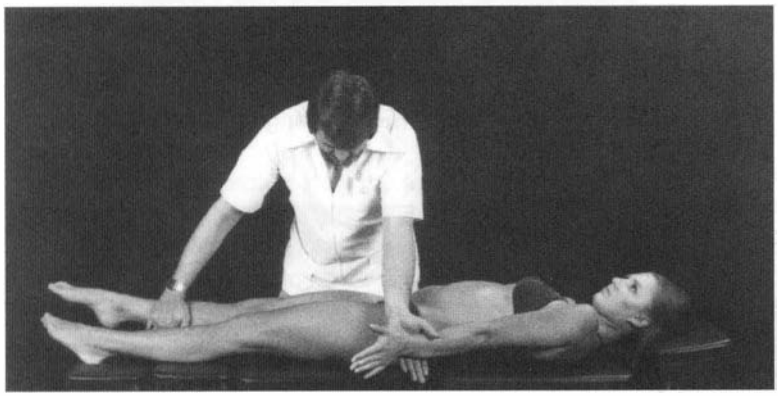
When the test is done supine, the patient attempts to hold the contralateral extremities against the table while the examiner lifts on the arm and leg in a direction of shoulder and hip flexion. The supine position is convenient because all the other tests are done supine, and the testing can be done with minimal patient movement.



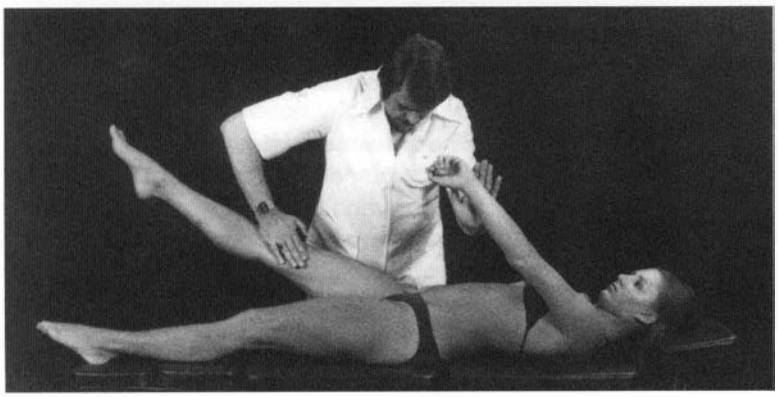
6—50. Contralateral shoulder and hip extensors.



6—51. Contralateral shoulder and hip abductors.



6—52. Contralateral shoulder and hip adductors.



6—53. Contralateral psoas major and pectoralis major.

Contralateral Shoulder and Hip Abductors

The supine patient abducts his contralateral arm and leg, keeping the elbow and knee in extension. The extremities are abducted approximately 30°. The examiner applies pressure to the distal arm and leg in a direction of adduction.

Contralateral Shoulder and Hip Adductors

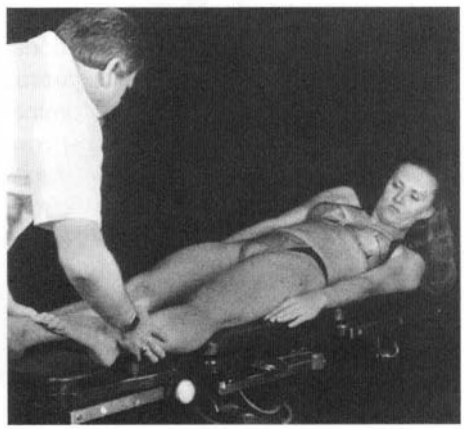
The supine patient holds his arm and contralateral leg in adduction, with the elbow and knee kept in extension. The examiner applies force to the distal arm and leg to abduct them.

Contralateral Psoas Major and Pectoralis Major

The psoas and pectoralis test is similar to the contralateral shoulder and hip flexor test, but it localizes testing more to the individual muscles of flexion. The supine patient raises his leg and contralateral arm into the positions for testing the psoas major and the general pectoralis major. Testing pressure is applied in the same manner as individual psoas and pectoralis major muscle testing.

Contralateral Gluteus Medius and Abdominals

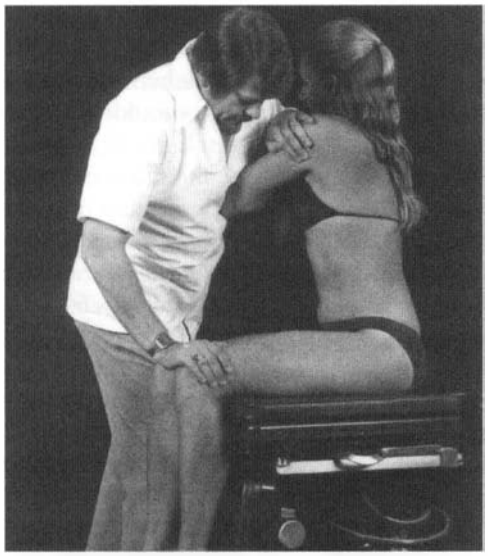
There are two tests for the contralateral gluteus medius and abdominals. One, with the patient supine, tests the gluteus medius with abdominal contraction. The supine patient begins a curling sit-up in which the shoulders are raised from the table. The trunk



6—54. Gluteus medius with abdominal contraction.

is turned to lift one shoulder higher from the table. This contracts the abdominals more on the side of higher shoulder elevation. A positive test is weakening of the contralateral gluteus medius.

The abdominals are best tested with the patient seated. The physician stands beside the patient, whose trunk is rotated toward the physician. The patient presses his knee against the examiner's leg. Testing pressure is applied to the shoulder to test the contralateral abdominals, while the knee on that side is stabilized. This more effectively tests the abdominals. Some failure of gluteus medius activity can be observed by the experienced examiner.



6—55. Contralateral abdominals and gluteus medius.

Treatment

When a gait test is positive, there will be an active acupuncture point associated with it. After the acupuncture point is stimulated, the gait test will be negative. Stimulation can be done by any method, e.g., digital pressure, acupuncture needles, electrical stimulation, and acu-aids.

Three gait complexes have meridian points located across the dorsum of the foot. The hip and shoulder adductors have points located lateral to the 5th metatarsal, while the shoulder and hip extensor complex has its point located close to the metatarsophalangeal articulation of the great toe on the medial aspect. The point for the psoas major and pectoralis major muscles is on the plantar surface of the foot (figure 6—56).

Four of the complexes have primary and secondary acupuncture points, which are the three meridians on the dorsum of the foot and the bladder meridian on the lateral aspect of the foot. On the illustration, the primary point is the larger dot where the involvement is usually found; if not, the secondary point should be evaluated. The active acupuncture point will have positive therapy localization. It can usually be located by palpation and is exquisitely tender. Treatment is generally applied by digital pressure, which is a rather hard digital manipulation of the point for approximately fifteen seconds. After treatment, therapy localization to the point should be negative.

Often there is a foot subluxation associated with the active acupuncture point. If present, it should be corrected. If foot dysfunction is not corrected, the active acupuncture point and positive gait test will probably return.

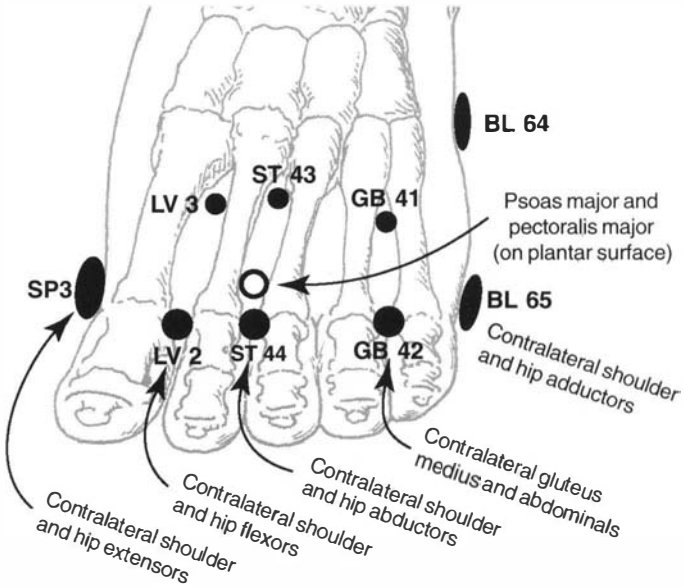
Memory Key

A memory key to help remember the location of the meridian points correlates with the first five letters of the city, Palo Alto, beginning on the medial side of the great toe:

Posterior = Shoulder and hip extensors
Anterior = Shoulder and hip flexors
Lateral = Shoulder and hip abductors
Oblique = Gluteus medius and abdominals
Adductors = Shoulder and hip adductors

The remaining point for the psoas major and pectoralis major — KI 1 — is the only meridian point on the plantar surface of the foot.

Gait testing and correction of dysfunction are indicated whenever a patient has a recurring subluxation or other dysfunction after walking, feels worse after standing or walking, or has apparent foot dysfunction. (See Chapter 11 for foot discussion.)



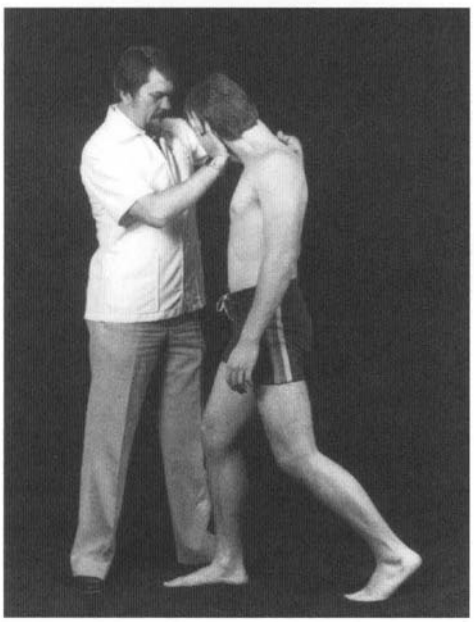
6—56. Meridian acupuncture points for treatment of gait mechanism as observed by manual muscle testing. The large dots are the primary points where involvement is usually found; the smaller dots are the secondary areas to evaluate for treatment. Treatment is hard digital stimulation over the meridian point. Negative therapy localization indicates adequate and effective treatment. There usually are foot subluxations or other foot dysfunction present which should be evaluated and treated for lasting correction.

Walking Gait Temporal Pattern

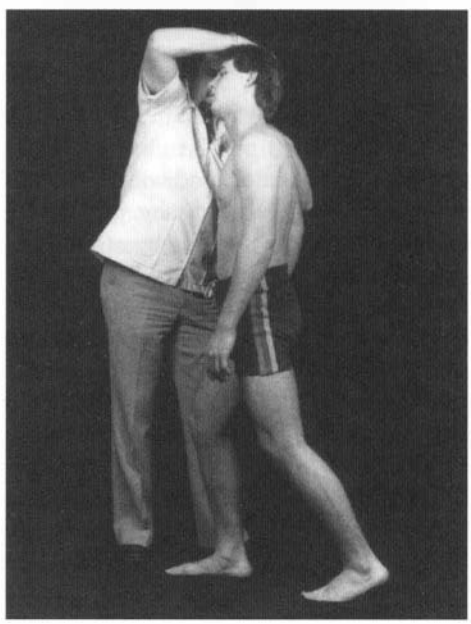
During walking and running, muscles from the shoulder girdle to the head have organized facilitation and inhibition in a manner similar to that described for the lower portion of the body. As the right leg and pelvis move forward, the left shoulder girdle moves forward as the shoulder flexes. Simultaneously, the head turns left in relation to the shoulder girdle. To accomplish this, the left sternocleidomastoid muscle is inhibited and the right upper trapezius and deep extensor muscles are inhibited, which keeps the head pointing forward rather than rotating right and left with the shoulder girdle. This organization has been described by Goodheart as the "walking gait."[32] He has developed a system of testing for normal facilitation and inhibition of the sternocleidomastoid, upper trapezius, and deep extensor muscles.

To test for normal facilitation and inhibition of the sternocleidomastoid, upper trapezius, and deep extensor muscles, the patient is placed in a simulated gait position similar to that of the demonstration of shoulder flexor and extensor facilitation and inhibition described on page 170. The patient is first tested with one leg forward, then with the other forward in gait position. The forward leg is the stance leg of gait and should carry most of the patient's weight. The trailing leg carries the remainder of the weight on the ball of the foot. The knees are bent in a simulated gait position. When the right leg is the stance leg, the left sternocleidomastoid muscle should test weak. The examiner should take care to stabilize the shoulder girdle when testing the sternocleidomastoid so that it operates from a stable base. With the right leg forward, the right upper trapezius and deep extensor muscles should test weak. Although this test is often referred to as an upper trapezius test, it is more of a general test of the neck extensors. The head is not rotated away from the side of testing, as in the standard upper trapezius test. It is most easily tested by stabilizing the patient's shoulder on the side of the test, with the examiner placing his arm over the vertex of the head, and grasping the posterolateral quadrant of the skull with his hand. The examiner applies force to bring the patient's head into lateral and anterior flexion, separating the shoulder and head on the side being tested.

The neck flexors and extensors are tested for normal inhibition with the appropriate phase of gait. First, test to determine that the muscles are strong in a weight-bearing position. If they are not, evaluate for the cause with the five factors of the IVF and correct before testing for gait inhibition. A normal test is for the sternocleidomastoid to test weak opposite the forward leg side, while the upper trapezius and deep extensors test weak on the side of the forward leg. A positive test is failure of this inhibition with the simulated gait posi-



6—57. To test the sternocleidomastoid in the gait position, the examiner must effectively stabilize the posterior thorax. The sternocleidomastoid should test weak on the trailing side.

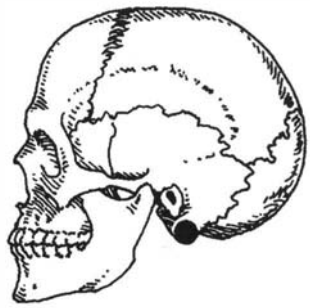


6—58. Testing the upper trapezius and deep cervical extensors in gait activity is accomplished by the examiner contacting the head to separate it from the shoulder in lateral and anterior flexion. The patient's head is not rotated away from the side of testing as in the upper trapezius test. The complex of extensor muscles should test weak on the forward leg side.

tion. When there is a fault, it appears to be limited to failure of inhibition. There has been no observation of the muscle that should be facilitated weakening with gait position, i.e., the sternocleidomastoid weakening on the forward leg side or the upper trapezius and deep extensors weakening on the trailing leg side.

When the muscles fail to weaken at the appropriate time in gait position, there is usually disturbance in the gait mechanism as described previously. There will probably also be an active stress receptor relating with the muscle. The applicable stress receptors are on the anterolateral portion of the mastoid process for the sternocleidomastoid, and on the frontal bone for the upper trapezius. The stress receptors are challenged with the patient in the neutral standing position to determine the direction of skin manipulation that causes the associated muscle to weaken. The patient is asked to take a deep inspiration, and the muscle is tested to determine if it strengthens. If it does not, the test is repeated with expiration. Usually the challenge will be abolished by inspiration. The stress receptor is treated with heavy manipulation of the tissue in the direction causing muscle weakness on the phase of respiration that abolished the challenge. This is usually repeated four or five times. The muscle should weaken during the appropriate phase of gait when the patient is re-tested to determine the effectiveness of treatment.

Gait muscle inhibition of the pelvis and lower extremities is more difficult to evaluate because of the testing problems. The hip flexors, primarily the psoas and rectus femoris, should be inhibited contralateral to the forward leg. This can be tested by placing the patient's heel on the side of a hi-lo adjustment table foot platform so that about half his body leans backward against the table. The leg on which he is standing is the stance (forward) leg, and the trailing leg is off the table and placed slightly in extension. The patient is stabilized against the table and further hip extension strength is tested, which should be weak. Failure of inhibition is corrected by stress receptor treatment.



6—59. Sternocleidomastoid stress receptor.

6—60. Upper trapezius stress receptor.

Further evaluation for proper facilitation and inhibition of the gait muscles can be done by evaluating the deep tendon reflexes. Foot subluxations are often the cause of improper gait muscle temporal pattern. All patients should at least initially be screened for foot problems by the foot shock absorber test, and for generalized weakening on weight bearing.

Ligament Interlink

Ligament interlink technique in applied kinesiology is based on the interaction of joints during gait. In quadruped walking, there is toe extension of the contralateral forelegs and hindlegs during the stance phase, and flexion during the swing phase. This correlation of motion is present throughout the extremity joints, including the elbows and knees, shoulders and hips. The same correlative motion is present in the biped, but it is less easily recognized in the distal articulations of the hand. When walking quickly in a relaxed manner, it can be observed that the wrist flexes and extends in unison with the contralateral ankle. The fingers also tend to flex and extend in unison with the toes of the contralateral extremity. The correlation between shoulder-hip and elbow-knee is obvious.

Goodheart[30] first observed the possibility of additional factors in the correlation of contralateral upper and lower limbs in a patient with severe inflammatory swelling of the knees, caused by rheumatoid arthritis. The patient could obtain relief in his knee by flexing his elbow. Upon extending or stiffening his elbow, the knee pain became worse.

Sometimes an enigmatic joint pain can be explained by the ligament interlink technique. In this situation, there is joint pain but no positive therapy localization, challenge, or other evidence of disturbed joint function or pathology. A positive ligament interlink test is when there is no positive therapy localization of the joint; however, when the contralateral correlating gait joint is simultaneously therapy localized, it becomes positive. An example is pain in an elbow joint that tests negative during all testing procedures, but becomes positive when there is simultaneous therapy localization to the elbow and the contralateral knee. In a positive ligament interlink case, both the elbow and knee will have negative therapy localization when done separately. If therapy localization is positive to either joint, determine the cause and correct it before further evaluating for ligament interlink.

In an effort to find some factor that would interrupt the positive two-handed therapy localization, Goodheart applied manipulation and other therapeutic efforts to the articulation(s). In general, these efforts were ineffective in eliminating the pain at the articulation. It was found that moving the hyoid interrupted the positive two-handed therapy localization. Goodheart hypothesized that moving the hyoid stimulates the proprioceptors of the hyoid muscles in some

way to influence the neurologic crossover mechanism connecting the two articulations.[30] A system was developed to treat the painful condition, using hyoid movement in conjunction with manual stimulation of one of the articulations.

Procedure

First the painful articulation is evaluated with therapy localization. If positive, the usual AK procedures of manipulation, muscle balancing, and others are performed to eliminate the positive finding. When therapy localization is negative, the joint is therapy localized simultaneously with the appropriate contralateral articulation. Weakening of a previously strong indicator muscle is positive indication of ligament interlink involvement.

The positive two-handed therapy localization is generally specific to associated ligaments. For example, elbow flexion correlates with contralateral knee flexion. Evaluate the action of the ligaments at the painful elbow, and therapy localize comparable ligaments in the knee. Considering the arm held in the anatomical position, the lateral aspect of the elbow correlates with the medial side of the knee because in this position the forearm moves anteriorly with elbow flexion, and the leg moves posteriorly with knee flexion. The correlation of the wrist-ankle, hand-foot is that the thumb equates with the great toe and the little finger with the 5th toe.

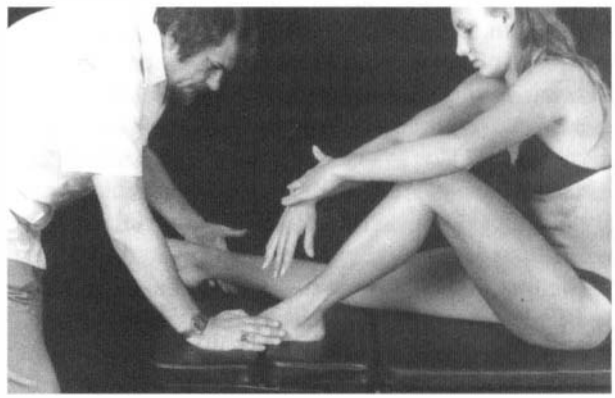
Therapy for positive ligament interlink is digital stimulation of one of the points of positive therapy localization while the hyoid is held toward the side being stimulated. The joint to stimulate is the one that is less tender. It is generally the joint contralateral to the joint with the patient's pain. In some cases the contralateral joint may be more painful. In any case, choose the less painful one.

Digital manipulation is applied with eight to ten pounds of pressure intermittently for thirty to forty seconds. Throughout the digital manipulation of the ligament, the patient holds the hyoid toward the side of manipulation. While administering the manipulation, the physician monitors the contralateral articulation for pain reduction.

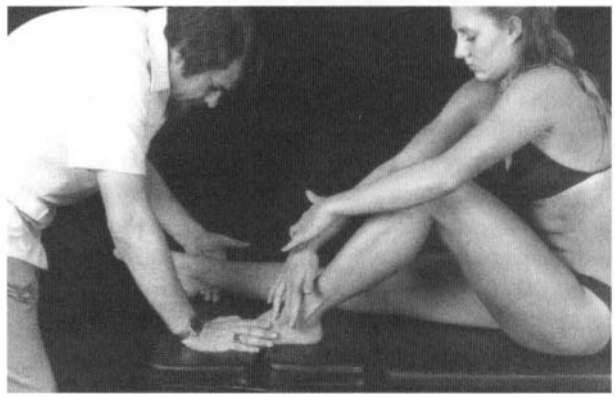
The contralateral joint correlation described is nearly always the pattern observed. Occasionally the pattern can be ipsilateral. The same two-handed therapy localization and treatment procedures are applicable. The reason for the unusual ipsilateral pattern is unknown. It does not appear to correlate with neurologic disorganization (switching). It may be failure of all the nerve fibers to decussate.

Occasionally one finds positive therapy localization to an articulation that cannot be effectively treated with the usual applied kinesiology techniques. In this case, evaluate the contralateral associated gait joint. If it also therapy localizes, use two-handed therapy localization. If the joints are associated, the two-handed therapy localization will be negative. In other words, both joints have positive therapy localization individually, but negative therapy localization together. The therapeutic effort is applied in exactly the same way as the usual correction for ligament interlink, described above.

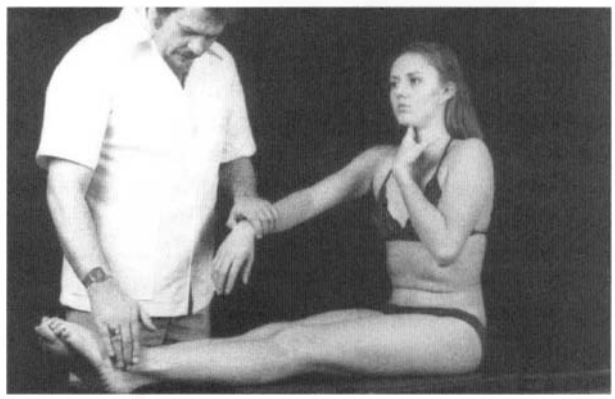
There is additional association of joints that is usually not regarded as being gait-associated. The sac-



6—61. Therapy localization to wrist while in position to move only slightly for second phase of test.



6—62. Second phase, two-handed therapy localization to wrist and ankle. Subject's arms are not touching the knee, which could add another variable.



6—63. Digital massage of ankle ligaments for wrist pain, while patient holds hyoid toward side of therapy on the ankle. Wrist contact by doctor is only for monitoring pain reduction.

roiliac articulations equate with the sternocostal articulations. Treatment to this association is sometimes effective in treating Tietze's syndrome. The xiphoid process and coccyx relate together and may be associated with general diaphragmatic involvement or specific problems, such as hiccups.

Sometimes there is pain at a vertebral level, but no evidence of a subluxation or fixation. The spinal column-ligament interlink relationship follows the Lovett reactor association of vertebrae (page 70). First the painful vertebra is therapy localized. If the therapy localization is negative, maintain it and therapy localize the Lovett reactor on the opposite side to determine if positive therapy localization develops. Therapy is applied to the less tender vertebra while holding the hyoid as usual toward the side being manipulated with digital pressure.

The temporomandibular joint has been clinically associated with any joint of the body. Its association is evaluated the same as any other two joints on a ligament interlink basis.

The ligament interlink approach is not applicable to all joint conditions. It does not substitute for proper therapy, which may include the correction of a subluxation, reduction of strain in the joint by correcting imbalance of the supporting musculature, and other procedures described throughout applied kinesiology.

When bilaterally therapy localizing the articulations, as many variables as possible must be ruled out. Place the patient in position to therapy localize both articulations with minimal movement. When the patient is therapy localizing the wrist and has to move a great distance to therapy localize the ankle, other variables — such as spinal movement — could influence the indicator muscle test.

PRYT Technique

The body is composed of modules. Major modules can be considered as the head, neck, shoulder girdle and thorax, pelvis, and arms and legs. Throughout the body are proprioceptors responsible for integration of the body modules. In-depth study of some of the proprioceptors indicates an extensive network and integration with the body.

The PRYT technique of applied kinesiology helps find subluxations and muscle dysfunction that cause inappropriate communication between the modules. The proprioceptors evaluated are in the neck and pelvic regions. The head-on-neck receptors are located in the upper cervical ligaments[53] and muscles. The small muscles of the neck may have up to 500 neuromuscular spindle cells per gram.[1] Movement of the eyes with head-on-neck movement is a natural integration and part of the overall organization of body modules.[42,76] Jirout[45] describes the intricate movement of the upper cervical vertebrae on head motion. Movement between the occiput, atlas, and axis vertebrae is sometimes very slight and intricately organized. The high concentration of neuromuscular spindle cells in the muscles of this area indicates the importance of proper movement.

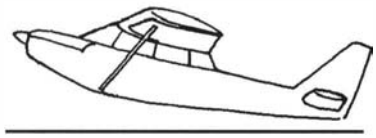
Centering reflexes in the pelvis have been described by Watkins.[87,88] The equilibrium synchronization technique evaluates these reflexes and their relationship to proper function of the cranial-sacral primary respiratory mechanism. The PRYT technique evaluates the integration of the pelvis and hips with motions different from the equilibrium synchronization technique.

The acronym "PRYT" comes from pitch, roll, yaw, and tilt. Pitch, roll, and yaw are nautical and aeronautical terms describing the position of a ship or an airplane in its relation to forward movement. The applied kinesiology PRYT examination evaluates the three-dimensional integration of body modules. The patient is put into a position to stimulate two sets of equilibrium proprioceptors. The basic premise is that a previously strong indicator muscle(s) should not weaken under these conditions. Clinical evidence supports the PRYT examination for finding faults, because a subluxation or muscular dysfunction is consistently found at one of the areas stimulated by the positive PRYT position; when corrected, the indicator muscle(s) no longer weakens.

Throughout PRYT testing, two areas that integrate body modules are put into a position and a previously strong indicator muscle is tested. For example, the head and neck may be flexed while the hips are simultaneously flexed. A positive test is weakening of the indicator muscle in the PRYT position; there is no weakening when only one of the areas is in the test position. In other words, it is not a positive PRYT test if hip flexion without head and neck flexion causes an indicator muscle to weaken. One should evaluate structures affected by the hip flexion and make corrections before PRYT testing is continued. Generally, the efficient method of PRYT testing is to first test the PRYT position. If positive, then test the individual movements making up the PRYT position. If these cause an indicator muscle to weaken, evaluate further and make corrections; then re-test for a positive PRYT test, which may or may not be present after the individual correction(s).

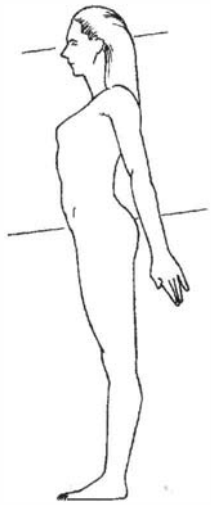
Pitch

Pitch is the position of an airplane in reference to its lateral axis, a line that parallels the wings. An airplane is positioned for climbing when pitch is increased; when it is decreased, an airplane is in a diving posi-



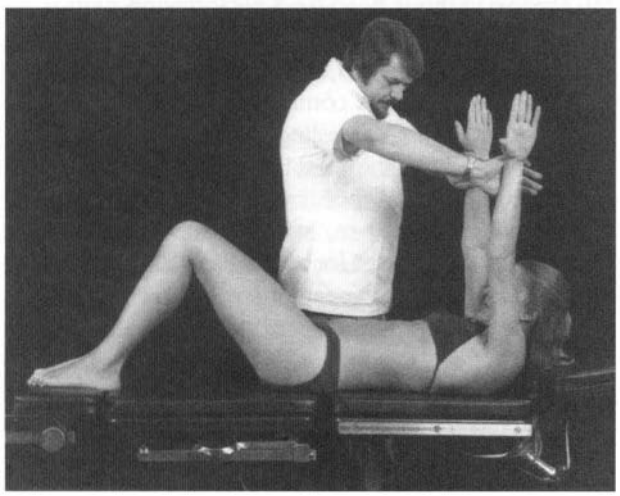
6—64. Pitch position for climb.

tion. In the body this relates to anterior or posterior positioning of the pelvis, head, or some other body module. Evaluation of pitch relates to the AP curves of the body. There will often be a limitation of hip abduction or flexion. The body modules are tested for integration of flexion and extension of the head and neck with the hips.



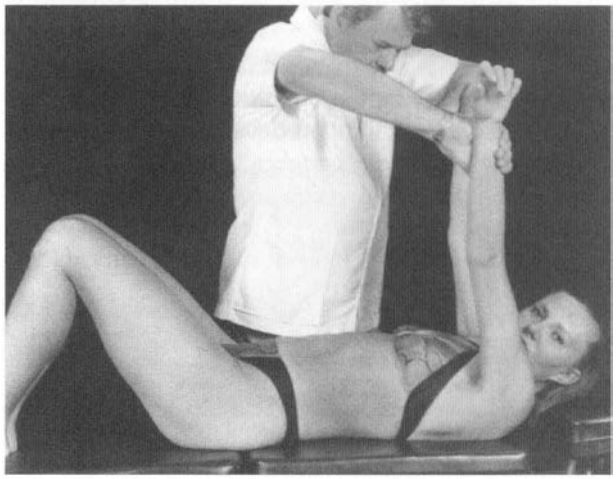
6—65. Body disorganization in pitch analysis.

Flexion. Pitch flexion is examined with the patient supine. The patient actively flexes his head and neck on his thorax, and the hips are flexed with the feet resting on the table. The bilateral pectoralis major (clavicular division) muscles are usually the strong indicator muscles tested for weakening. Most often the fault is found on flexion; in some cases extension can be positive.



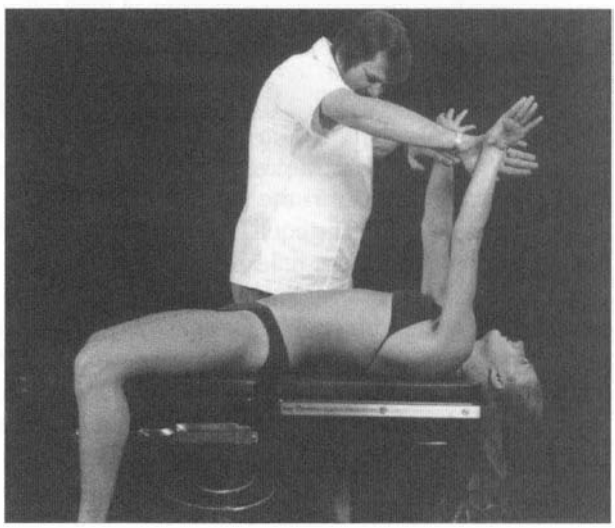
6—66. Position for testing pitch flexion.

Flexion and Cervical Rotation. Cervical rotation can be combined with the pitch flexion test to further evaluate joint movement in the cervical region. If a patient tests normal in the pitch flexion position, have him maintain that position and add cervical rotation first to one side and then the other; test each side individually. Weakening of the indicator muscle is a positive pitch flexion with rotation test.



6—67. Position for testing pitch flexion and cervical rotation.

Extension. Pitch extension is evaluated when the patient extends his head and neck as the hips are simultaneously extended. This is usually done by having the patient hang his head over the end of the table and drop both legs over the side. If the legs cannot be dropped over the side because of the table's width, a Dutch roll pillow can be placed under the pelvis to achieve hip extension. Again, previously strong bilateral pectoralis major (clavicular division) muscles are tested for weakening, indicating a positive test.



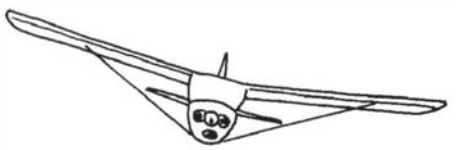
6—68. Position for testing pitch extension.

Treatment. Treatment is directed to the head-on-neck complex. The physician holds the patient's head with a broad contact while the patient makes a maximum effort to move the head into the position that tested positive, similar to the occiput-on-atlas flexion and extension correction described on page 96. It differs only in that there is no weakening of the head-on-neck position without the hip flexion or extension. Flexion with cervical rotation is treated in a manner similar to flexion or extension treatment. For rotation, the patient attempts to rotate and flex his head in the direction that tested positive while the physician resists his effort.

After the corrective effort there should no longer be positive findings in the position that tested positive. There is often a considerable increase in the patient's range of motion at the hip. If there is no increased hip abduction or flexion, evaluate for hypertonic and shortened muscles. Fascial release, intermittent cold with stretch, or other techniques may be necessary to return them to normal.

Roll

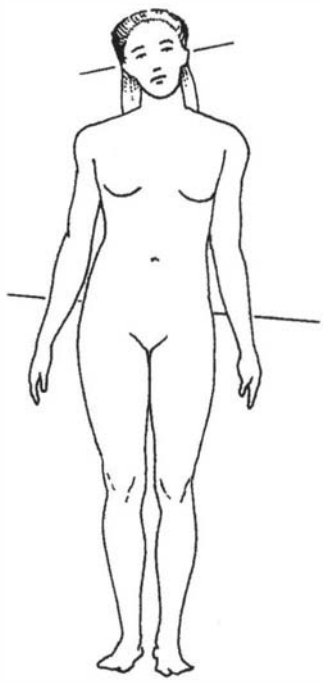
Roll refers to an airplane's position along its longitudinal axis. Looking at an airplane from the front, roll is seen as the wings not being level. Roll is observed in the body when modules are not level with the transverse plane.



6—69. Position of airplane in roll.

Roll was originally referred to by Goodheart as "oculobasic" technique.[30] "Oculo" represents the visual righting reflexes, and "basic" is the basic technique for sacral and spinal treatment. The characteristic posture of an individual with a positive roll pattern is failure of the body modules to align horizontally when viewed A to P, or P to A. A patient will typically have considerable postural strain and many areas of localized pain relating with the modular disorganization. The patient's gait will often have a characteristic lateral sway and an asymmetrical pelvic elevation.

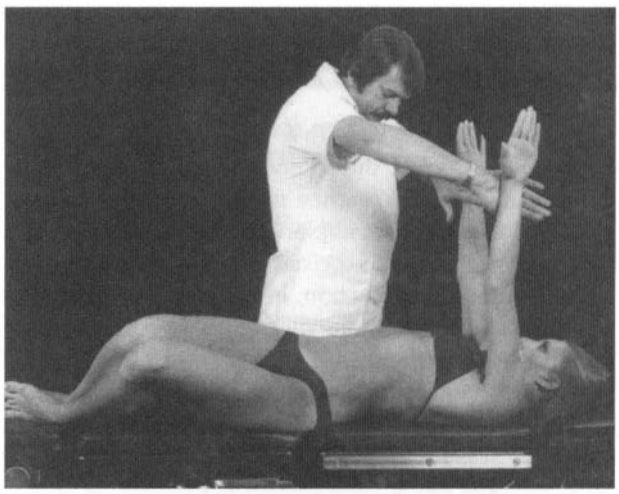
Examination. The examination tests the integration of pelvic proprioceptors with eye movement. Many of the testing procedures in applied kinesiology evaluate integration of the nervous system, and many of them overlap. Since the test for roll requires eye movement for final evaluation, it is best to rule out ocular lock and other evidence of neurologic disorganization before evaluating a patient in the roll position. In fact, correction of neurologic disorganization often eliminates a positive roll test.



6—70. Poor modular alignment typical of roll pattern.

A patient is evaluated supine, with his hips and knees flexed and the feet resting on the table. The bilateral pectoralis major (clavicular division) muscles should be strong in this position. During the roll test the patient rotates his knees to one side, placing torsion into the hips, pelvis, and lumbar spine. Weakening of indicator muscles is a positive test. Numerous muscles and ligaments are stretched in this position. Of particular note is the piriformis on the side where the knee crosses the midline. This position puts a stretch on the piriformis, a major sacral stabilizing muscle toward which corrective effort will be directed.

The second step of examination supports the



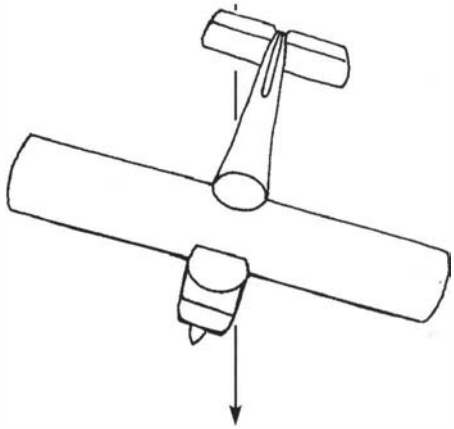
6—71. Position to test for roll. Lateral movement of the eyes in one direction or the other cancels weakening of the indicator muscle(s).

hypothesis that dysfunction is integrated with the equilibrium and body-organizing receptors. While the patient maintains the leg position, he turns his eyes to one side and then the other. If positive, one direction will cause the indicator muscles to regain strength even though the legs, hips, and pelvis are maintained in the rotated position. Eye involvement appears to indicate that the visual righting reflexes are involved in the complex.

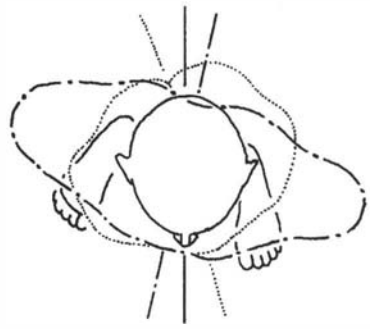
Correction. Treatment is directed toward the lateral apex of the sacrum, which is challenged with cephalad pressure by the examiner's thumb. While this static challenge is held, a previously strong indicator muscle (usually the hamstrings) is tested. If it remains strong, the patient is asked to turn his eyes in the direction that caused the indicator muscle to regain strength in the supine test. If the indicator muscle now weakens, this is the contact that will correct the roll condition. Both sides of the sacrum are challenged; usually only one will be positive. If both sides are positive, select the one causing maximum weakening. When the positive side has been determined, the challenge is repeated with the patient maintaining the lateral eye position. The indicator muscle is tested while the patient holds a deep inspiration and then an expiration. One phase of respiration will abolish the weakness that was present with the static challenge and lateral eye position. The phase of respiration that abolishes the challenge indicates the direction in which the sacrum is to be moved. If the weak muscle is eliminated on inspiration, correction is applied to the point of positive challenge in a cephalad, slightly anterior direction while the patient takes a slow, deep inspiration. This is repeated for approximately two minutes, during which the patient often feels relaxation in the trunk and neck muscles. If the weak muscle resulting from challenge and eye movement is neutralized by expiration, the apex of the sacrum is contacted at the point of positive challenge and pressed cephalad and slightly posteriorly during a slow expiration. After correction, re-evaluate the patient supine; there should be no weakening on hip, pelvic, and lumbar rotation.

Yaw

Yaw describes the rotation of an airplane along its vertical axis in reference to the direction of flight. The vertical axis of an airplane is from the nose to the tail. Yaw indicates that the airplane is not aligned with its horizontal flight direction. In the presence of a positive yaw condition, there will probably be head rotation in relation to the shoulder girdle, and the pelvis may be rotated over the foot position. Frequently associated with the yaw condition is a decreased flexion of the trunk, as in Adam's position. This is probably due to an inability of the lumbar vertebrae to rotate as flexion takes place, as described by Illi.[43] There are three yaw patterns, one examined with the patient supine and the other two with him prone.

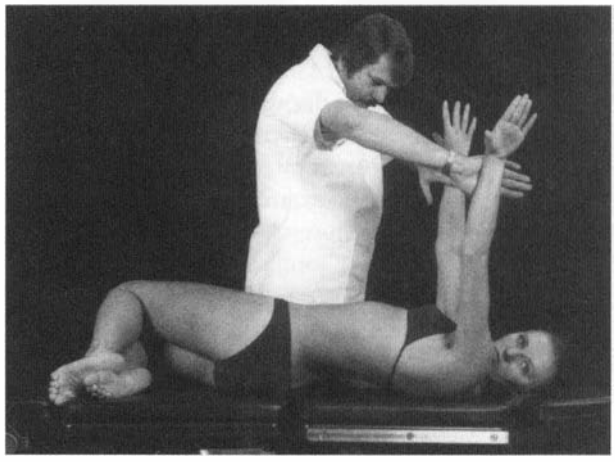


6—72. The arrow indicates the direction of flight. The airplane is yawed to its right.



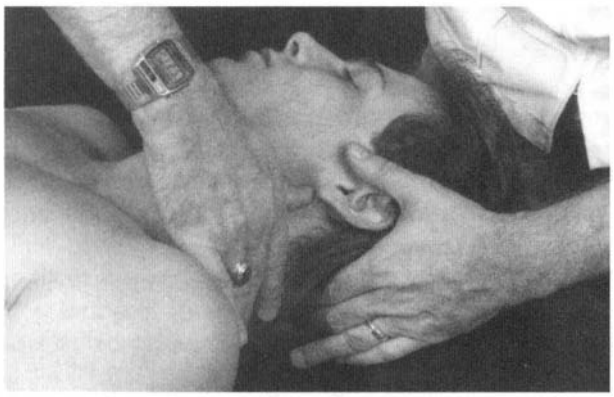
6—73. Superior view revealing modular displacement about the vertical axis of the body.

Yaw #1 — Occiput. The examination for yaw occiput is started with the supine patient's hips and knees flexed and the feet resting on the table. The patient then rotates the hips and pelvis to move the knees to one side, the same as for roll. In addition, the patient turns his head in the opposite direction, keeping



6—74. Testing position for yaw occiput.

it on the table. A strong indicator muscle is tested for weakening, which indicates a positive yaw occiput. The patient is tested in both directions. A positive test indicates failure of normal movement between the occiput and atlas. This can be considered a special type of occipital fixation because it only challenges positively with a two-handed fixation-type challenge; however, there is no bilateral psoas muscle weakness. The occiput is challenged anteriorly while the anterior portion of the atlas transverse is held stationary. The challenge will be positive on one side only, usually the side that was up when the patient's head was rotated during the test. The occiput is adjusted on the atlas. Contact is made on the most tender point of the inferior nuchal line of the occiput. The patient's neck and head are kept in a straight position, and the line of drive is from the



6—75. Challenge of atlas and occiput for yaw #1.

tender point to the glabella. After correction, the positive yaw occiput test should be negative.

Yaw #2 — Sacral. The patient is examined prone for yaw #2. The body modules are rotated by placing a DeJarnette block under a shoulder and another one under the opposite pelvis. This places torsion through the trunk and, if positive, will cause an indicator muscle to weaken. The patient is tested for rotation in both directions; only one will be positive. If the patient tests positive in both directions, some other factor — such as a sacral wobble or other pelvic or shoulder disturbance — is at fault. Evaluate the structures put into torsion from the test; correct and re-evaluate for yaw-sacral.

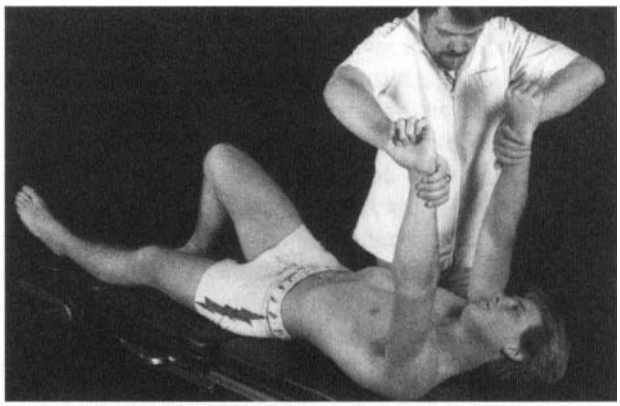
Correction is directed toward the sacrum. It is adjusted from posterior to anterior on the positive side. After correction, blocking at the shoulder and pelvis should no longer cause an indicator muscle to weaken.

Yaw #3 — Dorsolumbar. The test for yaw dorsolumbar is similar to that for yaw sacral. A DeJarnette block is put under the anterior superior iliac spine, and another under the opposite lower thoracic cage. This puts torsion into the dorsolumbar junction, revealing a fixation that is not related to bilateral lower trapezius weakness. The fixation may be anywhere in the lower thoracic or lumbar area and, is corrected in the manner described under fixations in Chapter 3.

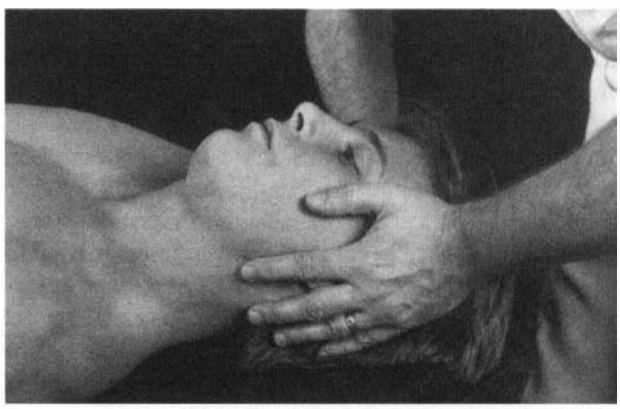
Tilt

Tilt is tested with the patient supine. A positive test is the patient's inability to laterally flex the head and neck while the knee and hip are flexed without an indicator muscle weakening.

The hip and knee are flexed on one side, and the foot rests on the table. The head and neck are laterally flexed to the side of hip flexion, and a strong indicator muscle is tested for weakening. Patients have a tendency to rotate the head as it is put into lateral flexion. This should be avoided as it often eliminates a positive test.



6—76. Test for right tilt pattern.



6—77. Correction is made by patient trying to laterally flex his head and neck while physician resists the effort.

The cause of a positive tilt pattern appears to be in the muscles of the cervical spine. Correction is obtained by the examiner stabilizing the head in a neutral position while the patient tries to laterally flex his head on his neck and shoulders. He is urged to make a maximum isometric contraction while the examiner resists any movement. The treatment is not as effective if the patient attempts to rotate the head and neck while laterally flexing them.

Equilibrium Reflex Synchronization

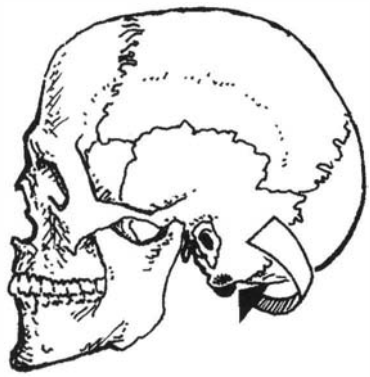
One of the major goals in examination and treatment is to organize a disorganized nervous system. Equilibrium proprioceptor synchronization is an examination technique that often helps find the cause of neurologic disorganization. The technique as originally described hypothesized that there are centering reflexes in the pelvis analogous to the cloaca of lower phylogenic levels; it was called cloacal synchronization.[28] Clinical evidence indicates there are equilibrium reflexes in the pelvis, and this technique proposes to evaluate their synchronization with the visual righting, head-on-neck, and labyrinthine reflexes. There are many techniques in applied kinesiology that appear to influence these reflexes.[88] Disorganization between the reflexes — for whatever reason — interferes with homeostasis and general integration of the body.

Active reflexes have specific therapy localization and a positive muscle testing pattern. When evaluating the reflexes with therapy localization, one must remember that therapy localization tells something is there but not **what**. The need for equilibrium reflex synchronization should be correlated both with therapy localization and the muscle-testing pattern.

Labyrinthine Reflexes

The labyrinthine receptors are located in the semicircular canals and utricles of the middle ear. Movement of the head stimulates the receptors, which influence the head righting muscles and postural muscles dealing with equilibrium. Animals subjected to a bilateral labyrinthectomy are unable to walk until one week after the surgery; then gait can only be accomplished on a broad-based leaning and staggering basis.

The point for therapy localization is at the digastric fossa of the temporal bone. This point is medial to the attachment of the splenius capitis and longus capitis, at the most superior section of the fossa. This point probably also therapy localizes some of the skin receptors involved with the head-on-neck and neck righting reflexes.



6—78. Therapy localization point for labyrinthine reflexes, medial to the mastoid process.

Head-on-Neck Reflexes

Head-on-neck reflexes are the mature tonic neck reflexes that were in the infant. They are located in the upper joints of the neck, especially at the atlantoaxial and occipitoatlantal articulations.[55] They are important in body orientation and motor coordination.[9]

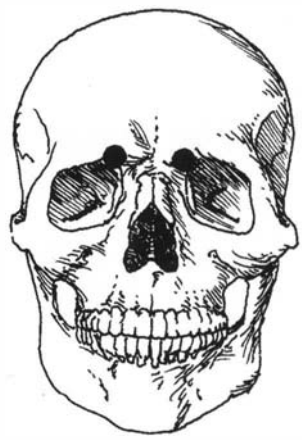
The head-on-neck reflexes are therapy localized at the occipitoatlantal, atlantoaxial, and sometimes at the axis-3rd cervical articulations.

6—79. Therapy localization points for neck righting reflexes.

Visual Righting Reflexes

The visual righting reflexes provide signaling that is important to optimal function of the body's organization and orientation in space. It is possible for a blind person to function adequately; however, note the disorganization of the body modules. The head is typically not placed on the neck, neck on trunk, and trunk on pelvis in a balanced manner.

The visual righting reflexes are therapy localized medial to the supraorbital notch. When a strong indicator muscle weakens from therapy localization to this



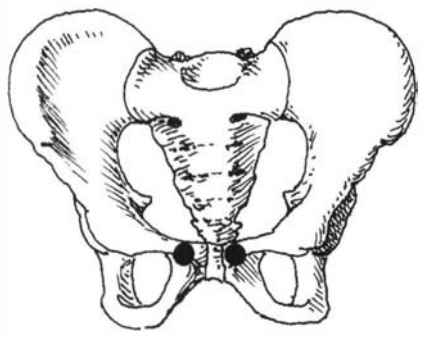
6—80. Therapy localization points for visual righting reflexes.

area, it will again become strong if the patient closes his eyes, even though the therapy localization is maintained. This takes the visual factor out of the test and helps determine that the weakening is actually due to the visual righting reflexes rather than some other factor, such as the peroneus brevis stress receptor located in the same area.

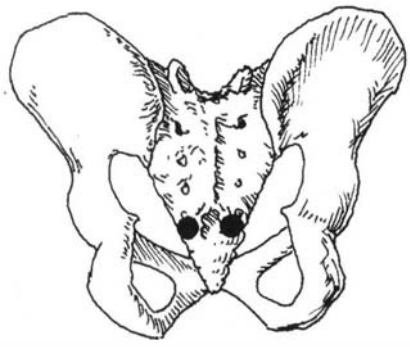
Pelvic Reflexes

The pelvic reflexes have not had much study by neurophysiologists. They have been used extensively in various chiropractic techniques where light to moderate, and sometimes heavy, stimulation is applied to the skin in the general area of the ischium, sacrum, coccyx, and even sometimes intra-anally. Watkins[87,88] makes a case for a perianal reflex being responsible for the success of all these techniques. He observes that stimulation to the medial aspect of the gluteus maximus causes a cow to move her pelvis toward the stimulation, whereas dermal stimulation to the lateral aspect of the gluteus maximus causes her to move away from the stimulation. The pelvic reflexes may be primitive centering reflexes. It is suspected that receptors for organization are present in the joints of the pelvis because of the effects that pelvic manipulation appears to have on these reflexes.

Beardall[2] specifically locates the cloacal reflexes, now called pelvic reflexes, from applied kinesiology



6—81. Therapy localization points for the anterior pelvic reflexes.



6—82. Therapy localization points for the posterior pelvic reflexes.

clinical evidence. The locations will have positive therapy localization when the reflex is active. The anterior pelvic reflex is on the anterior external surface of the superior ramus of the pubis, below the origin of the pectineus and lateral to the origin of the adductor longus along the superior border of the obturator foramen.

The posterior pelvic reflex is located where the sacrotuberous ligament attaches to the 4th and 5th transverse tubercles of the sacrum and the lateral margins of the coccyx.

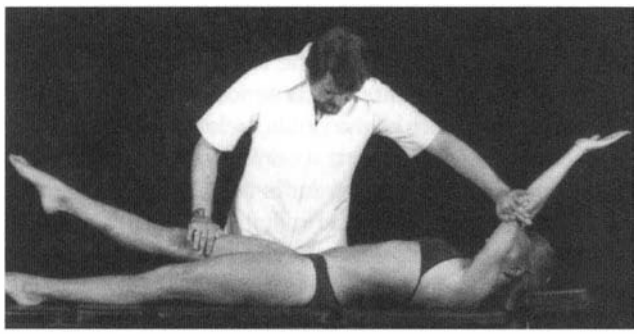
Muscle Testing

The best method to evaluate for equilibrium reflex synchronization is with group muscle tests developed by Beardall.[2] The tests consist of an arm and leg being brought together by either flexion or extension. This is done ipsilaterally and contralaterally for eight group tests. Reflexes on the anterior, i.e., visual righting and anterior pelvic reflexes, relate with shoulder and hip flexors, respectively. The labyrinthine and head-on-neck reflexes are considered posterior and relate with shoulder extensors. The posterior pelvic reflexes are associated with the hip extensors.

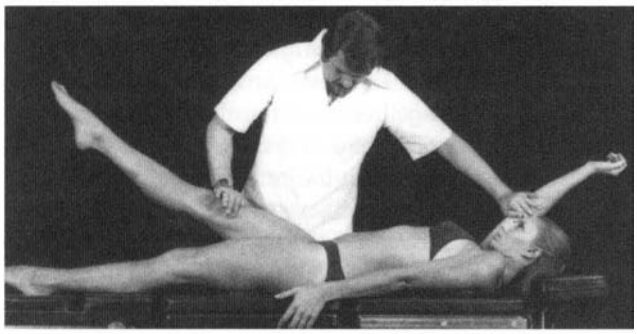
Two muscle groups are tested during each test, similar to gait testing. A positive test is when one or both muscle groups weaken when tested simultaneously, but each group is strong when tested by itself. As in gait testing, the efficient method is to test the two groups together; if one or both weaken, test each individually. If a group tested individually is weak, the muscles making up the group must be tested individually to determine which of the five factors of the IVF is responsible for the weakness. Treat as usual and then re-test the group for need of treatment with pelvic reflex synchronization technique.

Anterior Contralateral and Ipsilateral Apposition Test. The supine patient flexes his hip to raise his leg slightly from the table. For the contralateral test the opposite arm is raised to nearly 160° shoulder flexion; for the ipsilateral, the ipsilateral arm is raised to the same position. The examiner directs pressure on the leg toward hip extension and on the arm to further flex it, while the patient attempts to bring the extremities into apposition. The contralateral arms and legs are tested on both sides, as are the ipsilateral arms and legs, making four separate tests.

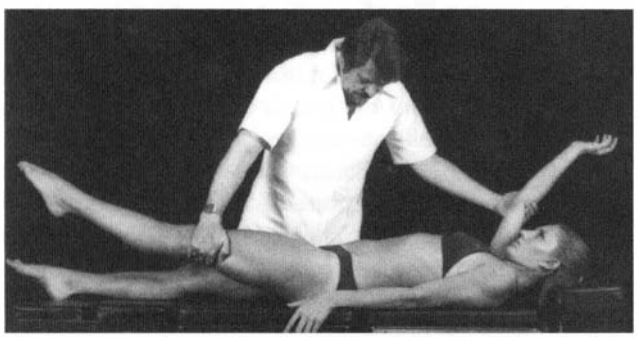
Posterior Contralateral and Ipsilateral Apposition Test. The patient duplicates the starting position for the anterior test by bringing the hip into slight flexion, raising the leg from the table, and placing the arm into 160° of shoulder flexion. The examiner directs pressure on the posterior aspect of the thigh to further flex the hip, while simultaneously directing pressure against the ipsilateral or contralateral posterior arm to bring it out of flexion. The patient attempts to bring the arm and leg into posterior apposition or, in other words, the hip toward extension and arm toward further flexion.



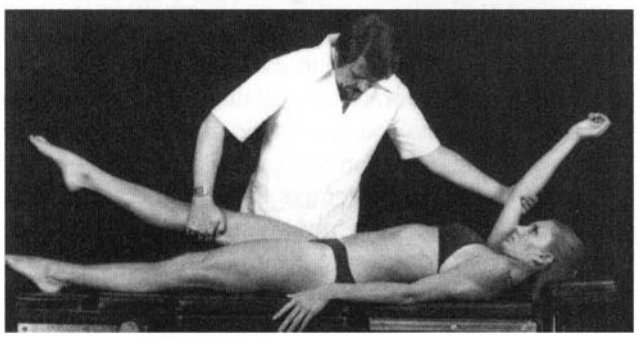
6—83. Anterior contralateral apposition test.



6—84. Anterior ipsilateral apposition test.



6—85. Posterior contralateral apposition test.



6—86. Posterior ipsilateral apposition test.

Therapy Localization

When there is a positive equilibrium reflex synchronization test, there should also be positive therapy localization to the associated reflex. For an anterior apposition test, the anterior pelvic and visual righting reflexes should have positive therapy localization, either contralaterally or ipsilaterally as indicated by the muscle test. When a posterior apposition test is positive, the posterior pelvic and labyrinthine or neck righting reflexes should have positive therapy localization. If there is no correlation between the muscle tests and reflex therapy localization, further evaluate the patient for other factors that might be therapy localized, or the possibility of muscle groups testing weak when tested individually.

Correction

There are two methods by which equilibrium reflex synchronization is obtained. Both use muscle testing and therapy localization to locate the area for treatment. One method uses gentle manipulative efforts coinciding with the patient's breathing; it apparently deals with the cranial-sacral primary respiratory system. The other method is simple two-handed reflex point contact.

The most effective method of obtaining equilibrium reflex synchronization is treatment to the cranial-sacral primary respiratory system.[86] This method is applicable when the group muscle weakening is eliminated by having the patient take a phase of respiration. When there is a positive test — one or both muscle groups weakening when tested together but not when tested individually — have the patient take a deep phase of respiration, either inspiration or expiration. When the patient holds one of the phases of respiration — usually inspiration — the muscles no longer test weak. This indicates that treatment to the cranial-sacral primary respiratory mechanism is needed and is the optimal method of correction. (This and further correlation to equilibrium reflex synchronization are discussed in Chapter 9 and elsewhere.[86]) When testing for respiratory correlation, be certain to have the patient take a deep phase of the respiration. A respiratory correlation is nearly always present with positive equilibrium reflex testing.

When respiratory correlation does not appear to be present with the positive combined muscle testing and therapy localization, reflex contacts can be held at the points of positive therapy localization. Electromagnetic energy patterns about the body are a controversial subject in the healing arts. They become somewhat more understandable after studying and working with the meridian system, especially in applied kinesiology. An attempt is often made to explain unusual therapeutic response by electromagnetic fields. Total understanding of these mechanisms must await instrumentation that can qualify and quantify these patterns that sometimes appear to be present. Beardall[2] promotes the hypothesis that there is an electromagnetic energy flow between the reflexes, an important aspect in maintaining normal function of the specific mechanisms. In other words, there is a flow of energy from the anterior pelvic reflex to the visual righting reflex, both ipsilaterally and contralaterally.

To eliminate the weakening of the dual muscle

group test, the positive reflex points are contacted simultaneously by the physician. The digital contact is held until the examiner feels synchronized pulsation at both points. This takes approximately twenty seconds in the average patient. The more debilitated the patient, the longer the contact must be maintained before pulsation is felt. Once pulsation is felt, the associated muscle groups should simultaneously test strong and the positive therapy localization of the reflexes should be abolished.

Treatment and subsequent synchronization of these centering and righting reflexes improve body organization. It is specifically indicated when an individual requires repeated applied kinesiology treatment to correct switching, or when conditions tend to recur when he runs or walks. This synchronization procedure rarely needs repeating unless there are recurrent problems with the cranial-sacral primary respiratory system.

Rib and Spinal Fixation ("Limbic Technique")

When there are positive findings with the PRYT technique, there are often fixations of the spine or ribs that may cause the PRYT condition to return. A frequent involvement is a fixation of the lower cervical vertebrae and the 1st rib. It can be evaluated by challenge and therapy localization, as generally used in the fixation technique described in Chapter 3. The most common area is the 7th cervical vertebra and its articulation with the 1st rib. The challenge is to contact the spinous process and 1st rib at its angle and separate them, testing a strong indicator muscle for weakening. There is no positive therapy localization unless therapy localized while the patient attempts to move the area. This is a constant finding in all fixations. There is often muscular weakness in the leg because of fixations in this area. There is no positive correlation of muscle weakness, such as in vertebral fixations described in Chapter 3. The frequency of weakness, in order, is the peroneus tertius, peroneus longus and brevis, tibialis anterior, and tibialis posterior. Upon correction of the cervical rib fixation, the weak muscles will strengthen if there is no other cause for the weakness.

The 7th cervical vertebra and 1st rib are adjusted away from each other in the direction of positive challenge. A single thrust with one hand on each structure will correct the condition, if the contact can be made. Often it is difficult to make the two-handed contact. The 7th cervical can be adjusted away from the rib, and then another adjustment can be applied to the rib away from the 7th cervical. There will often be an audible release in both adjustments, but it is not necessary for an effective correction.

The ribs and vertebrae should be further evaluated for fixations in a manner similar to that described. There is often a fixation between the 12th rib and 1st lumbar vertebra. Challenge for separation and test a previously strong indicator muscle for weakening. The basic rule — no positive therapy localization in fixations unless therapy localized with movement — is applicable.

Rib Pump Technique

With the introduction of the cranial-sacral primary respiratory mechanism into applied kinesiology, many additional conditions have been successfully treated. Progressively better methods of examining and treating the system have enhanced the effectiveness and stability of corrections.

At the upper portion of the cranial-sacral primary respiratory mechanism is the stomatognathic system, i.e., basically all structures from the shoulder girdle up. Recognizing that the stomatognathic system is a closed kinematic chain incorporating mandibular and cervical spine movement as intricately involved in cranial function, W.B. May,[52,53] a dentist who specialized in balancing the mandibular function to the cranium, stated, "If the cranium is a pump for cerebrospinal fluid, then the mandible is the pump handle."[54]

The sacrum and innominate bones make up the lower portion of the cranial-sacral primary respiratory mechanism. Movement of these bones must be intricately organized with cranial motion for optimal function. Connecting the upper and lower portions of the cranial-sacral primary respiratory mechanism is the firm dural attachment at the foramen magnum and the upper cervical vertebrae, with no further firm attachment until the 2nd sacral segment.

Thoracic Organization. Thoracic respiration influences the cranial-sacral primary respiratory mechanism. A weak muscle due to a cranial or pelvic fault will strengthen when a patient takes a specific phase of respiration that moves the cranium or pelvis toward improved function. Frymann[22] objectively measured cranial movement created by deep respiration. She observed the correlation between the three diaphragms, i.e., 1) cranial diaphragm: part of the intracranial dura mater that divides the superior compartment containing the cerebrum from the lower compartment containing the cerebellum, 2) diaphragm dividing the thorax from the abdominal cavity, and 3) pelvic diaphragm dividing the pelvic proper above and the ischiorectal fossae below.[21] Dysfunction of one of the diaphragms will be transferred to one or both of the other diaphragms, resulting in health problems of many kinds.

The organization of the three diaphragms is discussed more thoroughly in Chapter 12 on the thoracic diaphragm.

The integration of thoracic movement with the cranial-sacral primary respiratory mechanism leads to the conclusion that the system should be called the cranial-thoracic-sacral primary respiratory system. Wyke[91] has demonstrated types I, II and IV nerve receptors in the costal, vertebral, and sternal muscle articulations in the cat, and it is presumed they are the same in human anatomy. He proposes that if the normally symmetrical tonic reflexogenic influence from the type I costovertebral mechanoreceptors becomes asymmetrical from postural deviations, the paravertebral and intercostal muscle forces will in turn be asymmetrical. Rib motion in conjunction with the thoracic spine provides abundant afferent supply to the neuraxis.

Cerebrospinal Fluid. What other factors might be important regarding the movement of the thoracic cage? Goodheart[36] has proposed that proper movement of the thoracic cage provides an auxiliary pump for cerebrospinal fluid movement into the spinal nerve. There has been controversy regarding the movement of cerebrospinal fluid into the spinal nerves, although experiments to determine the movement date back to 1872.[92] In 1947 Somberg[74] found that cerebrospinal fluid passes only to the spinal ganglion under low pressure. This prompted a critical review by Hassin,[41] who took strong exception to Somberg's study. He states, "The assumption that a barrier exists at the proximal end of the spinal ganglion is a priori unsound, for the presence of a barrier along the course of the cerebrospinal fluid pathway would be a calamity. It would create a situation obtaining in the worst types of spina bifida, extra spinal tumors or meningeal and parameningeal inflammations." Citing several studies, he concludes, "1. The cerebrospinal pathways along which the spinal fluid escapes to the periphery extend from the subarachnoid space over the spinal roots, into the spinal ganglia, from which they emerge to follow the perineural spaces of the post-ganglionic nerve. 2. Macroscopically the spaces over and in the spinal ganglia are not demonstrable, just as similar spaces in the central nervous system, such as those of Virchow Robin, cannot be seen grossly but can be demonstrated under certain conditions microscopically." Steer and Horney[75] injected blue powder particulates into the lumbar subarachnoid space of pigs and sheep and found that the powder in the cerebrospinal fluid was transmitted by the nerves toward the periphery. Spinal nerve root nutrition supply is by diffusion from the cerebrospinal fluid and via the interneural vascular bed, as noted by Rydevik et al.[68] Kiwic[47] notes that the pre-lymphatic cerebral vessels and perineural lymphatic pathways around cranial and spinal nerves seem to be the most significant ones connecting the central nervous system and the lymphatic system.

Biologically Closed Electric Circuits. Most of applied kinesiology examination and treatment deals with improving communication throughout the body. This often deals with the nervous system. Receptors must supply accurate afferent supply, which in turn must be properly reacted to. Other less understood systems or mechanisms come into play, such as holographic memory. An additional method of communication is the biologically closed electric circuits (BCEC). Electric circuits have an ever-increasing importance in modern civilization. They are used to produce power, such as in electric motors, communication, and instrumentation. Only more recently have the biologically closed circuits' influence on structure and function come under more intense study. The first BCEC system identified is the vascular closed circuit (VIC).[63] Blood vessel walls have a high resistance; therefore the veins and arteries can function as relatively insulated conducting cables for ions. The vessels form conducting loops of various sizes. An external moving magnetic field can therefore induce a flow of ionic current in the loops.

The vascular interstitial closed circuit (VICC) is additional circulation to the blood and lymph. This circuit provides rapid bi-directional exchange of positive and negative ions and is a common mechanism involved in the fueling and maintenance of all organs. For example, an active muscle produces lactic acid and protons that make the muscle electro-positive in relation to blood, thus disturbing homeostasis. The voltage difference between muscle and blood represents an electrical field driving bi-directional flow of ions and cations. As equilibrium is restored, providing homeostasis, the muscle can again function normally.

Electrical conduction in the vascular interstitial closed electric circuit participates in the conduction of the motor supply to muscle. When the vessel(s) are ligated, muscle contraction ceases when the nerve is stimulated, as shown by Nordenström.[61]

Nordenström[60] has investigated the positive and negative ionic energy of the VICC system in correlation to the meridian system. The positive and negative collection of ions of the VICC may correspond to the Yin and Yang energies of the meridians; thus the electric energy driving the VICC system might correspond to the Chi energy in acupuncture. Nordenström measured the electric potential at 275-300 equally spaced locations in a dog's subcutis to a reference location. He concluded that the acupuncture meridians might be preferential pathways for ionic current through the VICC system.

Changes in the biologically closed electric circuit (BCEC) system are manifested when exposed to an external electro-motive force (EMF). Nordenström[62] demonstrated that moving a hand approximately 3 cm from a rat's tail changes the voltage reported between shielded electrodes in an abdominal vessel in relation to the peritoneum or subcutis. The change in voltage

is caused by the EMG polarization in the vessels of the rat's tail that extend as closed-circuit conducting vascular channels in the body. The direction of external EMF movement relative to the position of the vessels determines how the voltage gradients in the body are altered.

Nordenström[62] notes that the Chi energy of Chinese medicine is equivalent to the electrical energy of the body. The energy can secondarily be transferred via the chrona of the skin to another subject — in this case from the experimenter to the rat. This may explain the transfer of energy to a patient by a Qigong practitioner performing therapy. Nordenström notes, "Further, the meridians in acupuncture represent the preferential pathways for ionic flow in the subcutis of the vascular-interstitial closed circuits (VICC-system)."

Early in applied kinesiology's work with the meridian system it was found that the examiner could move his hand a few centimeters from the meridian pathway several times, and it would influence the meridian's associated muscle in many people. If done from the end to the beginning of the meridian it would cause the meridian's associated muscle to weaken temporarily. If the associated muscle was weak in the clear, moving the examiner's hand over the meridian from beginning to end would temporarily strengthen the associated muscle if the weakness was due to a deficient meridian. This is apparently the same energy transfer of electro-motive force that Nordenström demonstrated in the rat.

Thoracic Movement. The importance of proper thoracic cage movement is probably not yet completely understood, but we can make some assumptions for the health improvement attained by applying the rib pump technique.

Proper diaphragm function is needed to organize with the cranial and pelvic diaphragms. In order for the diaphragm to function properly there must be no restriction of the thoracic cage. (See diaphragm, Chapter 12.) It has been hypothesized in applied kinesiology[30] that the diaphragm mobilizes Chi in the meridian system. Certainly, there is significant evidence for this. When breathing is stopped there is more sensitive therapy localization to meridian points, and correcting diaphragm function often eliminates Chi imbalance within the meridians. Nordenström's studies of the VICC system help put this in perspective.

Rib motion is necessary for stimulation of the mechanoreceptors of the costovertebral and costosternal articulations. These receptors are an important afferent supply to the cerebellum and for cognition of the breathing status and dyspnea, if present.[91]

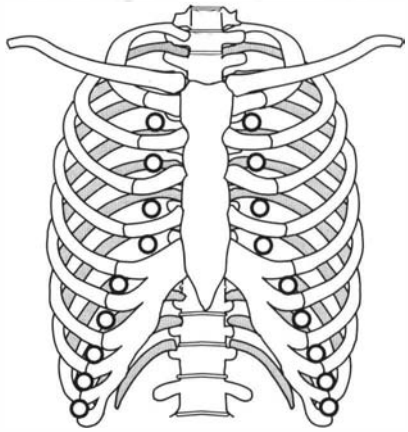
Mobilization of cerebrospinal fluid into the spinal nerves and lymphatic system may be enhanced by rib motion. No direct study of this possibility has been found.

Regardless of the reason(s), there are favorable results in patient function from application of the rib pump technique. It is another method of examination and treatment when considering the total complex a patient presents.

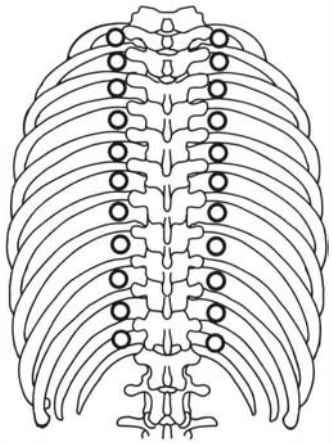
Examination and Treatment

Rib pump technique is administered primarily to improve movement of the thoracic cage. This improves stimulation to the mechanoreceptors at the costovertebral and costosternal articulations, may improve movement of cerebrospinal fluid along the nerve pathways and into the lymphatic system, and enhances activity in the biologically closed electrical circuits. The actual change in these functions may be speculative. Improved thoracic cage movement in conjunction with diaphragmatic technique, discussed in Chapter 12, improves respiration. Prior to application of rib pump technique, thoracic cage expansion and vital capacity can be measured to compare after treatment as an indication of effectiveness.

Strain/counterstrain. The need for strain/counterstrain technique can be determined by palpating the intercostal space near the sternum and vertebrae for tenderness. These tender points are treated with strain/counterstrain technique. Both the anterior and posterior locations should be monitored. There will



6—87. Anterior rib pump strain/counterstrain points.

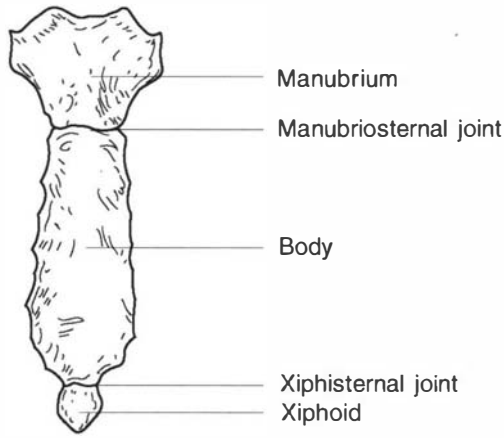


6—88. Posterior rib pump strain/counterstrain points.

be more tender points anteriorly than posteriorly. When a tender point is found anteriorly, flex the patient's head, neck, and possibly thoracic spine until the tender point is abolished or greatly diminished. While maintaining that position, have the patient take a deep breath and hold it while the tender point location is spread by a two-finger contact. Hold for about thirty seconds, then slowly return the patient to a neutral position. The tender point should remain abolished or greatly diminished.

Palpate for posterior pain in the intercostal spaces near the vertebrae for localized tender spots. There will be positive therapy localization, and the tenderness can be eliminated by positioning the patient in extension. Rotation and possibly lateral bending may be necessary in combination with extension to completely eliminate the tender spot. While maintaining the position that eliminated or greatly reduced the tenderness, have the patient exhale to the maximum and hold it for about thirty seconds while the tender spot is spread with a two-finger contact. Slowly return the patient to the neutral position; the tenderness should be abolished or greatly reduced.

Manubriosternal and Xiphisternal Joints. Dysfunction at the manubriosternal and xiphisternal articulations appears to be fixation; however, in this case there is positive therapy localization without the need to add movement. In the presence of positive therapy localization treat by percussion, which can be done by fingertip tapping or a percussion instrument.



6—89. Treat joints of the sternum with percussion.

Dural Tension

Walking and running are normally cross-patterning activities with the contralateral arm and leg moving forward together. Early in applied kinesiology it was found that cross-pattern crawling could enhance the neurologic control of muscles.[25] The detrimental effects of an improper gait can readily be demonstrated by having an individual lie supine and simulate homolateral walking, i.e., the arm and leg of the same side flexing at the shoulder and hip as if walking. Performing this pattern bilaterally five or six times will cause most individuals to test weak in all muscles until the nervous system reorganizes itself. Some people have an improper gait pattern, such as homolateral walking. Under normal conditions this is detrimental to the nervous system's organization every time an individual walks or runs. A poor gait pattern is often seen when children with learning disabilities run. There may be a homolateral pattern or flailing of the arms in an unorganized manner. Under certain conditions, training the child with cross patterning improves neurologic organization and general body function. The cross-pattern examination and therapeutic approach are discussed in Chapter 4.

From the experience of working with cross-pattern training and research done by educators,[13,14] it is obvious that a proper cross-pattern gait enhances neurologic organization and function while an improper one is detrimental to function.

Many individuals develop fatigue and other symptoms as the day progresses. They feel better after a night's rest, only to have the pattern repeat itself the next day. Similarly, some who embark on a physical fitness program of walking or running find that it does not enhance function as hoped, or they simply do not feel well doing the program. Most cease the activity, while others doggedly continue because they feel they should have the additional exercise.

The physical activities of walking and running under normal circumstances are excellent. Many people are under-exercised and can benefit greatly from the activity. Before embarking on this type of exercise, one must be sure that gait is proper. Sometimes the activities can be harmful to function.

Development of symptoms after walking or running is often related with a person having a longer stride on one side. This is a common gait fault in individuals with major health problems, as well as in those who appear healthy but are not functioning in an optimal way. After walking with the stride longer on one side, muscular weakness develops as observed by manual muscle testing. Antagonists to the weak muscle become hypertonic, resulting in axial torsion in the body. Goodheart[33] hypothesizes that the axial torsion culminates in torsion of the dura mater, inducing or increasing improper signaling of the nervous system. Breig,[6] a neurosurgeon in Sweden, has developed a system he calls "functional neurosurgery," which is designed to obtain relaxation of the soft tissues of the spinal canal. The primary aim of the surgery is to restore normal nerve conductivity by bringing the spine's bony components into harmony with its soft tissues, such as the dura and nervous structure. The surgical procedure

takes wedges out of the vertebrae to obtain harmony.

The dura mater is firmly attached superior to the circumference of the foramen magnum and the posterior surfaces of the bodies of the 2nd and 3rd cervical vertebrae. Inferiorly it attaches by the filum terminale to the dorsum of the 1st coccygeal segment.[90] It is generally considered that there is no attachment of the dura to the atlas, and minimal attachment between the 3rd cervical and coccyx. Some have observed firm attachment of the dura mater to the atlas in dissection.[15] Pressure and tension on the dura mater and spinal cord depend upon a proper relationship between the spinal column and the dura mater. There is some adaptability of the relationship between the two, but it is limited. Coccygeal motion appears to take up some of the slack in the dura mater with spinal movement. Goodheart's hypothesis for the dural torque technique of applied kinesiology is that there is a disrelation between the spinal column and dura mater, and torsion placed into the body from improper walking produces torsion of the dura mater, interfering with normal nerve function. In chiropractic the vertebral subluxation is often related to twisting or torsion of the dural sheaths that accompany the spinal nerves as they blend with the connective tissue sheaths in or slightly beyond the intervertebral foramina. Tension of the dura could also be responsible for back pain. Cyriax[10] stated in 1948 that back pain comes from pressure and irritation to the dura mater. He points out that there are receptors in the anterior but not the posterior portion of the dura.[11]

Goodheart[33] refers to dural torque as being an actual twisting of the dura. As this discussion proceeds, axial tension will be introduced. Dural tension is an all-encompassing term that will be used in this text.

Body Language

When testing a patient with applied kinesiology methods one may find no muscle weakness, yet the patient is symptomatic and the TS line indicates there should be muscle dysfunction. If the patient is asked to walk a relatively short distance and is then placed on the examination table, many areas of muscle weakness may be observed. After lying on the examination table for a short period, the dura mater tension is relieved and evidence of dysfunction will no longer be present. These individuals are often people who do not like to stand, sit, or walk for a long time. With prolonged sitting, as at a meeting, they have a tendency to get up and walk around periodically. When standing or walking, they want to change the activity. With prolonged walking, symptoms often worsen. These are people whose symptoms progress throughout the day. They feel worse on a running program and better after lying down for a while. This may be the reason why some individuals improve only after bed rest.

Children developing a scoliosis should be evaluated for dural tension. The rapid period of growth may exceed the ability of the spinal column, dura mater, and cord to adapt.

Most people who develop dural tension walk with a longer right step. The amount of increased stride on that side varies, but it is only a small amount. Most individuals do not observe the gait disparagement between sides. When the stride is long on the right, the left arm will usually swing further forward or the right arm will have less motion.

Relating Factors

General AK examination and correction are done before evaluating for dural tension. Patients with dural tension usually have some type of pelvic and often lumbar dysfunction. Among the factors that should be tested and cleared are pelvic categories I, II, and III, and any lumbar dysfunction; in addition, modular dysfunction in PRYT, gait, equilibrium reflex synchronization, and the cranial-sacral primary respiratory system should be evaluated and corrected, if necessary.

When dural tension develops from walking, many of the above corrections will immediately be lost. When they are again corrected and the patient is evaluated for dural tension and its cause corrected, there will no longer be a loss of the initial corrections when the patient walks.

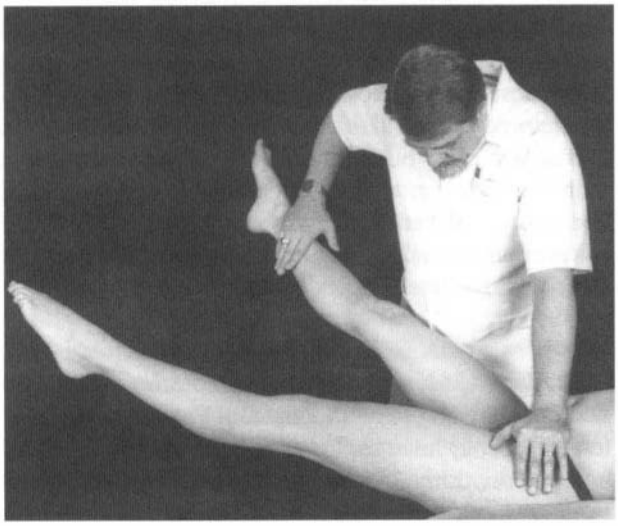
Examination

With the patient supine, evaluate internal thigh rotation. This is done by turning the legs in at the feet. There will be resistance on one side, with the contralateral leg turning in further. Increased internal thigh rotation is usually associated with weak piriformis and psoas muscles. When testing the piriformis and psoas muscles bilaterally, they will usually be equally strong on each side, even though there is increased thigh turn-in on one side. Immediately after walking there will be weakness of these muscles on the side of increased internal thigh rotation, but strength contralaterally. The weakness will go away after the patient lies on the examination table for a brief time, because the dura mater tension is relieved.

An individual who develops torsion while walking does so because of a longer stride on one side. The long stride is on the side of limited internal thigh rotation. It is postulated that the psoas is hypertonic on that side and literally pulls the leg further forward, increasing the stride on that side.

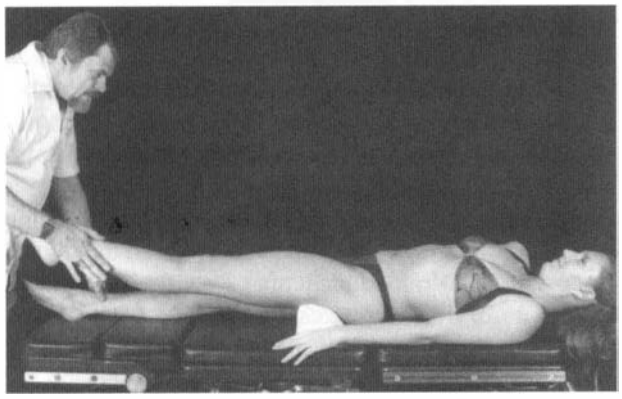
Another method for observing weakness of the piriformis and psoas muscles on the increased internal thigh rotation side, when they are strong in the clear, is to have the patient flex the contralateral hip 10° to lift the leg from the table. Lifting the leg increases the torsion, and the psoas and piriformis will now test weak.

A rapid method to evaluate for the presence of imbalanced stride is to place a DeJarnette block underneath the acetabulum and another one under the con-



6—90. The psoas and piriformis will test weak on the side opposite the long stride. If they do not test weak in the clear, they will when the leg of the long stride is held in hip flexion, as illustrated.

tralateral shoulder, placing torsion into the trunk. This simulates the torsion that develops during the long stride phase of gait. When a block is under the acetabulum on the side of long stride and one under the contralateral shoulder, almost any indicator muscle of the body will test weak.



6—91. Placing the patient into torsion with a DeJarnette block under a shoulder and another under the contralateral acetabulum will cause indicator muscles to weaken when dural tension is present.

There should be a correlation between the findings of the blocking and thigh rotation tests. The side that was blocked under the acetabulum to cause indicator muscles to weaken should be the limited internal thigh rotation side. If it is not, neurologic disorganization was missed and left untreated during earlier evaluation of the pelvis, equilibrium reflex synchronization, and PRYT, among other tests. After evaluating and correcting the neurologic disorganization, the leg rotation and blocking tests should correlate. Although supine testing is usually more convenient, the patient can also be evaluated when prone. Unless the patient has recently walked or there is some other problem present, the piriformis will be strong bilaterally. To evaluate for the side of long stride, have the prone patient move to the edge of the examination table and drop a leg over the edge. This places it into a flexion position similar to gait, as did lifting the leg with the patient supine. The contralateral piriformis will weaken if the leg dropped off the table is on the long stride side. Repeat on the opposite side. Only one side should be positive.

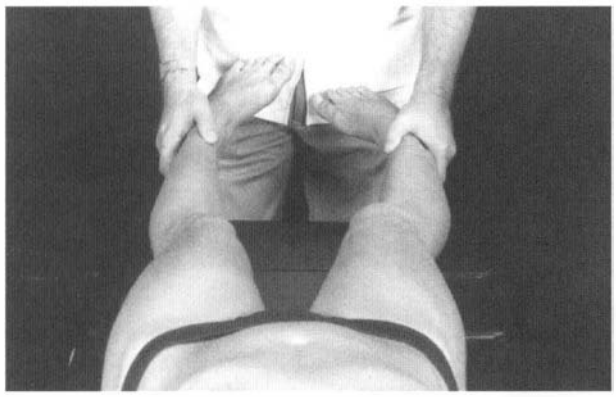
Some patients who develop body torsion while walking do not show positive tests as just described. Evidence that dural tension is developing is obtained after having the patient walk, sometimes for a considerable distance, and then re-evaluating function. If proper function is lost after walking, dural tension is probably present. The tests that failed to show it can be made positive by adding right and left brain activity during the test. This should routinely be done when there is suspicion of body torsion from walking, but test results are negative. Walking uses left and right brain activity, with the left brain controlling the forward leg and the right brain controlling the forward arm. Although it is recognized that walking takes place in decerebrate animals from the spinal locomotor generator,[58,66] there is obviously supraspinal activity in the intact biped. The test is performed with the patient on the blocks. He is asked to say the multiplication tables while the physician tests as before. The test is then repeated while the patient hums. Care must be taken that the patient is actually humming and not thinking or singing the words of a song. When a well-known tune such as "Happy Birthday" is hummed, many patients will think the words along with humming. A positive test with right or left brain activity is treated the same as one that did not require the added factor. Other factors must be taken into consideration for the cause of weakening, such as foot or gait problems.

Treatment

Change in an individual's walking pattern is necessary to eliminate the torsion that builds up as a result of taking a longer stride on one side. This can be accomplished by having the patient consciously take a longer stride on the opposite side. Dysfunction treated with three other AK procedures has a bearing on body torsion developing from improper walking. These are strain/counterstrain technique to pelvic muscles, iliolumbar ligament treatment, and the filum terminale cephalad lift technique. Correction of these problems may eliminate the need for the patient to walk with a longer stride on the opposite side.

Most individuals with an imbalanced stride will have a gluteus maximus that requires strain/counterstrain technique. About 15% will need the technique applied to the gluteus medius, and about 1-2% will require it at the piriformis. The strain/counterstrain technique is applied in the usual applied kinesiology

manner. After clearing the strain/counterstrain muscle dysfunction, blocking at the pelvis and shoulder will be negative, including when tested with right and left brain activity, if the change in walking pattern is not needed. Evaluate the patient for need of filum terminale cephalad lift and iliolumbar ligament techniques and correct if needed.



6—92. After correction, internal thigh rotation will be equal and the other tests normal.

If, after making any indicated corrections and ruling out foot, gait and other problems, there is still evidence of torsion when walking, the patient's gait must be modified. He is taught to take a longer stride on the side opposite that which tested positive with the block under the acetabulum. This is the side of excessive internal thigh rotation, and opposite the side of decreased thigh rotation.

The increase in stride length should be only one or two inches. Advise the patient that the change in gait should not be so much that others will notice a funny walking pattern. The length of time that a person must consciously change his gait varies from three to four days for mild problems to three or four weeks for moderate ones, and three to four months for severe disturbances. The patient should be examined periodically during this time. Approximately 10% of the people will shift to require a reversal of the conscious long stride pattern.

Spinal fixations are frequently present with dural tension. The patient should be routinely evaluated for upper cervical fixations that are associated with bilaterally weak gluteus maximus muscles. Unless the fixation is associated with weight bearing, the gluteus maximus will be bilaterally weak when evaluating for need of strain/counterstrain technique. If the patient feels worse after walking with the modified stride, there are usually mid-thoracic fixations.

The types of structural pain that might be caused from torsion building up from imbalanced walking are almost limitless. The patient may suffer from low back pain, neck pain, various types of leg pain, or almost anything. It might be beneficial to reassess the painful areas after the patient walks in his usual manner, and compare the pain when walking with a longer stride on the previously short side. If a treadmill is available, the location of tenderness can be monitored as the patient walks under either condition. With success in the procedure, limited or no pain will develop when the walking pattern is correct.

Filum Terminale Cephalad Lift Technique

The dura mater is firmly attached above to the foramen magnum, axis, and 3rd cervical vertebra.[90] By dissection observation, some indicate it is attached to the atlas.[15] Below it is attached firmly to the dorsum of the 1st coccygeal segment by the filum terminale. As the spine bends, the length of the intervertebral canal changes. Since the spinal cord and dura mater have a finite length,[6] there must be organization between the attachments of the dura superiorly and inferiorly. Goodheart[35] postulates that the coccyx is a "take-up mechanism" to maintain proper tension on the spinal cord. The filum terminale cephalad lift technique primarily relates to flexion and extension organization between the spinal column and the spinal cord. It must be pointed out, however, that the body functions in a spiral manner.[12] Illi[43] demonstrated that without spinal column rotation during forced flexion, excessive tension is put on the spinal cord.

The filum terminale cephalad lift technique appears to take tension off the dura mater, improving function. Treatment is directed to the coccyx and upper three cervical vertebrae and occiput. Need for this technique is determined after pelvic categories, sacral and vertebral subluxations, and the cranial-sacral primary respiratory mechanism are examined and treated, if necessary.

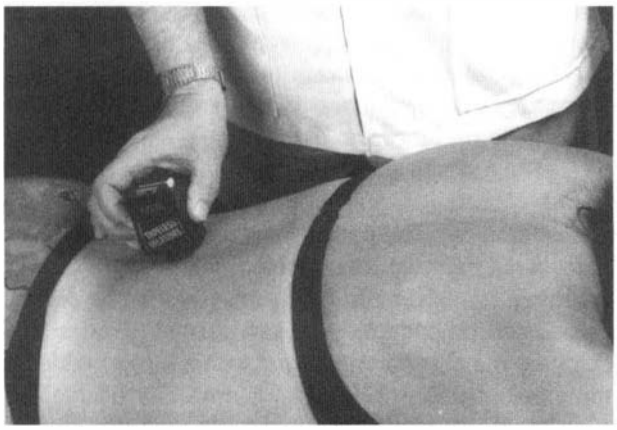
Examination

The first phase of examination is therapy localization to the coccyx and upper cervical vertebrae. Have the patient place his hand on the coccyx and test a strong indicator muscle for weakening; then repeat the procedure on the upper cervical vertebrae. There should be no weakening of a previously strong indicator muscle; if there is, look for some other factor that needs treatment first.

Indication to apply the filum terminale coccygeal cephalad lift technique is positive therapy localization at the coccyx when the patient applies an inferior traction, but no therapy localization without the traction. In addition, there will be positive therapy localization of the upper three cervical vertebrae when cephalad

traction is applied with the therapy localization. Again, there should be no positive therapy localization without the cephalad traction. If there is positive therapy localization without the traction, determine the reason and make correction before continuing with the examination. There may be positive findings with traction after the correction.

The coccygeal cephalad lift can be done in a standing, seated, or prone position. It is usually applied with the patient prone, which is easiest. In the position in which the patient is to be treated, measure the spinal length from the coccyx to the external occipital protuberance, using a rolling ruler. There should be a change in spinal length following treatment.

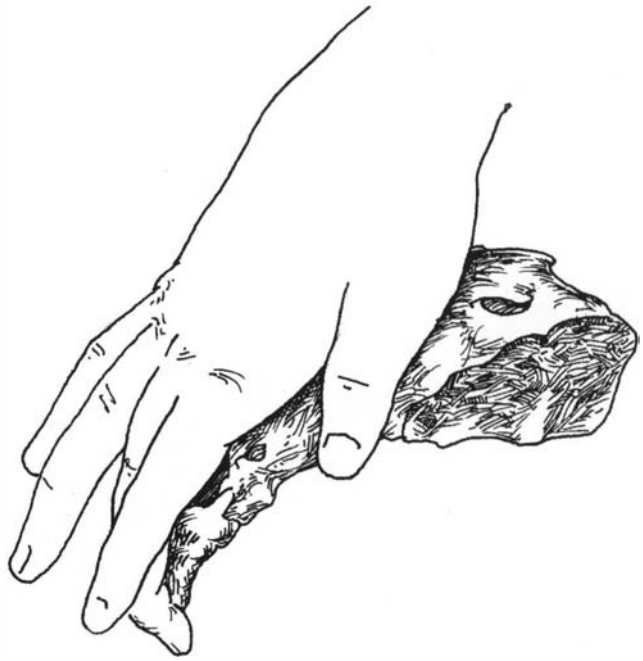


6—93. Measuring length of the spine from the tip of the coccyx to the external occipital protuberance with a rolling ruler.

Treatment

Treatment will be applied simultaneously to the coccyx, upper cervical vertebrae, and occiput. The occipital contact was modified by Goodheart[35] from the cranial osteopathic technique "CV-4,"[84] originally developed by Sutherland.[77] CV-4 refers to compression of the 4th ventricle. In the osteopathic CV-4 technique, the physician cradles the supine patient's occiput between the thenar eminences of his two hands. The contact is medial to the occipitomastoid sutures, which should not be contacted. During sphenobasilar extension, the occipital squama folds inward on itself. The physician's thenar eminences follow this movement with extension of the skull, and during flexion resist the widening of the occiput. This is continued until the cranial primary respiratory motion temporarily ceases, called the "still point."

To demonstrate the change that takes place from treating the still point, Goodheart ties three tennis balls together with tape. Two of the tennis balls are placed under the supine patient's occiput, simulating the thenar eminences of the physician's hands. The third tennis ball makes no contact; it is more for stabilization of the other balls to keep them from rolling. When the patient's occiput rests on the tennis balls, the internal rotation of the leg becomes balanced in dural tension evaluation.



6—94. The physician's finger wraps around the coccyx to make full contact. Pressure is primarily in a cephalad direction.

With the patent prone for treatment, the most critical contact point in the coccygeal cephalad lift technique is at the coccyx. The optimal contact and vector of pressure give maximum relief to tenderness in the cervical muscles. First evaluate the bilateral cervical muscles simultaneously to find the most tender area. It is probable that this tenderness is due to strain of the dural attachment in the cervical region. The coccyx is contacted with the physician's finger curving to match the curve of the coccyx so that full contact is made. The full contact is important. A digital contact with the tip of the finger does not produce as effective results. Press on the coccyx with the full-finger contact in a cephalad direction, with 12-14 lbs of force. Take care that the direction of pressure is cephalad and not anterior. Re-evaluate the tenderness in the cervical muscles. When the optimal vector is obtained, tenderness in the cervical muscles will be greatly reduced or absent. The optimal vector is usually almost directly cephalad, varying only 1-3°. In some instances, the optimal vector may be up to 10° away from midline. When the optimal vector has been reached, hold the heavy pressure; with the other hand contact C1, 2, and 3 with the middle, ring, and little fingers, respectively. Loosely contact the occiput with the index finger and thumb. Have the patient take a slow, deep inspiration and then exhale. As the patient exhales, gently squeeze the index finger and thumb together on the occiput. As the patient takes the next inhalation, maintain the pressure on the

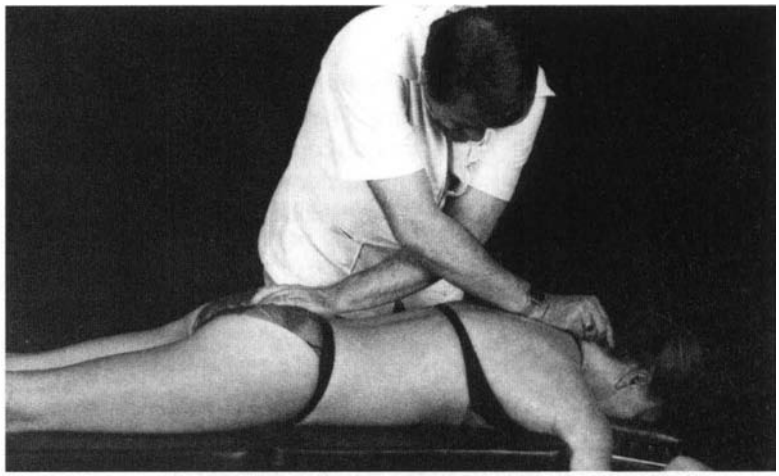
occiput while inferiorly moving the upper three cervical vertebrae. Repeat with four or five respirations while continuing to hold the cephalad lift on the coccyx throughout.

Following treatment there should be no tenderness in the cervical muscles, or it should be greatly reduced. The positive therapy localization with traction should be negative on the coccyx and upper cervical vertebrae.

Again measure the spine from the coccyx to the external occipital protuberance with the rolling ruler. There should be a change in the length of the spine. Usually it shortens about 1". In approximately 10% the spine lengthens by about 1", but the amount can be greater. If less than 1", evaluate the patient for spinal fixations that are often present. After correcting the fixations, again treat with the filum terminale cephalad lift technique, which will usually give better results.

Moving from prone to sitting or standing does not cause a loss of the spinal length change; however, if dural tension develops with walking, the incorrect long-step manner causes a loss of the length change. The corrected long-step gait, as indicated by dural tension evaluation, does not cause a loss of the spinal length change.

Routinely the cervical muscles are evaluated for reduction of tenderness to determine the vector of pressure on the coccyx. Other painful areas of the body can also be evaluated. For example, if an individual has a painful knee, spinal, or other area, one can monitor the localized pain with digital pressure while contacting the coccyx. Optimal pressure on the coccyx will often reduce that pain, as well as that in the cervical muscles.



6—95. Double-hand contact for filum terminale cephalad lift technique. Constant cephalad pressure on coccyx. Loose contact on the occiput as patient inhales, gently squeeze the occiput as he exhales. Maintain the pressure on the occiput with the next inhalation and move the upper three cervical vertebrae inferiorly. Repeat the process four or five times.

Cerebellum

The cerebellum is the largest part of the hindbrain and occupies most of the posterior cranial fossa. The tentorium cerebelli separates the cerebellum from the overlying cerebrum. The cerebellum has several unusual characteristics. It is the most oxygen-sensitive tissue in the nervous system, making it highly sensitive to minor shifts in the blood's ability to transport oxygen. Although the cerebellum is only one-tenth the volume of the brain, it contains one-half of the neurons. The cerebellum cannot initiate motor movement of the body, yet it is responsible for control of the muscles. To do this it receives afferent impulses from other parts of the brain and from the body mechanoreceptors. It monitors muscle function and makes corrective adjustments in motor activities to correspond with the intention of the motor signals elicited by the motor cortex and other parts of the brain. The extensive fiber connections of the cerebellum indicate that it may be able to influence almost any other part of the brain and regulate all muscle activity. According to Brodal,[7] "It may be essential for the full perfection of a number of bodily functions. The cerebellum is, however, not essential to life. In fact, individuals who are born without a cerebellum do not betray themselves in daily life by any obvious defects." How this adaptation takes place without a cerebellum is unknown, because destruction of portions of the cerebellum is readily recognized by muscle dysfunction.

The cerebellum instantaneously monitors the status of the body by comparing the information from the mechanoreceptors with what is intended by the motor system. When the afferent information does not correlate with the intention of the motor impulse, the cerebellum sends corrective signals into the motor system to increase or decrease the levels of activation of the specific muscles. Precise movement of the body is coordinated by the cerebellum. It receives afferent supply from the neuromuscular spindle cells, Golgi tendon organs, large tactile receptors of the skin, and the joint receptors. The most important afferent pathways from the periphery are the dorsal spinocerebellar tract, ventral spinocerebellar tract, and similar tracts from the

neck and facial regions. The spinocerebellar tracts can transmit impulses at velocities up to 120 m/sec, which is the most rapid conduction of any pathway in the central nervous system.

Cerebellar disease, failure of proper input from the mechanoreceptors, or disturbance of the afferent pathways can cause numerous clinical conditions.

1. Action tremor or intention tremor results from failure of cerebellar feedback to dampen the muscular movements. Normally the cerebellum prevents overshooting the intended movement by the cerebrum. Failure of the cerebellum in this action is eventually corrected by conscious centers of the cerebrum. The correction results in overshooting and again correction, followed by overshooting results in the tremor.
2. Dysmetria is the inability to stop motion at a specifically intended point. Dysmetria is much more pronounced in rapid movements than in slow ones.
3. Dysdiadochokinesia is the inability to perform rapidly alternating movements. This can be demonstrated by having a patient with cerebellar damage turn one hand upward and downward at a rapid rate. Failure in this action results in a series of jumbled movements.
4. Hypotonia causes moderate decrease in tone of the peripheral musculature on the side of lesion. After several months compensation takes place, and in some cases a hypertonic state may ultimately result.
5. Ataxic gait. To compensate for the staggering, unstable gait the patient walks with the feet spread apart. The instability may result in falling.
6. Dysphonia may be present, with slurred or explosive speech.

Belli[4] and Goodheart[37] have presented applied kinesiology methods to find and correct areas in which there is disturbed afferent supply from the periphery affecting the cerebellum's ability to maintain muscle tone. Of particular interest regarding postural balance and low back pain are the spinal extensor muscles. In the presence of weak spinal extensor muscles, have the patient tap the fingers of each hand together with eyes closed; this appears to concentrate cerebellum activity. The muscles will test strong while tapping if there is cerebellar involvement. Any body area that is dysfunctioning and providing limited or improper mechanoreceptor afferent supply to the cerebellum can be at fault. The dysfunction will most often be found in the upper cervical and thoracic areas.

The cerebellum depends on proper afferent supply from the periphery to monitor movement. The cervical spine and thoracic mechanoreptors are of particular importance in cerebellar control of muscle balance for postural control. Guyton and Hall[40] place the joint receptors of the neck among the most important proprioceptive information for maintenance of equilibrium. Wyke[91] describes types I and II mechanoreceptors and type IV nociceptors in the costovertebral and costotransverse joint capsules and considers their role in regulation of posture.

Involvement of the thorax is most often the result of abdominal muscle weakness. If the abdominal muscles are weak in the clear, correct the dysfunction using the five factors of the IVF. The most common cause of weakness in the clear is sagittal suture jamming, described in Chapter 9.

A type of abdominal muscle weakness recently found occurs when the abdominal muscles are strong in the clear but weaken after repeated muscle activation, patient induced (RMAPI), or after the muscles are stretched.[37] Even though the muscles test strong in the clear it appears that the body recognizes the muscle dysfunction, perhaps within the cerebellum, and overcompensates by contracting the muscles, pulling the thoracic cage anteriorly and inferiorly. In an effort to regain structural balance there is repositioning of the cervical spine and head, with tension centered in the suboccipital area. Cervical range of motion will be restricted, especially when tested passively as described in the cervical compaction technique in Chapter 3.

A quick method to determine if the abdominal muscles are the cause of thoracic and cervical restriction is to test active and passive cervical range of motion while the patient holds his arm over his head. This stretches the thoracic cage up on that side, and the cervical range of motion will improve if the abdominal muscles are responsible for the limitation. The involvement may be bilateral or unilateral. If there is improved cervical range of motion, test the abdominal muscles for the type of weakness. The muscle may weaken to RMAPI requiring origin/insertion technique. If due to muscle stretch reaction treat with fascial release, B_{12}, or folic acid as appropriate. If the weakness is from maximum muscle contraction, strain/counterstrain technique is appropriate with glycine or fatty acids as needed.

If the abdominal weakness is unilateral there will probably be an anterior atlas on that side. If bilateral, there will probably be bilateral atlas anteriority. Test the atlas with the primary atlas technique (PAT) and correct, if necessary.

After correcting the abdominal muscles there will often be an increase in cervical range of motion both actively and passively, especially in flexion combined with rotation. If there is still limited range of motion, examine and treat with cervical compaction technique. Failure to obtain full active and passive range of motion following these corrections indicates probable quadratus lumborum involvement.

To determine quadratus lumborum involvement have the patient lean laterally 10–15° away from the

side of limited cervical rotation, stretching the ipsilateral quadratus lumborum. While in this position, cervical range of motion will improve if the quadratus lumborum is involved. This is applicable to range of motion in flexion, lateral flexion, and extension. Most often the cervical range-of-motion limitation is in flexion combined with rotation.

The quadratus lumborum will usually be weak only after RMAPI or muscle stretch response; treat accordingly. In the case of RMAPI weakness that requires origin/insertion technique, recognize that the quadratus lumborum may have two layers of muscle fibers.[19,90] The muscle originates from aponeurotic iliolumbar ligament fibers and the internal lip of the iliac crest. It inserts into the medial half of the lower border of the 12th rib and by four small tendons to the apices of the upper four lumbar vertebrae, and sometimes to the transverse process or body of the 12th thoracic vertebra. If a superficial layer is present, it originates from the upper borders of the lower three or four lumbar transverse processes to the lower margin of the anterior surface of the 12th rib.

Correcting the thoracic and cervical areas will usually eliminate the cerebellar associated weakness identified by finger-tapping with the eyes closed. If there is still strengthening of a weak muscle by the patient tapping his fingers together, evaluate for mechanoreceptor deafferentation throughout the spine and rest of the body.

CHAPTER 6 — REFERENCES

1. Abrahams, V.C., "Neck muscle proprioception and motor control," in *Proprioception, Posture and Emotion*, ed D. Garlick (Kensington, NSW: Univ New South Wales, 1982).
2. Beardall, A.G., "The cloacal synchronization technic." Proceedings of Winter Meeting, ICAK, San Diego, 1977.
3. Beardall, A.G., "Additional gait tests," *Chiro Econ*, Vol 19, No 5 (Mar/Apr 1977).
4. Belli, R., "Cerebellum dysfunction and low back pain — a neurological model," *ICAK-USA News Update* (Apr 1993).
5. Blaich, R.M., "Ligament stretch-adrenal stress syndrome related to knee disorders." Proceedings of Summer Meeting, ICAK, Detroit, 1980.
6. Breig, A., *Adverse Mechanical Tension in the Central Nervous System* (New York: John Wiley & Sons, 1978).
7. Brodal, A., *Neurological Anatomy in Relation to Clinical Medicine*, 2nd ed (New York: Oxford Univ Press, 1969).
8. Bronston, G.J., "The M.C.R. Test," *J Current Podiatric Med* (Jan 1987).
9. Cohen, L.A., "Role of eye and neck proprioceptive mechanisms in body orientation and motor coordination," *J Neurophysiol*, Vol 24 (Jan 1961).
10. Cyriax, J., "Fibrositis," *Br Med J*, Vol 2 (Jul 31, 1948).
11. Cyriax, J., "Dural pain," *Lancet*, Vol 1 (Apr 29, 1978).
12. Dart, R.A., "Voluntary musculature in the human body — the double-spiral arrangement," *Brit J Phys Med*, Vol 13 (1950).
13. Delacato, C.H., *The Diagnosis and Treatment of Speech and Reading Problems* (Springfield, IL: Charles C. Thomas Publisher, 1963).
14. Delacato, C.H., *Neurological Organization and Reading* (Springfield, IL: Charles C. Thomas Publisher, 1966).
15. Denton, D.G., *Craniopathy and Dentistry* (Los Angeles: privately published, 1979).
16. Deutsch, G., "An adrenal screen test to determine adrenal insufficiency." Proceedings of Winter Meeting, ICAK, San Juan, Puerto Rico, 1975.
17. Ducroquet, R., J. Ducroquet, & P. Ducroquet, *Walking and Limping — A Study of Normal and Pathological Walking* (Philadelphia: J.B. Lippincott Co, 1968).
18. Durlacher, J.V., "Injury susceptibility in hypoadrenia." Proceedings of Summer Meeting, ICAK, Detroit, 1977.
19. Dvorak, J., & V. Dvorak, *Manual Medicine Diagnostics* (New York: Thieme-Stratton, Inc, 1984).
20. Foredom Electric Company, The, 16 Stony Hill Road, Bethel, CT 06801.
21. Frymann, V.M., "The core-link and the three diaphragms," *Acad Appl Osteo Yr Bk* (1968).
22. Frymann, V.M., "A study of the rhythmic motions of the living cranium," *JAOA*, Vol 70, No 9 (May 1971).
23. Fulford, R.C., *Touch of Life* (New York: Simon & Schuster, 1996).
24. Garrick, J.G., "Frequency of injury, mechanism of injury, and epidemiology of ankle sprains," *Am J Sports Med*, Vol 5, No 6 (1977).
25. Goodheart, G.J., Jr., "Cross-pattern crawling and muscle spasm," *Chiro Econ*, Vol 11, No 5 (Mar/Apr 1969).
26. Goodheart, G.J., Jr., "Gait and associated problems," *Chiro Econ*, Vol 18, No 1 (Jul/Aug 1975).
27. Goodheart, G.J., Jr., *Applied Kinesiology 1976 Workshop Procedure Manual*, 12th ed (Detroit: privately published, 1976).
28. Goodheart, G.J., Jr., *Applied Kinesiology 1977 Workshop Procedure Manual*, 13th ed (Detroit: privately published, 1977).
29. Goodheart, G.J., Jr., *Applied Kinesiology 1978 Workshop Procedure Manual*, 14th ed (Detroit: privately published, 1978).
30. Goodheart, G.J., Jr., *Applied Kinesiology 1979 Workshop Procedure Manual*, 15th ed (Detroit: privately published, 1979).
31. Goodheart, G.J., Jr., *Applied Kinesiology 1980 Workshop Procedure Manual*, Vol 1, 16th ed (Detroit: privately published, 1980).
32. Goodheart, G.J., Jr., *Applied Kinesiology 1982 Workshop Procedure Manual*, 18th ed (Detroit: privately published, 1982).
33. Goodheart, G.J., Jr., *Applied Kinesiology 1983 Workshop Procedure Manual*, 19th ed (Detroit: privately published, 1983).
34. Goodheart, G.J., Jr., *Applied Kinesiology 1984 Workshop Procedure Manual*, 20th ed (Detroit: privately published, 1984).
35. Goodheart, G.J., Jr., *Applied Kinesiology 1985 Workshop Procedure Manual*, 21st ed (Detroit: privately published, 1985).
36. Goodheart, G.J., Jr., *Applied Kinesiology 1990/91 Workshop Procedure Manual*, 26th ed (Grosse Pointe Woods: privately published, 1991).
37. Goodheart, G.J., Jr., *Applied Kinesiology 1994/95 Workshop Procedure Manual*, 30th ed (Grosse Pointe Woods: privately published, 1995).
38. Goodheart, G.J., Jr., *Applied Kinesiology 1997-98 Workshop Procedural Manual* (Grosse Pointe Woods, MI: privately published, 1998).
39. Gowitzke, B.A., & M. Milner, *Understanding the Scientific Bases of Human Movement*, 2nd ed (Baltimore: Williams & Wilkins, 1980).
40. Guyton, A.C., & J.E. Hall, *Textbook of Medical Physiology*, 9th ed (Philadelphia: W.B. Saunders Co, 1996).
41. Hassin, G.B., "The cerebrospinal fluid pathways," *J Neuropathol Exp Neurol*, Vol 6 (Apr 1947).
42. Hellebrandt, F.A., M. Schade, & M.L. Carns, "Methods of evoking the tonic neck reflexes in normal human subjects," *Am J Phys Med*, Vol 41 (1962).
43. Illi, F.W., *The Vertebral Column — Life-Line of the Body* (Chicago: National Coll of Chiro, 1951).
44. I.M.P.A.C., Inc., P.O. Box 535, Salem, OR 97308-0535.

45. Jirout, M.J., "The dynamics of the craniocervical junction on lateral inclination of the head and neck," *ChiroEcon*, Vol 26, No 4 (Jan/Feb 1984).
46. Jones, L.H., *Strain and Counterstrain* (Newark, OH: Amer Acad of Osteopathy, 1981).
47. Kiwic, G., K. Slusarczyk, & R. Slusarczyk, "The central nervous system and the lymphatic system. Lymphatic drainage of the cerebrospinal fluid (Abstract)," *Neurol Neurochir Pol*, Vol 32, No 3 (May/Jun 1998).
48. Klepper, G.N., "The relationship of aerobic muscle fatigue findings to laboratory measurements of iron," *A.K. Review*, Vol 3, No 1 (Summer 1992).
49. Korr, I.M., "Proprioceptors and somatic dysfunction," *JAOA*, Vol 74, No 7 (Mar 1975).
50. Maffetone, P.B., "Diagnosing aerobic and anaerobic excess and deficiency." Proceedings of Summer Meeting, ICAK, Washington, DC, 1987.
51. Maffetone, P., *Everyone is an Athlete* (New York: David Barmore Publisher, 1989).
52. May, W.B., "Little-known dental ailment is causing millions to suffer needlessly from an amazing variety of chronic health problems, Part I," *Healthview Newslet*, Vol 1, No 7 (1976).
53. May, W.B., "A little-known dental ailment is causing millions to suffer needlessly from an amazing variety of chronic physical problems, Part II," *Healthview Newslet*, Vol 1, No 8 (1976).
54. May, W.B., DDS, personal communication.
55. McCouch, G.P., I.D. Deering, & T.H. Ling, "Location of receptors for tonic neck reflexes," *J Neurophysiol*, Vol 14 (May 1951).
56. Mennell, J.M., "'Spray-and-stretch' treatment for myofascial pain," *Hosp Physician* (Dec 1973).
57. Mennell, J.M., "The therapeutic use of cold," *JAOA*, Vol 74 (Aug 1975).
58. Miller, S., & P.D. Scott, "The spinal locomotor generator," *Exp Brain Res*, Vol 30 (1977).
59. Munro, R.R., "Electromyography of the masseter and anterior temporalis muscles in subjects with potential temporomandibular joint dysfunction," *Aust Dent J*, Vol 17, No 3 (Jun 1972).
60. Nordenström, B.E.W., "An electrophysiologic view of acupuncture: Role of capacitive and closed circuit currents and their clinical effects in the treatment," *Am J Acupunc*, Vol 17, No 2 (Jun 1989).
61. Nordenström, B.E.W., "I. Neurovascular activation requires conduction through vessels," *Physiol Chem Phys & Med NMR*, Vol 21, No 4 (1989).
62. Nordenström, B.E.W., "Hand movements above the unshielded tail of a shielded rat induces differences in voltage inside the animal," *Am J Acupunc*, Vol 20, No 2 (1992).
63. Nordenström, B.E.W., "Impact of biologically closed electric circuits (BCEC) on structure and function," *Integrative Physiolog Behavioral Sci*, Vol 27, No 4 (Oct-Dec 1992).
64. Page, L.E., "Structural factors in headache," *Am Acad Osteo Yr Bk* (1951).
65. Page, L.E., "The role of the fascae in the maintenance of structural integrity," *Acad Appl Osteo Yr Bk* (1952).
66. Pearson, K., "The control of walking," *Sci Am*, Vol 235, No 6 (Dec 1976).
67. Rolf, I.P., *Rolfing: The Integration of Human Structures* (Santa Monica, CA: Dennis-Landman Publishers, 1977).
68. Rydevik, B. et al., "Diffusion from the cerebrospinal fluid as a nutritional pathway for spinal nerve roots," *Acta Physiol Scand*, Vol 138, No 2 (Feb 1990).
69. Schmitt, W.H., Jr., "The ligament stretch-adrenal stress syndrome summary of clinical investigation." Proceedings of Summer Meeting, ICAK, Detroit, 1977.
70. Schmitt, W.H., Jr., "Injury recall technique: Dealing with the history of injury and trauma." Proceedings of Summer Meeting, ICAK, Dearborn, MI, 1990.
71. Schmitt, W.H., Jr., "A neurological rationale for injury recall technique." Proceedings of Summer Meeting, ICAK-USA, New Orleans, 1999.
72. Selye, H., *Stress Without Distress* (New York: New American Library, 1974).
73. Simons, D.G., "Electrogenic nature of palpable bands and 'jump sign' associated with myofascial trigger points," in *Advances in Pain Research and Therapy*, Vol 1, ed J.J. Bonica & D.G. Albe-Fessard (New York: Raven Press, 1976).
74. Somberg, H.M., "The relation of the spinal subarachnoid and perineurial spaces," *J Neuropathol*, Vol 6 (Apr 1947).
75. Steer, J.C., & F.D. Horney, "Evidence for passage of cerebrospinal fluid along spinal nerves," *Can Med Assoc J*, Vol 98, No 2 (Jan 13, 1968).
76. Stejskal, L., "Postural reflexes in man," *Am J Phys Med*, Vol 58, No 1 (1979).
77. Sutherland, W.G., *The Cranial Bowl* (Mankato, MN: privately published, 1939).
78. Travell, J., "Referred pain from skeletal muscle," *NY State J Med*, Vol 55 (Jan 1, 1955).
79. Travell, J., "Mechanical headache," *Headache*, Vol 7, No 1 (Apr 1967).
80. Travell, J., "Myofascial trigger points: Clinical view," in *Advances in Pain Research and Therapy*, Vol 1, ed J.J. Bonica & D.G. Albe-Fessard (New York: Raven Press, 1976).
81. Travell, J., & S.H. Rinzler, "The myofascial genesis of pain," *Postgrad Med*, Vol II, No 5 (May 1952).
82. Travell, J.G., & D.G. Simons, *Myofascial Pain and Dysfunction — The Trigger Point Manual* (Baltimore: Williams & Wilkins, 1983).
83. Travell, J.G., & D.G. Simons, *Myofascial Pain and Dysfunction — The Trigger Point Manual. The Lower Extremities*, Vol 2 (Baltimore: Williams & Wilkins, 1992).
84. Upledger, J., & J. Vredevoogd, *Craniosacral Therapy* (Chicago: Eastland Press, 1983).
85. Walther, D.S., "Upper extremity peripheral nerve entrapment," in *1982 Selected Papers of the International College of Applied Kinesiology*, ed W.H. Schmitt, Jr. (Detroit, MI: ICAK, 1982).
86. Walther, D.S., *Applied Kinesiology, Volume II — Head, Neck, and Jaw Pain and Dysfunction — The Stomatognathic System* (Pueblo, CO: Systems DC, 1983).
87. Watkins, R.J., "Anthropology in reflex technics," *J National Chiro Assoc* (Aug 1948).
88. Watkins, R.J., "Perianal postural reflex technic," *Chiro Econ*, Vol 17, No 6 (May/Jun 1975).
89. Webber, T.D., "Diagnosis and modification of headache and shoulder-arm-hand syndrome," *JAOA*, Vol 72 (Mar 1973).
90. Williams, P.L., & R. Warwick, eds, *Gray's Anatomy*, 36th Br ed (Philadelphia: W.B. Saunders Co, 1980).
91. Wyke, B., "Morphological and functional features of the innervation of the costovertebral joints," *Folia Morphologica*, Vol XXIII, No 4 (1975).
92. Yates, P.O., "The passage of cerebrospinal fluid along spinal nerves," *Developmental Med & Child Neurol*, Vol 10 (1968).

Chapter 7

Meridian Therapy

7—1. Numerous ancient Chinese illustrations give evidence that spinal manipulation, nutrition, herbs, and acupuncture constituted the health care of the Chinese.

Introduction

Meridian therapy is thousands of years old, but it has only recently become well-known in North America. In the context of this discussion, the term "meridian therapy" is more appropriate for describing what is commonly called acupuncture. The term "acupuncture" is derived from the Latin "acus" (needle) plus the English "puncture,"[37] indicating that a needle puncture is necessary in the application of the treatment system. Actually, classic work with the meridian system by the Chinese and others uses various methods of stimulating the points on the meridians, including stimulation with the finger, fish bones, sharpened bamboo sticks, and moxibustion (the burning of an herb). In addition to these and other methods of stimulating acupuncture points, structural manipulation, nutrition, and herbology are used. This text will use the term "meridian therapy" for acupuncture treatment. The points along the meridian will be called "acupoints" or "acupuncture points," but it should be understood that the stimulation is not necessarily by needle.

There is a considerable difference in the way acupuncture is practiced in various countries. In Korean acupuncture, many needles are used in comparison with other forms. There is a distinct difference between Chinese and Japanese acupuncture. The principles presented here will primarily follow the classic Chinese philosophies,[30,34] with the exception of the applied kinesiology additions.

It is impossible to know how knowledge of the meridians was originally established. It seems probable that some of the basic knowledge was developed by those with higher sense perception, charting the meridians by the auras they saw. Although the visualization of auras is not scientifically understood, it has nevertheless been established as a valid phenomenon.[17]

Today, thousands of years after the establishment of acupuncture as a method of treatment, its laws and principles are better understood on a scientific basis. Sophisticated electronic instruments yield information about the meridian system heretofore unknown. As we understand more and more about the meridian system, additional methods of balancing it, with or without the use of needles, become apparent.

Understanding the body language of the meridian system increases one's ability to understand dysfunction that causes symptoms when there is no pathologic process to diagnose. A physician unaware of the meridian system misses much body language that gives clues to understanding health problems.[21] Applied kinesiology adds to the ability to read this body language.

Meridian therapy was introduced into applied kinesiology by Goodheart in 1966, before there was much interest in the subject in the United States.[8] There have been contributions by many people to the applied kinesiology application of meridian therapy as its use has increased. The primary contribution applied kinesiology has made to meridian therapy is in diagnosis and understanding why there may be imbalance of energy in the system.

When working with the meridian system, it is necessary to understand the balance of energy before attempting to change it. There are numerous books giving formulae for treating different conditions with acupuncture[6,20,33,40]; in this writer's opinion, they should be correlated with a diagnostic system. Failure to know how the energy balance is changed is a potential cause of iatrogenic conditions. There are two flaws in the use of formulae: 1) the individuality of each patient, and 2) a diagnosis must be made, which is the weakest link in the health care delivery system. The "cookbook" approach has been developed primarily by observing what is effective in improving a patient's condition, then listing the points for use on other individuals. What works for one patient may be wrong for another patient with apparently the same symptoms. The cookbook approach primarily treats symptoms; those in natural health care recognize this as a less than desirable method.

A primary aspect of meridian therapy is the concept that energy goes through twelve bilateral meridians, and the energy should be balanced within the system. This energy, called Chi (or Qi, both pronounced "chee"), is electromagnetic. As will be explained later, applied kinesiology helps one understand the nature of this energy.

The discussion presented here will introduce the meridian system and give a brief overview of diagnostic methods, both classic and those more recently developed in the Western world. Specifically, this discussion will add applied kinesiology contributions to the diagnosis and treatment of imbalance within the meridian system. A complete discussion of the meridian system and its application in Eastern and Western cultures is a massive study in itself. Philosophical and religious doctrines are interwoven in the complete understanding of classic acupuncture. Only a very small portion will be presented here. The student is encouraged to make a more thorough study, which will greatly increase the ability to read body language.

Evaluation of energy balance in the meridian system and actual treatment to the meridians are the only things about the meridian system that are new to chiropractic. Chiropractors have been affecting the energy level in the meridians since the profession first began in 1895. As we proceed with our study, we will find many ways in which chiropractic manipulation of the spine and extremities influences the meridian system. Many of the reflex points used in various chiropractic techniques coincide with acupuncture points.

As the Western world has begun to recognize the value of Oriental philosophies and treatment of health, so Orientals are picking up techniques from the Western world to add to their approach. Feldman and Yamamoto[7] state that "...a substantial chiropractic and orthopedic

approach to bone and muscle manipulation has developed in the Orient. This approach complements and further extends the treatment of the meridians."

There are many parallels between meridian therapy and chiropractic. The practitioners of meridian therapy have been using chiropractic principles, and chiropractic has been affecting the meridians; the philosophies of health parallel as well. Meridian therapy works to balance and release the "life force" within the body. This sounds like a chiropractic principle that has been put forth by some, doesn't it? Both are dedicated to the prevention of disease, rather than treating the symptoms of pathology. Both use natural approaches to enable the body to heal itself, rather than trying to control body function. This writer once heard a medical doctor who had adopted acupuncture into his practice state that he is now practicing constructive rather than destructive medicine.

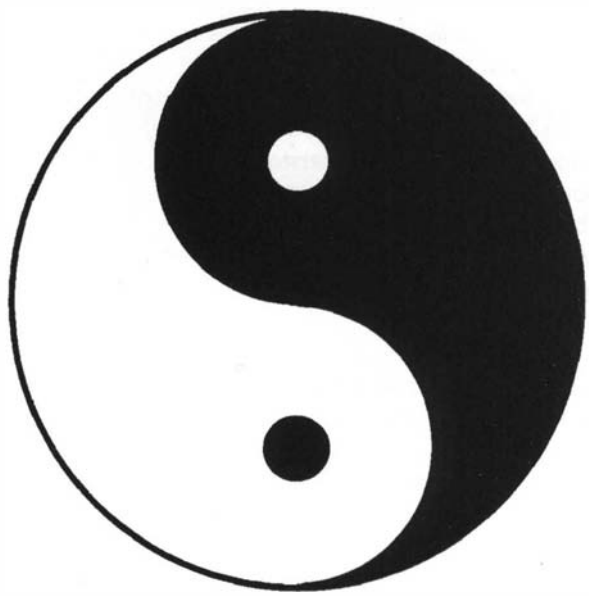
The meridian system is the fifth of the five factors of the IVF. Goodheart[9] found that muscles which tested weak were sometimes associated with imbalance of energy within the meridian system. The meridian-muscle association closely followed the previously developed muscle-organ/gland association.[35] For example, the subscapularis previously associated with the heart is influenced by the heart meridian. The tensor fascia lata, associated with the large intestine, is influenced by the large intestine meridian. The other muscle-organ/gland associations mostly relate to the expected meridians.[9]

Another close association of the meridian system with chiropractic is the associated points along the spine on the bladder meridian. There tends to be an active associated point adjacent to a subluxated vertebra or, conversely, a subluxation adjacent to an active associated point. The age-old question of which came first, the chicken or the egg, seems applicable; however, when one becomes knowledgeable about meridian imbalance, it is usually clinically evident which is primary. Subluxations tend to recur when the meridian system remains imbalanced, and balancing of the system is not as effective if subluxations are not corrected. Subluxations of the vertebrae are not the only type influencing the meridian system. Often there will be a blockage of Chi where a meridian crosses over an extremity subluxation, such as in the foot.

Yin and Yang

In Chinese culture, everything in the universe is classified as predominantly Yin or Yang. These terms are expressions of opposites throughout the universe and signify the fundamental duality in the universe. Although opposites they are ultimately unified, because whether classified as Yin or Yang there is always some of the opposite present. The Taiji, or great polarity, has been adopted by many as the symbol of meridian therapy. It is divided in half, with black indicating Yin and white, Yang. The two colors swirl into each other and each contains a portion of the other, indicating the presence of Yang in Yin and Yin in Yang. An example is male and female. The male is Yang, yet he contains some Yin or female hormones, as females — who are Yin — contain some male hormones.

Oriental philosophy teaches that there must be a balance between the forces of Yin and Yang for health to exist. Balance between the forces is always changing with time, weather, seasons, and the other rhythms of nature. The meridians of the body are divided equally between Yin and Yang.



7—2. Taiji.

YIN	YANG
In Nature	
Night	Day
Cloudy day	Clear day
Autumn	Spring
Winter	Summer
North	South
West	East
Lower	Upper
Interior	Exterior
Cold	Hot
Water	Fire
Dark	Light
Moon	Sun

YIN	YANG
In The Body	
Body interior	Body surface
Chest/abdomen	Spine/back
Female	Male
Dirty fluid	Clean fluid
Cloudy fluid	Clear fluid
Solid	Hollow
Inner	Outer
Plain	Splendorous
Soft	Hard
Parasympathetic	Sympathetic
Right	Left

YIN	YANG
In Disease	
Chronic	Acute
Non-active	Virulent
Weak	Powerful
Decaying	Flourishing
Feels cold	Feels hot
Hypothermia	Fever
Moist	Dry
Retiring	Advancing
Lingering	Hasty
Water	Fire
Soft	Hard
Empty	Full

7—3.

Meridians

There are twelve major bilateral meridians (sometimes called channels), each with a beginning and ending location and acu-points along its course. Acu-points are locations of lowered electrical resistance and can be located with instruments that measure resistance. Meridian charts have lines connecting the meridian points. The exact physiological mechanism of connection between points is unknown. At one time it was reported that the meridians had been traced by injecting radioisotopes; however, the report was false. There is conjecture that the meridians may be connected by an electromagnetic harmony between them. An analogy of the connection between a radio transmitter and receiver may be applicable.

Meridians affect what they are named after and where they go. The meridian points are numbered from one to the end point, in the direction of energy flow. Chi travels from the end of one meridian to the beginning of the next.

The meridians are usually referred to by abbreviations, which vary among authors. The abbreviations used here will be as follows:

LU = LUng	BL = BLadder
LI = Large Intestine	KI = KIdney
ST = STomach	CX = Circulation seX
SP = SPleen	TH = Triple Heater
HT = HearT	GB = GallBladder
SI = Small Intestine	LV = LiVer

Muscle/Meridian Association

LUNG	Deltoids (middle, posterior, and anterior), serratus anticus, levator scapula, and coracobrachialis. Provisional for flexor pollicis longus and brevis
LARGE INTESTINE	Hamstrings, tensor fascia lata, and quadratus lumborum
STOMACH	Pectoralis major (clavicular division), biceps brachii, brachialis, brachioradialis, supinator, pronator teres, pronator quadratus, opponens pollicis, adductor pollicis, opponens digiti minimi, sternocleidomastoid, neck extensors, and medial neck flexors
SPLEEN	Lower trapezius, middle trapezius, latissimus dorsi, triceps brachii, and anconeus
HEART	Subscapularis
SMALL INTESTINE	Quadriceps, abdominals, flexor digiti minimi brevis
BLADDER	Tibialis anterior, peroneus tertius, peroneus longus and brevis, extensor hallucis longus and brevis, and sacrospinalis
KIDNEY	Psoas, iliacus, and upper trapzeius
CIRCULATION SEX	Sartorius, gracilis, adductors, gluteus maximus, gluteus medius and minimus, piriformis, gastrocnemius, soleus, tibialis posterior, flexor hallucis brevis, and flexor hallucis longus
TRIPLE HEATER	Infraspinatus, teres minor, occasionally sartorius and gracilis
GALLBLADDER	Popliteus
LIVER	Pectoralis major (sternal division), rhomboids
CONCEPTION VESSEL	Supraspinatus
GOVERNING VESSEL	Teres major

As more experience has been gained in applied kinesiology work with the meridian system, some of the muscle/meridian associations have changed. The changes and provisional associations are primarily for muscles that are not usually treated with meridian therapy.

7—4.

The numbered acu-points are then associated with their meridians by the abbreviation, e.g., ST 36, LU 1, and so on.

Each meridian is classified as Yin or Yang. One will note an association with the characteristics of Yin and Yang previously listed. Yin meridians are associated with solid organs, Yang with hollow organs. This is quite clear in the Yang meridians, as the gallbladder, small and large intestines, stomach, and bladder are hollow, and it is also quite clear in the Yin meridians — liver, spleen, lung, and kidney. One may think of the heart, which is Yin, as a hollow organ because of its chambers, but its muscle mass makes it solid. The triple heater is defined as three fires — respiration, digestion, and genital/urinary; the association made as fire is Yang. The circulation sex meridian is Yin; it is associated for easy memory with the female's more complex hormonal and reproductive system. The Yin meridians all begin or end on the chest, and the Yang meridians all begin or end on the head.

Circulation of Energy

Chi flows over a meridian in only one direction. The beginning of the meridian is acu-point number one, and the points are progressively numbered to the final point. When the arms are above the head, all meridians that flow up are Yin, and those flowing down are Yang.

The direction of flow from a low to a high number can be observed with applied kinesiology manual muscle testing. If one passes his hand over the course of a meridian opposite the direction of energy flow several times, an associated muscle will temporarily test weak. For example, the lung meridian begins on the anterior portion of the chest close to the shoulder, and ends on the thumb. The experiment is begun as the examiner places his hand close to the subject's thumb where the lung meridian ends (see lung meridian chart on page 245). He then follows the course of the meridian to its beginning, withdraws his hand from the body, and repeats the action several times. In most cases the deltoid muscle, associated with the lung, will temporarily test weak. The movement of the hand against the meridian flow appears to disrupt the electromagnetic field of the meridian. All individuals will not test weak following this procedure. Those with a high energy level appear to maintain energy balance, in spite of the disrupting force.

Following the meridian's path with the examiner's hand in this manner is called "running the meridian." When an individual's muscle has been weakened in this manner, it will return to normal in a relatively short time, depending on the subject's general energy level. When the return to normal is slow, it can quickly be normalized if the examiner runs the meridian from beginning to end. Running the meridian in the direction of energy flow is sometimes done to enhance meridian function.

Energy enters the system at the lung meridian, flowing down the arm to where it ends on the thumb. Energy from the lung meridian goes to the large intestine meridian, beginning on the index finger and flowing up the arm and ending on the head. The energy from the large intestine meridian then goes to the stomach meridian, which proceeds down the front of the body and down the leg, ending on the foot. The energy then goes to the spleen meridian, which travels up the leg. The energy continues to travel from meridian to meridian until it has passed through all twelve. There are short connecting meridians or channels between the end of one meridian and the beginning of the next. The connection is not always from the last acu-point to the first one of the next meridian. There are also connecting channels from some meridians to specific acu-points. Some of these are indicated on the accompanying charts.

Each meridian has a two-hour peak energy time, called the horary period, making a twenty-four-hour circulation of energy. Twelve hours after the horary period the meridian has its lowest energy level. The energy travels through a repetitive pattern of two Yin meridians, two Yang meridians, two Yin meridians, and so on. In the twelve-hour cycle, each Yin meridian has as its opposite a Yang meridian, and each Yang meridian is opposed by a Yin meridian.

The midday/midnight effect refers to the opposing relationship of meridians on the twenty-four-hour circulation of energy. For example, the lung meridian has its horary period (highest energy) from 3:00-5:00 a.m. Since the bladder meridian is twelve hours opposite the lung, it has its lowest energy from 3:00-5:00 a.m.

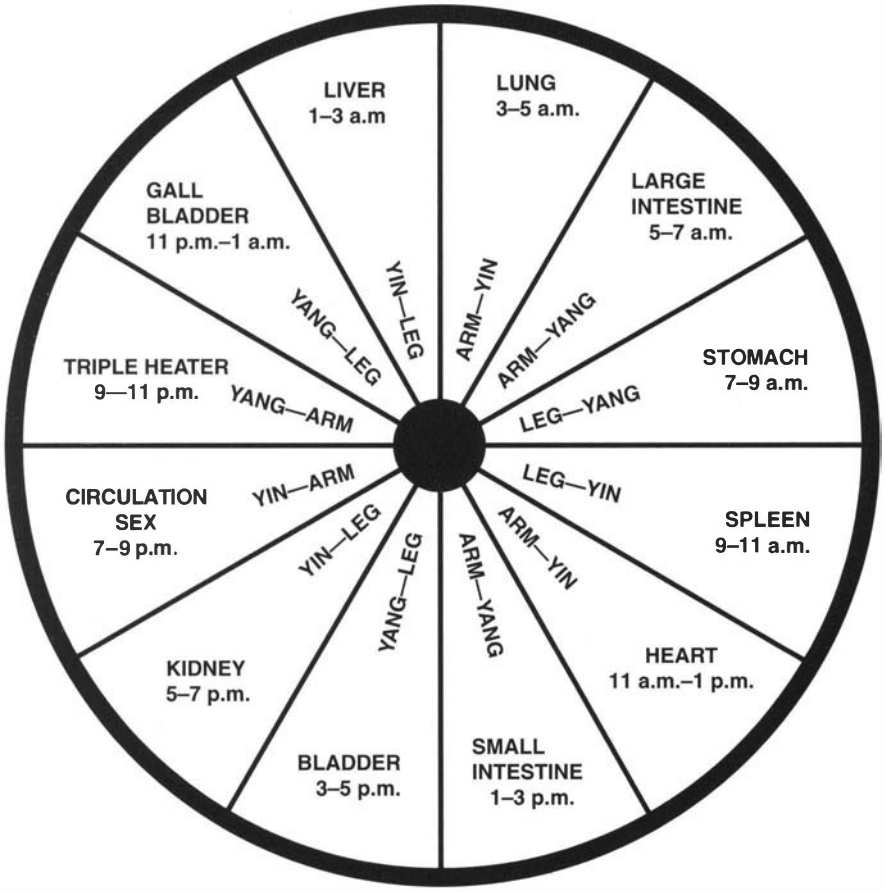
The twenty-four-hour circulation of energy can be used as a diagnostic tool. If a patient consistently awakens with a headache between 3:00 a.m. and 5:00 a.m., one would think of excessive energy in the lung meridian or a deficiency in its twelve-hour opposite, the bladder meridian.

There are two important midline meridians that course up the front and back of the body. The meridian in front is the conception vessel, and the one in back is the governing vessel.

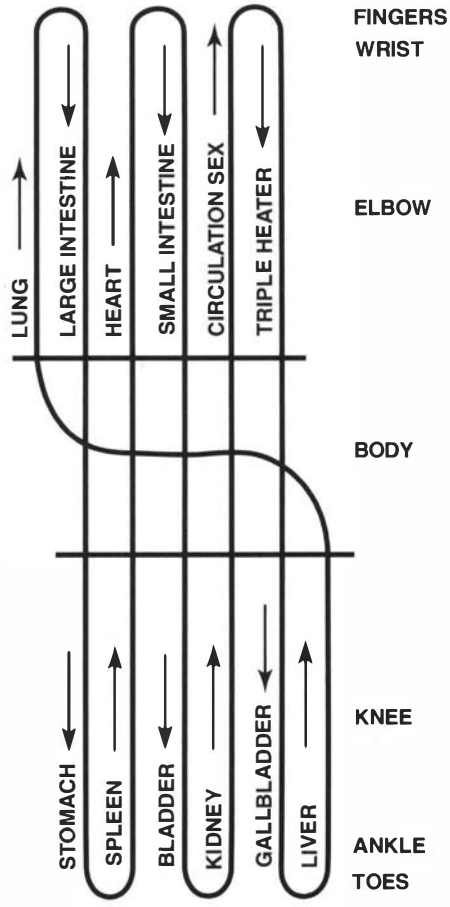
There are hundreds of meridians described in the various forms of acupuncture. This book will deal primarily with the twelve bilateral meridians, some connecting

meridians or channels that connect the energy from meridian to meridian or specific acu-points, the governing vessel, and the conception vessel.

Prior to the meridian charts is an introduction to the major types of acu-points. Accompanying each chart is information regarding classification of the meridian, location of acu-points, and indications for point usage. Some of the information relates to classic point usage, e.g., HT 7 for emotional conditions. This is not to be construed as a "cookbook" approach to meridian therapy. Use the diagnostic methods of applied kinesiology and/or classic meridian therapy to determine if points should be stimulated.



7—5. Chi (energy) enters the meridian system at the lung meridian. The lung meridian's highest energy is at 3:00-5:00 a.m. The high energy moves around the meridians in a twenty-four-hour circulation pattern.



7—6. Chi flows in one direction: down an arm, up the arm, through the body, down a leg, up the leg, continuing until through all twelve meridians.

Types of Meridian Points

Each meridian has numerous points which, when stimulated, react in various ways. Nine points will be considered here: alarm, associated, entry/exit, horary, hsi, luo, sedation, tonification, and source. In addition, each meridian has five command points relating with the five-element law, which will be discussed later. All of these points except two are located between the elbows and fingertips, or between the knees and toe tips. The two exceptions are TH 10 and ST 34.

Occasionally authorities will differ on acu-point location, the numbering of points, and what a specific point represents. Applied kinesiology helps determine where the acu-points are by therapy localization and the effectiveness of stimulating the points. In preparing this text many authorities were reviewed,[16,19,28,29,42] and the most common description and location consistent with AK findings are presented here.

An acu-point usually has only one function, but occasionally it serves more than one purpose, e.g., it may be both a sedation point and a source point. A more comprehensive discussion of examination and point use follows with the different subjects after this introduction.

Alarm Point

In classic acupuncture an alarm point is considered diagnostic and therapeutic. Only the alarm points for the lung, liver, and gallbladder meridians are on their respective meridians. The alarm points are basically located over the organ with which they are associated, e.g., the heart alarm point is over the heart and the large intestine alarm point is over the large intestine. For the twelve meridians, six alarm points are located on the conception vessel, two on the gallbladder, two on the liver meridian, and one each on the lung and stomach meridians.

Associated Point

There are fourteen associated points, one for each meridian and for the conception and governing vessels. They are located along the spine on the bladder meridian. Stimulation of associated points will affect either the bladder meridian or the meridian with which it is associated, depending upon the law of deficiency.

An associated point can be active as a result of the meridian it represents being imbalanced. If active there will be positive therapy localization of the point. Stimulation of the point will help balance its associated meridian.

Of possibly greater significance is the relationship between associated points and vertebral subluxations. When an associated point is active, the adjacent vertebrae should be challenged and therapy localized to determine if a subluxation is present. Each vertebra should be challenged with rebound and interosseous challenges. Failure to correct the subluxation may cause recurrence of the meridian imbalance, even though it was effectively balanced with other techniques. On the other hand, when a meridian is consistently out of balance, it is clinically observed that subluxations tend to recur until the meridian has been balanced.

One of the main reasons chiropractors have been influencing the meridian system unknowingly is the effect that correcting subluxations has on the adjacent associated point. By understanding the meridian system, one can affect the body purposely rather than by accident and, when necessary, treat the meridian system to prevent recurring subluxations.

KI 27, the last point on the kidney meridian, is the "house of associated points." It affects all the associated points. As indicated elsewhere in applied kinesiology literature,[35,36] it is an important point for the spinal column and general neurologic disorganization.

Entry or Exit Point

On the fingertips and toe tips are entry or exit points for each meridian, also called tsing and Akabane points. The points are located near an intersecting line drawn from the proximal aspect of the fingernail or toenail and the side of the nail, about a "rice-grain's width" from the nail. The location is called the nail point. Stimulation of these points helps transmit energy from one meridian to the next, if there is a blockage in the continuity. They are points used in Akabane measurement of the meridian system. Some use this measurement to evaluate the balance within the twelve major meridians; others consider it a more meaningful measurement of the muscle meridians, which are not discussed in this text.

Horary Points

The horary period consists of the two hours during which a meridian has its highest energy level. For example, the lung horary period is 3:00-5:00 a.m. Stimulation to the horary point is often effective when a symptomatic pattern, such as pain, routinely develops during the horary period. One should also consider the possibility of deficiency in the twelve-hour-opposite meridian as a cause of the symptomatic pattern. The horary point is the element point of the meridian. For example, the lung belongs to the element metal. The metal point on the lung meridian is LU 8, so the horary point for the lung meridian is LU 8.

Hsi Points

Hsi points, pronounced "she," can be considered the supercharger points because they have a strong effect on the meridian. They are stimulated when energy levels are very low.

Luo Points

Luo points, pronounced "low," are sometimes called the "connector" or "connecting" points. They are used

to balance energy between meridians under certain conditions. When Chi is too high in a meridian and its bilateral counterpart is low, stimulation of the luo point on the deficient meridian will balance the energy. In the five-element law, stimulation of the luo point will balance energy between "coupled meridians." In applied kinesiology, the luo point is used when energy is held or blocked in a meridian, causing several following meridians to be deficient. (Examination for the AK approach to balance energy in the superficial circulation of energy is presented on page 279.)

Sedation Points

Sedation points lower the energy in an overactive meridian. A muscle that tests strong in the clear should temporarily test weak when the sedation point is stimulated. If function is normal, meridian balance will return within a short time and the muscle will again test strong in the clear. Under some circumstances there may be a failure of the associated muscle to weaken with stimulation to the sedation point which is abnormal. There are many reasons this can occur that are outside the scope of this text. Sedation point stimulation is a rather simplistic way to balance meridian energy. In most cases there are better methods to balance the energy, e.g., using the five-element law or some other method.

Tonification Points

The tonification points are sometimes called stimulation points because they increase energy in the meridian. The term "stimulation point" is discouraged because it is easily confused with stimulating a point. When a muscle associated with the meridian tests weak because of deficiency in the meridian, it will strengthen with stimulation to the tonification point. When several meridians are out of balance, there is probably a better method of balancing the system by the five-element law or some other means.

Source Points

Source points affect the entire meridian. They are four times more active than any other point on the meridian. There is a system for electrically measuring the energy balance in the meridian system called Ryodoraku (row do rock' oo) measurement. The measurement is taken at the source points located on the wrists and ankles/feet. The source points are sometimes called Nakatani points after Dr. Nakatani, developer of the Ryodoraku system.

Methods of Point Stimulation

In North America it is probable that needle stimulation of the points on the meridians is used less than a combination of other methods of stimulation. Most common are mechanical and electrical stimulation. Electrical stimulation is done with various types of instruments producing electrical waves designed specifically to influence the meridian system. The points on the meridian can be localized with some of these instruments, and computer programs can help analyze the system.

Most applied kinesiology practitioners do not use needles to influence the meridian system. Indeed, those who choose to use needles should be well aware of potentially disastrous effects, from transmitting AIDS or causing a pneumothorax to penetrating the gut and causing peritonitis. Use of needles without a wide range of knowledge contributes significantly to the possibility of iatrogenic problems. This author has used needles in the past, and gets results that are just as effective without them in the applied kinesiology techniques.

Law of Stimulation

In classic acupuncture there are numerous guidelines for producing the desired effect from stimulation to an acu-point. Most are in relation to the use of needles, i.e., insert slowly or rapidly, withdraw slowly or rapidly, angle of needle, movement of needle, and others.

A general overview of the law of stimulation is that stimulation will first cause activity, followed eventually by sedation, and finally by anesthesia. In most cases of applied kinesiology application to the meridian system, activity of the point is desired. Overstimulation may lose the effect desired.

When applying stimulation to the sedation point of a meridian, one is stimulating the point to lower the meridian's energy. Overstimulation does not sedate the meridian; rather, it loses the sedation point's ability to lower the energy in the meridian.

Active acu-points usually have positive therapy localization in the clear. When stimulation is successful, therapy localization should be negative.

Law of Deficiency

The law of deficiency is described as the body responding homeostatically to stimulation; that is, the body has a natural tendency to use any stimulus in its most beneficial aspect.[29] Stimulation of an acu-point can result in one or more of many effects. The response given priority depends upon the deficiency need and whether it can be met by the applied stimulus. If there is no need, there is an inclination to ignore the stimulus. This should not be interpreted to mean that no harm can be caused by the improper stimulation of acu-points. As Goodheart

points out in his lectures, one should evaluate for the need, supply the therapy, and observe the results. This should be a basic criterion for all therapy.

Digital Pressure

Finger pressure or massage of a meridian point — often called acupressure — is a common and effective approach.[4] A complete method of finger-pressure treatment not directly related to the meridian system is "Shiatsu."[26] In Japan there are schools that teach this method.

Acu-points requiring stimulation are usually very tender and can often be palpated as rather small, localized "mushy" areas. The tenderness is a diagnostic factor and is one method of locating points for treatment. The tender points are called ah-shi points because as the acupuncturist goes over the tender point the patient says "Ah-shi," meaning yes in Chinese. Sometimes these are referred to as ah-so points, meaning yes in Japanese.

Tapping

A method of stimulating acu-points that has been found very effective in applied kinesiology is digitally tapping the point. The fingertip sharply strikes the acu-point hard enough to bounce off but not hard enough to cause the patient pain. The point can usually be sufficiently stimulated with fifteen to twenty taps.

Teishein

The teishein is considered a non-piercing needle for acu-point stimulation. It is an instrument that has a dull-pointed rod about 1 mm in diameter, protruding from a barrel which contains a spring that allows the rod to slide into the barrel and be returned by the spring expansion. Acu-points are stimulated by the rod striking the acu-point. As the end of the rod strikes the skin its initial contact is acute, but the force is then absorbed by the rod sliding into the barrel. Stimulation with a teishein is usually done with five quick impacts, a short rest, then repeating with five more until twenty-five impacts have been made.

Moxibustion

Moxibustion is the burning of an herb. The leaves of the herb are finely ground and made into a pellet or punk. The pellet is placed over an acu-point and lit with punk to burn and raise a small blister, or the lit punk is held close to the skin to raise the blister. This method is not widely used in Western culture, but one will often read of it in the literature. In the classic use of acupuncture, points were stimulated by burning with red-hot irons. An individual who had undergone considerable acupuncture treatment would have numerous scars over the body. The burning of herbs for inhalation is common in the practice of acupuncture.

Acu-aids

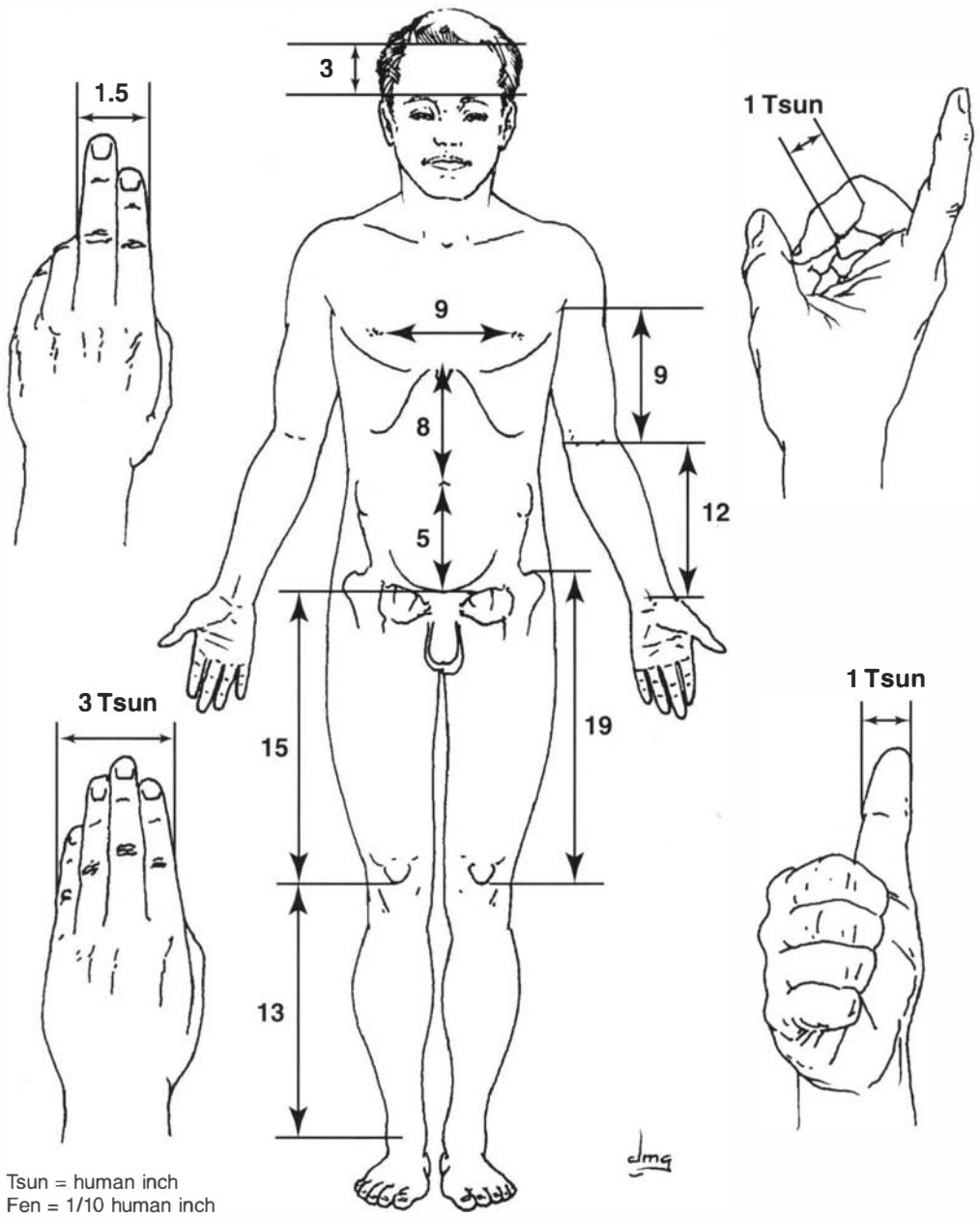
Acu-aids are small steel balls attached to the skin with tape. They are usually supplied with about one hundred of the small patches and balls attached to a plastic sheet. The patches are pulled off the sheet as needed to apply to the patient. This type of stimulation is good when continuous mild stimulation is desired.

Body Measurements

To locate acu-points it is necessary to have a measurement system that varies with the size of the individual. This is accomplished by the "human inch," called a "tsun," sometimes written "cun." Both are pronounced "sun." It is also referred to as the standard Chinese inch (SCI). Some meridian charts place a measurement scale along the body to help locate the acu-points.

One tsun is the width of the thumb or the distance between the lateral creases of the interphalangeal joints of the middle finger when the finger is mostly flexed. One-and-one-half tsun is the width of the index and middle fingers at the interphalangeal joints; three tsun is the width of the four fingers at the interphalangeal joints. One "fen" is one-tenth of a tsun. It is generally used as a measure of needle penetration.

Accompanying the meridian charts are methods of locating some of the more important points.[16,42] Often measurement is given in the human inch. When location is described by using a portion of the examiner's body for measurement, one must make conversion for a disparagement between patient and physician size.



7—7. Standard Chinese measurements.

Main Meridians

Following are the twelve main bilateral meridians. They are presented in the circulation of energy order. The location of acu-points is taken from several sources; a general consensus is used.[2,5,6,18,19,20,22,28,29,33,42,43] Included with each meridian is a word description of point location for some of the major active points. The Chinese name — and sometimes its translation — is given for some of the points. Although the classic acupuncture use is given for some of the points, one should not consider the description as promoting a "cookbook" approach. When considering these points for the symptomatic pattern described, therapy localize or otherwise measure the activity of the point to determine if treatment is indicated. There are many connecting channels between the meridians, organs, and acu-points. Some of the most important ones are shown to illustrate the interaction of the system.

Lung — LU — Yin — Metal

Begins on the anterior chest and ends at the radial nail point of the thumb. Energy enters the body at the umbilicus (Shrine of God) and travels over the connecting channel to CV 12, then goes into the diaphragm and to the lungs, travels up to the thyroid, then to ST 12, and connects with LU 1. A channel branches from LU 7 to connect with LI 1.

Muscle association: levator scapula, serratus anticus, deltoid, coracobrachialis (provisional for flexor pollicis longus and brevis).

LU 1 Begins one tsun below the clavicle medial to the apex of the coracoid process, usually in a definite depression.

Alarm point for lung meridian. This point is often tender to the touch in respiratory conditions.

LU 5 On the elbow crease about one tsun lateral to the biceps tendon.

Sedation point

LU 6 Five tsun distal to LU 5, lateral to the flexor carpi radialis.

Hsi point

LU 7 Two tsun up from wrist crease on thumb side of the anterolateral forearm. Point can be located by the patient approximating his thumb webs. The point is at the end of the opposite hand's index finger.

Luo point

Used for migraine.

LU 8 On the most lateral aspect of the styloid process of the radius.

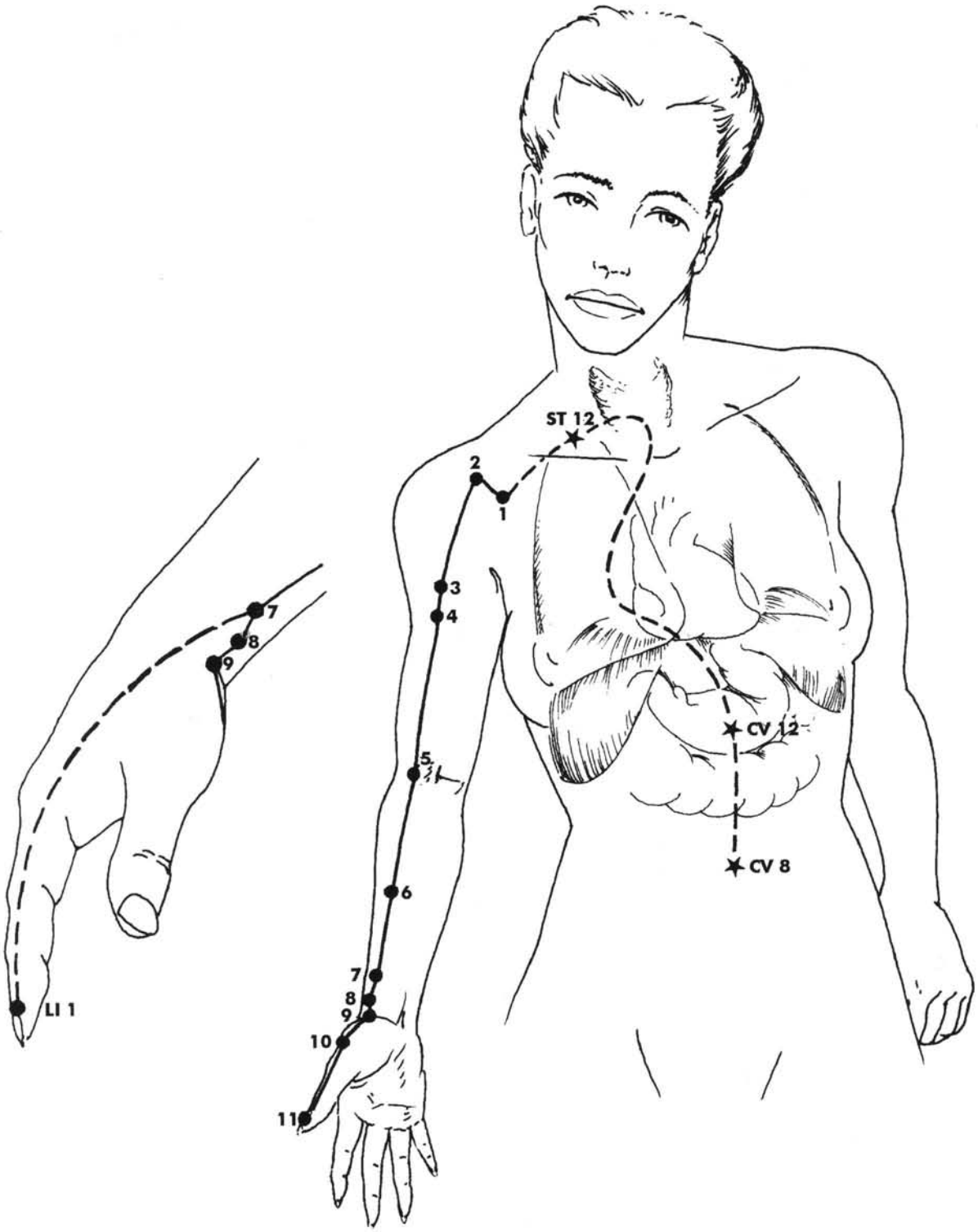
Horary point (3:00-5:00 a.m.)

LU 9 At anterolateral palmar wrist crease.

Source point (Nakatani point for Ryodoraku measurement).

Tonification point

BL 13 **Associated point** between T3-4 transverse processes.



7—8. Lung meridian

Large Intestine — LI — Yang — Metal

Begins at the radial nail point of the index finger and ends at the nasal sulcus. A channel branches to connect the meridian with ST 12, goes through the lung to connect with ST 25. It also branches to GV 14.

Muscle association: hamstrings, tensor fascia lata, quadratus lumborum.

LI 1 Radial nail point of the index finger.

Horary point (5:00-7:00 a.m.)

LI 2 Radial side of the index finger, distal to the base of the proximal phalanx.

Sedation point

LI 4 (Hoku or Hegu, meeting of the valleys) Located at the end of the crease formed when the thumb and forefinger are held together.

This point is often used in acupuncture. It has a special effect for everything in the head and neck. Stimulation is always directed toward the pisiform bone.

Used for dental anesthesia.[15]

Source point

LI 5 Radial side of wrist crease. Nakatani point for Ryodoraku measurement. This is the only Nakatani point that is not the source point.

LI 6 Three tsun proximal to the wrist crease on the lateral aspect of the radius.

Luo point

LI 7 Five tsun proximal to the wrist crease on the lateral aspect of the radius.

Hsi point

LI 11 Located at lateral elbow crease with the elbow in full flexion. Enhances and gives strength to LI 4.

Tonification point

LI 15 On the lateral shoulder directly under the acromion process in the depression made by abduction of the arm.

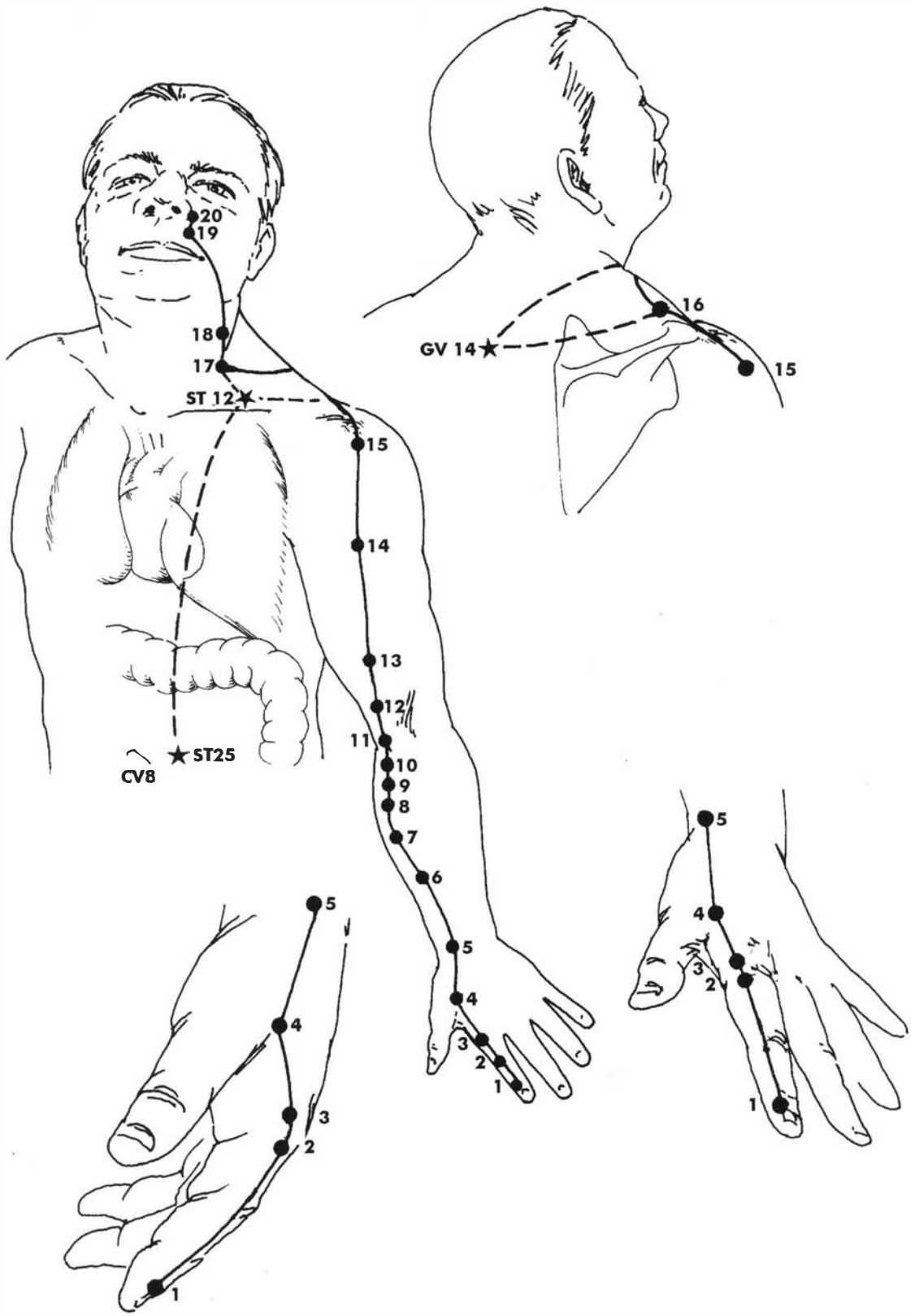
Excellent for various types of shoulder pain.

LI 20 "Welcome fragrance" at the lower portion of the nasal sulcus, one fen from the nose.

Affects the pituitary and is often used for pain.

ST 25 **Alarm point** for the LI. One and one-half tsun lateral to the umbilicus.

BL 25 **Associated point** between L4-5 transverse processes.



7—9. Large intestine meridian

Stomach — ST — Yang — Earth

Begins below the eye and ends at ST 45 lateral nail point of the second toe. A channel branches at ST 12 to cross the diaphragm and connect with the stomach. From ST 42 there is a connecting branch to SP 1.

Muscle association: pectoralis major (clavicular division), biceps brachii, brachialis, brachioradialis, supinator, pronator teres, opponens pollicis, adductor pollicis, opponens digiti minimi, sternocleidomastoid, deep neck extensors, medial neck flexors, pronator quadratus.

ST 1 (Receive tears) On the infraorbital ridge directly below the center of the pupil, with the eyes looking straight ahead.

ST 4 Four fen lateral to the corner of the mouth.

Used for facial pain.

ST 5 Inferior border of the mandible, anterior to the masseter.

ST 6 Inferior border of the mandible, five fen anterior to the angle.

ST 7 Inferior to zygomatic arch in depression anterior to the mandibular condyle. The depression disappears when the mouth is opened.

ST 5, 6, and 7 are used to surround the dragon in cases of facial pain, tic douloureux, and Bell's palsy, among others.

ST 12 (Chieuh pien, half a bowl) In the supraclavicular fossa on the nipple line.

Important for almost everything because most meridians have a direct connection with this point.

ST 17 In center of nipple.

ST 25 One and one-half tsun lateral to the umbilicus.

Alarm point for the large intestine meridian.

ST 34 Two tsun above the knee crease on lateral aspect of the rectus femoris.

Hsi point

ST 36 (Tsusanli, stomach three mile) Lateral to tibial tubercle, three tsun down from depression inferior to the lateral patella when the knee is flexed to 45°.

An important, frequently used point for abdominal and infectious processes. Increases phagocytosis. Used with SP 6 for low resistance and infection from low autoimmune function.

Horary point (7:00-9:00 a.m.)

ST 40 Anterolateral surface of leg, eight tsun proximal to the lateral malleolus.

Luo point

ST 41 Middle of anterior flexure of the ankle.

Tonification point

ST 42 Located in depression formed by junction of 2nd and 3rd cuneiform and navicular bones.

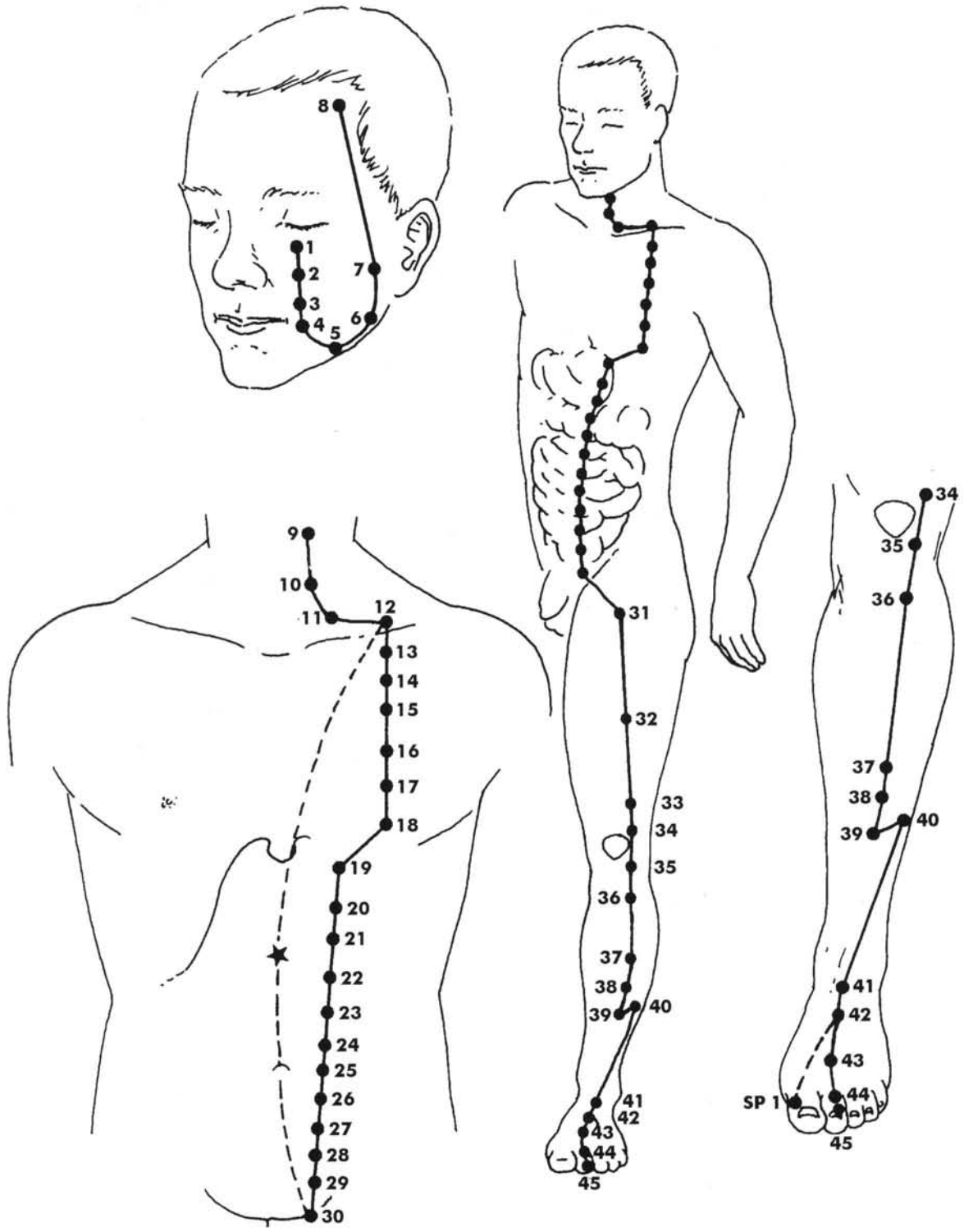
Source point (Nakatani point for Ryodoraku measurement).

ST 45 Lateral nail point of the second toe.

Sedation point

CV 12 **Alarm point** for ST, halfway between CV 8 (umbilicus) and CV 15 (tip of xiphoid process).

BL 21 **Associated point** between T12-L1 transverse processes



7—10. Stomach meridian

Spleen — SP — Yin — Earth

Begins at the medial nail point of great toe and ends on the lateral chest. Connecting channels to CV 3, 4, and 12, LV 14,GB 24, HT 1, LU 1, and ST 12. The connecting channel continues to disperse at the base of the tongue. From the stomach region the energy branches to the heart meridian.

Muscle association: lower trapezius, middle trapezius, latissimus dorsi, triceps brachii, anconeus.

SP 1 Medial nail point of great toe.

SP 2 Medial edge of great toe, just distal to the base of the proximal phalanx.

Tonification point

SP 3 On medial foot just proximal to the metatarsal head.

Source point (Nakatani point for Ryodoraku measurement).

Horary point (9:00-11:00 a.m.)

SP 4 Medial edge of foot, just proximal to the base of the first metatarsal bone.

Luo point

SP 5 At hollow, anterior and inferior to the medial malleolus.

Sedation point

SP 6 (Sanyinchiao, three Yin meeting) On medial leg, three tsun proximal to the medial malleolus posterior to the edge of the tibia.

Frequently used for female or male reproductive problems, e.g., hormonal imbalance, dysmenorrhea, and others.

Used with ST 36 for autoimmune conditions.

SP 8 Two and one-half tsun down from SP 9

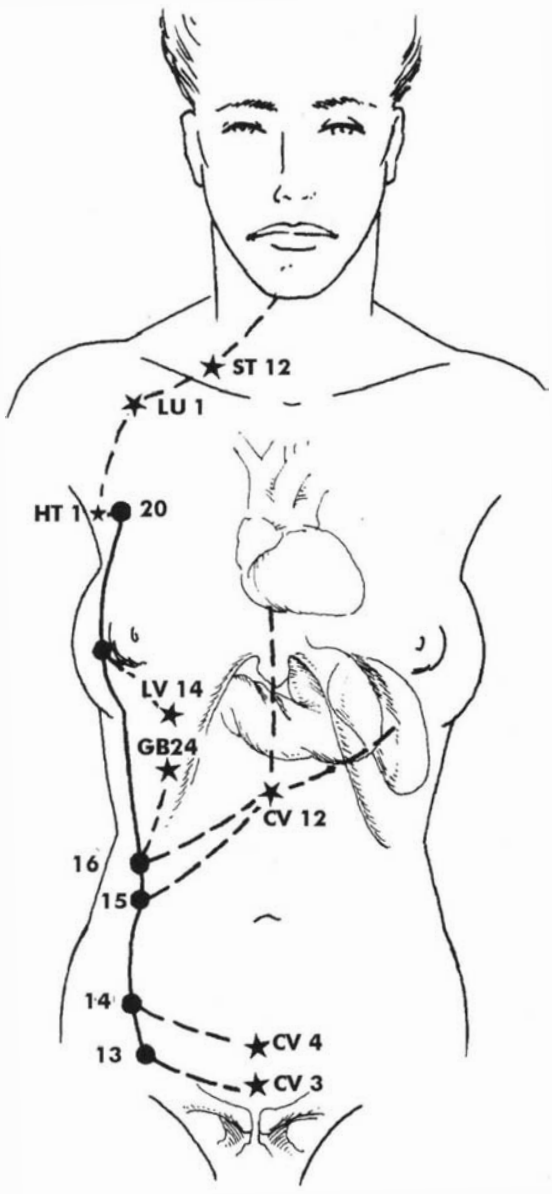
Hsi point

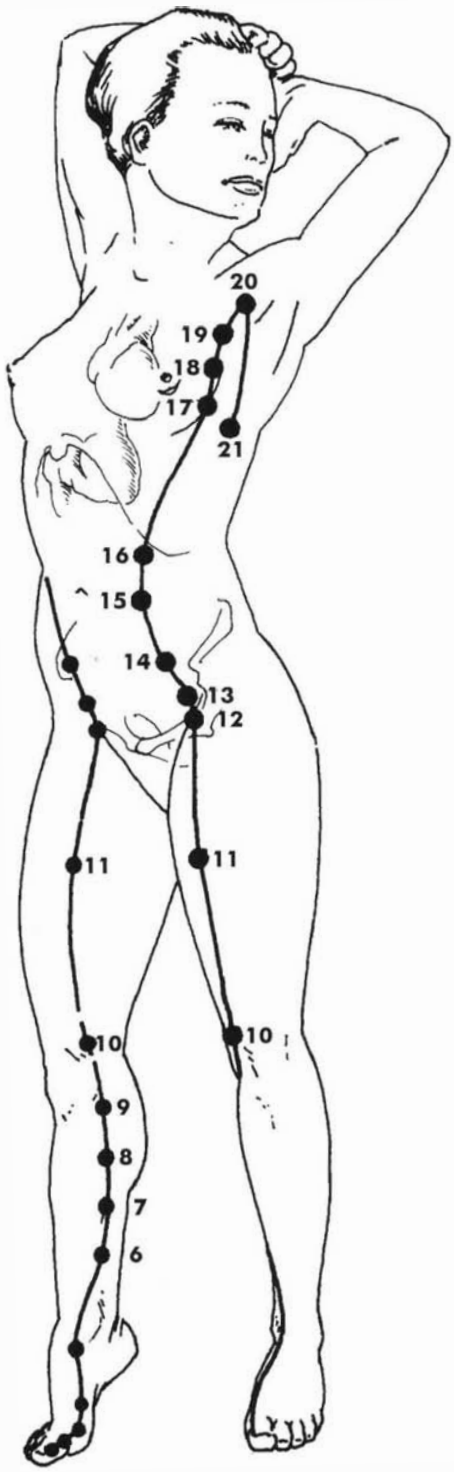
SP 9 Inferior, posterior border of the medial condyle of the tibia.

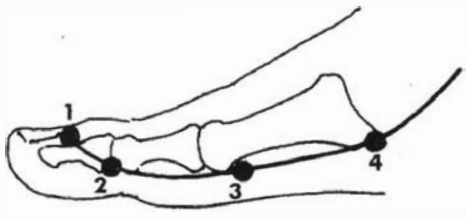
SP 21 (**Great luo point,** great enveloping) On lateral thorax at level of sixth intercostal space on the mid-axillary line.

LV 13 **Alarm point** for SP, at the end of the eleventh rib.

BL 20 **Associated point** between T11-12 transverse processes.







7—11. Spleen meridian

Heart — HT — Yin — Fire

Begins in the axilla and ends at the radial nail point of the fifth finger. Connecting channels to the lungs, ST 12, tissues around the eyes, CV 14, and CV 8.

Muscle association: subscapularis.

HT 1 In the axilla.

HT 3 At end of medial crease formed by flexing the elbow.

Used for emotional problems.

HT 5 One tsun proximal to HT 7.

Luo point

HT 6 Five fen proximal to HT 7.

Hsi point

HT 7 (Shenmen, spirit gate) On ulnar side of wrist crease.

Used for insomnia.

Source point (Nakatani point for Ryodoraku measurement).

Sedation point

HT 8 Palmar surface of hand on the distal transverse crease between the fourth and fifth metacarpal bones.

Horary point (11:00 a.m.-1:00 p.m.)

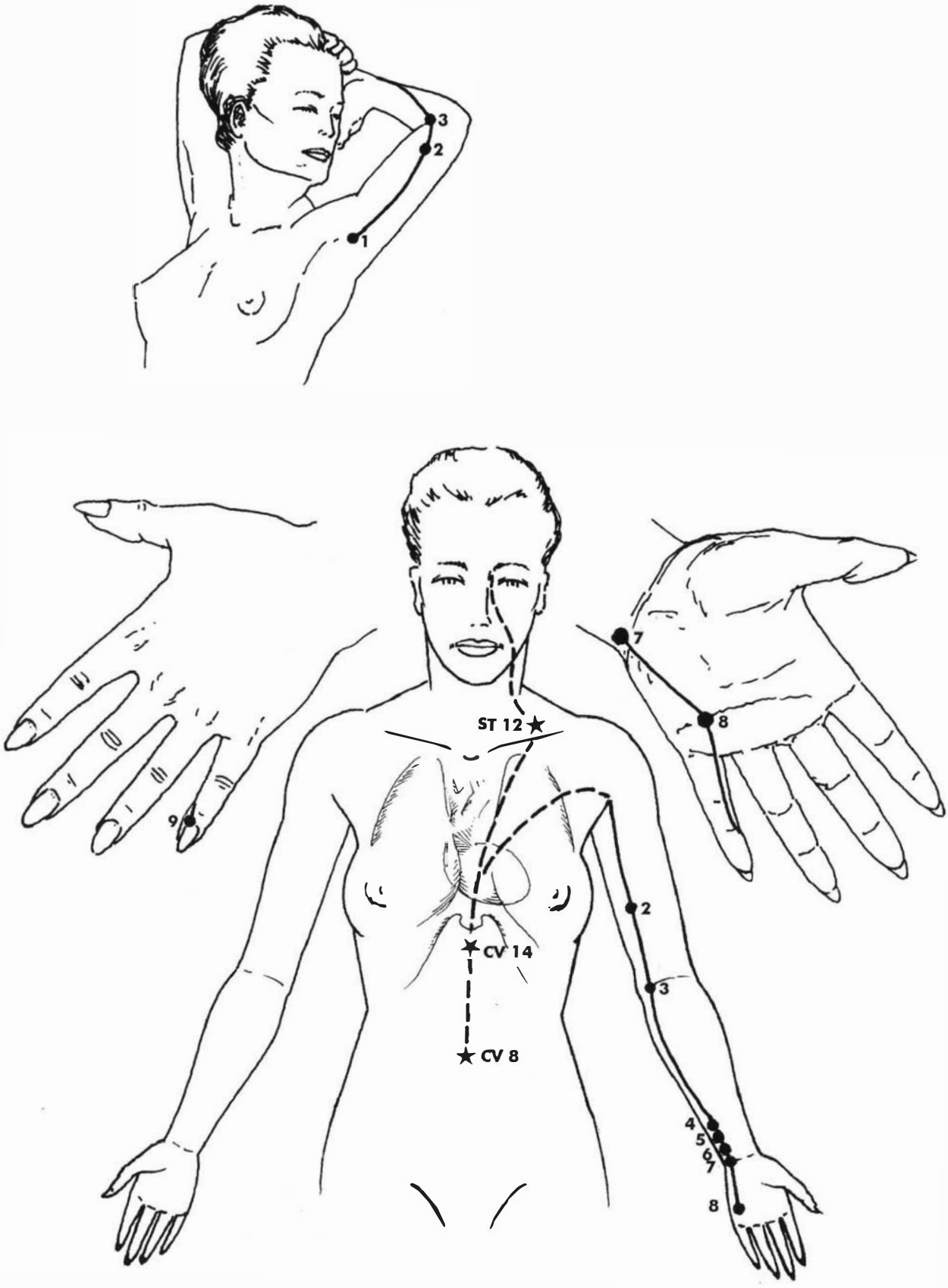
HT 9 At radial nail point of the fifth finger.

Used in cardiac disorders.

Tonification point

CV 14 **Alarm point** for HT, one tsun below tip of xiphoid process.

BL 15 **Associated point** between T5-6 transverse processes



7—12. Heart meridian

Small Intestine — SI — Yang — Fire

Begins at the ulnar nail point of the fifth finger and ends anterior to the tragus of the ear. Connecting channels to GV 14, ST 12, heart; crosses the diaphragm to the stomach and then reaches the small intestine. There is also a connecting branch to CV 17, 12, and 4.

Muscle association: quadriceps, abdominals, flexor digiti minimi brevis.

SI 1 Ulnar nail point of the fifth finger.

SI 3 Ulnar edge of hand at crease from palm when fist is made.

Used for shoulder pain and for low back pain when used in combination with BL 62.

Tonification point

SI 4 At dorsal wrist crease on ulnar side.

Source point (Nakatani point for Ryodoraku measurement).

SI 5 Ulnar edge of hand at distal ulnar bone.

Horary point (1:00-3:00 p.m.)

SI 6 Ulnar surface of forearm, just proximal to the head of the ulna.

Hsi point

SI 7 Posterior forearm, five tsun proximal to the wrist crease on the medial border of the ulna.

Luo point

SI 8 In the fossa between the olecranon and medial epicondyle of the humerus.

Sedation point

SI 10 Below the lateral aspect of the spine of the scapula (acromion process) in a definite depression.

Used with SI 3 and SI 12 for shoulder problems.

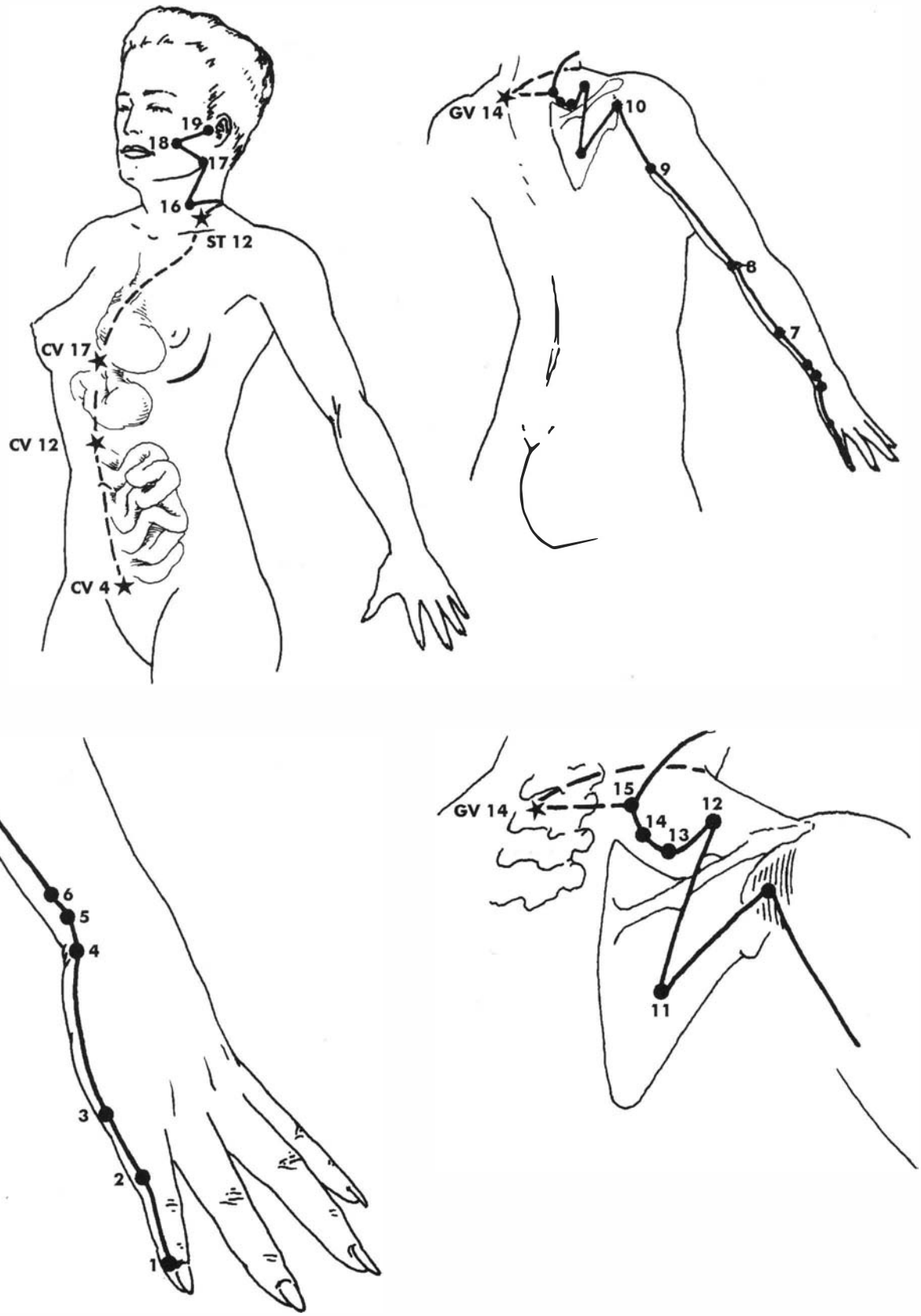
SI 12 One tsun superior to the superior edge of the center of the scapular spine.

SI 19 (Listening palace) Anterior to the tragus of the ear in the depression formed when the mouth is open.

Used for deafness, tinnitus, TMJ syndrome, and facial pain. Always treat with the mouth open.

CV 4 **Alarm point** for SI, two tsun above superior border of symphysis pubis.

BL 27 **Associated point** at level of first sacral foramen.



7—13. Small intestine meridian

Bladder — BL — Yang — Water

Begins at the medial canthus of the eye and ends at the lateral nail point of the little toe. Longest meridian of the system. There is a connecting channel from BL 49 to GB 30 and then to BL 54. BL 67 connects to KI 1. Goodheart postulates that the reason for the double channel of the bladder meridian may be to provide a reference and object beam for holographic function.

Muscle association: tibialis anterior; peroneus tertius, longus, and brevis; sacrospinalis; extensor hallucis longus and brevis.

BL 1 (Eyes bright) Begins one fen medial and superior to the medial canthus of the eye.

Used for eye disorders.

BL 11 Two tsun lateral to the median line of the body, level is between first and second thoracic vertebrae transverse processes.

Master point for any bone condition, e.g., osteoporosis and fracture healing, among others.

Associated Points

BL 13-21 are two tsun from the midline, BL 21-28 are two and one-half tsun from the midline. Authorities vary slightly regarding the level of some associated points. Use therapy localization and palpation to find the exact location.

Point level	Associated point	Point level	Associated point
BL 13 between T3-4 transverse	LU	BL 20 between T11-12 transverse	SP
BL 14 between T4-5 transverse	CX	BL 21 between T12-L1 transverse	ST
BL 15 between T5-6 transverse	HT	BL 22 between L1-2 transverse	TH
BL 16 between T6-7 transverse	GV	BL 23 between L2-3 transverse	KI
BL 17 between T8-9 transverse	CV	BL 25 between L4-5 transverse	LI
BL 18 between T9-10 transverse	LV	BL 27 level of 1st sacral foramen	SI
BL 19 between T10-11 transverse	GB	BL 28 level of 2nd sacral foramen	BL

BL 50 Midpoint of the gluteal fold. Used in sciatica.

BL 51 Middle of posterior thigh, midway between gluteal fold and crease of knee.

BL 54 (Weichung, commanding center, middle of man) Center of popliteal surface on knee crease.

Used in low back and knee pain, relaxes paraspinal muscles.

Called BL 40 on some charts.[16]

BL 57 Between lateral and medial bellies of the gastrocnemius, nine tsun proximal to the lateral malleolus.

Used in low back and knee pain, relaxes paraspinal muscles.

BL 58 Posterolateral leg, eight tsun proximal to the lateral malleolus between the gastrocnemius and soleus muscles.

Luo point

BL 60 (Kunlun) Halfway between lateral malleolus and Achilles tendon.

Used for release of endorphins and pain control, low back and leg pain.

BL 63 Lateral foot, just proximal to the fifth metatarsal base.

Hsi point.

BL 64 Lateral foot, distal to the fifth metatarsal base.

Source point (Nakatani point for Ryodoraku measurement).

BL 65 Lateral foot, proximal to the fifth metatarsal head.

Sedation point

BL 66 Lateral foot, distal to base of proximal phalanx.

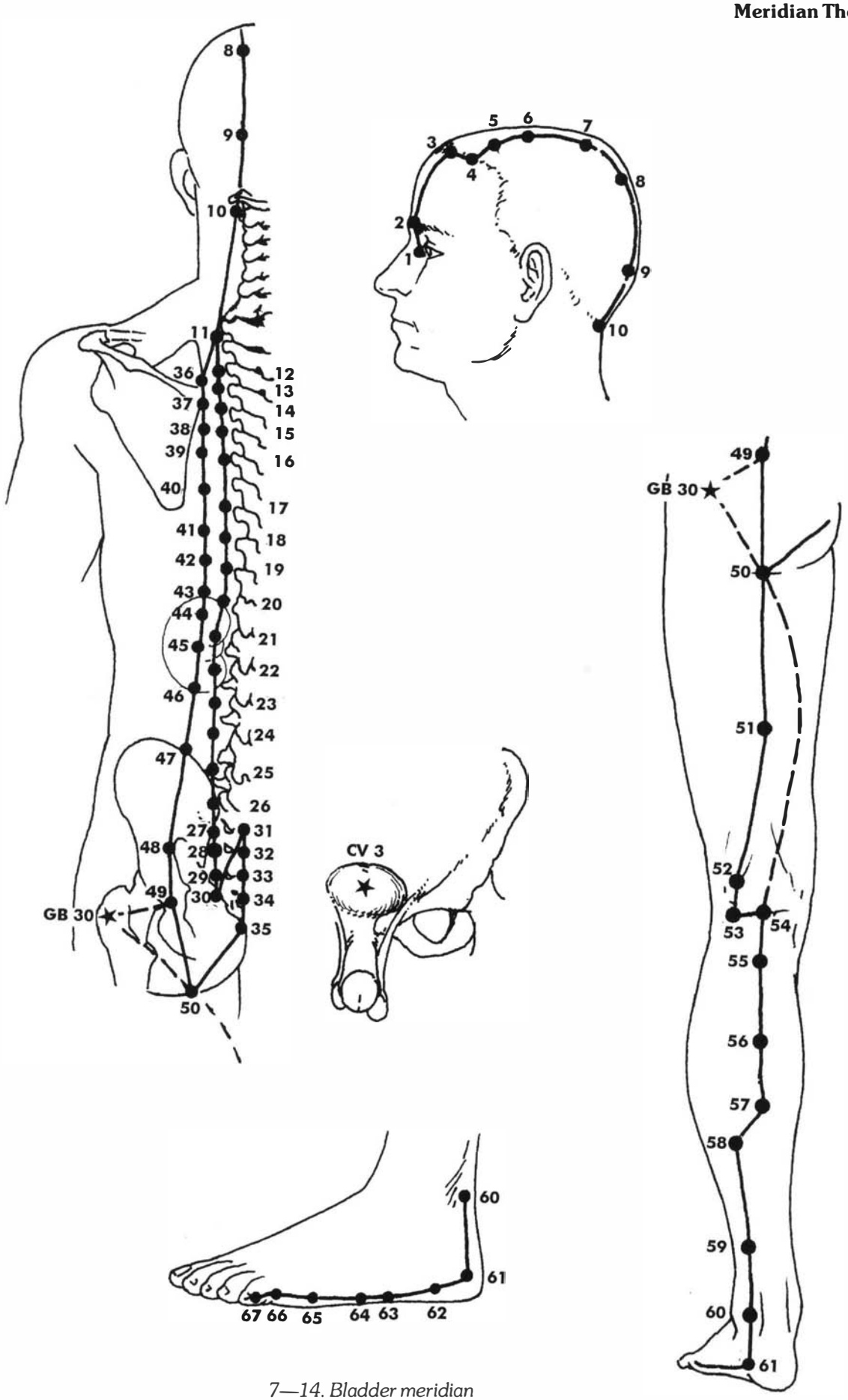
Horary point (3:00-5:00 p.m.)

BL 67 On lateral nail point of little toe.

Used for conditions involving the head, especially headache.

Tonification point

CV 3 **Alarm point** for BL, one tsun above CV 2



7—14. Bladder meridian

Kidney — KI — Yin — Water

Begins on the plantar surface of the foot and ends on the chest at the junction of the first rib, clavicle, and sternum. There is an intersection with SP 6. A connecting channel goes into GV 1, coursing along the spine to the kidney, liver, lungs, heart, and other organs. A channel from the kidney traverses the liver and diaphragm, and then enters the lung to end at the root of the tongue.

Muscle association: psoas, iliacus, upper trapezius.

KI 1 (Bubbling spring) Plantar surface of foot between the second and third metatarsal bones at the crease made by toe flexion at the metatarsophalangeal joints.

Used for chronic pain and hypertension.

Sedation point

KI 3 Posterior and slightly proximal to medial malleolus.

Source point (Nakatani point for Ryodoraku measurement).

KI 4 Posterior to medial malleolus.

Luo point

KI 5 One tsun distal to KI 4 on the superior edge of calcaneus.

Hsi point

KI 6 Distal to tip of medial malleolus.

KI 7 Three tsun proximal and slightly posterior to the medial malleolus.

Tonification point

KI 9 (House guest) Five tsun proximal to the inferior tip of the medial malleolus.

KI 10 At medial end of knee crease when knee is bent.

Used for knee pain.

Horary point (5:00-7:00 p.m.)

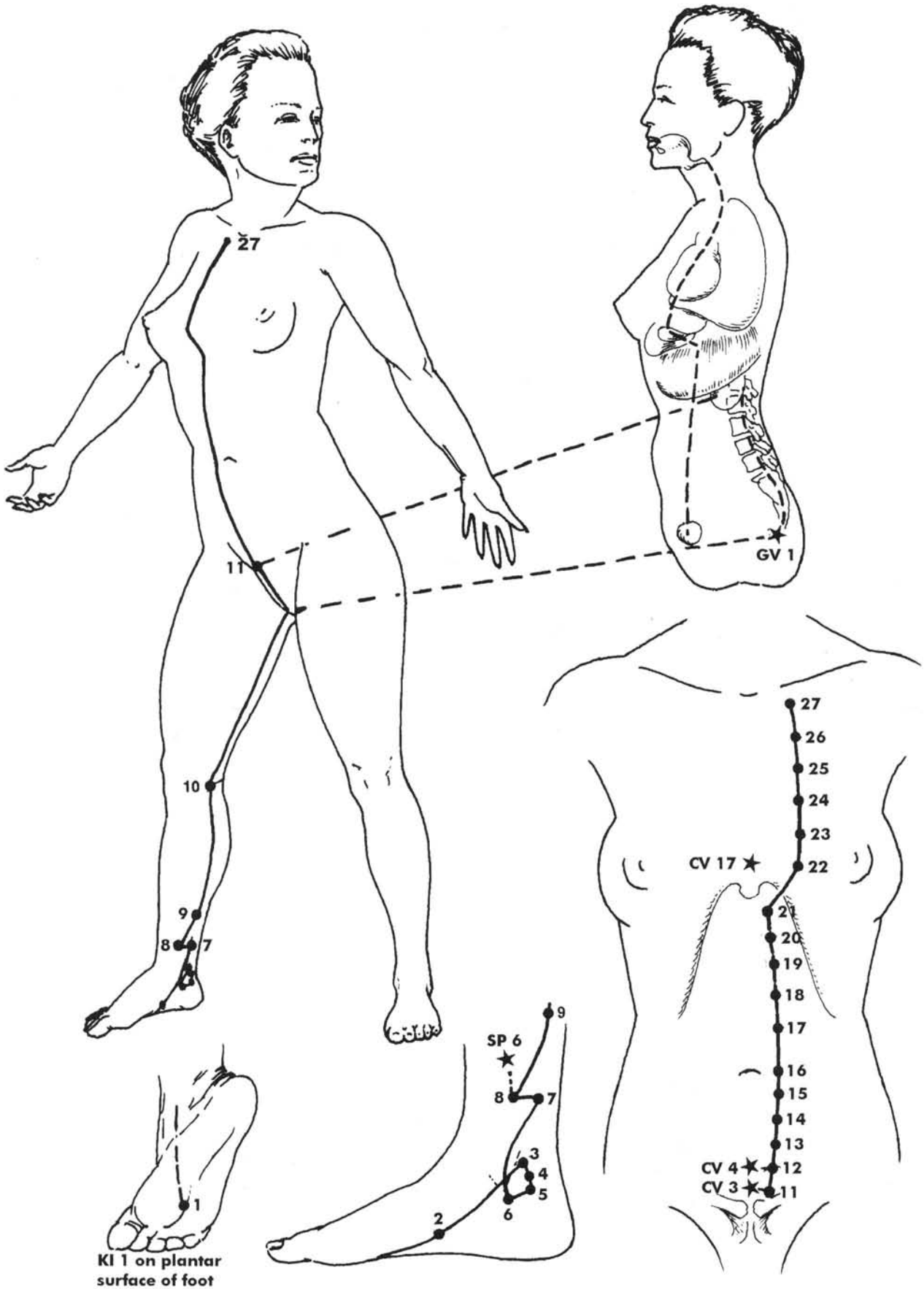
KI 27 At the junction of the first rib, clavicle, and sternum (two tsun from midline).

Used with CV 8 (Shrine of God) to evaluate and effect neurologic organization in applied kinesiology.

Home of the associated points; stimulation affects the entire associated point channel.

GB 25 **Alarm point** — located at end of twelfth rib.

BL 23 **Associated point** between L2-3 transverse processes.



7—15. Kidney meridian

Circulation Sex — CX — Yin — Fire

Also called Pericardium (P) and Heart Constrictor (HC). Begins on the chest one tsun lateral to the center of the nipple (fourth intercostal space) and ends at the radial nail point of the middle finger. A connecting channel goes to the heart, diaphragm, and reproductive organs and glands. There is connection with CV 7, 12, and 17. A branch goes from CX 8 to TH.

The circulation sex meridian deals with circulation of blood and sex hormones.

Muscle association: sartorius, gracilis, adductors, gluteus medius and minimus, piriformis, gluteus maximus, gastrocnemius, soleus, tibialis posterior, flexor hallucis longus and brevis.

CX 1 One tsun lateral to the center of the nipple.

CX 3 On the crease from elbow flexion on the ulnar side of the biceps tendon.

Used for any inflammatory process of the elbow.

CX 4 Anterior forearm between radius and ulna, five tsun proximal to the wrist crease.

Hsi point

CX 5 Anterior forearm between radius and ulna three tsun proximal to the wrist crease.

Used for psoriasis.[1]

CX 6 Anterior forearm between tendons of palmaris longus and flexor carpi radialis muscles, two tsun proximal to wrist crease.

Used in mental disorders, neurosis, and other similar problems.

Master point for the abdomen.

Luo point

CX 7 Middle of the wrist crease between tendons of the palmaris longus and flexor carpi radialis muscles.

Source point (Nakatani point for Ryodoraku measurement).

Sedation point

CX 8 Palmar surface of hand between second and third metacarpal bones, on the proximal transverse crease.

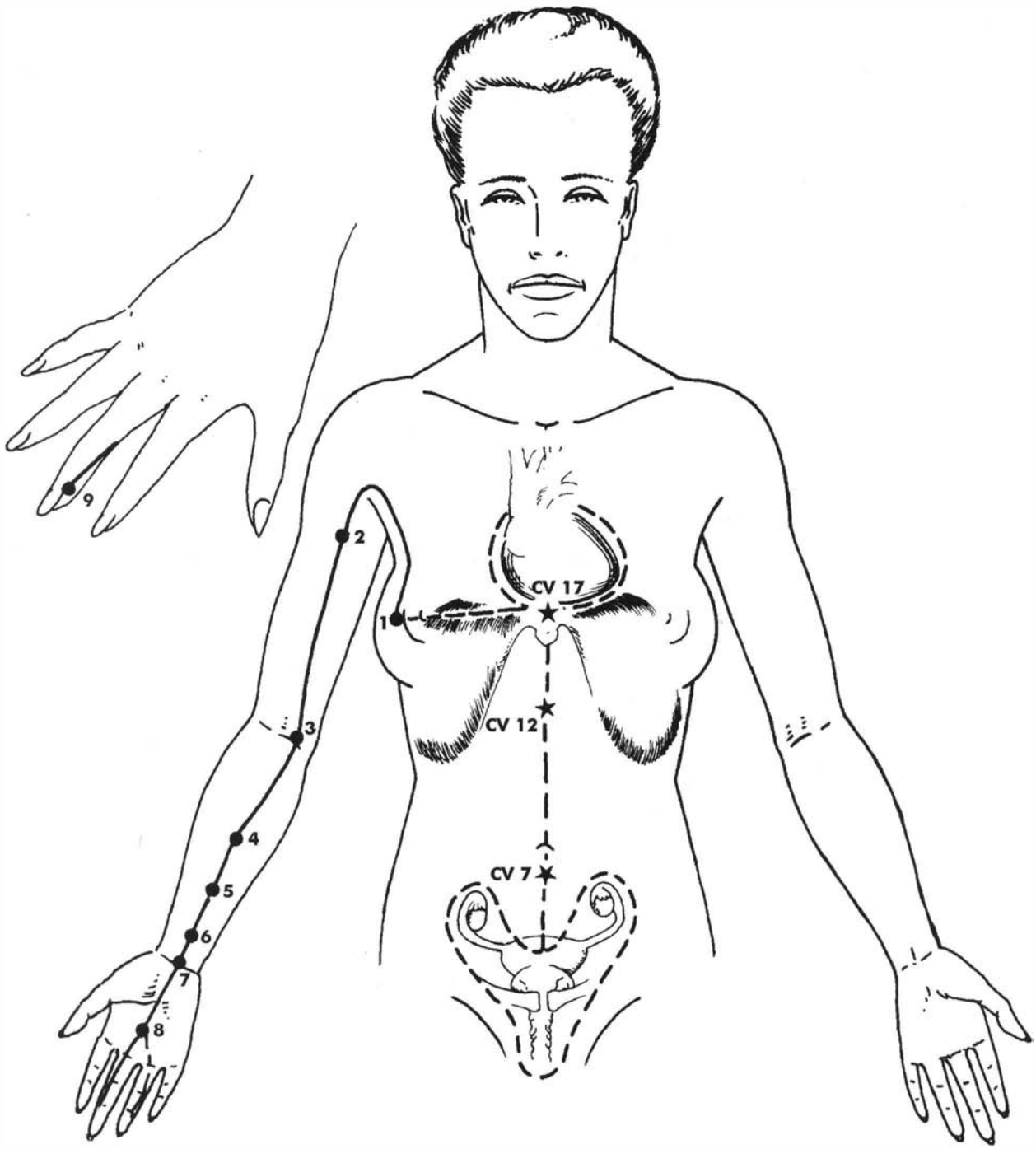
Horary point (7:00-9:00 p.m.)

CX 9 Radial nail point of the middle finger.

Tonification point

CV 17 **Alarm point** for CX, two tsun above the inferior tip of the xiphoid process.

BL 14 **Associated point** between T4-5 transverse processes.



7—16. Circulation sex meridian

Triple Heater — TH — Yang — Fire

Also called the Triple Warmer (TW) and Tri Heater. Begins at the ulnar nail point of the ring finger and ends lateral to the eyebrow. A connecting branch goes to SI 12, ST 12, GB 21, and GV 14. There is a connection to points on CV. It has been found to associate with the thyroid and thymus in applied kinesiology.

Muscle association: infraspinatus, teres minor, occasionally sartorius and gracilis.

TH 1 Ulnar nail point of the ring finger.

TH 3 Dorsal surface of the hand proximal to and on ulnar side of fourth metacarpal head.

Used for hearing problems.

Tonification point

TH 4 On the exact center of dorsal wrist crease.

Affects metabolism in general.

Source point (Nakatani point for Ryodoraku measurement).

TH 5 Two tsun proximal to posterior wrist crease between the radius and ulna bones.

Master point for the upper extremity; used for symptomatic treatment of the fingers, wrist, elbow, shoulder, and neck.

Luo point

TH 6 Posterior forearm between the radius and ulna, three tsun proximal to the wrist crease.

Horary point (9:00-11:00 p.m.)

TH 7 Posterior forearm five fen medial to TH 6.

Hsi point

TH 10 One tsun proximal to the olecranon process.

Used in elbow conditions.

Sedation point

TH 17 Directly posterior to the earlobe in a depression between the mastoid process and mandible.

Used for hearing problems, including tinnitus.

TH 21 Anterior to the superior edge of the tragus of the ear, slightly above SI 19.

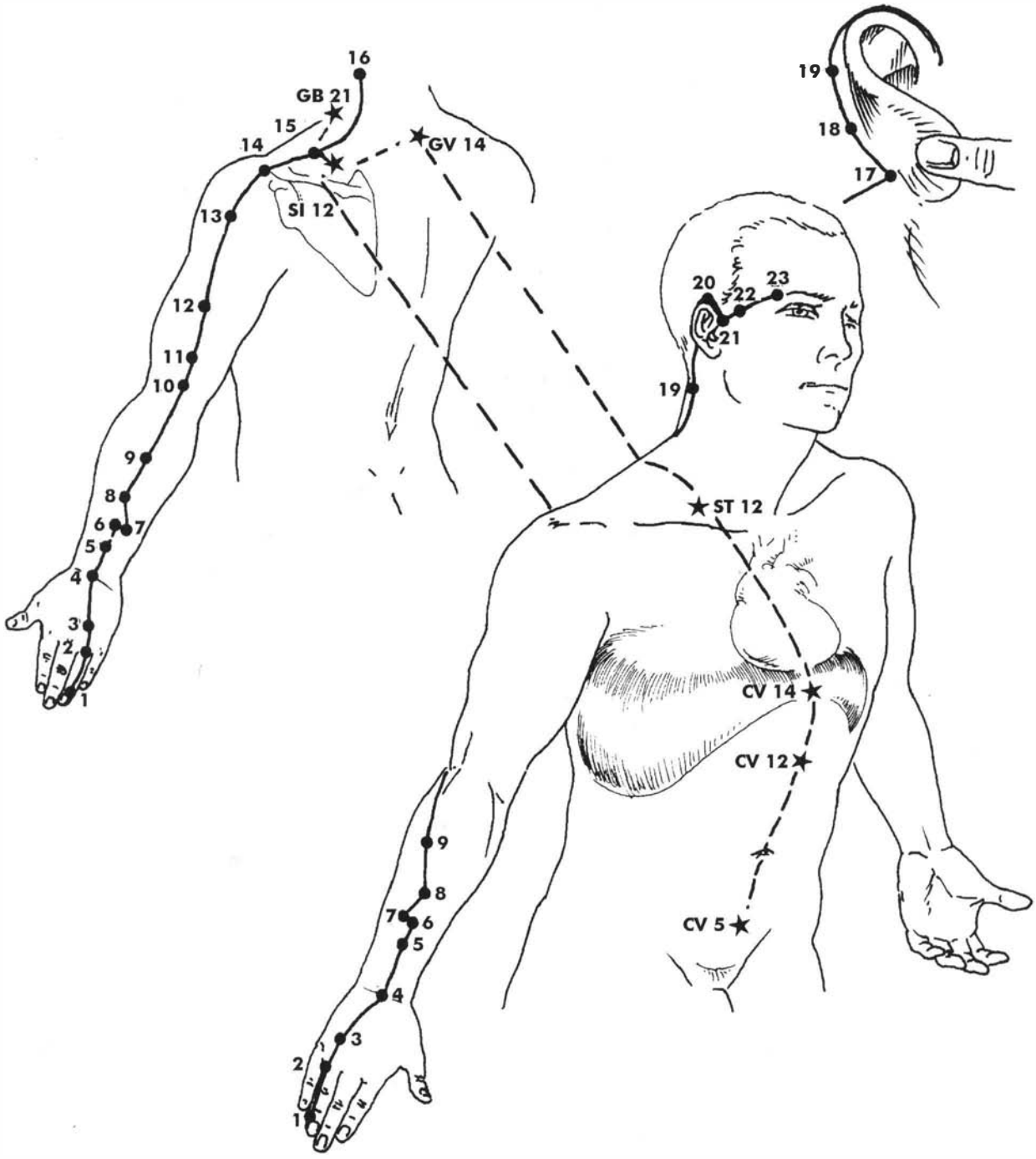
Used in facial pain, hearing, TMJ, and others.

TH 23 At the lateral edge of the eyebrow, directly above GB 1.

Used in any eye condition.

CV 5 **Alarm point** for TH, three tsun above superior border of the symphysis pubis.

BL 22 **Associated point** between L1-2 transverse processes.



7—17. Triple heater meridian

Gallbladder — GB — Yang — Wood

Begins lateral to the eye and ends at the lateral nail point of the fourth toe. There are several connecting channels to connect this meridian with the gallbladder, liver, and heart. It has a branch to ST 12 and a branch from GB 41 to LV 1.

Muscle association: popliteus.

GB 1 One-half tsun lateral to the lateral eye canthus.

Used for eye conditions.

GB 2 Slightly anterior to the inferior border of the tragus. A depression appears when the mouth is opened.

Used in hearing problems, facial pain, TMJ, and others.

GB 14 Directly above the pupil of the eye, one tsun above the supraorbital notch.

Used for sinus and frontal headaches.

GB 20 In depression between external occipital protuberance and the mastoid process. Lateral to the upper trapezius.

Used for occipital headaches and all cervical problems, including torticollis.

GB 24 Two tsun below LV 14 (alarm point for the LV) directly below the nipple.

Alarm point for GB.

GB 30 Posterior to the greater trochanter of the hip over the sciatic notch.

Primary point for hip (coxa) pain, used for back and/or leg pain.

GB 31 On lateral thigh between vastus lateralis and biceps femoris muscles, seven tsun proximal to the knee crease. When the arm is at the side, the point is level with the middle fingertip.

Used with GB 30 for sciatica and/or hip pain.

GB 34 Anterior and slightly distal to the fibular head.

Master point for the lower extremity.

GB 36 Seven tsun proximal to the lateral malleolus tip on the anterior border of the fibula.

Hsi point

GB 37 Five tsun proximal to the lateral malleolus on the anterior border of the fibula.

Luo point

GB 38 Four tsun proximal to the lateral malleolus on the anterior border of the fibula.

Sedation point

GB 40 Anterior to the lateral malleolus, posterior to the tendon of the extensor digitorum longus.

Source point (Nakatani point for Ryodoraku measurement).

Often has major effect on the general body because of the wide distribution of the GB meridian.

GB 41 Dorsal foot, where the bases of the fourth and fifth metatarsal bones join.

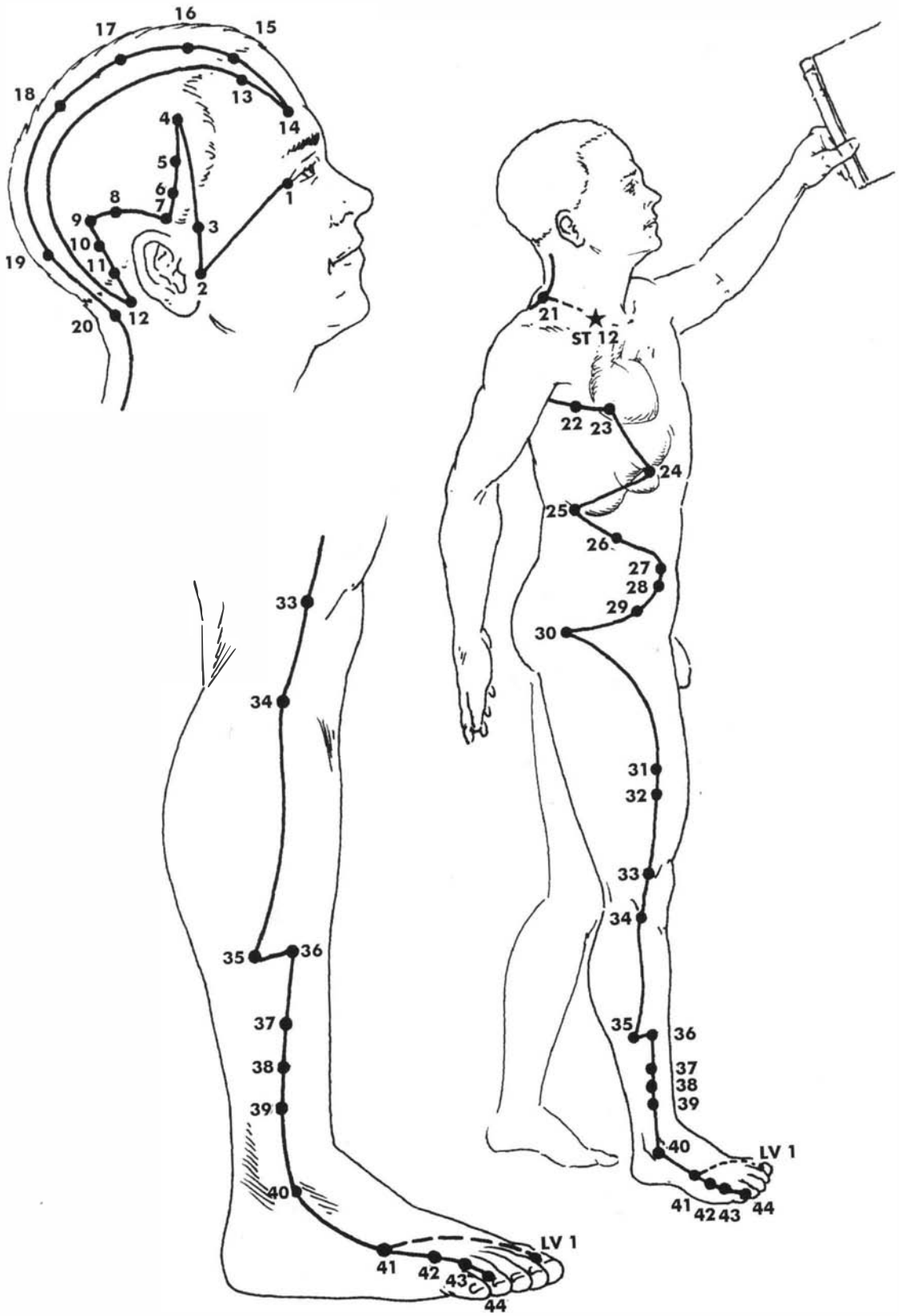
Horary point (11:00 p.m.-1:00 a.m.)

GB 43 Where the skin joins the fourth and fifth toes.

Used with HT 3 for mental depression.

Tonification point

BL 19 **Associated point** between T10-11 transverse processes.



7—18. Gallbladder meridian

Liver — LV — Yin — Wood

Begins at lateral nail point of great toe and ends on the thoracic cage at the seventh intercostal space. Connection to SP 6, CV 2, CV 3, and CV 4. It is considered by many that the meridian crosses to the opposite side at CV 2. Homolateral muscles are affected by therapy localization to the alarm point (LV 14), e.g., the left pectoralis major (sternal division) is strengthened with TL to the left LV 14 if weakness is due to liver meridian deficiency. From LV 14 there is connection to ST 12 and the eye.

Muscle association: rhomboid major and minor, pectoralis major (sternal division).

LV 1 Lateral nail point of the great toe.

Horary point (1:00-3:00 a.m.)

LV 2 Where the skin joins the first and second toes.

Used to relax muscles throughout the body.

Sedation point

LV 3 Over angle formed by the first and second proximal metatarsal bones joining.

Used for eye and muscle problems.

Source point (Nakatani point for Ryodoraku measurement).

LV 5 Five tsun above the tip of the medial malleolus on the posterior edge of the tibia.

Luo point

LV 6 Seven tsun above the tip of the medial malleolus on the posterior edge of the tibia.

Hsi point

LV 8 In the depression between the medial condyle of the femur and the tendon of the semimembranosus muscle.

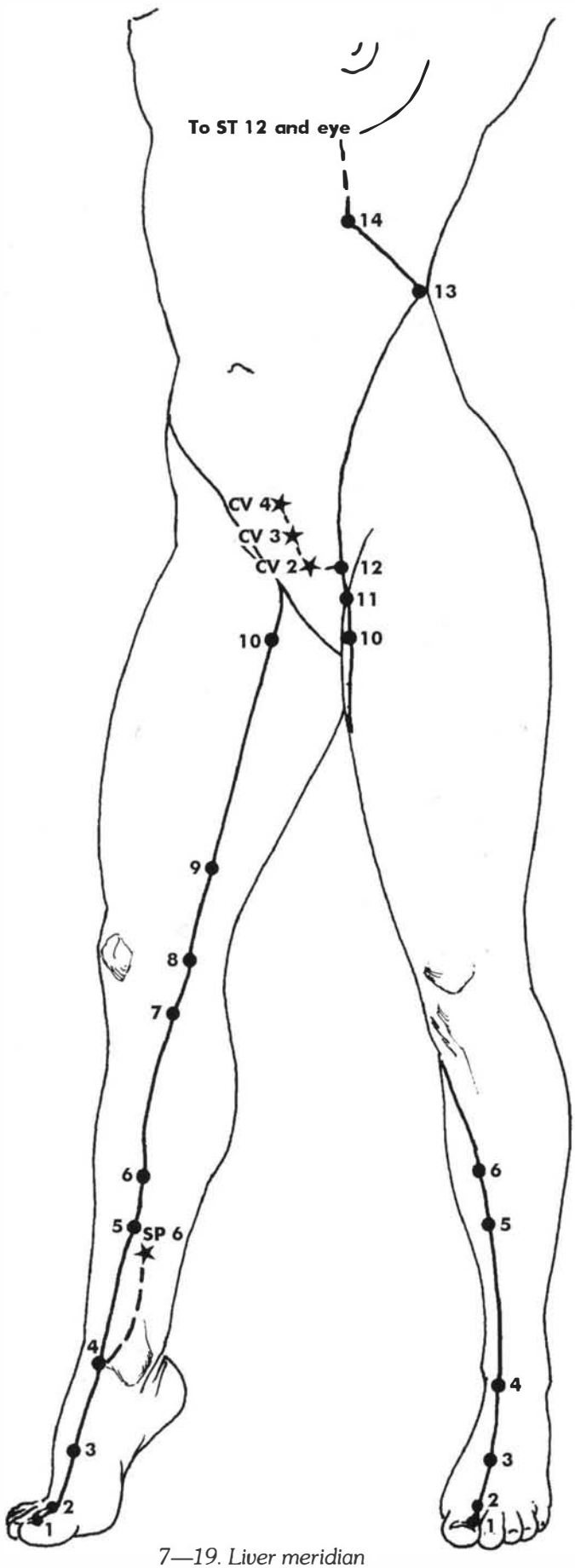
Used for knee pain; is a point frequently indicated for use in the applied kinesiology Melzack-Wall technique for pain control.

Tonification point

LV 14 Upper edge of the eighth rib directly inferior to the nipple.

Alarm point for LV

BL 18 **Associated point** between T9-10 transverse processes.



7—19. Liver meridian

Conception Vessel — CV

Begins on the center of the perineum and ends in depression between lower lip and chin.

Muscle association: supraspinatus.

CV 1 Center of the perineum, between the anus and scrotum in the male and the anus and posterior labial commissure in the female.

CV 2 Superior edge of the symphysis pubis.

A contact point used in applied kinesiology for CV-GV switching.

Used in any pelvic cavity problem.

CV 3 One tsun above CV 2.

Alarm point for BL.

CV 4 One tsun above CV 3.

Alarm point for SI.

CV 5 One tsun above CV 4.

Alarm point for TH.

CV 8 (Shrine of God) Center of navel.

Chinese philosophy considers Chi to enter the body here at birth and leave from here at death.

Considered to be a powerful effect point, especially in children's disorders.

CV 12 Halfway between CV 8 (umbilicus) and CV 15 (tip of xiphoid process).

Alarm point for ST.

CV 14 One tsun below the tip of the xiphoid bone.

Used in heart conditions and hiatal hernia.

Alarm point for HT.

CV 15 At the inferior tip of the xiphoid bone.

CV 17 Two tsun above the inferior tip of the xiphoid process.

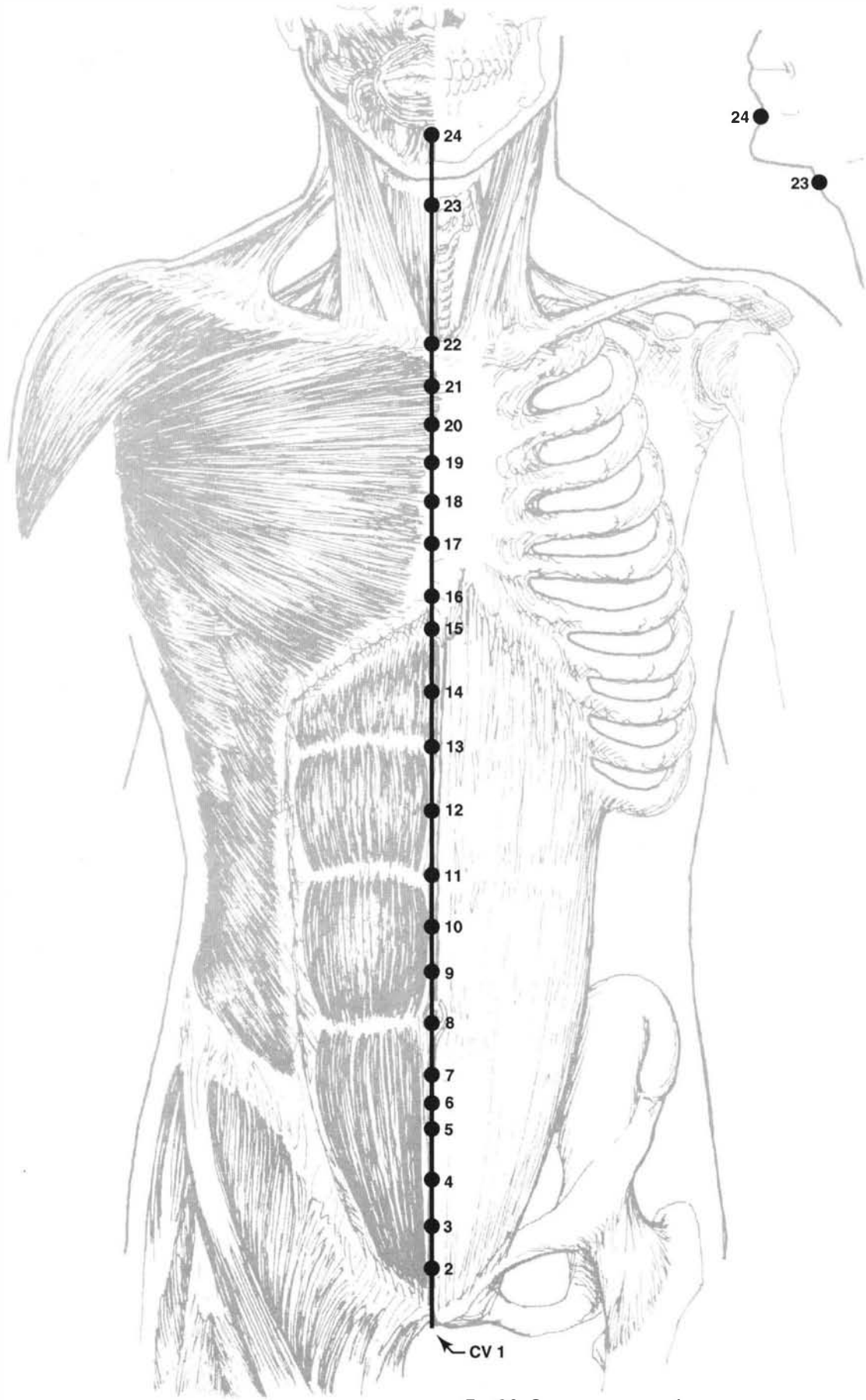
Alarm point for CX.

Influences circulation and sex hormones.

Used in respiratory problems.

CV 24 (Water ditch) In depression between the lower lip and chin.

A contact point used in applied kinesiology CV-GV switching.



7—20. Conception vessel

Governing Vessel — GV

Begins at the tip of the coccyx and ends on the inside of the lip at the insertion of the frenulum.

Muscle association: teres major.

GV 1 Tip of the coccyx.

GV 4 Between spinous processes of L2-3.

Used for adrenal stimulation and low back pain.

GV 14 Between spinous processes of C7 and T1.

Frequently stimulated with ST 12 because most of the meridians pass through these points.

GV 20 (Cure of one hundred diseases, one hundred meeting palaces or places) At the extreme top and center of the head where an imaginary line straight up from the ears crosses the sagittal suture.

Used for hemorrhoids. Use caution in stimulating with a teishein and on children, as it can cause blackouts. Electrical or laser stimulation produces no ill effect.

GV 23 One tsun above the natural hairline.

Used to rapidly open sinuses.

GV 25 At the tip of the nose.

Used to sober an inebriated person.

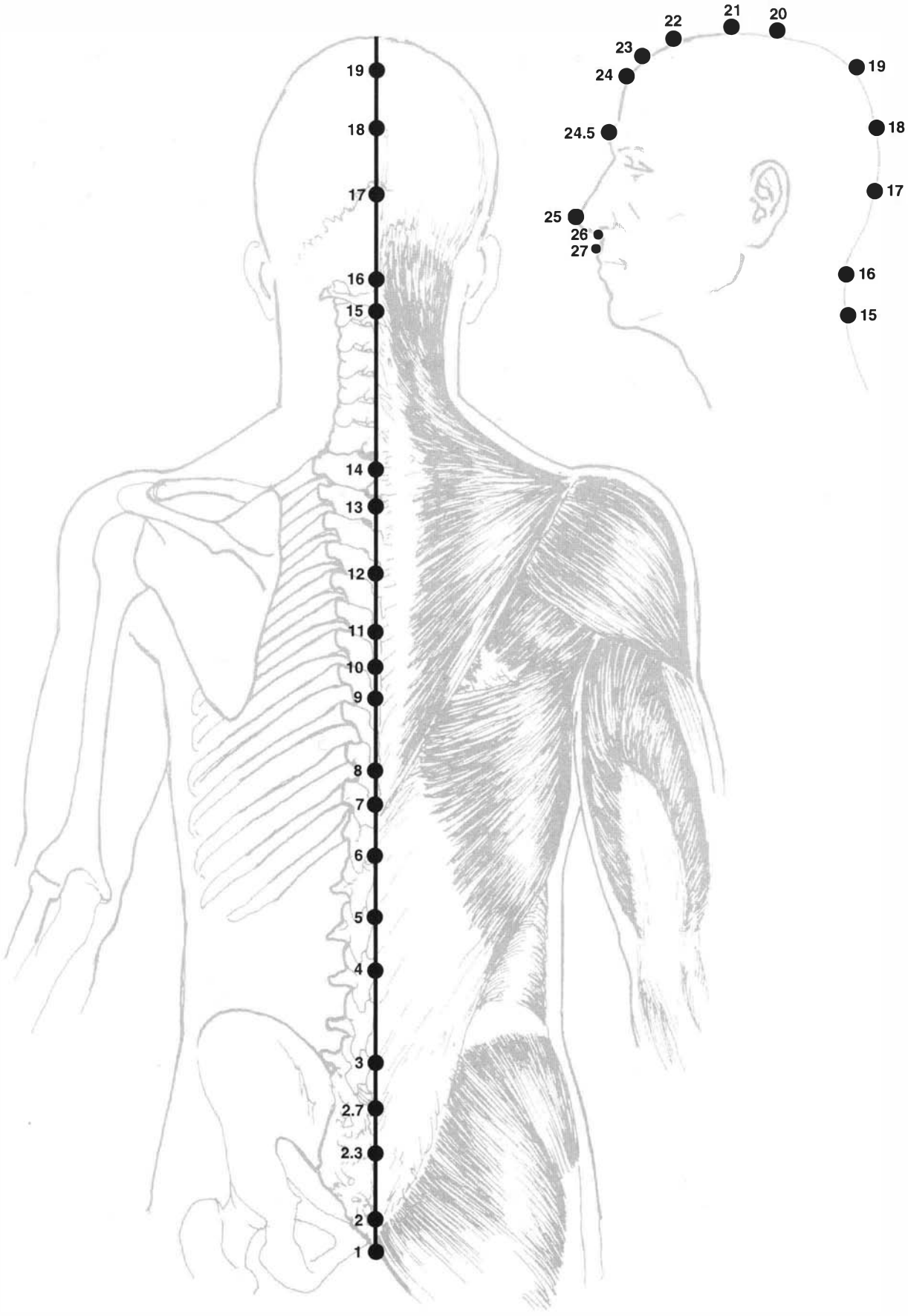
GV 26 Inferior to the nose in the philtrum.

An emergency point for fainting and shock.

GV 27 At the junction of the philtrum and upper lip.

A contact point used in applied kinesiology CV-GV switching.

GV 28 On the gingiva, inferior to the insertion of the frenulum.



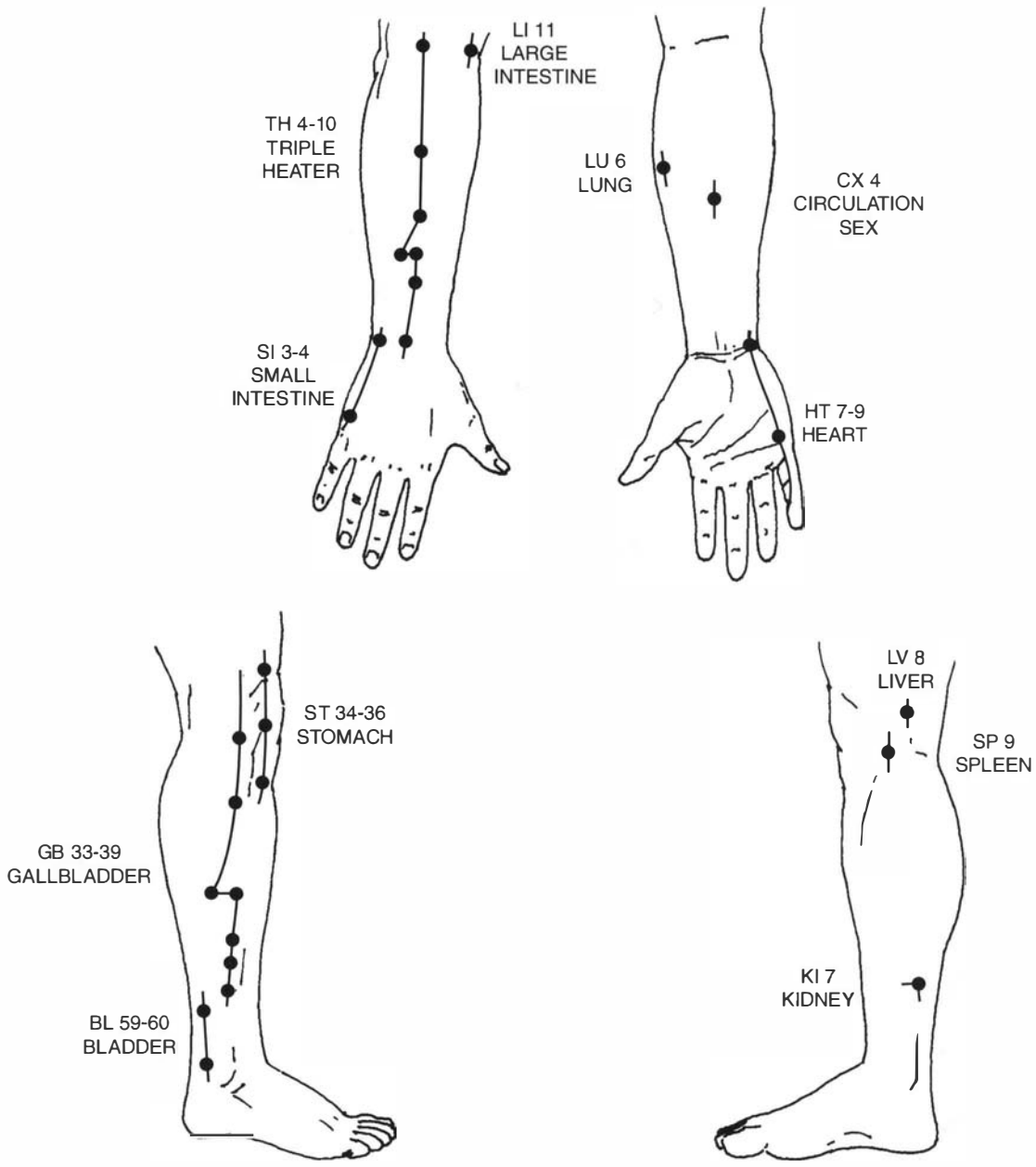
7—21. Governing vessel

Thermal Examination

Each meridian has a location that is susceptible to thermal changes with meridian over- or underactivity. When the area is cool, the meridian is underactive; when warmer than the surrounding tissue, it is overactive. The thermal difference may first be brought to the physician's attention by the patient's complaint of the area feeling hot or cold. The physician may observe it by comparing the area with the surrounding tissue, using the back of his hand. It can also be measured by thermocouple, with the exact temperature being recorded.

As with other indications of meridian over- or underactivity, the thermal areas indicate how the muscles associated with the meridian will probably function. When the thermal area is hot, the muscles of that meridian are likely to be hypertonic; when the area is cold, they will test weak with manual muscle testing. Not all muscles associated with the meridian will test weak or strong as indicated, because there is a considerable interaction of energy within the meridian system by the coupling meridians.

Meridian/Thermal Area



7—22.

Symptomatic Pattern of Meridian Imbalance

Part of the classic acupuncturist's method of diagnosing the balance of the meridian system is assessing an individual's symptomatic pattern. Although this method is not widely used in applied kinesiology for diagnosis and is not recommended here, it adds to a physician's ability to read body language and understand a patient's condition.

The primary methods of diagnosis used in applied kinesiology are thermal evaluation, pulse and alarm point therapy localization, and manual muscle testing. Measurement of the Ryodoraku and Akabane points is not a part of applied kinesiology, but it is recommended for a more thorough diagnosis and understanding of the meridian system.

There are symptoms associated with over- or underactivity of a meridian that should correlate with the diagnosis of the meridian's balance. As one's knowledge of the meridian system increases it will readily be seen that many aspects of these symptoms fit with the pattern of the meridian as being Yin or Yang, and with the characteristics of the meridian's association in the five-element law.

Knowledge of symptoms helps in questioning a patient about his condition. In most cases, the symptoms do not indicate the method of treatment; rather, they help understand the patient's condition. They often explain the complaint of thermal variations. Correlate the thermal area examination with pulse diagnosis, alarm points, and muscle tests.

Meridian	Thermal Area	Meridian Excessive — Warm	Meridian Deficient — Cool
Lung	**LU 6**	Chest heavy, shortness of breath, heavy cough, and copious amounts of mucus.	Patient feels like he is coming down with a cold; symptoms of sneezing, sniffles, cough, and chills.
Large Intestine	**LI 11**	Shoulder pain, dizziness, and shoulder pain associated with constipation.	Gas, dry lips, and constipation.
Stomach	**ST 34-36**	Overeating, warm feeling, and pain at lateral thigh.	Loss of appetite, no taste for food, legs seem weak.
Spleen	**SP 9**	Patient wants to rest, appetite will vary, patient will want something to eat but does not know what he wants.	Patient will crave sweets, have loss of memory, be sleepy during day and wake up during night; excessive gas.
Heart	**HT 7-9**	Patient talks a lot, has dry mouth, feels heaviness in chest.	Overwhelming fatigue, fear, or dread.
Small Intestine	**SI 3-4**	Pain at sphenoid sinus and down sides of the neck, shoulders, and arms; noisy bowel but no flatulence.	Head hurts on one side with pain around ears; gas which is odorless.
Bladder	**BL 59-60**	Spinal pain concentrated around waist and down both legs.	Frequent urination; general spinal pain from shoulder to coccyx.
Kidney	**KI 7**	Patient has a lot of intestinal activity, including peristalsis and gas movement but not necessarily flatulence. The urine has a dark color. The patient has a great deal of energy.	Lacks courage, timid, lacks self-assurance, lacks sexual interest.
Circulation Sex	**CX 4**	Heavy head feeling with headaches, upper abdominal distress, light sleep with dreaming.	Sleeps poorly, fear of heights, head feels heavy.
Triple Heater	**TH 4-10**	Patient has trouble hearing, although audiometer test is normal.	Patient cold all over, sphenoid sinus pain.
Gallbladder	**GB 33-39**	Head and stomach seem heavy, pain and cramps along the anterolateral abdominal wall.	Relative acidosis which causes patient to sigh; has chills, dizziness, and also tendency to stumble.
Liver	**LV 8**	Patient excitable and cries easily, starts on several different projects at once and goes in different directions at one time; has an obsession to keep going until everything is done.	Visual disturbances, cannot use eyes for a long period; has short temper and will fly into rage over nothing; dizziness and clumsiness present.

Alarm Points

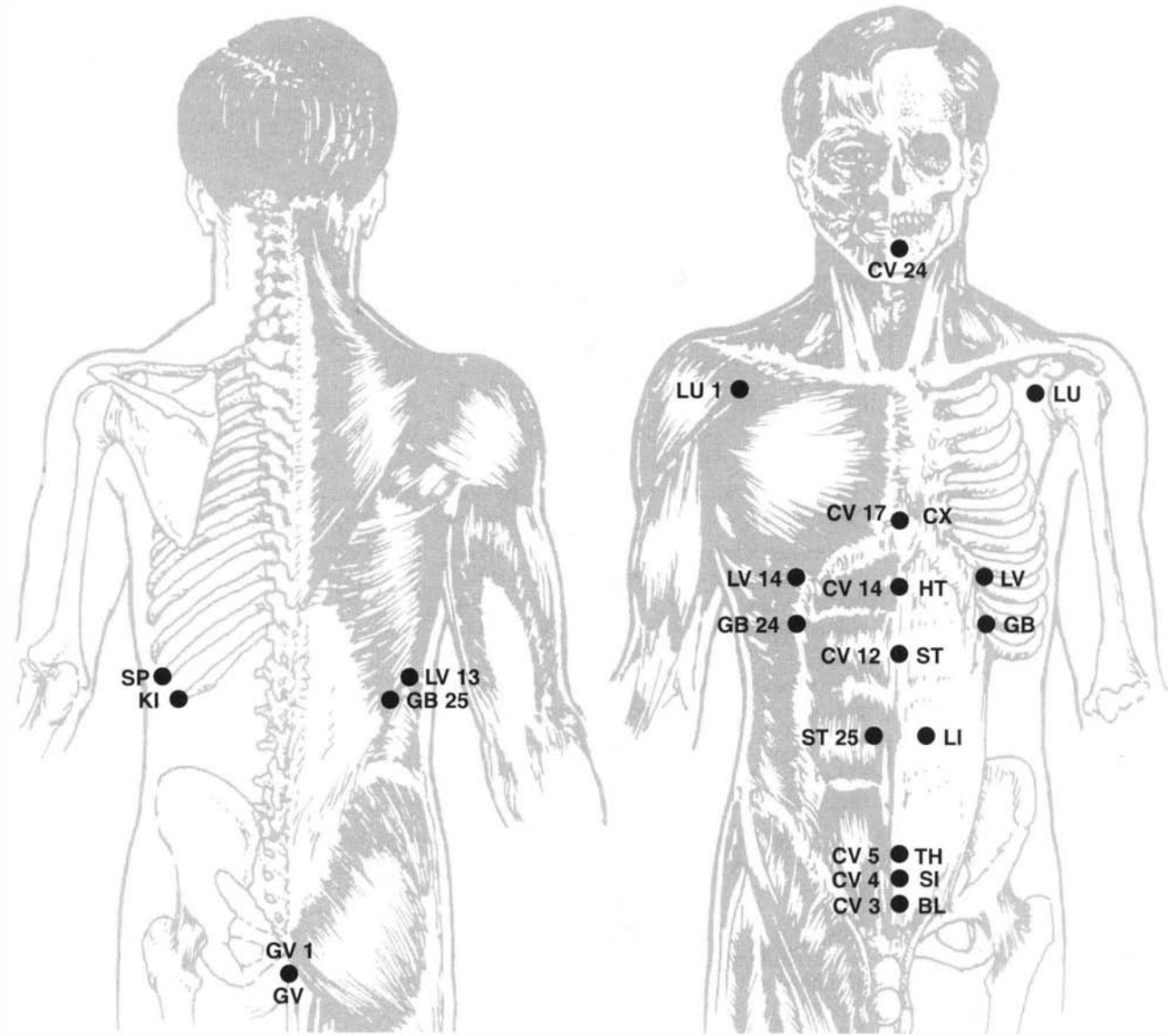
Each of the twelve meridians has an alarm point. Only the lung, liver, and gallbladder meridians have their alarm points on their meridians. In addition to their own alarm points, the liver meridian has the spleen alarm point and the gallbladder meridian has the one for the kidney meridian. Six alarm points are on the conception vessel.

In classic acupuncture an alarm point is both diagnostic and therapeutic. The diagnostic aspect is very important in applied kinesiology. When a meridian is out of balance, the alarm point is tender. When a patient complains of spontaneous pain at an alarm point, the meridian is probably overactive. When there is tenderness on palpation but no spontaneous pain, the meridian is probably underactive.

If a meridian is out of balance with the rest of the system, its alarm point will be active to therapy localization. A muscle that tests weak in the clear can be evaluated for meridian dysfunction by therapy localizing the alarm point and re-testing the muscle. If deficiency in the meridian is the cause of the weakness, the muscle will now test strong. Other diagnostic methods using therapy localization of the alarm points are described with the circulation of energy and five-element law.

An alarm point can be challenged; the examiner sharply taps it with his fingertip. If the meridian is involved, a previously strong indicator muscle will weaken temporarily.

Elimination of positive therapy localization and challenge of the alarm point indicates an improvement in the meridian's energy balance, but it does not totally rule out an additional involvement. There is considerable interplay among the meridians, and all methods of diagnosis should be used.



7—24. Alarm points. The labels on the figure's right are the meridian point numbers and on the left are the meridan alarm point names.

Pulse Diagnosis

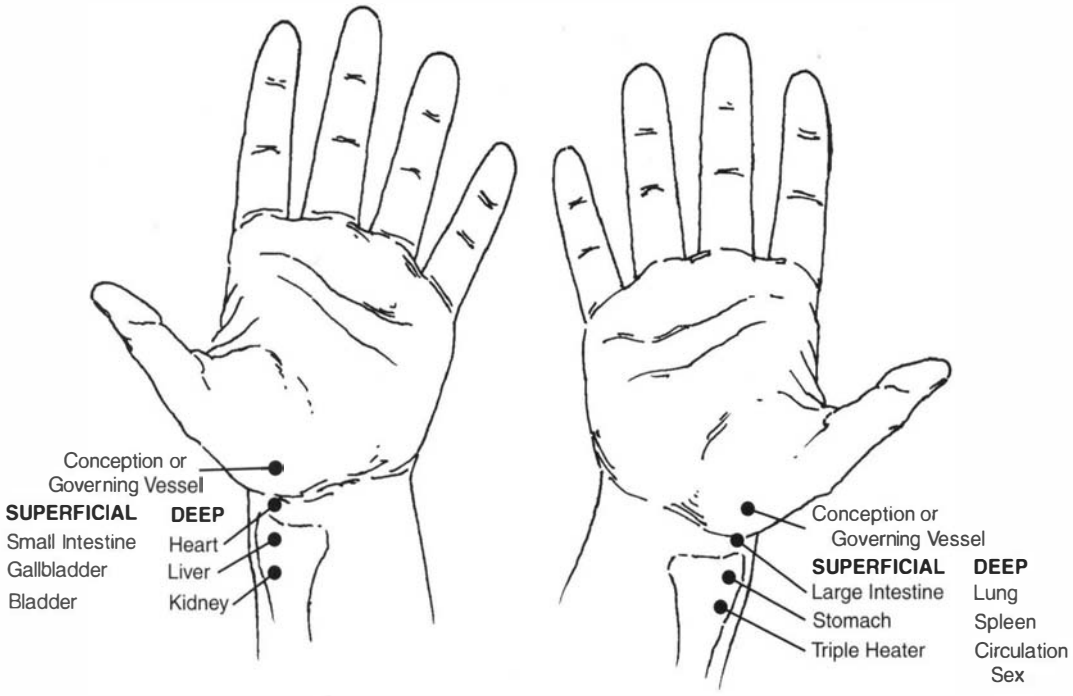
In classic acupuncture diagnosis, twelve pulses are used to determine the balance of Chi in the meridians. There are six pulses on each radial artery near the wrist; three pulses are superficial, and three are deep. Each pulse represents a different meridian. Goodheart[10] describes an additional pulse point for the conception and governing vessels, located distal to the standard distal point.

The Western physician is familiar with the radial pulse only as a location for taking the pulse rate. Most have probably noticed that the pulse can often be better felt at a slightly more distal or proximal location. This is probably the result of more or less contraction of the vessel's muscle fibers, and is the same thing responsible for the different pulses felt by the classic acupuncturist.

Variation of pulses has been picked up with pressure transducers and recorded on strip recorders. This objective method has confirmed there are three separate pulses that have a shallow and deep characteristic when the external pressure on the artery changes.[3] Sixteen types of pulses have been objectively recorded.[3] The objective methods of recording the pulses have not yet been introduced into clinical practice.

It takes many years and great care for the classic acupuncturist to learn to palpate the pulses. The patient's wrists are placed on a special pillow to cause a slight dorsiflexion. The pulses are palpated for special characteristics, such as rhythm, volume, tension, irregularity, intermittence, rolling, thready, wiry, and other factors. First the superficial pulses are felt to compare the strength, rate, and special characteristics. When there is balance of energy in the meridians, the characteristics of the pulses are the same. If one pulse is stronger than the others, an excess of Chi in the meridian represented by that pulse is indicated. A weak pulse represents a lack of Chi.

The deep pulses are then evaluated by placing a stronger pressure on the artery, but not strong enough to occlude it. The deep pulses represent the Yin meridians and the superficial pulses are Yang. The superficial and



7—25. Pulse points.

deep pulses at the same location are coupled meridians; that is, they share the same element on the five-element law.

Few in the Western world use pulse palpation to diagnose the energy in the meridians. More recently developed techniques to evaluate balance of Chi in the meridians — such as muscle testing described in this text, and electrodiagnosis — have mostly replaced pulse diagnosis in the conventional manner.

Pulse points can be therapy localized by having the patient put his finger over a pulse point while the physician tests a previously strong indicator muscle for weakening. The pulse points are directly over the radial artery, with the center pulse point at the radial apophysis. The first and third points are adjacent to the center one by approximately the width of the patient's forefinger. The conception or governing vessel point is one tsun distal to the most distal conventional point.

It has been stated that light therapy localization tests the superficial meridian, while heavier therapy localization evaluates the deep pulse point. There does appear to be a difference in therapy localization with different levels of pressure; however, it is difficult to distinguish between the two. It is probably best to therapy localize with different pressures and, when positive, use other diagnostic methods to determine which — or if both — of the superficial and deep pulses are involved.

The usual method for therapy localizing the pulse points is to first use a screening test for all the points on one wrist. The distance between the pulse points is approximately the width of the patient's fingers. The pulses are therapy localized with the patient's other hand. Lightly put the patient's middle finger on the radial artery at the level of the radial apophysis, with the index, ring, and little fingers on the adjacent pulse points. Keep the fingers close together but not touching. Test a previously strong indicator muscle for weakening, then evaluate again with deeper pressure. This screening test evaluates all the pulse points on one wrist. If the indicator muscle weakens, determine which pulse point is involved by therapy localizing each one. Of course, more than one point on the wrist can test positive.

Pulse point diagnosis in applied kinesiology is most effective in determining deficient meridians. When therapy localization is positive over a pulse point, test the associated muscles for confirmation of the involvement. For example, when therapy localization over the middle point on the left wrist is positive, test the pectoralis major (sternal division) and popliteus muscles, associated with the liver and gallbladder meridians respectively. The muscles are tested bilaterally. If one of the muscles tests weak because of deficiency in the meridian, it will test strong with therapy localization to the meridian's alarm point.

When there is no positive therapy localization to the pulse points, subclinical involvement can be brought out by having the patient stop breathing before the test. This appears to lower the general energy in the meridian system, with loss of the diaphragm's mobilizing effect on Chi. Under normal conditions there will still be no positive therapy localization to the pulse points. When asking the patient to stop breathing, it is usually best to say, "Don't take a breath, but stop breathing." The average individual tends to immediately take a deep breath when asked to stop breathing.

When there is positive therapy localization to the conception or governing vessel point, evaluate the alarm points and associated muscles to determine if the conception vessel or governing vessel is involved. The conception vessel (Yin) and governing vessel (Yang) are not as often involved because few people have an all-Yin or all-Yang condition.

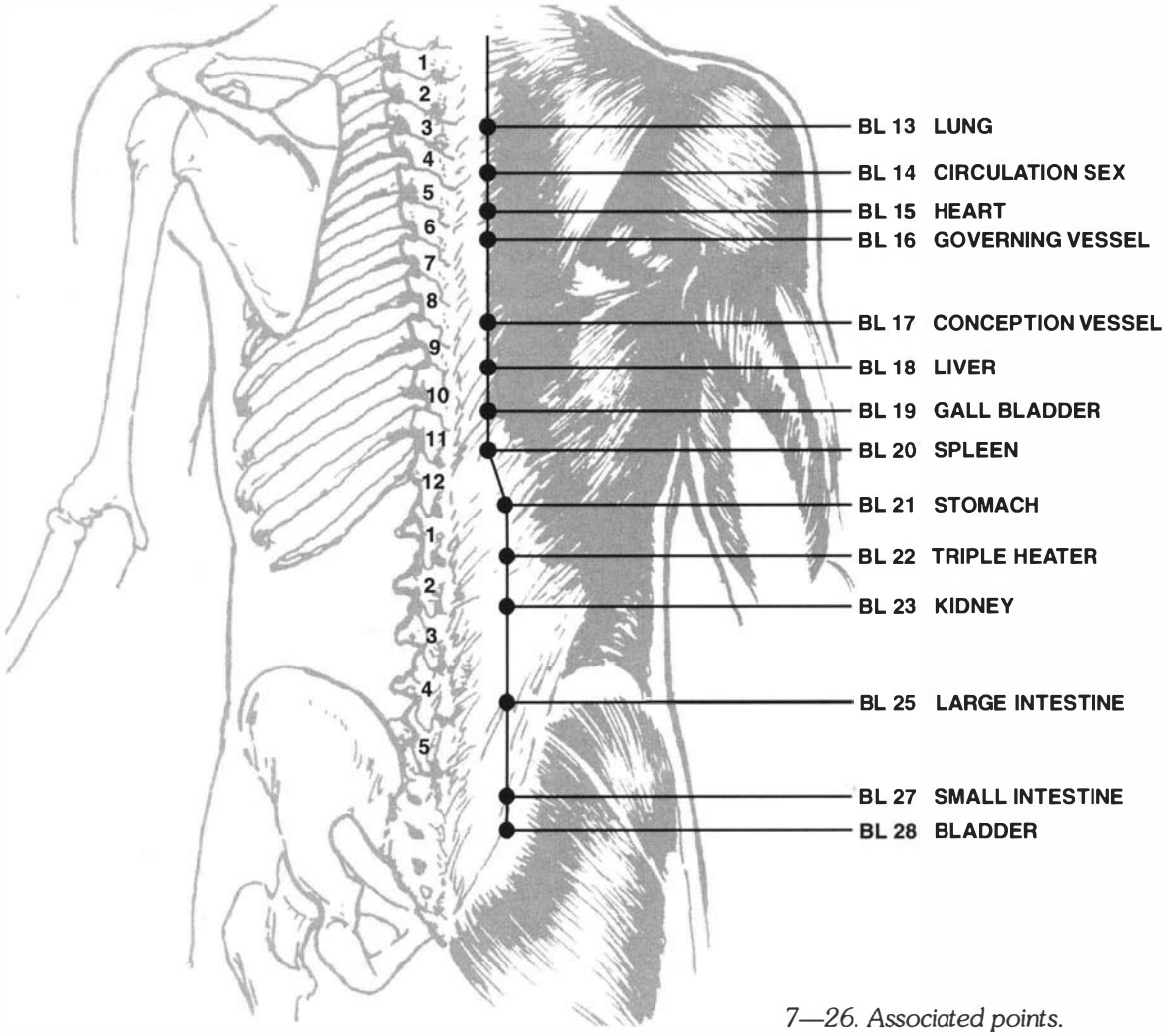
Associated Points

There is an associated point for each of the twelve bilateral meridians and for the conception and governing vessels. They are located on the bladder meridian two tsun from the midline down to L1; from there down they are two and one-half tsun from the midline.

There is a close correlation between the associated points and the function of the adjacent vertebrae. When a meridian's Chi is out of balance, its associated point will be active and very often cause a vertebral subluxation at its level of the spine. For example, when the lung meridian is overactive, BL 13 will be active. It will be positive to therapy localization and probably cause a subluxation of T3 or T4. The subluxation may have a rebound or holographic challenge. It may therapy localize with one hand or require the two-handed challenge of the holographic subluxation.

The interplay between the associated point and the subluxation appears to be of a reflex nature. If the subluxation is corrected but the meridian is not balanced, the subluxation often returns. Those unaware of the diagnosis and correction of the meridian system may balance it by accident so that the subluxation does not return. In fact, the subluxation may be the primary factor causing the meridian imbalance. This is a two-way street; the meridian imbalance can cause a subluxation, or the subluxation can cause meridian imbalance.

When a health problem is chronic, there are usually many factors contributing to it. The problem may

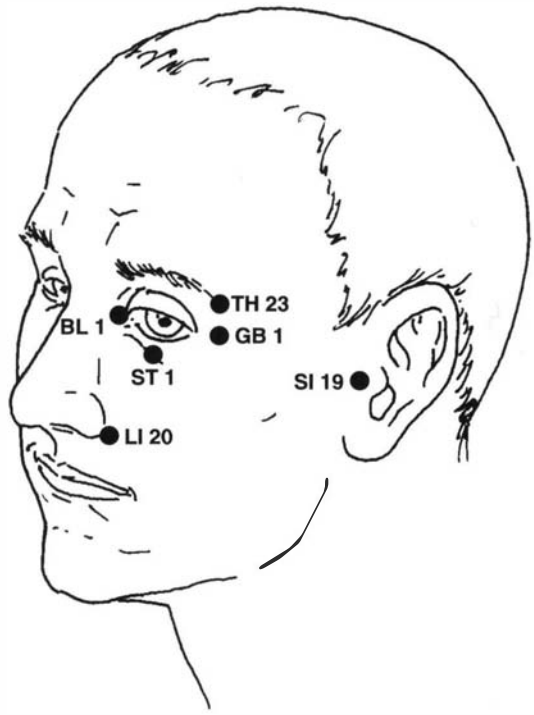


7—26. Associated points.

originate from only one cause, but as it persists other parts of the controlling mechanism become involved. Good procedure is to examine and correct all systems. When the primary method of treatment is directed to the meridian system, evaluate for subluxations adjacent to the associated points of the imbalanced meridians and correct them when necessary. When the primary system of correction is adjusting subluxations, evaluate the meridian system to determine if additional correction to it is necessary.

Beginning and Ending Technique

A meridian technique that has developed in applied kinesiology is called the "Beginning and Ending Technique" (B and E). It consists of tapping the beginning or ending acu-points of the Yang meridians (those beginning or ending on the head) under certain circumstances. Application of the technique often causes dramatic changes in temperature, oral pH, vitamin C, and blood sugar levels within two or three minutes. Goodheart[13] postulates that the reason for these immediate changes is that the Yang meridians are intimately involved with temperature regulation of the pituitary gland, which by way of the hypothalamic-pituitary axis provides for internal adjustments of body chemistry and regulation of various functions. The concept is that temperature regulation, especially cooling of the pituitary gland, is by blood circulation and the paranasal sinuses, with an important



7—27. Yang beginning and ending points on the head.

control by the meridian system. Goodheart supports this concept with the observation that when B and E technique is performed, temperature at the axillae and occipital areas rises, while there is a drop in temperature at the glabella. The areas are simultaneously measured by thermocouples attached to the areas prior to treatment, then observed during and immediately after treatment.

B and E technique is applied when there is negative pulse point therapy localization that becomes positive when the patient puts his eyes into distortion (see page 43). If therapy localization to the pulse points is positive in the clear, the meridian system should be balanced before applying B and E technique.

When therapy localization is negative to all four pulse points on a wrist, continue the therapy localization and have the patient put his eyes into distortion. If weakening occurs, it indicates that B and E technique would be of value. Determine which meridian is involved by continuing the therapy localization, with EID, to the individual pulses. Further confirmation of the need for B and E technique is obtained when the muscle(s) associated with the positive meridian react to the eyes into distortion in the same manner. Test the muscle(s) associated with the Yang meridian of the pulse point bilaterally. The involved muscle will usually be strong in the clear but will weaken with EID. While continuing EID, determine which phase of respiration abolishes the weakness. The beginning or ending of the meridian located on the head is then tapped vigorously while the patient holds the phase of respiration that abolished the weakness. While tapping the beginning or ending point of the meridian, temperature at the glabella will lower. If one also monitors the axillary temperature or the skin over the occiput, one will usually observe an elevation of temperature.

An example of the sequence of evaluation and treatment when the pulse points have negative therapy localization in the clear is as follows. The patient's postural carriage is with the head tilted to the left. With the eyes down and to the left, the pulse points on both wrists are screened; positive therapy localization is found on the left. Individual pulse-point therapy localization is positive only at the most proximal point, which is bladder and kidney. Since eyes into distortion always deals with the Yang meridians, the bladder muscles — peroneus longus and brevis, peroneus tertius, and tibialis anterior — are tested. If all other factors have been cleared prior to testing for EID, the muscles will be strong in the clear. One or all of the muscles will weaken with EID, either unilaterally or bilaterally. Find the phase of respiration that causes the muscle to regain strength while EID is held. Finally, vigorously tap the beginning or ending meridian point on the head. In this case, BL 1 is located slightly medial and superior to the medial canthus of the eye. Of course, one should take care not to strike and injure the eyeball. If the response is inadequate, it may be necessary to tap the other end of the meridian simultaneously. These are all located at a nail point of a finger or toe. To maintain precision tapping, it may be necessary to have an assistant tap one end of the meridian while the physician taps the other.

Most often the treatment does not need repeating. Hypothalamic nutritional support is routinely used and seems to help prevent any need for further treatment. Goodheart[14] routinely evaluates for and corrects any cranial dysfunction found. Cranial faults entrapping the jugular vein at the jugular foramen can lower venous drainage, possibly contributing to poor circulation around the pituitary gland.

B and E technique has been clinically effective in improving chemical regulation in the body and sensory function.

Circulation of Energy

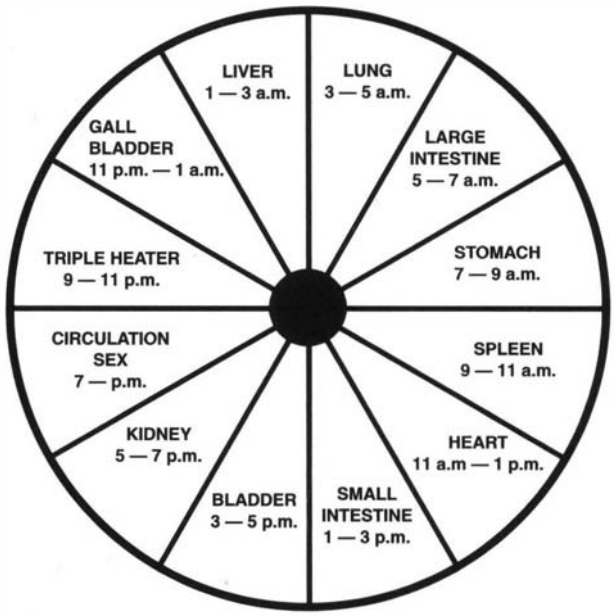
As discussed previously, energy enters the meridian system at the lung meridian and travels in a wave down an arm, up an arm, through the body, down a leg, up a leg, through the body, and continues the cycle in an arm. The meridians, or channels of the body, can be likened to a series of irrigation channels with water flowing continuously. Each meridian has a period when its energy is highest, with the high energy going on to the next meridian successively until all meridians are covered in twenty-four hours. The high-energy period is called the horary period.

The horary period is in relation to the position of the sun. The time on the clock is only correct if you are in the middle of the time zone in which you operate. For example, if your location is close to the western border of the Eastern time zone, your sun time is one-half hour behind; therefore 3:00 a.m. would actually be 2:30 a.m. During daylight saving time, an additional adjustment must be made for the one-hour lag of the sun. In the summer, the western border of the Eastern time zone sun time would be 1:30 a.m. The two adjustments for the western border of the Eastern time zone would make the high energy time of the lung meridian from 1:30-3:30 a.m., instead of from 3:00-5:00 a.m. as is listed on the chart of circulation of energy.

Optimal health is balance in the meridian system, with a continuing rise and fall of the energy pattern according to the twenty-four-hour circulation of energy. Many factors can disrupt this pattern and cause poor health symptoms. A temporary disruption takes place when an individual travels rapidly from one time zone to another, such as in jet airplane travel. Jet lag affects some individuals more than others. Those who have a high energy level in the meridian system, with little or no interference to its innate balancing mechanism, have minimal symptoms following even long journeys. The best treatment for an individual suffering from jet lag is to balance the meridian system and improve its energy level.

Midday/Midnight Effect

The midday/midnight effect refers to the opposing relationship of meridians on the twenty-four-hour circulation of energy. For example, the lung meridian has its highest energy from 3:00-5:00 a.m. Since the bladder meridian is twelve hours opposite the lung, it has its lowest energy from 3:00-5:00 a.m. The high and low periods of a meridian's energy are the times when symptoms are most likely to develop as a result of imbalance in that meridian. If an individual consistently awakens with a headache at 2:00 a.m., the problem is likely in the liver, which has its highest energy at that time, or in the small intestine, which has its lowest energy. Examine the organ relationship with all five factors of the IVF. Although evidence to examine the organs is obtained by evaluating the meridian system, meridian therapy is not necessarily the optimal therapeutic approach. Consider all the factors of applied kinesiology, such as subluxations, lymphatic drainage, and nutrition, among others, as well as meridian therapy.

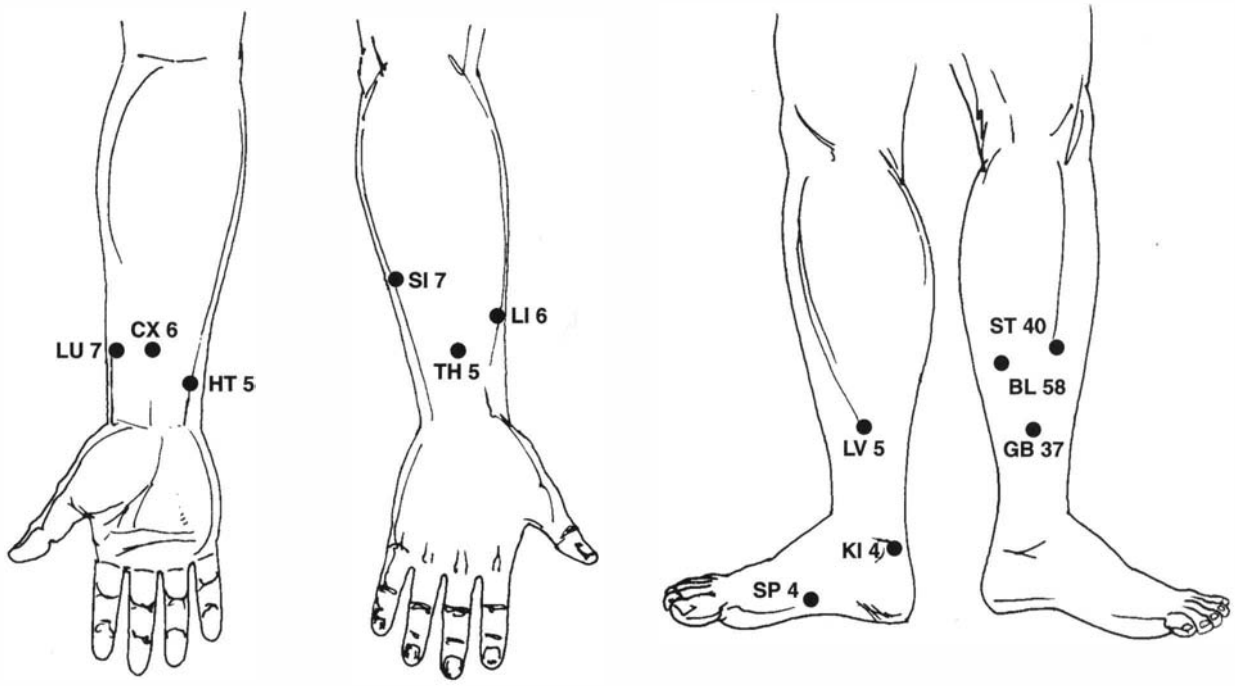


7—28.

Mother/Child Effect

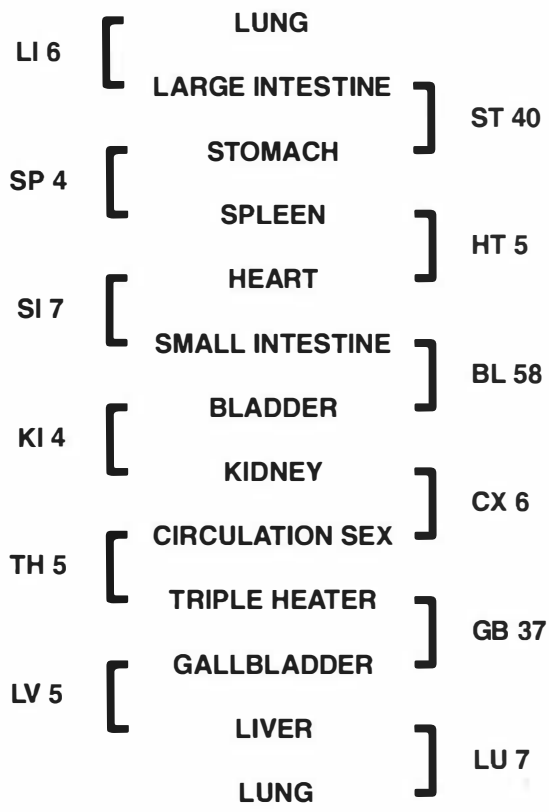
There is a relationship of the adjacent meridians in the twenty-four-hour circulation of energy that is known as the mother/child effect. A meridian that gives its energy to the next one is known as the mother, and the meridian receiving the energy is called the child. This relates in Chinese philosophy to the mother nourishing her child, and the child receiving from its mother. Thus the lung meridian is the mother of the large intestine meridian, and the large intestine meridian is the child of the lung meridian. The large intestine meridian, in turn, is the mother of the stomach meridian, and the stomach meridian is the child of the large intestine meridian, and so on.

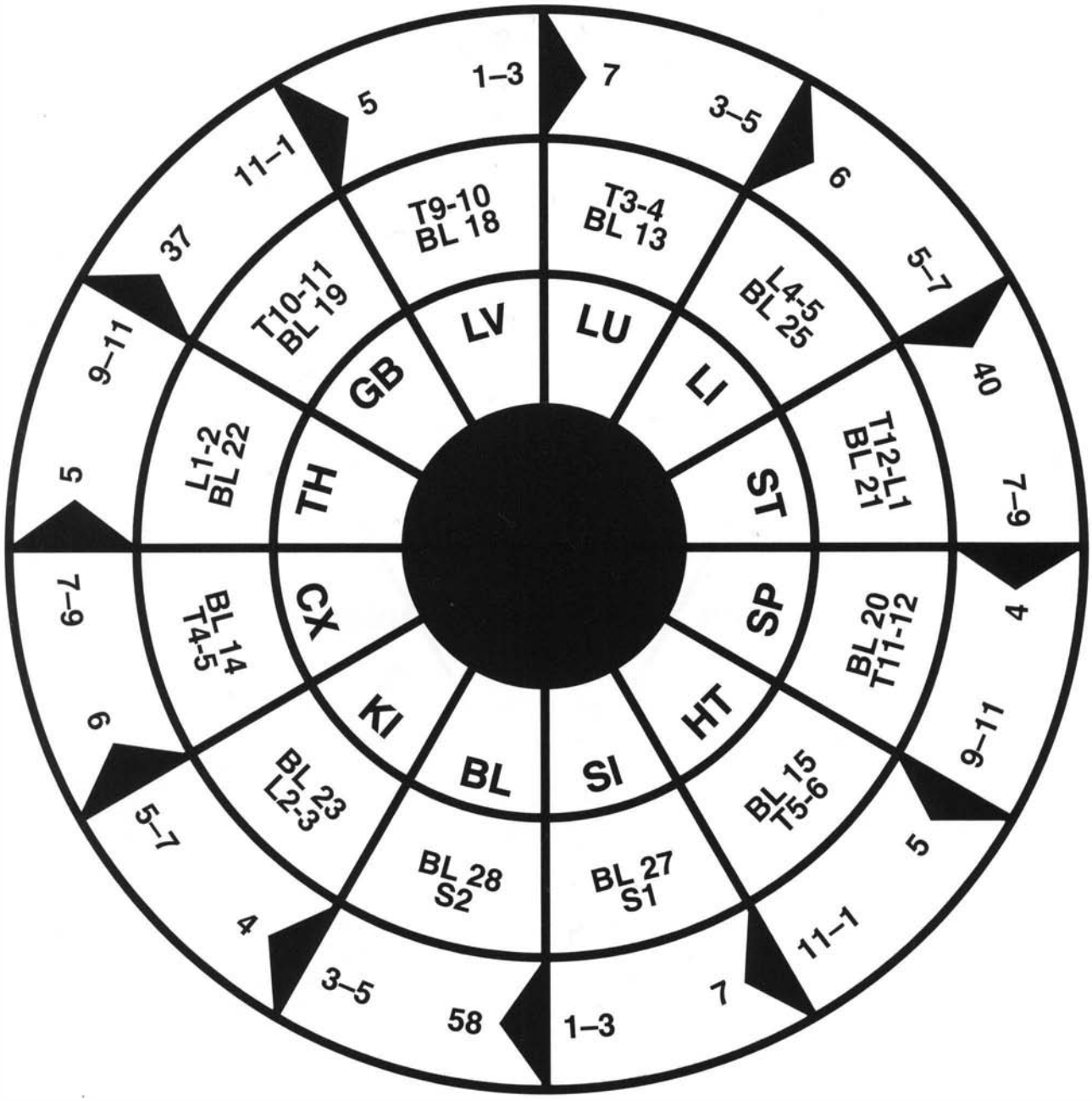
When several meridians are deficient within the sequence of the twenty-four-hour circulation of energy, it is often because energy is blocked at a meridian so that the flow cannot continue. The meridian holding the energy is considered the mother meridian to the sequential child meridian. If the mother meridian cannot give its energy to the child, there will be a continuing circulation of energy deficiency in the meridians until the energy can be restored by the coupling meridians, most of which have not been discussed here. The rapidity with which the energy is restored depends upon the efficiency with which an individual's meridian system is working.



7—29. The location of the luo points for each meridian is given above. The point on the first deficient meridian in the twenty-four-hour circulation of energy is stimulated to "bring the energy through." The associated weak muscle of the deficient meridian(s) should then test strong. Failure to strengthen indicates some other of the five factors is involved.

7—30. Luo points to bring energy through when held in a meridian. If the muscles of the small intestine test strong and the bladder, kidney, and circulation sex muscles test weak, therapy localize the small intestine alarm point. If the muscles in the circulation of energy order become strong, stimulate BL 58.





7—31. This twenty-four-hour clock gives the horary period, luo point, and associated point and its location for each meridian. For example, the lung meridian's horary period is 3-5 a.m., the luo point is LU 7, and the associated point is BL 13, located between T3 and T4 vertebrae. When energy is blocked in a meridian, causing the succeeding meridian to be weak, stimulate the luo point of the first deficient meridian following the blockage. A subluxation is probable at either T3 or T4 when the lung meridian is involved. The subluxation is revealed by vertebral challenge.

There are several methods by which the meridian holding the energy can be discovered. One method is to test muscles associated with the deficient meridians in reverse order of circulation until a muscle is found that tests strong. For example, if the latissimus dorsi associated with the spleen meridian tests weak, test the pectoralis major (clavicular division) associated with the stomach. If it is weak, go backward on the clock and test the tensor fascia lata, associated with the large intestine. If the TFL tests weak, go backward on the clock and test the deltoid muscle, associated with the lung. If it tests weak, test the pectoralis major (sternal division) associated with the liver. If it tests strong, that is where the energy is probably blocked. To confirm this, therapy localize to the liver alarm point and re-evaluate the muscles that tested weak. If the liver meridian is actually responsible for holding energy and causing the others to be deficient, all the muscles will test strong with the liver alarm point therapy localization. The method to "bring the energy through" is to stimulate the luo point on the first deficient meridian — in this case LU 7. After effective stimulation, the muscles of the lung, large intestine, stomach, and spleen should test strong; in most cases they will. If a muscle fails to strengthen, evaluate it for other of the five factors of the IVF.

Although stimulating the luo point of the first deficient meridian in the blockage of circulation of energy will nearly always bring the energy through, it may not be a permanent correction. One should consider why the energy was held in the excessive meridian. It may be a natural process; the body is holding energy to combat some type of health problem. In the example of energy being held in the liver meridian, one may need to supply nutritional support or some other treatment to help correct liver detoxification. There may be a mechanical blockage of circulation of energy through the meridian. Foot subluxations are a common cause of blockage where meridians pass in close proximity. A scar across the path of a meridian is another common blockage of meridian energy. Energy can often be brought through a scarred area with the usual methods of acupuncture stimulation directly over the scar, and daily massage with vitamin E cream. Consider other factors of the meridian system, such as a subluxation close to the meridian's associated point.

Then and Now Technique

Sometimes a patient will give indication of meridian imbalance by complaining of a symptomatic pattern developing at a specific time every day or night, yet no imbalance of the indicated meridian can be found to fit the pattern. For example, the patient may wake up with a headache at approximately 2:00 a.m. every morning, yet the liver, which has its highest energy at that time, does not appear to be involved, nor does the small intestine meridian, which has its lowest energy at that time. By the time the patient gets up in the morning, the headache is gone and does not return again until the next morning around 2:00 a.m.

As so often happens, the patient is being examined at the doctor's convenience. Normally it is not reasonable for the doctor to make a house call at 2:00 a.m., or for the patient to go to the doctor's office at that time. Yet if the patient were examined during the headache period, a completely different pattern would probably be observed. Goodheart[12] developed a system that ties the time of examination into the time of symptoms.

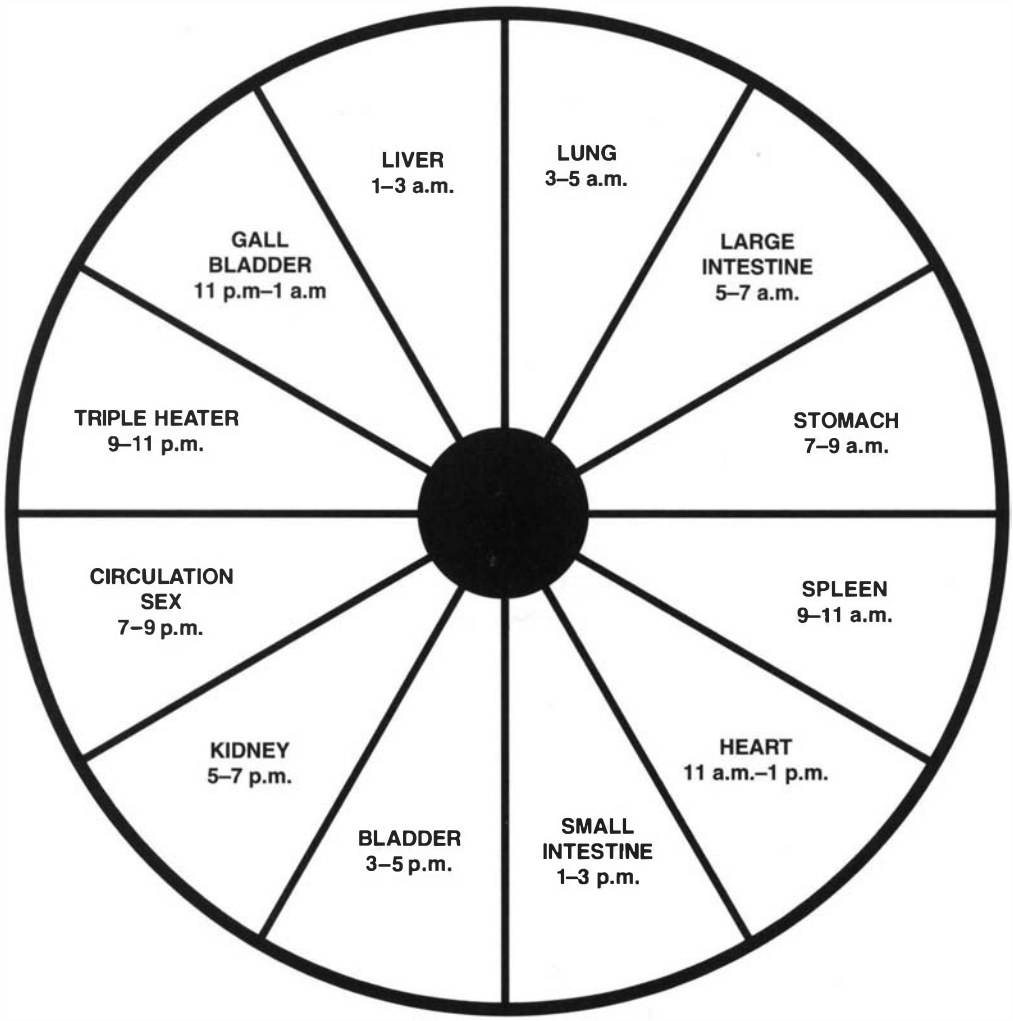
In the event that the meridian indicated by the time of symptoms (the liver or small intestine in the example) does not have positive alarm point therapy localization or a weak associated muscle, the possible involvement can be observed by tying it in with the time of examination. First the alarm point of the meridian that has its highest energy level at the time of examination is therapy localized. In the event that it does not show positive therapy localization, the alarm points of the meridian timed with the symptoms and the meridian timed with the examination are therapy localized simultaneously. For example, headaches develop at 2:00 a.m., the liver meridian's high-energy time. The examination is conducted at 10:00 a.m., the high-energy time for the spleen meridian. The alarm point for the liver (LV 14) and the alarm point for the spleen (LV 13) are therapy localized simultaneously. Positive two-point therapy localization indicates disturbance in the liver meridian at the time of its highest energy level, but not at the time of examination. The energy can be balanced by stimulating a luo point associated with the imbalance.

The proper luo point can be on any of the twelve meridians; it is the one that cancels the positive two-point therapy localization of "then and now." The luo point will be on the side of involvement and usually close to one of the meridians being tested. If the alarm point is on the conception vessel, test the muscles associated with the meridian to determine the side of involvement. SP 4 is more frequently found to be the luo point indicated than any other, regardless of what the then and now times are.

The involved luo point is found by a sharp tapping challenge while the two-point therapy localization is held. The indicator muscle weak with the two-point therapy localization will strengthen when the proper luo point is tapped.

Example as above: symptoms, 2:00 a.m., and examination about 10:00 a.m. A strong indicator muscle weakens when the alarm points for the liver and spleen are simultaneously therapy localized. First challenge the luo point for the spleen (SP 4), then re-test the indicator muscle. If the muscle doesn't strengthen, proceed to the heart luo point (HT 5). If the muscle is still weak, proceed to the small intestine luo point (SI 7), and continue on until the luo point is found that strengthens the indicator muscle weakened by the simultaneous alarm point therapy localization. When the appropriate luo point is found, stimulate by tapping for twenty to sixty seconds. There will usually be extreme tenderness at the luo point, which diminishes as the tapping continues. Continue tapping until therapy localization is abolished. Other stimulation methods can be used.

Adjacent subluxations can confuse the pattern observed during the tapping of an acu-point. For example, SP 4 is on the medial edge of the foot, just proximal to the base of the first metatarsal bone. One can inadvertently challenge mid-foot subluxations or cause a shock absorber effect, which keeps the indicator muscle weak even though adequate stimulation to SP 4 has been accomplished. On the other hand, subluxations of the foot may be what caused SP 4 to become active in the first place. Correction of the subluxation, if present, is mandatory for permanent correction of the acu-point.



7—32.

Tonification and Sedation Points

Each of the twelve bilateral meridians has a tonification and a sedation point. These are stimulated to increase or decrease the meridian's energy. The points can be stimulated individually or combined with additional acu-points to increase effectiveness. The combination of points is achieved when the main tonification or sedation point is touched simultaneously with another point, followed by the stimulation of two additional points. Most often the main point is used individually. When a continuous effect is desired, acu-aids are used on the main point, or they can be applied to all four points.

The tonification and sedation points are stimulated when one simply wants to bring additional energy into a meridian or reduce excessive energy. When there is a complex imbalance of energy within the system, it is usually better to balance the system with the five-element law or applied kinesiology's approach to the circulation of energy.

If a muscle tests weak because of deficiency in its meridian, it will become strong with therapy localization to the alarm point or to the tonification point. Treatment to strengthen the muscle is stimulation of the tonification point. Applied kinesiologists frequently stimulate the tonification point by tapping it with their fingertips. Any of the stimulation methods previously discussed are effective.

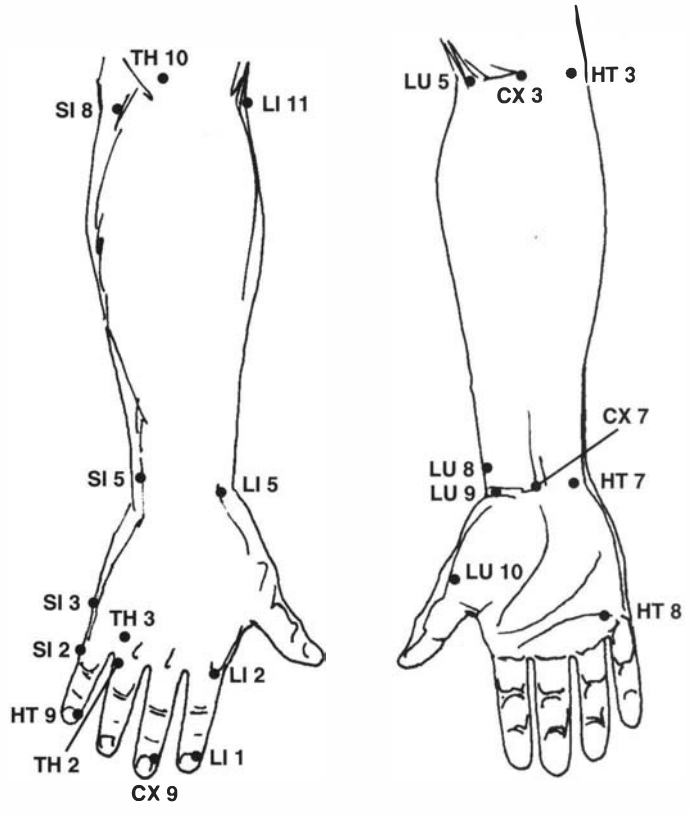
Stimulation of a sedation point is done in a manner similar to that for a tonification point. It is used to lower meridian energy and is sometimes effective in helping reduce hypertonicity of muscles associated with the overactive meridian.

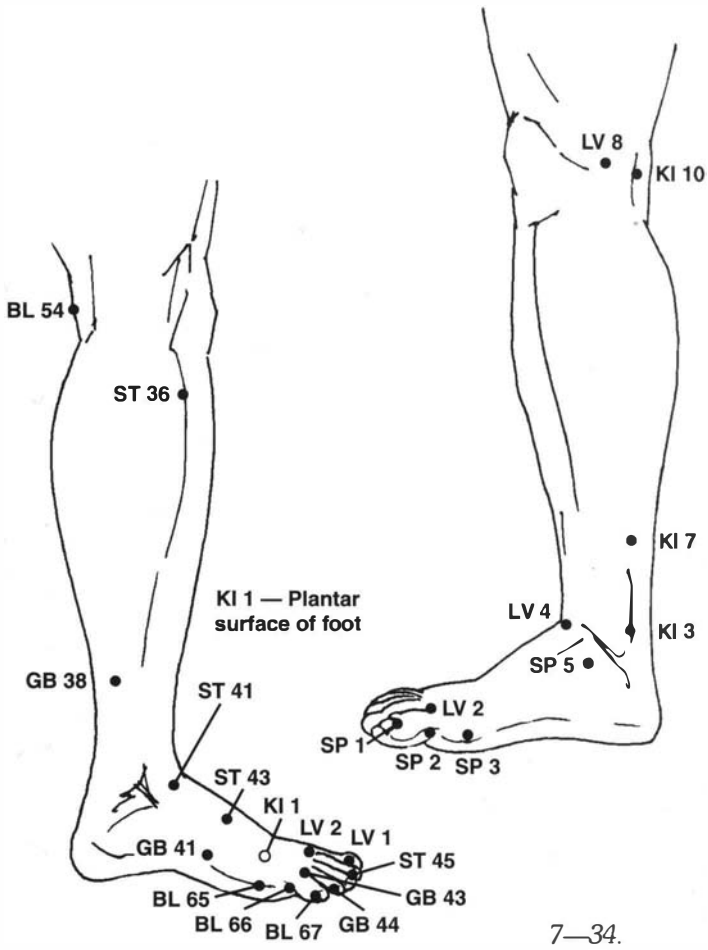
When all four points are used for tonification or sedation, the physician begins by simultaneously contacting the first two points and observing for the development of a simultaneous pulsation. Then the second two points are contacted until simultaneous pulsation is felt. For continuous stimulation to the set of tonification or sedation points, acu-aids can be put on all four points and left for two or three days. The patient can remove the acu-aids at home. If one is lost, all should be removed.

The main tonification/sedation point is larger, and the luo point is a star on the charts from Figure 7—35 through 7—58. For word description and accuracy of point location, consult the charts on pages 245 to 271.

	TONIFICATION				SEDATION			
	First		Second		First		Second	
LU	LU 9	SP 3	LU 10	HT 8	LU 5	KI 10	LU 10	HT 8
LI	LI 11	ST 36	LI 5	SI 5	LI 2	BL 66	LI 5	SI 5
ST	ST 41	SI 5	ST 43	GB 41	ST 45	LI 1	ST 43	GB 41
SP	SP 2	HT 8	SP 1	LV 1	SP 5	LU 8	SP 1	LV 1
HT	HT 9	LV 1	HT 3	KI 10	HT 7	SP 3	HT 3	KI 10
SI	SI 3	GB 41	SI 2	BL 66	SI 8	ST 36	SI 2	BL 66
BL	BL 67	LI 1	BL 54	ST 36	BL 65	GB 41	BL 54	ST 36
KI	KI 7	LU 8	KI 3	SP 3	KI 1	LV 1	KI 3	SP 3
CX	CX 9	LV 1	CX 3	KI 10	CX 7	SP 3	CX 3	KI 10
TH	TH 3	GB 41	TH 2	BL 66	TH 10	ST 36	TH 2	BL 66
GB	GB 43	BL 66	GB 44	LI 1	GB 38	SI 5	GB 44	LI 1
LV	LV 8	KI 10	LV 4	LU 8	LV 2	HT 8	LV 4	LU 8

7—33.

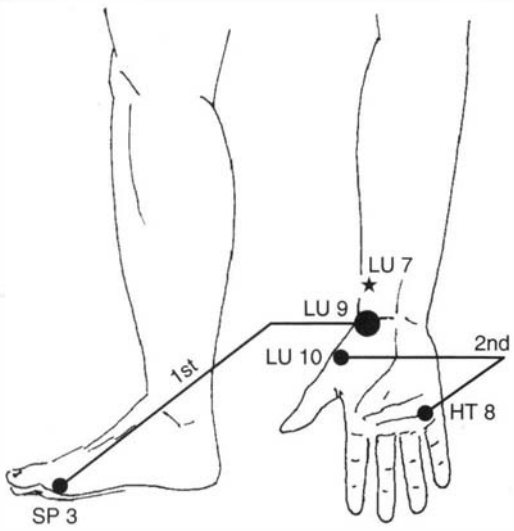




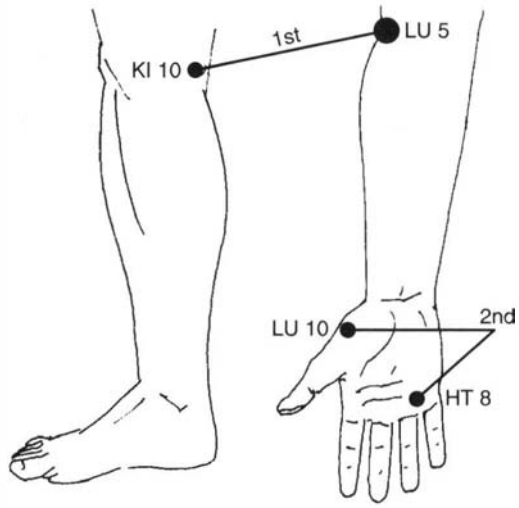
7—34.

Tonification | **Sedation**

LUNG
3 — 5 a.m.

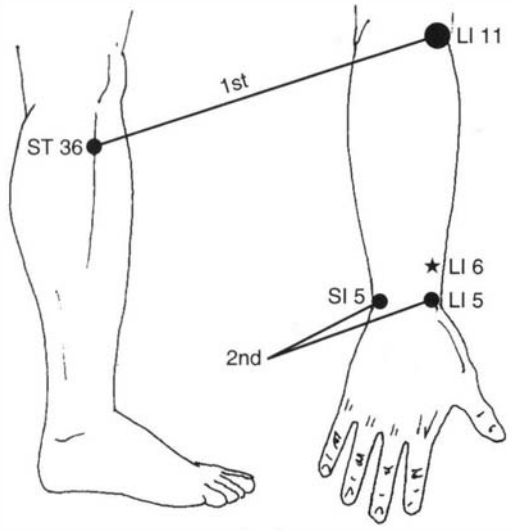


7—35.

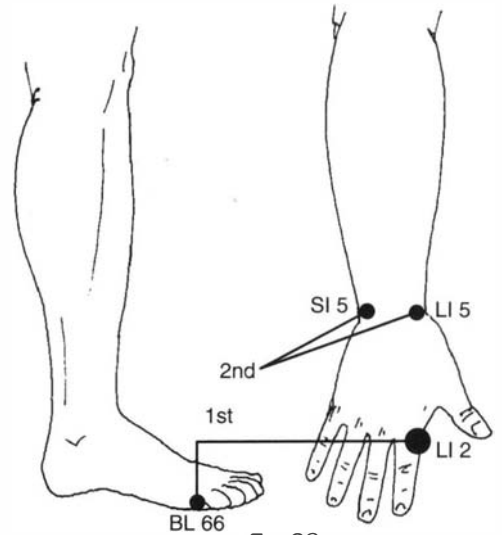


7—36.

LARGE INTESTINE
5 — 7 a.m.

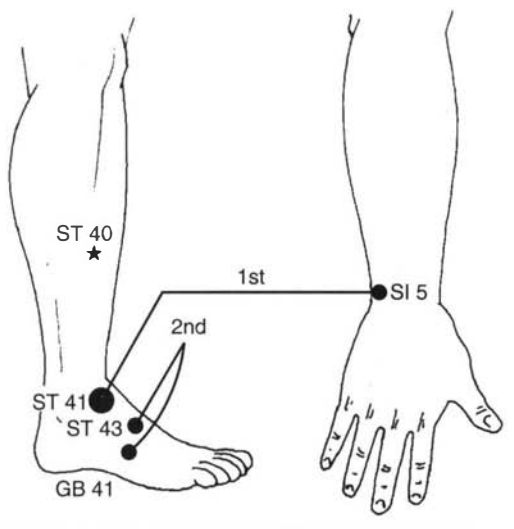


7—37.

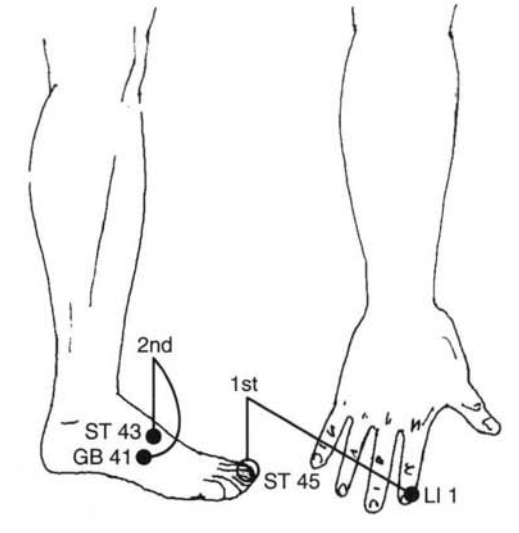


7—38.

STOMACH
7 — 9 a.m.



7—39.

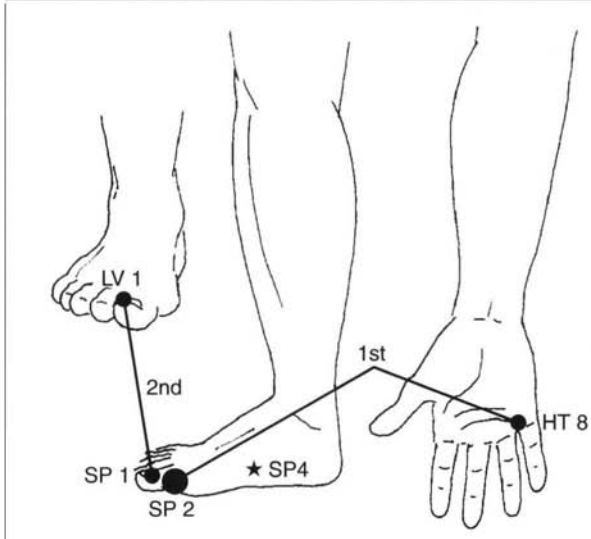


7—40.

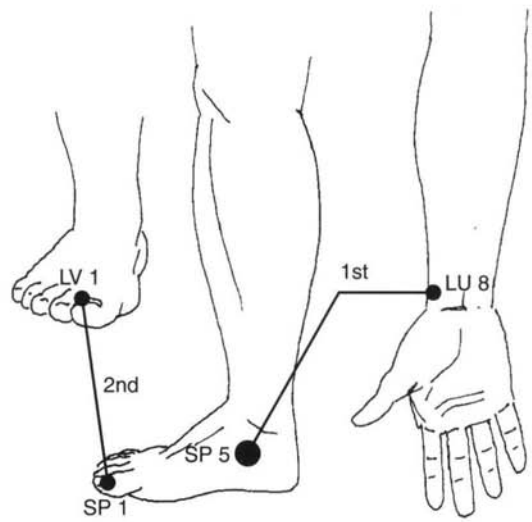
LUO POINTS

Tonification | **Sedation**

SPLEEN
9 — 11a.m.

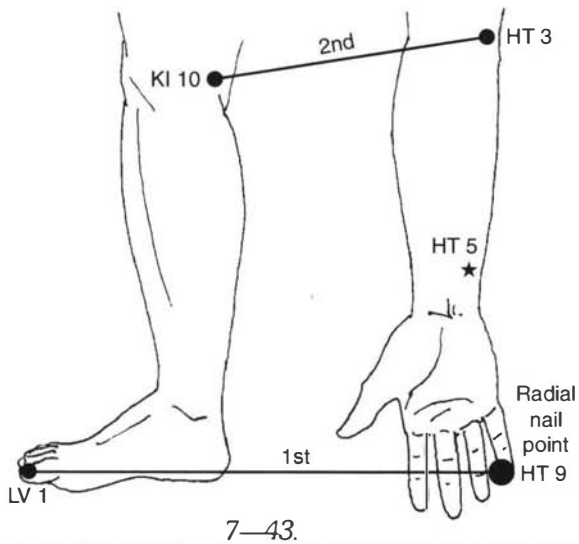


7—41.

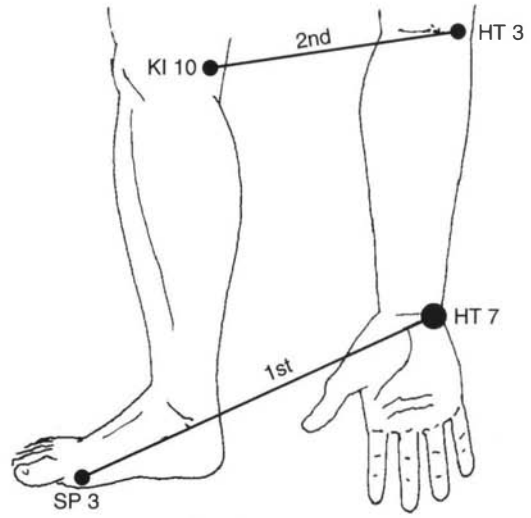


7—42.

HEART
11 a.m. — 1 p.m.

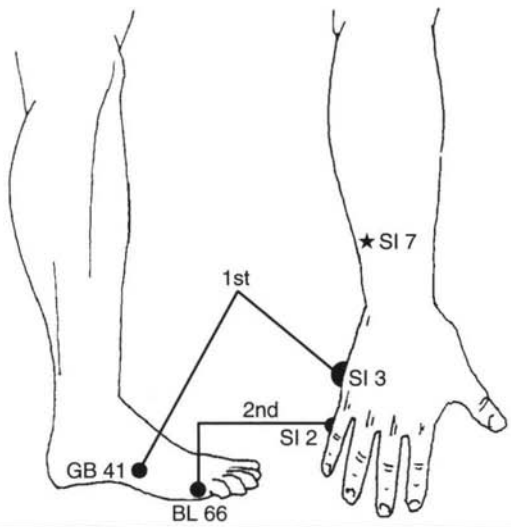


7—43.

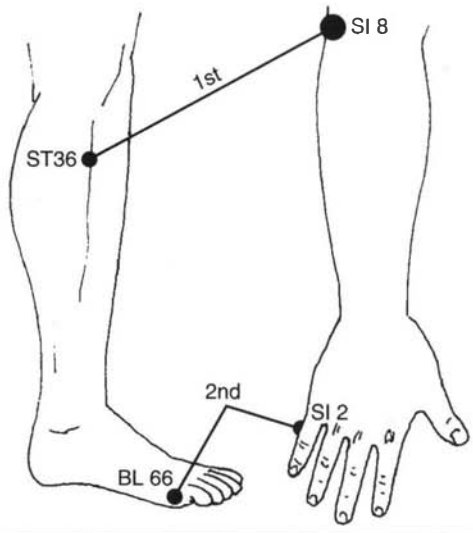


7—44.

SMALL INTESTINE
1 — 3 p.m.



7—45.

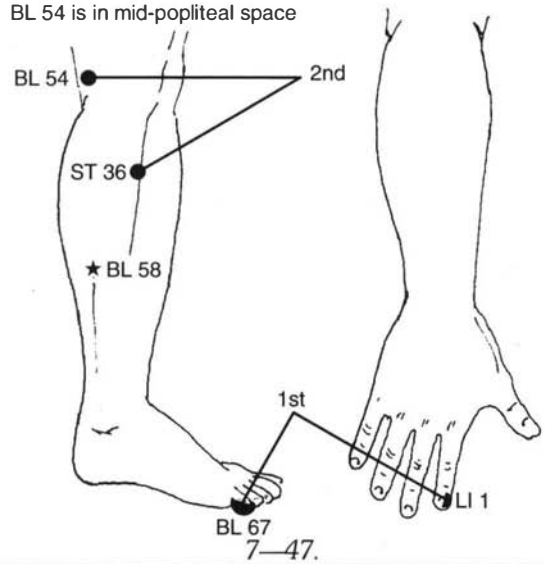


7—46.

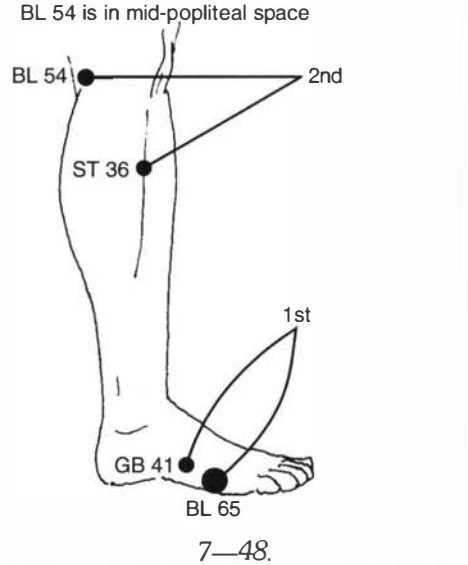
LUO POINTS

Tonification | **Sedation**

BLADDER
3 — 5 p.m.

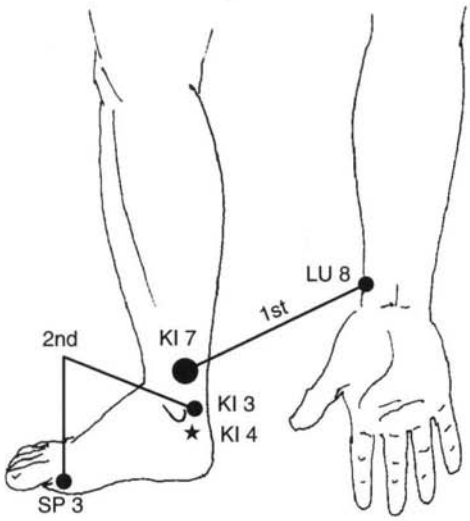


7—47.

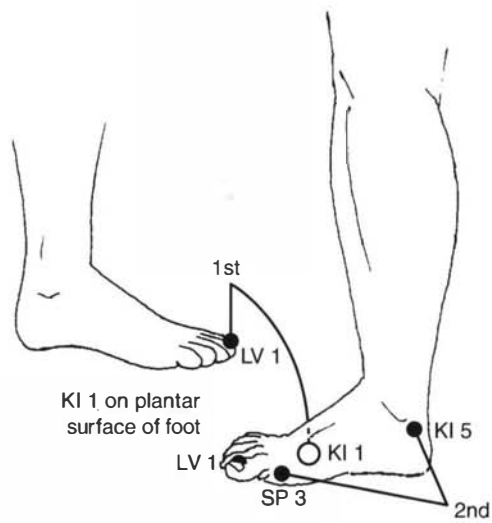


7—48.

KIDNEY
5 — 7 p.m.

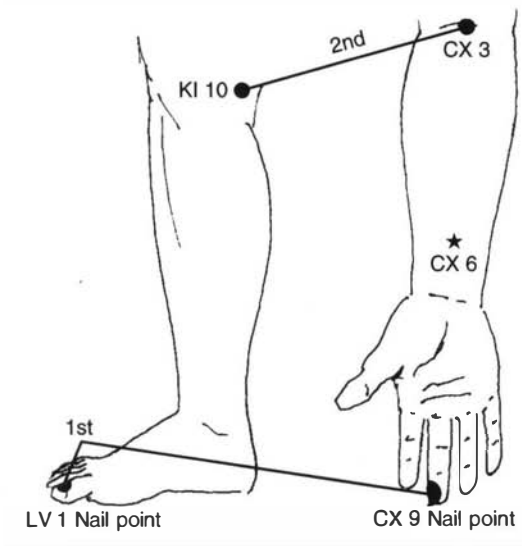


7—49.

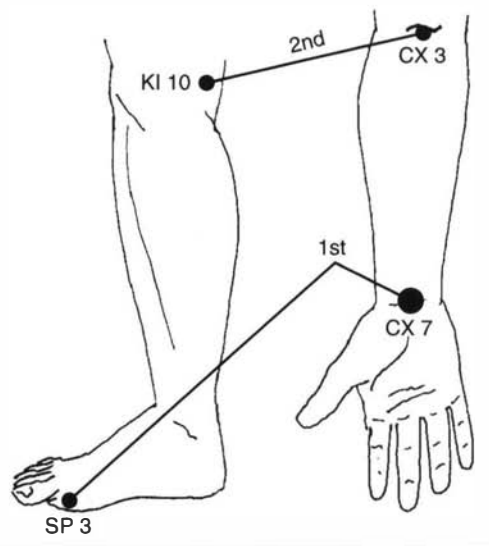


7—50.

CIRCULATION SEX
7 — 9 P.M.



7—51.

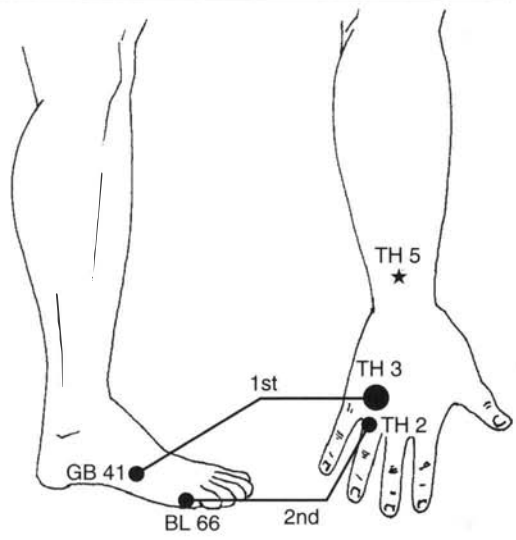


7—52.

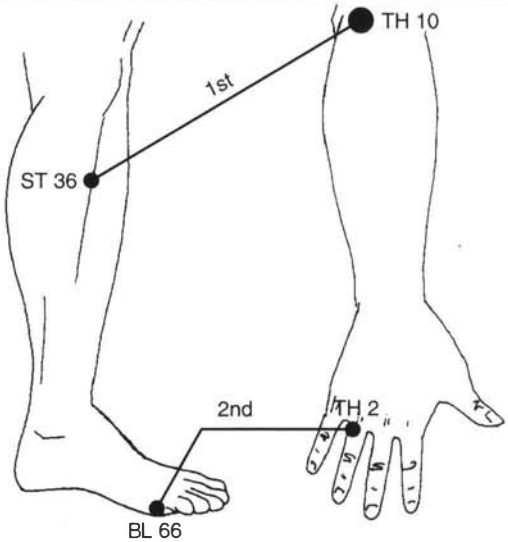
LUO POINTS

Tonification **Sedation**

TRIPLE HEATER
9 — 11 p.m.

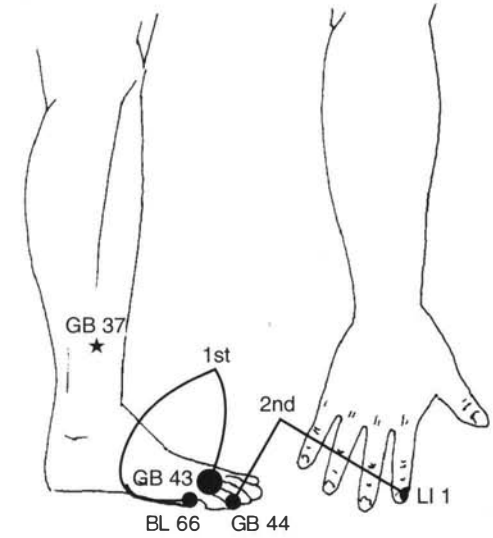


7—53.

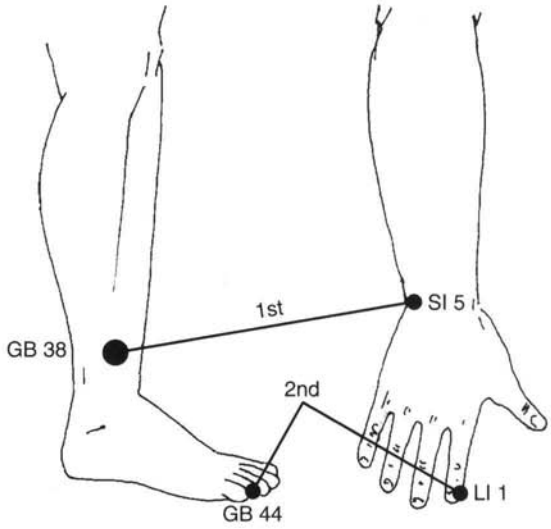


7—54.

GALLBLADDER
11 p.m. — 1 a.m.

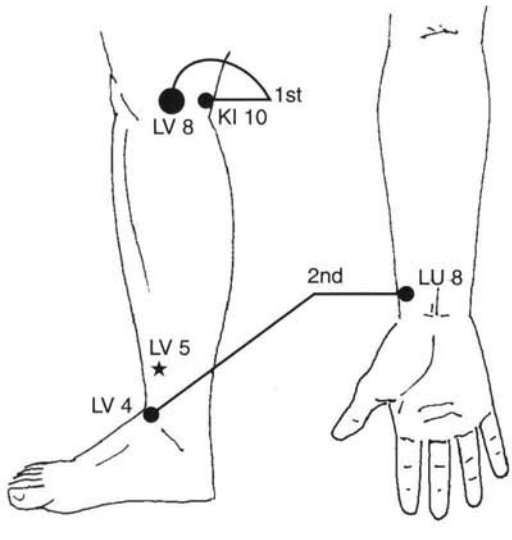


7—55.

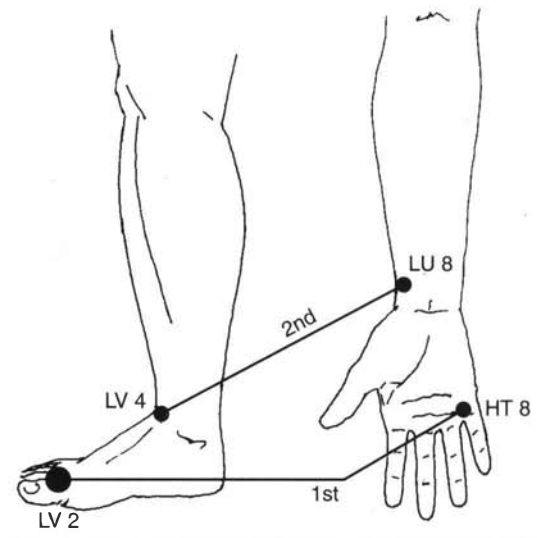


7—56.

LIVER
1 — 3 a.m.



7—57.



7—58.

LUO POINTS

Antenna Effect on Acu-Points

Most authorities agree that the energy characteristics of the meridian system are electromagnetic. Goodheart[9] observed an antenna effect of the acu-points, which can easily be demonstrated by various types of stimulation to the tonification and sedation points. The primary sedation point for the lung meridian is LU 5, located on the elbow crease about one tsun lateral to the biceps tendon. Accurately placing an acu-aid on this point will cause most individuals to develop weakness in a previously strong deltoid muscle. To effect this weakening, it is necessary that the acu-aid be accurately placed on LU 5. If an individual has a balanced meridian system, LU 5 will not show positive therapy localization. One may need to place the acu-aid several times until the exact acu-point is located. Acu-points are very small, and accuracy is essential in effective stimulation.

In most individuals, the deltoid will weaken as soon as an acu-aid is placed on the acu-point. In individuals with high energy levels, the acu-aid may need to be in place for thirty to sixty seconds for the energy to be reduced in the lung meridian, effecting deltoid weakness. On some occasions a muscle may weaken with the acu-aid placement on LU 5 which is abnormal. There are many reasons this can occur that are outside the scope of this text.

An interesting aspect found by Goodheart[9] is that with an acu-aid in place, the deltoid muscle will remain weak unless it is covered with a piece of lead or some other substance, such as certain ceramics, through which electromagnetic energy cannot pass. When the acu-aid on LU 5 is covered with lead, the deltoid muscle will immediately test strong in most individuals. Some persons with a low energy level may need several seconds to regain strength in the deltoid. As soon as the lead is removed from the acu-aid, the deltoid muscle will again test weak. This indicates that the acu-aid acts as an antenna for electromagnetic energy rather than stimulating the skin receptors, because the acu-aid still stimulates the skin when the lead is placed over it. When an acupuncture needle is used for stimulation, the same canceling effect is present when lead is placed over the needle.

The same effect of the acu-aid and lead can be observed when a muscle is weak because of a deficient meridian. This is easier to demonstrate because the exact location of the tonification point can be found by therapy localization. Put an acu-aid on the tonification point and the muscle will test strong, but it will lose its strength when lead is put over the acu-aid.

The effect of the physician contacting the first two points of tonification or sedation is cancelled if he allows his body to come in contact with an object that is grounded. For example, if the doctor contacts the first two points but allows his leg to touch a metallic surface on a grounded examination table, no effect will develop from the contact, nor will simultaneous pulsation develop.

Applied Kinesiology Use of Melzack-Wall Gate Theory in Pain Control

Many patients seek a doctor's assistance because of pain. Pain, in most cases, is a friend because it indicates that something is wrong in the body; however, pain can become an enemy after the alarm has been recognized, or when there is pain for no apparent reason. In most cases a patient sees pain as an enemy; he wants it treated immediately. It becomes difficult to determine the role of pain. Sometimes it is, indeed, a friend, and at other times the greatest of enemies. Melzack[24] reports an interesting comment Leriche made: "Defense reaction? Fortunate warning? But as a matter of fact, the majority of diseases, even the most serious, attack us without warning. When pain develops...it is too late...the pain has only made more distressing and more sad the situation already long lost...in fact, pain is always a baleful gift, which reduces the subject of it, and makes him more ill than he would be without it." Still, we must remember that in most cases, pain is a warning sign of something happening in the body that is not correct. Pain is also beneficial in determining the type of problem a patient has, but one must always remember that the basic underlying cause of the pain may not be in the location mentioned. A common thought that Goodheart uses in his lectures is, "Where it is, it ain't." There are many puzzles about pain that have not, until recently, been explained at all satisfactorily. In fact, there is considerable controversy over the current explanations of the cause of pain. The most generally accepted model of pain is thoroughly discussed in Melzack's book, *The Puzzle of Pain*.[24] Take, for instance, situations where, from all indications, tremendous pain should be present; however, the individual involved experiences no pain. In some Indian cultures, individuals hang from hooks inserted into their skin with ropes attached and exhibit no pain. Others install skewers in their flesh to be torn away with no pain. There are cultures where women have absolutely no pain with childbirth.

During World War II, some soldiers showed no pain from severe wounds; their response to injury was relief, thankfulness for escaping alive from the battlefield, and even euphoria. Yet they were certainly able to feel pain! In normal situations these soldiers complained as vigorously as other men at an inept venipuncture.

There is no doubt that mental attitude has some control over pain. The mind certainly affects pain in the emotionally worked-up native during a tribal dance and the emotion-ridden victim of war. Emotional control is also found in normal development into adulthood. If a family makes a great fuss about ordinary cuts and bruises, a child will grow up overreacting to pain; the individual raised in a family wherein little sympathy is shown, even toward serious injuries, will not.

Among the most interesting and confusing types of pain are phantom limb pain and causalgia. These conditions can, and frequently do, develop immediately after amputation or injury to a nerve, and can persist for years even though surgical intervention is attempted.

Phantom limb pain can be as simple as a tingling feeling and a definite feeling of shape that resembles the real limb before amputation, or it can be a severe, terrible pain persisting long after the injured tissues have healed. Phantom limb pain is more likely to develop in patients who suffered pain in the limb prior to amputation. The pain can sometimes be permanently abolished by injecting a local anesthetic into the stump tissues or nerves, even though the anesthesia wears off within hours. Some soldiers found they could eliminate phantom limb pain by hitting the end of the stump with a rubber hammer.

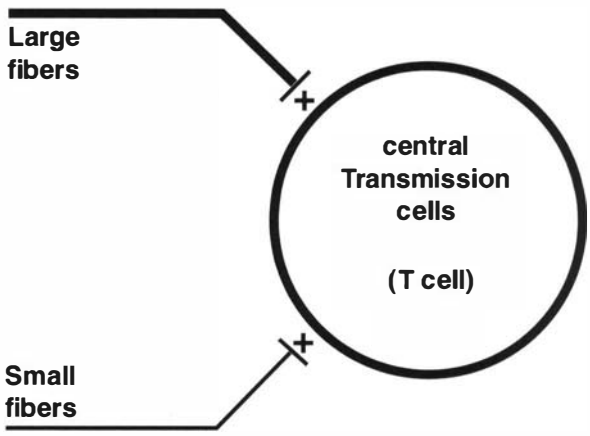
The burning, severe pain of causalgia is characteristically associated with rapid injury to the nerve, such as caused by high velocity missiles (bullets). Causalgia pain remains long after the tissues have healed from the actual injury. Peripheral surgery is used, but all too often it is not successful. In fact, operations have been performed for causalgic pain at nearly every site in the sensory pathway, from peripheral receptors to somatosensory cortex. Some initial results are encouraging, but the tendency is for the pain to return.

So far in our discussion of pain we see an interesting paradox; people who should have severe pain have none, and people who should have no pain suffer greatly. This is not consistent with the theory of pain that has, basically, been accepted for years. The specificity theory was first offered by Descartes in 1644. It proposes that a specific pain system carries messages from pain receptors in the skin to a pain center in the brain. This theory has undergone evolution, especially since the 19th century. Although many new additions to the theory have been presented, they still do not explain the many variables to pain or the common failure of surgical intervention to control pain. Certainly there has to be some higher center control of pain.

In 1965 Melzack and Wall[23] described their new gate control theory of pain. The basic theory explains our knowledge of incongruous pain. It proposes a mechanism within the cord for switching nerve impulses on and off, known as a gate to open or close flow. The following diagrams and explanations build the Melzack-Wall Gate Control Theory of Pain step by step. This is the early version of their theory; it has since been modified. Techniques are currently being developed in AK to incorporate recent developments in pain neurophysiology.[32]

Figure 7—59. Nerve impulses are brought from the receptors to both large and small fibers. These fibers have different characteristics.

Large Fibers	**Small Fibers**
Myelinated	Unmyelinated
A fibers	C fibers
Fast conduction (up to 120 meters/seconds)	Slow conduction (down to 1 meter/second)
Have receptors that react to low and moderate intensity	Have receptors that react to low, moderate, and high intensity stimuli

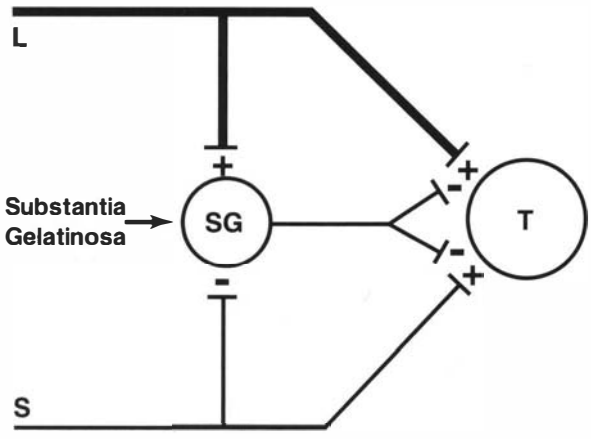


7—59.

When there is a stimulus to a receptor field, both large and small fibers conduct impulses. As the stimulation becomes noxious, such as pain stimulus, the small fibers conduct with greater intensity due to their receptors' characteristic reaction to high intensity stimuli.

The large and small fibers activate the transmission cells (T cells) that project information to the brain. The T cells are located in the spinal cord dorsal horns, apparently in lamina 5. They fire when a certain threshold of stimulation is reached.

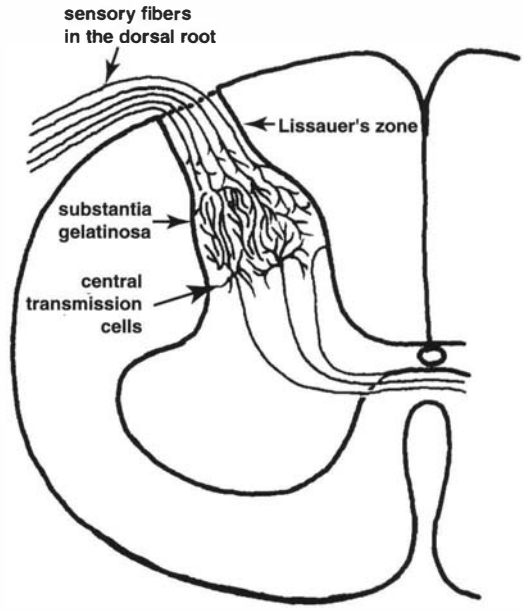
Figure 7—60. The large diameter fibers and the small diameter fibers give off branches to the substantia gelatinosa (figure 7—60). The substantia gelatinosa is located in the dorsal horn lamina 2 and 3. As a functional unit it extends the length of the spinal cord on each side. Its cells connect with one another by short fibers; they influence each other at distant sites on the same side by means of Lissauer's tract (figure 7—61), and on the opposite side by means of commissural fibers that cross the cord. The substantia gelatinosa therefore receives afferent input from large and small fibers. Its cells connect with one another to different levels of the spinal cord, and they communicate with the contralateral side. The spinal gate mechanism appears to be at the substantia gelatinosa.



7—60.

Activity in the large fibers stimulates the substantia gelatinosa; activity of the small fibers inhibits it. Activity of the substantia gelatinosa inhibits activity of the T cell. Thus the balance of activity between the large and small fibers either activates or deactivates the substantia gelatinosa which, in turn, either allows activity at the T cell or inhibits activity there. When the T cell is inhibited, information received by the cell cannot be transmitted to the brain.

Adaptation to mild and moderate stimuli, primarily conducted by the large nerve fibers, is accomplished by this gate mechanism. For example, when you sit down in a chair to read a book, your nervous system soon adapts to the pressure of sitting in the chair, your hand lying in your lap, and the pressure of the book in your hand. These mild pressure stimuli are conducted primarily by the large

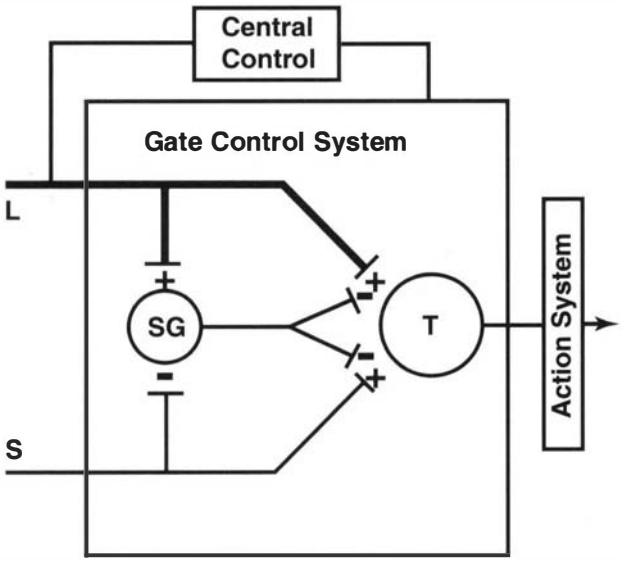


7—61.

fibers to both the substantia gelatinosa and the T cells. Upon first conduction, the T cells transmit information to the brain that is interpreted as pressure. With continued stimulation, the substantia gelatinosa sends impulses of an inhibiting nature to the T cells. This reduces the information about the sitting position that is being transmitted to the brain, so the body (large fibers) adapts to the stimulation. If, on the other hand, you sit on a sharp tack, there would not only be transmission by the large fibers but also increased transmission by the small fibers, which are activated more by high intensity stimulation. The activity of the small fibers stimulates the substantia gelatinosa and the T cells. The immediate response is transmission by the T cells to the brain, informing it of the noxious stimuli. The continued inhibitory stimulation of the substantia gelatinosa would turn its activity off, giving no inhibitory action to the T cells; consequently, the T cells would continue to transmit information of a pain stimulus to the brain. This is the manner in which the gate is held open by pain stimuli.

Figure 7—62. Continued sitting on the tack and the subsequent transmission of the T cells trigger the action system required by the pain. The activity will be (1) perceptual information, giving location, magnitude, and spatiotemporal properties of the noxious stimulus; (2) motivational tendency toward escape or attack; or (3) cognitive information based on analysis of past experience and probable outcome of different response strategies. The interplay of these three activities could then influence motor mechanisms responsible for the complex pattern of overt responses that characterize pain. Also added to the model is the central control mechanism.[25] By this mechanism central activities, such as anxiety or excitement, may open or close the gate for inputs from

any part of the body. The central control cortical projections and reticular projections explain how higher central nervous system processes — such as attention, anxiety, anticipation, and past experience — exert a powerful influence on pain processes.



7—62.

Applied Kinesiology Use of the Gate Theory in Control of Pain

Goodheart[11] observed the serendipitous, dramatic reduction of pain in a patient while he was tapping a meridian tonification point. In an effort to evaluate what was taking place, he tapped the sedation point of the same meridian and the severe pain returned. When the tonification point was tapped again, the pain dramatically diminished. Recognizing that this could be a method of stimulating the large fibers and thus affecting the spinal gate mechanism to control pain, he systematically searched for an examination procedure and stimulation technique that would consistently affect transmission of pain impulses. The following procedure, developed by Goodheart, is consistently effective in controlling pain. The procedure is not designed to take the place of accurate and effective treatment to correct conditions that cause pain; it is designed to help control pain from trauma, and pain that is out of proportion to the circumstances.

1. Determine the level of the patient's pain. This is usually accomplished by pressing on the painful area. When pressure elicits pain, the doctor should note the amount of pressure and the location so he can reproduce it at a later time. Likewise, the patient should grade the severity of the pain on a scale of 1-10 for comparison after treatment.
2. Therapy localize the pulse points to find the positive pulse point. Usually, only one pulse point will be active on therapy localization; occasionally there may be two. If no pulse point therapy localizes, have the patient quit breathing for ten seconds prior to testing; this slows down meridian activity. Make sure the patient does not take a breath prior to the cessation of breathing. Another factor that may prevent the pulse point from therapy localizing is a lateral atlas subluxation. The most common meridian involved is the liver; the second most common is the spleen.
3. Locate the weak muscle associated with the pulse point. For example, if the liver-gallbladder pulse point is active, check the popliteus bilaterally and the pectoralis major (sternal division) bilaterally. Usually only one muscle will be weak. This may or may not correlate with the side on which the pain is located. More than one active point is usually associated with medication usage.
4. Confirm that the muscle weakness is associated with the active pulse point by therapy localizing the alarm point of the associated muscle. If the muscle is weak because of the meridian involvement, it will strengthen with this therapy localization.
5. Using therapy localization, locate exactly the first tonification point of the involved meridian on the side of the weak muscle. When the point is accurately therapy localized, the weak muscle involved with that meridian will strengthen. For example, if the pectoralis major (sternal division) is weak on the right, therapy localization of LV 8 on the right should cause it to strengthen.
6. Tap the primary tonification point rapidly for thirty to forty seconds. Often, but not always, the point will be exquisitely tender. Re-test the muscle involved with the meridian. Tapping is the proper stimulus because a steadier type of stimulation would cause adaptation; the fibers being stimulated are large fibers that adapt to constant stimulation. If tapping is adequate stimulation, the muscle will now test strong.
7. Continue tapping on the primary tonification point for one and one-half to two minutes. The most productive tapping is when there is a bony backup to the tonification point. If possible, direct the tapping to obtain a bony backup.
8. Pain is usually reduced by at least 50% and often more. Periodically recheck the pain reduction. Continue tapping until maximum reduction is obtained. In rare cases when there is less than 50% relief, continue tapping until some observation is made to discontinue, i.e., failure of further pain reduction or a physiological response such as autonomic nervous system change.
9. If pain reduction is not to the level of 90%, further tapping of the tonification point is done simultaneously with tapping of the associated point for the involved meridian. If tapping of the tonification point is discontinued because of a physiologic response, allow the patient to rest for a while and then simultaneously tap the tonification point and the associated point. In the case of the liver meridian, the

associated point is BL 18, about the level of T10 vertebra and two tsun away from the midline.

10. After adequate change is observed, have the patient suck a ribonucleic acid (RNA) tablet. The muscle involved with the meridian may weaken again, but the pain will not return. If the muscle weakens, have the patient suck the appropriate nutritional factor for the weak muscle simultaneously with RNA. When you've found the nutritional factor that again strengthens the muscle when tested with RNA, have the patient use that nutritional support. For example, with the liver meridian either vitamin A, bile salts, or liver extract is usually the factor that will gain a response.
11. Patients can further stimulate the points at home. Mark the tonification and associated points on the patient with a non-water-soluble felt-tip pen. Instruct the patient in the stimulation procedures to be used as needed for control of pain. Of course, the patient will need an assistant in most cases to tap the associated point. If the patient fails to obtain relief at home, one of two factors is usually responsible: 1) the patient is not stimulating the points accurately, hard enough, or long enough, or 2) the points have changed. Occasionally — especially with treatment — the meridian requiring tonification stimulation will change. One should periodically re-evaluate the patient to determine that the same meridian is involved.
12. Failure of the Melzack-Wall technique to reduce pain is probably due to a lack of neurotransmitters and co-factors. Schmitt[31] has described an excellent applied kinesiology method for evaluating the neurotransmitters and determining the nutrients necessary for normalization. Another factor that may cause less than adequate results with the Melzack-Wall technique is tapping at an improper frequency. It is often necessary to reduce the tapping rate. Two to four Hz appear to be the most productive. Great care should be taken to accurately locate and stimulate the point.

Stimulation of an auricular point adds to the effectiveness and permanence of pain relief. Stimulate the point for the organ associated with the meridian found by pulse diagnosis. In the example given where the tonification point for the liver meridian was stimulated, the appropriate auricular point is the liver and not the point that coincides with the location of pain.

The length of time pain is relieved by AK Melzack-Wall technique varies considerably among individuals. The relief may be as short as one hour, but it usually lasts considerably longer, from four to twelve hours. Conditions such as shingles usually require stimulation twice a day. In some cases, such as a phantom limb pain where there should be no pain, the relief is sometimes permanent after the first treatment.

Use of the above procedure has been very satisfactory in the control of severe pain. It is most effective with a traumatic type of pain, such as fractures, sprains, and tooth extraction, among others. It is also effective in conditions where an obvious problem needs correction; however, it should only be used for control of pain while correction is being accomplished. The AK Melzack-Wall therapy appears to have some therapeutic effect in addition to reducing pain, but it will obviously not correct a sacroiliac or a foot subluxation; in fact, an adjacent subluxation may be a contributing factor to the problem, such as a knee subluxation in the case of LV 8, or extended pronation causing strain on the first ray and activating SP 2. Take care that the patient avoids physical activities that ordinarily would be limited by pain, causing further injury to the traumatized area.

Moderate success has been obtained with transcutaneous electrical nerve stimulation (TENS) after the initial effect has been obtained by the tapping method. Some continuous relief may also be obtained by applying acu-aids to the area and having the patient periodically rub them.

Five Elements

Ancient Chinese philosophy relates everything on earth to one of five elements: fire, earth, metal, water, and wood. Some objects can easily be classified as one of the five elements, such as lumber is wood, the sun is fire, bricks relate to earth, a knife blade is metal, and milk relates to water. Many things, however, are a combination of the five elements. A coal fire is classified as earth for the coal, fire for the heat, and metal for the gasses.

Knowledge of the five-element law gives a physician a great advantage in reading body language to help understand a patient's problem. Each meridian belongs to one of the five elements, and each element is associated in a different way with various factors such as sounds, emotions, climates, seasons, and taste (see chart 7—63). When the patient's signs and symptoms are dominated by one of the five elements, the meridian and organ association is a clue to the underlying cause of the patient's health problem.

This author has had many experiences in which relationships in the five-element law provided the clue to finding the basic underlying cause of a health problem. A woman with chronic, severe headaches was being seen on the third office visit, with no improvement in her condition having been obtained. She stated, "There's another factor about my headaches I haven't told you. I've mentioned it to other doctors but they just laughed at me. Finally I quit telling doctors about it, but it's of vital concern to me. You seem different from other doctors and interested in what I tell you. My most severe headaches come on after I laugh a lot. In fact, I don't want to go

out socially where it is probable there will be a lot of laughing, because I know I'll end up with a severe headache." The sound of laughter is related with the fire meridian. After hearing this, I evaluated her meridian system and found the heart meridian extremely overactive. Balancing the meridian system to bring the energy in the heart meridian down to normal eliminated her headaches, after years of using medication and chiropractic to no avail.

There are many things patients tell doctors that are ignored simply because there does not seem to be any relationship to the condition. Think of the number of times a patient has stated, "I'm worse when I get cold." "I just seem to want to cry all the time since I've had this condition." "I feel much better when I have a sauna (dry), but am much worse with a steambath." In addition to what patients tell you, observation indicates which meridians may be out of balance. The patient who has excessive fear about your examination and treatment probably has an imbalance of the kidney or bladder meridian, which is associated with the element water. Such individuals generally have poor willpower, again relating to the kidney or bladder meridian. When one is familiar with the relationships in the five elements, a major step is accomplished in understanding why patients act and feel as they do. It is interesting to observe how the personality and symptoms of an individual change as the meridian system is balanced.

FIVE ELEMENT ASSOCIATIONS

	Fire	Earth	Metal	Water	Wood
Yin	**HT — CX**	**SP**	**LU**	**KI**	**LV**
Yang	**SI — TH**	**ST**	**LI**	**BL**	**GB**
Sense	Speech	Taste	Smell	Hearing	Sight
Color	Red	Yellow	White	Black	Green
Smell	Burnt	Sweet	Fleshy	Putrid	Rancid
Fluid	Sweat	Lymph	Mucus	Saliva	Tears
Sound	Laughter	Song	Sob	Groan	Cry
Emotions	Joy	Worry	Grief	Fear	Anger
Season	Summer	Midsummer	Autumn	Winter	Spring
Climate	Heat	Moisture	Dryness	Cold	Wind
Orifice	Ears	Mouth	Nose	Anus-urinary	Eyes
Direction	South	North	West	North	East
Taste	Bitter	Sweet	Piquant	Salty	Acid
Psychic	Conscience	Ideas	Animal spirit	Will, ambition	Spirit
Energy	Psychic	Physical	Vital	Will power	Blood
Strain	Walking	Sitting	Lying down	Standing	Eyes
Planet	Mars	Saturn	Venus	Mercury	Jupiter
Controls	Taste — tongue	Tactile — lips	Smell — nose	Hearing — ears	Eyes
	Pulse	Flesh — lips	Skin	Bones — hair	Muscles
	Complexion		Body hair		Nails

7—63.

The elements earth, metal, water, and wood have two meridians that are "coupled." A couple is composed of a Yin and a Yang meridian that belong to a specific element. The associations are as follows: stomach and spleen are earth, lung and large intestine are metal, bladder and kidney are water, and liver and gallbladder are wood.

The element fire has two sets of coupled meridians: the heart/small intestine and the triple heater/circulation sex. There is a relationship between these coupled meridians known as the "Emperor-Prime Minister law." The heart and small intestine meridians are considered the emperor, which dominates the circulation sex and triple heater meridians (the prime minister).

The five elements and their meridians are arranged in a diagram with specific organization (see figure 7—67). Coupled meridians are contained in a circle arranged on a larger circle. The fire element is diagrammed with the two coupled meridians side by side, but one should visualize the emperor (HT and SI) as being on top and dominating the prime minister (CX and TH) underneath. The Yang meridians are on the outside of the large circle; the Yin are on the inside in keeping with the characteristics of Yin and Yang. The meridians to the right of center are the right pulses; to the left are the left pulses.

Element	Yin	Yang
Wood	Liver	Gallbladder
Fire	Heart	Small Intestine
Earth	Spleen	Stomach
Metal	Lung	Large Intestine
Water	Kidney	Bladder
Fire	Circulation Sex	Triple Heater

Sheng (Creation) Cycle

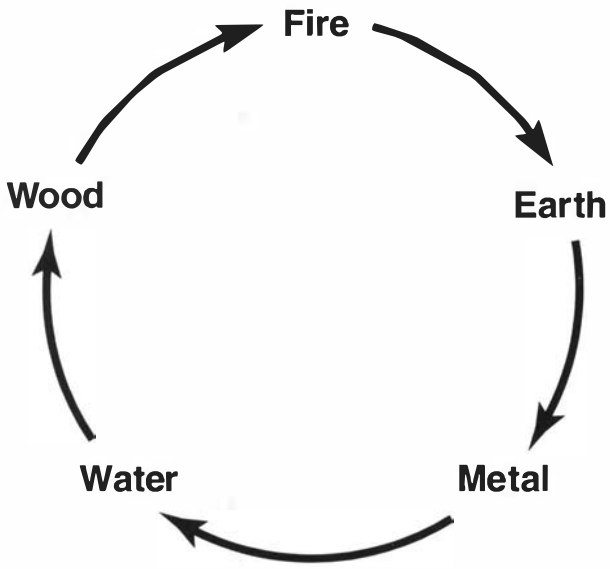
There is a creation cycle in the five-element law that is continuous. Wood creates fire by burning, fire creates earth by the ash left from burning, earth creates metal (such as iron ore), metal produces water when heated by becoming molten like water, water creates wood by feeding plants, and so the cycle continues. The flow of creation is indicated by arrows around the diagram's large circle.

The mother/child effect is applicable in the creation cycle. Fire is the mother of earth, and earth is the child of fire, while earth is the mother of metal, and metal is the child of earth, and so on through the cycle.

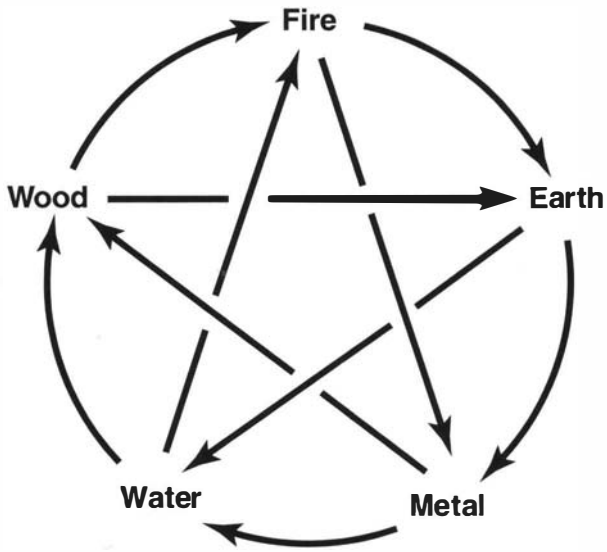
Ko (Destruction) Cycle

The Ko cycle is the destruction or controlling aspect of the five-element law. The flow of the Ko cycle is indicated in the diagram by arrows inside the large circle. Wood destroys earth by breaking up the soil and covering it, earth destroys water by confining and damming it, water destroys fire by extinguishing it, fire destroys metal by melting it, and metal destroys wood by cutting it.

To read body language, it is important to understand the Ko cycle. High activity in a meridian may cause deficiency in the meridian affected by the Ko cycle. For example, if an individual consistently has deficiency in the spleen meridian, the cause may be the consumption of too much alcohol, putting a heavy load on the liver. The meridian system may hold energy in the liver meridian to help handle the condition.



7—64. Creation cycle.



7—65. Ko cycle.

Meridian Balancing with the Five-Element Law

The goal throughout treatment of the meridian system is to balance the energy in the meridians. As indicated earlier, even classic acupuncturists do not use stimulation of the meridian points alone to effect this balance. Herbs, nutrition, and manipulation are used in addition to, or in place of, point stimulation. In the Western application of meridian treatment, the meridians are often evaluated by measuring resistance at the Akabane[39] or Nakatani[41] points. The energy of the meridians is charted, and the methods discussed below and others are used to balance energy within the system. Applied kinesiology provides additional diagnostic methods to determine the point to stimulate or other treatment necessary to obtain balance.

Luo points. As noted in the twenty-four-hour circulation of energy, luo points are used in applied kinesiology to bring energy through when circulation is blocked, or to balance energy between bilateral counterparts. Luo points are also used to balance energy between coupled meridians in the five-element law. For example, the lung and large intestine are coupled meridians of the element metal. If the tensor fascia lata, associated with the large intestine meridian, tests weak and the deltoid, associated with the lung, tests strong, therapy localize to the lung alarm point (LU 1) to see if the tensor fascia lata strengthens. If it does, it provides applied kinesiology indication of excess energy in the lung meridian and deficiency in the large intestine meridian. The rule in the use of the luo point is to always stimulate the luo point on the deficient meridian. In this case, LI 6 would be stimulated to balance the energy between the lung and large intestine. The reason for always stimulating the luo point of the deficient meridian is to bring the energy across to the desired meridian. The luo point is also capable of transferring energy between the midday/midnight meridians. If the

bladder meridian is deficient, stimulation of the luo point on the lung meridian may erroneously transfer energy to the bladder meridian, which is the twelve-hour counterpart of the lung meridian.

Transferring energy between bilateral counterparts is accomplished in a manner similar to that for coupled meridians. If the deltoid tests weak on the left and strong on the right, and therapy localization to the right lung alarm point causes the left deltoid to strengthen, one would again stimulate the luo point on the deficient meridian. Thus LU 7 on the left side would be stimulated to balance energy between the meridians. When coupled or bilateral counterpart meridians have excess energy in one meridian and a deficiency in the other, it is called a split. When there are numerous splits throughout the body, SP 21 is stimulated. SP 21 is the great luo point that affects all the luo points in the body. Numerous splits are usually discovered when the meridians are electrically evaluated at the Akabane[39] or Nakatani[41] points.

Command points. Each meridian has five command points, one for each of the five elements (see Figure 7—66). They are used to transfer energy according to the five-element law.

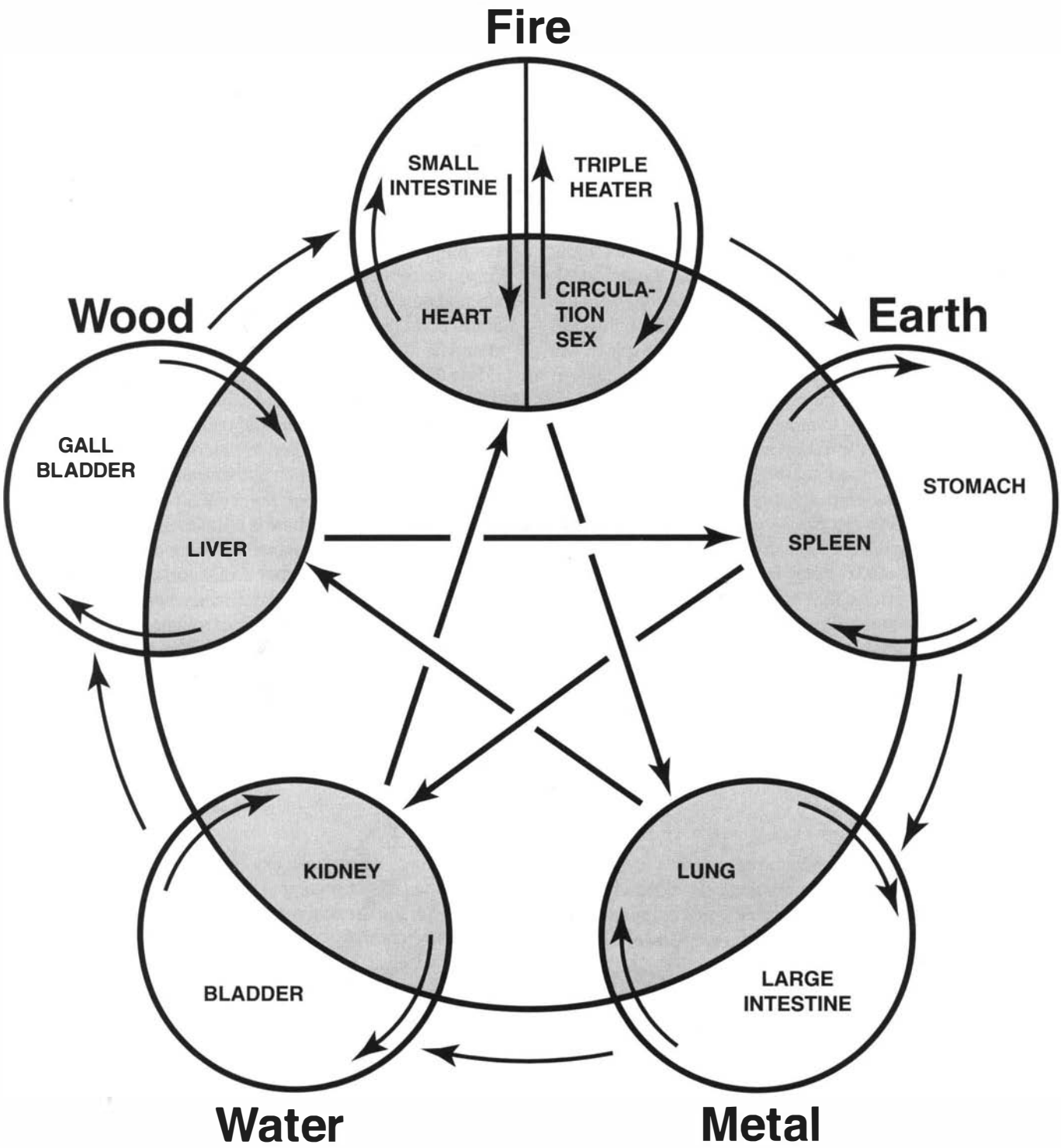
In the Sheng or creation cycle, the mother/child effect is applicable in energy movement as in the twenty-four-hour circulation of energy. Since the stomach (earth) is the mother of the large intestine (metal), energy held in the stomach meridian cannot be transferred to the child or the large intestine meridian, so it then becomes deficient. This can be determined in a manner similar to that used for coupled and bilateral counterpart meridians. If the tensor fascia lata, associated with the large intestine, is weak and therapy localization to the stomach alarm point causes it to strengthen, energy must be transferred from the stomach to the large intestine. This is accomplished by stimulating a command point on the deficient meridian. The command point to use is the element of the excess meridian, which is the earth command point of the large intestine (LI 11). When using command points with the creation cycle, one must always transfer in the direction of creation; only Yang can be transferred to Yang, or Yin to Yin.

There is a method for transferring energy in the creation cycle when the deficient meridians are not both Yin or Yang. This method is rarely used when meridian balance is evaluated only with applied kinesiology techniques. It is more applicable when the entire meridian system is evaluated electronically. In applied kinesiology evaluation, if the large intestine meridian is deficient — indicated by a weak tensor fascia lata — and is strengthened by therapy localization to the spleen alarm point, there is evidence of excess spleen energy and a deficiency in the large intestine. Since the spleen is Yin and the large intestine Yang, energy must first be borrowed from the stomach meridian and then replaced from the excess spleen meridian. This is accomplished by stimulating the earth command point (LI 11) on the large intestine meridian to borrow energy from the stomach meridian. Now the stomach meridian is deficient. The luo point on the stomach meridian (ST 40) is then stimulated to replace

Chart of Command Points

Meridian	Fire Point	Earth Point	Metal Point	Water Point	Wood Point	Luo Point	Source Point
Lung	10	9	8	5	11	7	9
Large Intestine	5	11	1	2	3	6	4
Stomach	41	36	45	44	43	40	42
Spleen	2	3	5	9	1	4	3
Heart	8	7	4	3	9	5	7
Small Intestine	5	8	1	2	3	7	4
Bladder	60	54	67	66	65	58	64
Kidney	2	3	7	10	1	4	3
Circulation Sex	8	7	5	3	9	6	7
Triple Heater	6	10	1	2	3	5	4
Gallbladder	38	34	44	43	41	37	40
Liver	2	3	4	8	1	5	3

7—66.



7—67. Five-element interaction.

energy in the stomach meridian from the spleen meridian (from Yin to Yang).

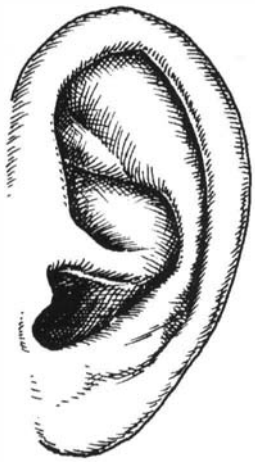
When a muscle or organ fails to respond to the treatments generally used in applied kinesiology, or does not hold its correction, there may be overactivity in the meridian associated by the Ko cycle. An example is deficiency in the spleen meridian, indicated by a weak middle or lower trapezius that fails to respond or hold its correction. If the pectoralis major (sternal division) tests strong, the liver meridian may be overactive and, by way of the Ko cycle, cause destruction to the spleen meridian. This can be determined by therapy localizing the wood element point on the spleen meridian (SP 1). If the middle or lower trapezius muscle strengthens to the therapy localization, stimulation of SP 1 will transfer energy from the liver meridian to the deficient spleen meridian; thus the destructive effect of the liver meridian on the spleen will be discontinued and the muscles will be strong. Quite often the improvement will be rapidly lost, which indicates a liver problem needing treatment. It may require improvement of its lymphatic drainage, blood supply, or nutritional support, or there may be a subluxation or some other factor associated. Dietary change — particularly regarding fats and alcohol — may be the proper treatment for lasting results. Note that in the Ko cycle, as with the Sheng, excessive energy is transferred only in the direction of the arrows that indicate energy flow, and the appropriate command point is always stimulated on the deficient meridian. In the Ko cycle, the command point to stimulate is the element point of the excess meridian on the deficient one.

Other methods for transferring energy with the five-element law are the double Sheng and double Ko cycles. These are more applicable when the entire meridian system is charted by electrical evaluation of the Akabane[39] and Nakatani[41] points. Their use is outside the scope of this text.

The answer for patients with resistant conditions is often found by applying the law of the five elements. As indicated previously, applicable treatment may be other than acu-point stimulation. A patient's primary complaint may be ankle weakness. Examination readily reveals that the peroneus longus and brevis test weak and are not giving lateral support to the ankle. The muscles generally respond adequately to the usual applied kinesiology techniques, but they are weak on the next office visit and there has been no improvement of the condition. If the muscles strengthen on therapy localization to the large intestine alarm point (ST 25), the approach described above is to stimulate the metal command point on the bladder meridian (BL 67). The peroneus longus and brevis should then test strong, but sometimes they do not hold the correction. In this case, one should consider the pathway of the large intestine meridian to determine if there is any mechanical blockage, such as extremity subluxations or scars. Also, the associated point (BL 28) should be considered to determine if there is a subluxation. BL 28 is at the level of the second sacral foramen; a sacroiliac disturbance is probable. Further evaluation should be done of the large intestine itself. Determine whether there may be colon stasis as a result of lack of roughage, imbalance of flora, or any of the other usual factors that cause disturbance in the large intestine. Often, supplying the proper nutrition to support the excess meridian immediately causes the muscles associated with the deficient meridian to test strong.

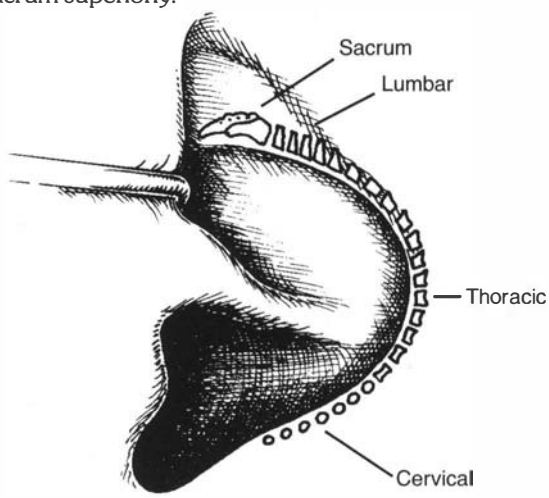
Auriculotherapy

There is a homuncular representation of the entire body within the ear. The embryologic development of the ear is early, at the 4 mm stage.[38] Acu-points representing the entire body are located as if the ear were an upside-down fetus. The cephalic extremity, or head, is equivalent to the lobe of the ear. The antihelix represents the spine, with the cervical region located inferiorly and the sacrum superiorly.



7—68.

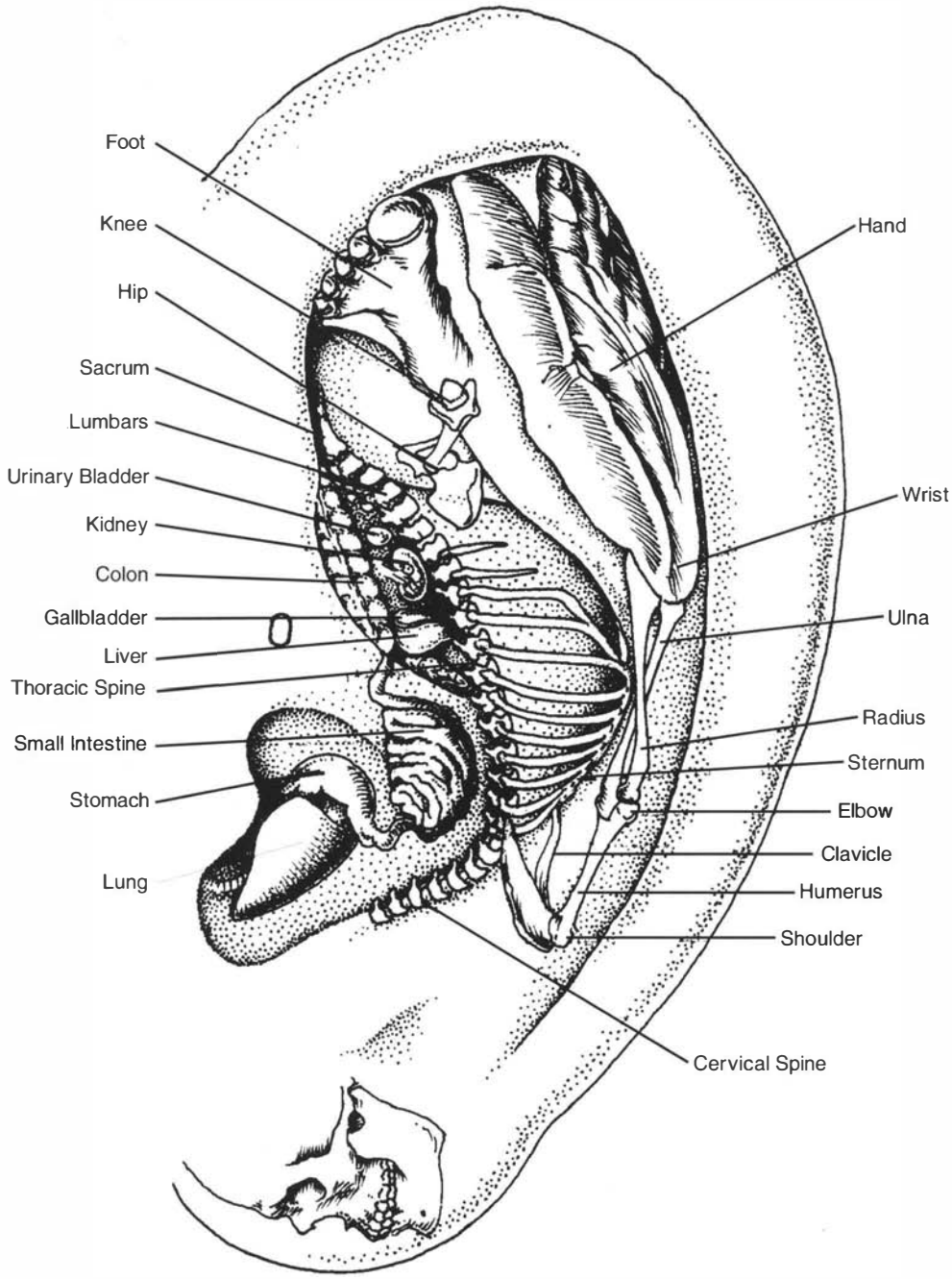
7—69.



7—70.

The importance of the ear on the body's energy patterns can readily be recognized by the effect it has on muscle function. In a high percentage of cases, when the ear is covered with lead all ipsilateral muscles test weak with manual muscle testing. The contralateral muscles remain strong. This is called the "lead square effect." Contrary to what one would expect, when both ears are covered with lead no weakness develops. The effect of lead weakening a muscle can be eliminated if one first stretches the ear in all directions. This is done by gently stretching around the outer portion of the ear as if one were trying to enlarge it. After the stretching procedure, there is no weakening when lead is applied over the ear, at least for a while.

There is an apparent correlation between the ear and the cranial-cervical mechanism. On those affected by the lead square, a general indicator muscle will weaken with a certain head position and phase of respiration. The combination that weakens the indicator muscle will be turning the head and neck to the right or left with inspira-



7—71.

tion or expiration. Finding the combination requires trial and error. When the combination is found, it cannot be repeated if the ear is first stretched as described above.

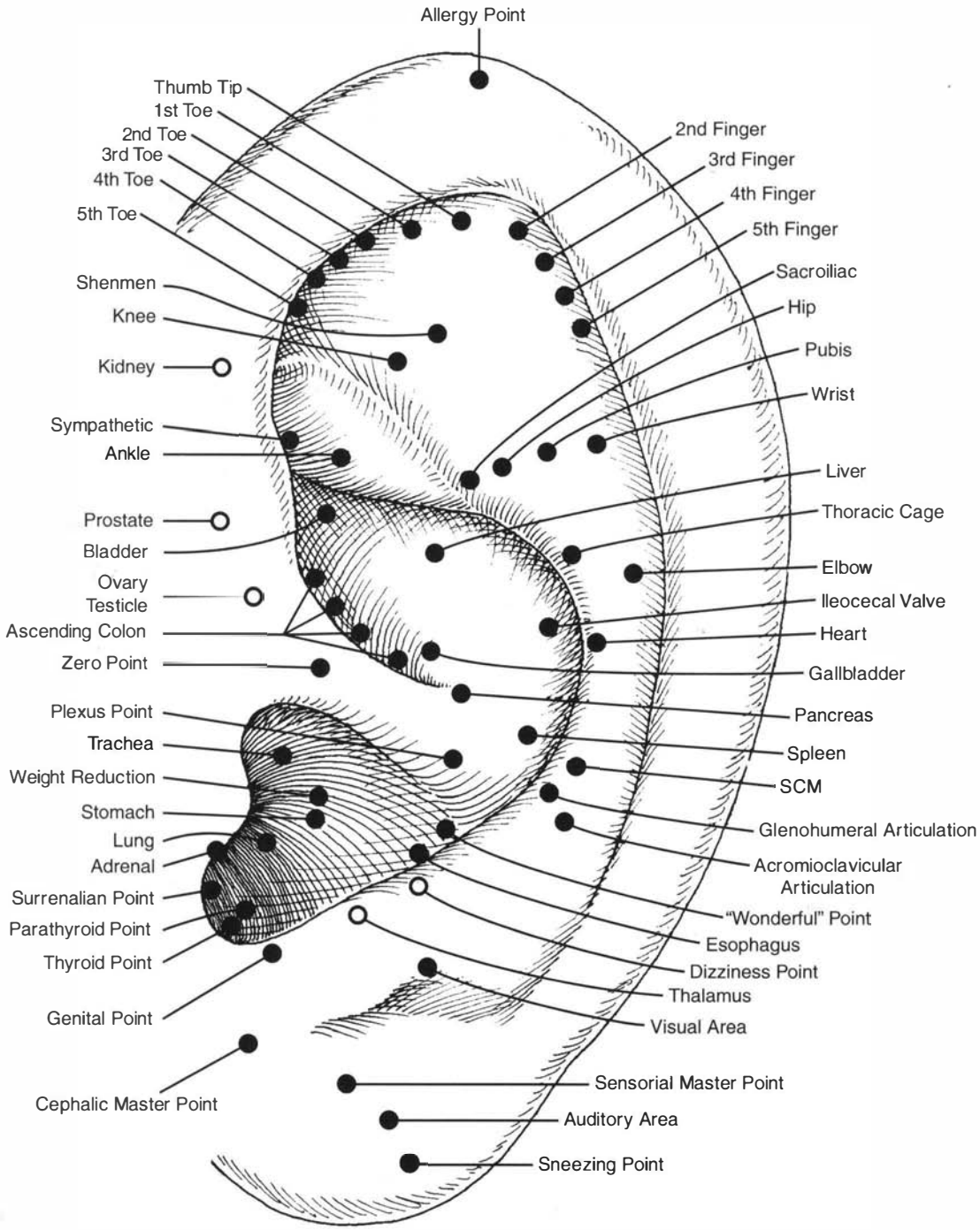
Numerous auriculotherapy charts exist, all varying to some degree. Nogier[27] of France is the principal individual who introduced auriculotherapy to the Western world. The accompanying charts of the ear are a combination of Nogier's work with some points from Chinese charts. Although the charts primarily reflect what is observed in applied kinesiology, therapy localization is the main factor in determining the exact location of a point that will affect the body. Use the muscle-organ association and find the acu-point that strengthens a weak associated muscle.

The acu-points in the ear can be stimulated by many of the same methods used for acu-points on the general body. Acu-aids are used by many when constant stimulation is desired. This is often done for habit control, i.e., to stop smoking or lose weight. Some use indwelling staples or special tacks for constant stimulation; when this is done great care must be taken because severe infections have developed, some even requiring partial amputation of an ear.

The antenna effect previously described is demonstrated in the ear. A needle or acu-aid may stimulate a point, strengthening a weak associated muscle. The muscle will weaken when the needle or acu-aid is locally covered with lead; avoid covering the entire ear.

A method frequently used in applied kinesiology to find active auricular points and to stimulate them is by using a cotton swab with a wooden stick. When a cotton swab moistened with saline solution touches an active acu-point, either a strong indicator muscle will weaken or a weak muscle associated with the point will strengthen. It is unknown if the cotton swab acts as an antenna, or if therapy localization energy passes from the patient's hand through the applicator stick to the point.

Palpation of the antihelix with a small, blunt instrument will find specific areas of tenderness that correlate with the locations of spinal subluxations. There appears to be no correlation with spinal fixations. Chronic spinal involvement will radiate tenderness from the antihelix into the helix. The area of spinal tenderness in the ear will correlate with positive challenge and therapy localization of the spine, indicating subluxations. The areas in the ear will have positive therapy localization. These are best therapy localized by having the patient hold a blunt, metallic instrument (a paper clip serves well) on the point of the ear, then testing a previously strong indicator muscle for weakening. The spinal locations in the ear can be stimulated in the same way as any other auricular point. The stimulation seems to augment spinal adjustment in resistant cases.



7—72. The hollow circles are underneath and are not observable externally.

CHAPTER 7 — REFERENCES

1. Amaro, J.A., International Academy of Clinical Acupuncture Seminars, Denver, CO 1987.
2. Austin, M., *Acupuncture Therapy*, 2nd ed (New York: ASI Publishers, Inc, 1975).
3. Broffman, M., & M. McCulloch, "Instrument-assisted pulse evaluation in the acupuncture practice," *Am J Acupunc*, Vol 14, No 3 (Sep 1986).
4. Cerney, J.V., *Acupuncture without Needles* (W. Nyack, NY: Parker Publishing Co, Inc, 1974).
5. Chinese Traditional Medical College of Shanghai, *Anatomical Charts of the Acupuncture Points and 14 Meridians* (Shanghai, China: Shanghai People's Publishers, 1976).
6. Chu, L.S.W., S.D.J. Yeh, & D.D. Wood, *Acupuncture Manual — A Western Approach* (New York: Marcel Dekker, Inc, 1979).
7. Feldman, R., & S.Yamamoto, "Massage in Oriental medicine: Its development and relationship to 'Ki,' or vital energy," in *The Kirlian Aura*, ed S. Krippner & D. Rubin (Garden City, NY: Anchor Press/Doubleday, 1974).
8. Goodheart, G.J., Jr., "Chinese lessons for chiropractic," *Chiro Econ*, Vol 8, No 5 (Mar/Apr 1966).
9. Goodheart, G.J., Jr., "The antenna concept in chiropractic meridian therapy," *Chiro Econ*, Vol 17, No 5 (Mar/Apr 1975).
10. Goodheart, G.J., Jr., *Applied Kinesiology 1975 Workshop Procedure Manual*, 11th ed (Detroit: privately published, 1975).
11. Goodheart, G.J., Jr., *Applied Kinesiology 1977 Workshop Procedure Manual*, 13th ed (Detroit: privately published, 1977).
12. Goodheart, G.J., Jr., *Applied Kinesiology 1980 Workshop Procedure Manual*, Vol 1, 16th ed (Detroit: privately published, 1980).
13. Goodheart, G.J., Jr., *Applied Kinesiology 1981 Workshop Procedure Manual*, Vol 1, 17th ed (Detroit: privately published, 1981).
14. Goodheart, G.J., Jr., *Applied Kinesiology 1982 Workshop Procedure Manual*, 18th ed (Detroit: privately published, 1982).
15. Hwei, C., "Acupuncture for tooth extraction: A report 1000 cases." Paper presented at The Republic of China Delegation to the Fourth World Congress of Acupuncture, 1975.
16. Jiasan, Y., *The Way to Locate Acu-Points* (Beijing, China: Foreign Languages Press, 1982).
17. Karagulla, S., *Breakthrough to Creativity — Your Higher Sense Perception* (Santa Monica, CA: DeVorss & Co, Inc, 1967).
18. Mann, F., *The Meridians of Acupuncture* (London: Wm. Heinemann Med Books, 1964).
19. Mann, F., *Atlas of Acupuncture Points and Meridians in Relation to Surface Anatomy* (London: Wm. Heinemann Med Books, 1966).
20. Mann, F., *The Treatment of Disease by Acupuncture*, 2nd ed (London: Wm. Heinemann Med Books, 1967).
21. Mann, F., *Acupuncture: Cure of Many Diseases* (Tao Books & Publications, 1972).
22. McGarey, W.A., *Acupuncture and Body Energies* (Phoenix, AZ: Gabriel Press, 1974).
23. Melzack, R., & P.D. Wall, "Pain mechanism: A new theory," *Science*, Vol 150, No 3699 (Nov 19, 1965).
24. Melzack, R., *The Puzzle of Pain* (New York: Basic Books, Inc, 1973).
25. Melzack, R., "Myofascial trigger points: Relation to acupuncture and mechanisms of pain," *Arch Phys Med Rehabil*, Vol 62 (Mar 1981).
26. Namikoshi, T., *Shiatsu* (Tokyo, Japan: Japan Publications, Inc, 1969).
27. Nogier, P.F.M., *Treatise of Auriculotherapy* (France: Maisonneuve, 1972).
28. O'Connor, J., & D. Bensky, trans/ed, *Acupuncture — A Comprehensive Text* (Chicago: Eastland Press, 1981).
29. Pennell, R.J., & G.D. Heuser, *The "How to" Seminar of Acupuncture for Physicians* (Independence, MO: IPCI, Inc, 1973).
30. Porkert, M., *The Theoretical Foundations of Chinese Medicine — Systems of Correspondence* (Cambridge, MA: The MIT Press, 1974).
31. Schmitt, W.H., Jr., "But what if there's no water in the hose?" *Chiro Econ*, Vol 29, No 6 (May/Jun 1987).
32. Schmitt, W.H., Jr., "The functional neurology of pain and pain control clinical applications and new techniques." Proceedings of Summer Meeting, ICAK, Dearborn, MI, 1988.
33. Tan, L.T., M.Y.-C. Tan, & I. Veith, *Acupuncture Therapy — Current Chinese Practice* (Philadelphia: Temple Univ Press, 1973).
34. Veith, I., *The Yellow Emperor's Classic of Internal Medicine*, new ed (Berkeley, CA: Univ of California Press, 1972).
35. Walther, D.S., *Applied Kinesiology, Volume I — Basic Procedures and Muscle Testing* (Pueblo, CO: Systems DC, 1981).
36. Walther, D.S., *Applied Kinesiology, Volume II — Head, Neck, and Jaw Pain and Dysfunction — The Stomatognathic System* (Pueblo, CO: Systems DC, 1983).
37. *Webster's Third New International Dictionary of the English Language* (1976).
38. Williams, P.L., & R. Warwick, eds, *Gray's Anatomy*, 36th Br ed (Philadelphia: W.B. Saunders Co, 1980).
39. Wing, T.W., *The Theory of Applied Electro-Acupuncture and Technic of Non-Needle Electro-acupoint Therapy* (Pomona, CA: See-Do Press, 1976).
40. Wong, E., *Wong's A.P.P. Action Points for Acupuncture* (Denver: Technique Seminars, Inc, 1975).
41. Wong, E., *Wong's A.P.P. Ryodoraku System* (Not given: A.P.P. Technique Seminars, 1975).
42. Worsley, J.R., *Traditional Chinese Acupuncture, Vol 1 — Meridians and Points* (Great Britain: Element Books, Ltd, 1982).
43. Worsley, J.R., *Acupuncture Charts* (South Colton, NY: Acupuncture, 1983).

Chapter 8

Muscles — Testing and Function

Chapter 8

The Science and Art of Manual Muscle Testing

Manual muscle testing has been described by many as a method of evaluating nerve function.[22,23,26,36] Many of the testing techniques were developed to evaluate disability from poliomyelitis,[6,11,31,35,49] with the main purpose of evaluating motor loss. Muscle function was graded, ranging from paralyzed with no palpable contraction to normal strength.[1,25] From this background, manual
testing as used in applied kinesiology developed. The current applied kinesiology use of manual muscle testing varies considerably from the historic testing of paralyzed muscles. It is a much more discernible type of muscle testing to determine how muscle function is adapted by the nervous system. Schmitt[51] has used the phrase "muscle testing as functional neurology,
scribes applied kinesiology use of manual muscle testing.

There have been many attempts to objectively quantitate the muscle test, with much effort during the epidemic era of poliomyelitis. Newer methods of quantitating muscle function take many directions. Sophisticated instruments,
strength, both isometrically and eccentrically. Some instruments are basically fixed transducers; the test consists of pushing against the transducer.[38,45] Numerous hand-held transducers have been developed. These are units that,
between the examiner's hand and the subject's appendage being tested. There is a constant effort to upgrade the hand-held unit. One unit is interfaced with a computer program that gives statistical analysis for repeated testing reliability.[21] Using this muscle testing method, Unger[56] found a significant increase in strength of the pectoralis (sternal and clavicular divisions tested separately), anterior deltoid,
fascia lata,
lowing the correction of a category II pelvic fault by sacro occipital technique.

Another hand-held instrument is designed to respond only to perpendicularly applied forces.[33] Still another measures force and angle of the unit, and time is integrated into the findings.[40] All these features are important,
none of these methods have been capable of measuring the manual muscle test as used in applied kinesiology.

Many have attempted to quantitate manual muscle testing by measuring force with dynamometers that fit between the examiner's hand and the subject.[10,34,39,42,43] Most of these units are only as good as the operator conducting the test. There are numerous variables in a manual muscle test,
the perception of strength.

Another method of testing strength being produced is with electromyography.[7,13,30,44] This is one of the research methods the International College of Applied Kinesiology is using to better understand the manual muscle test. Six independent studies are combined in a report by Leisman et al.[29] that supports the manual muscle test as used in AK as follows. (1) Muscles identified as weak using applied kinesiology manual muscle testing methods are in a fundamentally different state than those identified as strong. (2) Muscles that test weak are fundamentally different from fatigued muscles. (3) The weakness identified by the AK manual muscle test is not attributable to fatigue. (4) Applied kinesiology muscle testing procedures can be objectively evaluated via quantifying the neurologic electrical characteristics of muscles. (5) The course and effect of applied kinesiology treatment can be plotted objectively over time.

8—1. Modified Cybex II used in applied kinesiology research.

Ongoing research is being done to understand what is happening within the nervous system. A study by Leisman et al.[28] measured the way the central nervous system (brain) is functioning when muscles test strong versus when they test weak. Clear, consistent, and predictable differences were identified in the brain between weak and strong muscle test outcomes. This supports the idea that manual muscle testing outcome changes reflect changes in the central nervous system.

Applied kinesiology use of manual muscle testing as an indicator of the nervous system's function is aptly illustrated in numerous studies. Rybeck and Swenson, in a controlled blind study, evaluated the effect of subjects chewing sugar by testing the latissimus dorsi manually and against a force transducer.[47] The latissimus dorsi weakened significantly over the control group when measured by manual muscle testing, but it did not against the force transducer. Blaich and Mendenhall[9] compared manual and Cybex II muscle testing and found that the two are statistically independent. Blaich[8] found only a 40% correlation between the Cybex II and manual muscle testing when evaluating and correcting sacral respiratory faults. Nicholas et al.[41] have had considerable experience with manual muscle testing in a sports medicine clinic. They state, "What is measured manually cannot be measured by the Cybex alone."

In this author's experience, there is a close correlation between the Cybex II dynamometer and manual muscle testing when the cause of muscle weakness is peripheral nerve entrapment, such as an intervertebral disc. When other factors — such as the cranial-sacral primary respiratory mechanism, active reflexes, or imbalanced meridian energy — are at fault, correlation is poor between manual muscle testing and the Cybex II dynamometer. It appears that the major difference between testing against fixed transducers — whether isometric or concentric — is that the muscle is required to simply produce power; in manual muscle testing, the muscle is required to adapt to the changing pressure of the examiner's force. This requires effective function of the gamma system adjusting the neuromuscular spindle cell, and proper interpretation of its afferent supply and response by the neuraxis.

The manual muscle test, as generally described, starts with the examiner asking the patient to resist as he applies force to the patient. With the examiner's application of force, a sensation of muscle locking is perceived. With this perception, the examiner increases his testing pressure to overcome the patient's contraction, taking the muscle into an eccentric contraction; that is, the muscle is lengthened by the examiner's pressure while the patient continues to attempt to stop the movement. It appears that a major factor in this type of test is the ability of the patient's nervous system to lock the muscle against the examiner's pressure, and to continue adapting the muscle to meet the changing demands of the examiner's testing force. Often the examiner perceives a muscle as weak because it is late in adapting to his changing pressure. If the examiner applies pressure very slowly, allowing additional time for the muscle to adapt to it, the muscle will be perceived as strong.

It is amazing that the time factor of the manual muscle test is not considered more thoroughly. As early as 1917, Ryan and Agnew[46] proposed that the product of force and time was the significant factor. Nicholas et al.[40] convincingly demonstrated the influence of time on the test. They state, "It has been generally assumed that manual muscle tests are tests of 'strength'; that is, of the force with which the patient resists the tester. Our data indicates that time required to move the limb through a certain range of motion multiplied by the average force of resistance applied during that range was the factor most highly correlated with the tester's perception of deficits in strength." When a muscle is in voluntary isometric contraction, EMG reveals that additional muscle fibers contract at low forces; when the force increases, the rate of firing becomes the predominant mechanism to increase strength.[37] Tension, velocity, and electrical activity are interdependent. Integration of the electrical record provides a composite measure of the number of active fibers and their frequency of excitation.[7] This indicates the need of proper neurologic control for the muscle to meet the changing pressure demands of the manual muscle test.

A manual muscle test that takes the muscle from isometric to eccentric contraction is called the "break test technique" by the group at the Institute of Sports Medicine and Athletic Trauma. This type of manual muscle testing more closely parallels that of applied kinesiology than any other.[33] Nicholas, a member of that group, conceived a hand-held instrument that measures the force during the test. The application of the unit has been studied primarily in testing muscle groups, such as abduction, adduction, flexion, and extension.[33] When applied to more individualized muscle testing, as applied kinesiology does, it is hoped that this type of instrument will help quantitate manual muscle testing, especially if combined with the timing of the test.

Presently the best "instrument" to perform manual muscle testing is a well-trained examiner, using his perception of time and force with knowledge of anatomy and physiology of muscle testing. With this combination there has been positive agreement in some interexaminer reliability studies. Conable and Hanicke[1] found 78.2% agreement between two trained muscle testers when each was blind to the other's findings. After correcting cranial faults and ocular lock, there was 100% agreement between them.

This brings up an important factor regarding the results of manual muscle testing. The subject being tested may change the parameters of the test unknown to the examiner. For example, when cranial faults are

present the phase of respiration that the patient takes or holds during the test has a bearing on muscle strength. In fact, individuals with disturbance in the cranial-sacral primary respiratory mechanism will often innately take and hold the phase of respiration that gives optimal function to the muscle. In other respects, subjects who have muscles that are poorly controlled by the nervous system will innately attempt to change the test parameter in any way, such as recruiting other muscles, in an attempt to perform the test adequately.

In a double-blind study, Jacobs[24] found 81.9% agreement between two testers. There was no control of other factors — such as cranial faults and ocular lock — as in the Conable/Hanicke study. Some studies that have evaluated multiple muscles for interexaminer reliability have found significant agreement between examiners for some muscles and not for others.[12,27] Other interexaminer reliability studies have been done that report good interexaminer reliability.[32,43,48,55] The quality of the study design varies considerably.

One of the unique factors of applied kinesiology is the numerous methods of treatment found that affect muscle strength as perceived by the manual muscle test. Any controlled research in applied kinesiology must take this into account. If one studies the effect of chewing nutrition on muscle strength, other possible causes of a weak muscle must be considered. If a muscle is weak because of a subluxation, dysfunctioning neuromuscular spindle cell, cranial faults, or whatever, one cannot expect the nutrition associated with the muscle to strengthen it. These factors must be controlled, as well as performing proper muscle testing to rule out test parameter changes.

Factors that Influence Manual Muscle Testing

Consistent timing is imperative in comparing one muscle test with another. The most crucial portion of applying pressure in a muscle test is at the beginning. Schmitt[51,52,53] reports a difference when the action of the muscle being tested is initiated before or after the doctor's application of pressure. This is termed "patient-induced" or "doctor-induced" muscle testing. Schmitt originally referred to the doctor-induced muscle test as a gamma I test and the patient-induced test as a gamma II test, referring to the activity within the neuromuscular spindle cell. The doctor-induced test is discussed earlier in this chapter and is the most common type of testing. It is applicable to most of the techniques in this introductory text. The starting position is the same for the patient-induced test; the patient develops full isometric contraction against the doctor's resistance. When the patient's full force is exerted the doctor increases the counteracting force to overcome the patient's strength, changing the patient's contraction from isometric to eccentric. Later Schmitt described a third test in which the patient and doctor are positioned to do the muscle test, and the doctor instructs the patient to push against his testing hand. As soon as force is felt from the patient, the doctor applies his testing force. This is concentric to eccentric patient contraction.

At this point the terms were changed to G-1 for doctor-induced test and G-2 for patient-induced to the maximum force test, and G-2s or G-2 submax for the third type of testing. Recently, a third change in terminology has been made to type 1, type 2, and type 3, respectively.[54]

Any combination of weakness can exist in a muscle at the same time. The type(s) of positive test 1, 2, and 3 give additional neurologic information about muscle weakness.[54]

Type 1 test is used as a general screening test. Most of the techniques in Chapters 2, 3, 6, and 7 of this text are used to correct this type of weakness. This includes spinal manipulation of subluxations, joint manipulation, reflex and trigger point treatment, and meridian point stimulation, among other treatments. When type 1 testing is positive, it is applicable to further test with types 2 and 3.

Type 2 test indicates suprasegmental (supraspinal) problems. These include chemical imbalances such as nutritional needs and hypothalamic monitored activities, e.g., electrolyte imbalances, autonomic imbalance, and stomatognathic system problems.

Type 3 weakness relates to the withdrawal reflexes following injury, allergy and hypersensitivity type reactions, systemic functional endocrine imbalances, and visual motor problems such as functional problems with accommodation reflexes.

Type 1 testing is done by the doctor asking the patient to resist as he increases his testing pressure to take the patient's muscle into eccentric contraction; this is doctor-induced muscle testing. The test is not one of matching muscle strength. The doctor is feeling for the patient's nervous system's ability to adapt the muscle action to the testing pressure. When the muscle test is normal, the doctor feels a rapid locking against his testing pressure. When first learning the art of muscle testing, it helps to try testing muscles at different initial speeds of pressure application.

The patient's head position affects muscle test results due to the tonic neck and tonic labyrinthine reflexes.[50] These reflexes are present in the newborn and remain throughout life. As the child matures, other more sophisticated reflexes develop from input, such as the eyes and mechanoreceptors throughout the body.

The tonic neck reflexes (TNR) are from the mechanoreceptors of the upper cervical vertebrae. There are predictable muscle facilitation and inhibition patterns with head-on-neck movement that are observed most actively in the decerebrate, labyrinthinectomized animals and in human infants.[19] With head and upper neck extension the upper extremities (forelimbs) extend and the lower extremities (hindlimbs) flex. This is the neurologic basis for gluteus

maximus weakness in the presence of upper cervical fixation. Flexion of the head and upper cervical vertebrae causes upper extremity flexion and lower extremity extension.

Head rotation and tilting also affect the extremities through the tonic neck reflexes. Rotation or tilting results in facilitation of the extensors and inhibition of the flexors to the side of movement. On the contralateral side of head movement there is opposite reaction, i.e., facilitation of the flexors and inhibition of the extensors.

Muscle facilitation and inhibition from the tonic neck reflexes can be seen in many actions. When a cat looks up to observe a bird in a tree there is facilitation of the forelimbs and inhibition of the hindlimbs. This tends to move the cat into a seated position that is more appropriate for observing the bird. On the other hand,

8—2. Head and cervical extension cause forelimb facilitation and hindlimb inhibition.

looking down to eat causes the opposite action to lower the front part of the body toward the food.

Head tilting or rotation equates with head-on-neck motion. When one is falling the head turns toward the direction of fall and appropriate muscle facilitation develops to resist the fall.

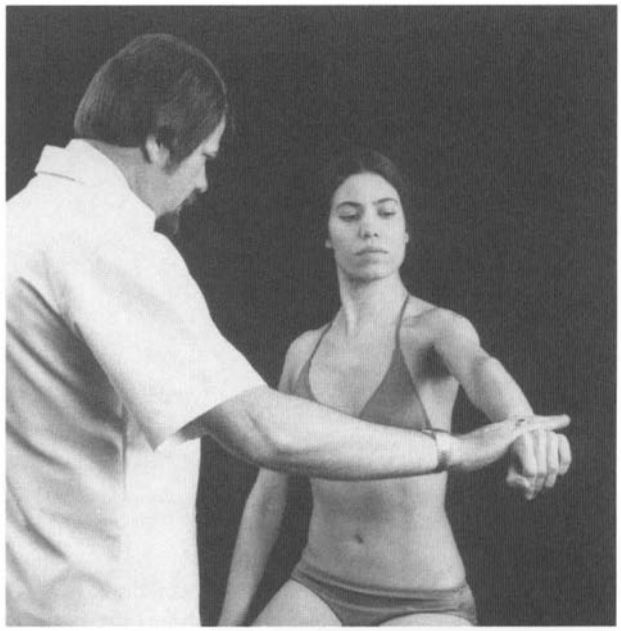
The tonic labyrinthine reflexes (TLR) are also present and active at birth. As the infant matures, more important righting reflexes from the visual receptors, semicircular canals, and remote mechanoreceptors supersede the importance of the TLR, but the latter remain constantly active throughout life.

The TLR are activated only by the relation of the head to gravity and not by the relation of the head to the neck or rest of the body. There are four distinct head positions that cause facilitation and inhibition of muscle patterns: (1) Face up (supine), extensors of all four limbs are facilitated. (2) Face down (prone), flexors of all four limbs are facilitated. (3) Ear up, inhibition of the ipsilateral extensors and facilitation of the flexors. (4) Ear down, facilitation of the ipsilateral extensors and inhibition of the flexors. In the ear up and down positions there is reverse facilitation and inhibition contralaterally.

When the TLR are functioning properly, there will be no weakness of an extensor when tested supine and no weakness of a flexor when tested prone. Often the TLR are aberrant, and an extensor weakness may be found when supine or a flexor weakness when prone. In this case there is a TLR fault. If there is no TMJ fault, the patient can clench his teeth; this will override the TLR. A TLR fault often relates to dysfunction in the stomatognathic system or the tilt pattern of PRYT technique.

A major objective of manual muscle testing in applied kinesiology is to isolate the muscle being tested to the maximum. In all tests — with the exception of the muscles that move the distal phalanges of the fingers and toes — there is some synergism taking place. The starting position for the muscle test is that which places the muscle being tested at the greatest advantage, with the synergists at a disadvantage. When the prime mover being tested is weak, the patient's natural reaction is to shift the test position to recruit synergistic muscles. An expert muscle tester learns as much or more from observing what the patient does as from the perception of force produced by the tested muscle. An example is how the side-lying patient with a weak gluteus medius will posteriorly rotate the pelvis on the side being tested to align the tensor fascia lata for hip abduction.

The muscle being tested must operate from a stable base. In most cases this requires the examiner to stabilize the structure from which the muscle originates. The patient may attempt to shift the base from which the tested muscle originates, or there may be failure of the patient's muscles to stabilize the base. In the first instance, the patient may rotate the pelvis to recruit adductor muscles when the psoas muscle is

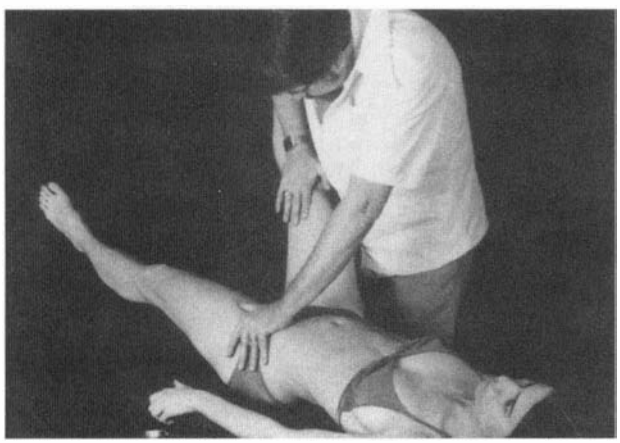


8—3. A general test such as the "arm pull-down" test is not specific and gives poor ability to reproduce findings and provide accurate information.

tested and shows weakness. The rotation of the pelvis toward the side being tested aligns the adductors to be more active in the test. It is necessary for the examiner to stabilize the pelvis on the contralateral side from the test. If the examination table has thick, soft cushions, pelvic stabilization may be almost impossible. Pelvic rotation is allowed by compression of the cushion on the side of the test.

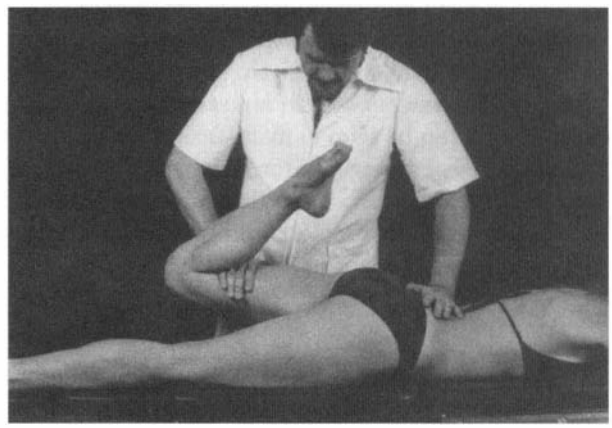
An example of the patient's muscles failing to stabilize the base is seen when there are weak abdominal muscles when the sternocleidomastoid muscle is tested. It may appear weak until the examiner stabilizes the thorax, giving the sternocleidomastoid muscle a stable base from which to operate.

The results of the muscle test may be influenced if the patient experiences pain during the test. When attempting to produce maximum muscle power, it may be pain that causes the muscle to let go rather than fatigue or muscle weakness.[16] Usually it is obvious when pain causes the muscle to test weak; however, one should advise the patient to indicate when the test is painful. The presence of pain does not mean that the test cannot be performed; in fact, additional valuable information can often be gained. For example, when one of the rotator cuff muscles tests weak with shoulder pain, the patient can often perform the test very well, without pain, when therapy localizing to the neurolymphatic reflex, holding a certain phase of respiration, or adding some other applied kinesiology factor. This not only helps the examiner determine the cause of shoulder dysfunction, it is also strong positive feedback to the patient that the doctor understands the condition.

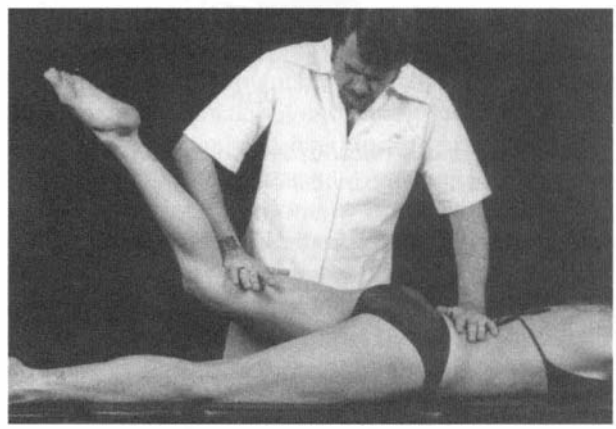


8—4. The examiner's hand stabilizing over the anterior superior iliac spine can cause pain and, consequently, inaccurate results.

Good muscle testing procedure considers several aspects of the test. When putting the patient into the starting position, observe whether he makes an effort to change the position to recruit synergistic muscles. For example, when the prone patient's hip is extended with the knee flexed for a gluteus maximus test, observe whether there is an effort to extend the knee, recruiting hamstring function. The patient with a strong gluteus maximus can easily hold the leg in the starting position without extending the knee. The same observation can be made when the examiner first begins to apply pressure. If muscle weakness is present, there is often an effort by the patient to extend his knee or rotate his pelvis.



8—5. Maximum knee flexion may limit hip extension if the rectus femoris is short. The position helps take the hamstrings out of the test.



8—6. Patient will tend to straighten leg to recruit more hamstring synergism.

A common observation among those who have made interexaminer reliability studies of manual muscle testing is that the examiner must be knowledgeable about physiology, anatomy, and the factors that change the test parameters. This is the scientific aspect of manual muscle testing. Added to this is the examiner's developed art in perceiving time and force.

The scope of this text is adequate as an introduction to manual muscle testing. The applied kinesiology student is encouraged to continue study of the subject[25,33,57] and to attend workshops on muscle testing.

Provisional neurolymphatic, neurovascular, and meridian association are listed for some muscles in the following material because there has been little requirement for treatment with those aspects. The muscle usually responds to other treatment methods.

Piriformis

Origin: anterior surface of sacrum between — and lateral to — anterior sacral foramen, capsule of sacroiliac articulation, margin of greater sciatic foramen, and sacrotuberous ligament.
Insertion: superior border of greater trochanter of femur.
Action: rotates thigh externally, abducts thigh when limb is flexed.
Test:

Sitting: The patient's knee is flexed to 90°, and the thigh is externally rotated. Pressure is directed toward the distal leg to internally rotate the thigh while the patient resists.

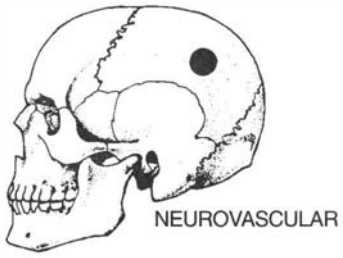
Prone: The patient flexes the knee to 90° and externally rotates the thigh. The thigh is stabilized by the examiner while pressure is directed to the lower leg to internally rotate the thigh.

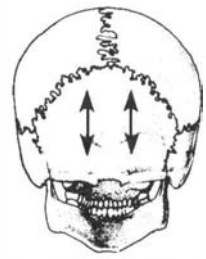
Nerve supply: Sacral plexus, L5, **SI, 2**.
Neurolymphatic:

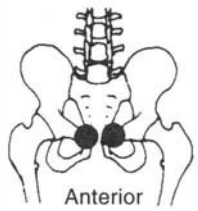
Anterior: upper symphysis pubis.

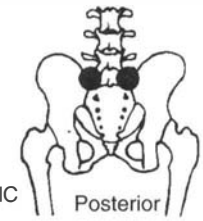
Posterior: between posterior superior iliac spine and L5 spinous.

Neurovascular: on parietal eminence, posterior aspect.
Nutrition: vitamin A, male or female endocrine concentrates or nucleoprotein extracts.
Meridian association: circulation sex.
Organ/gland association: reproductive organs or glands.





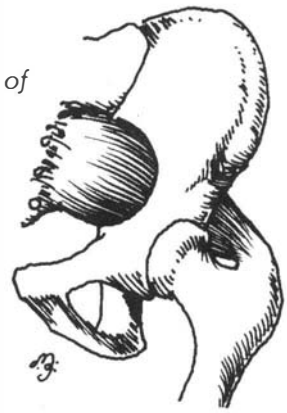


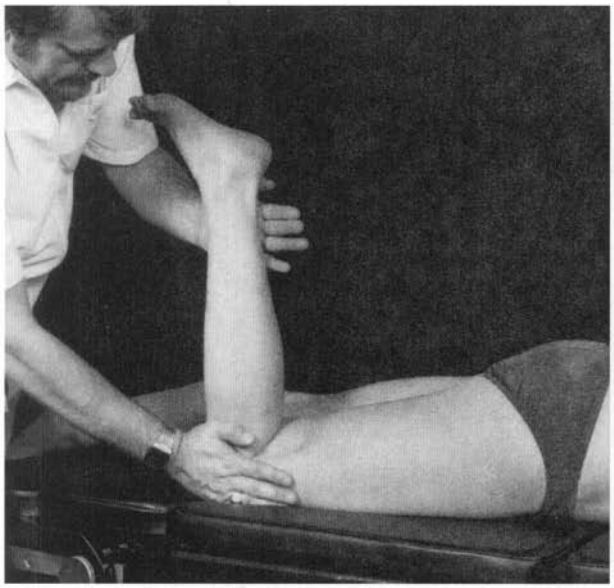


NEUROLYMPHATIC

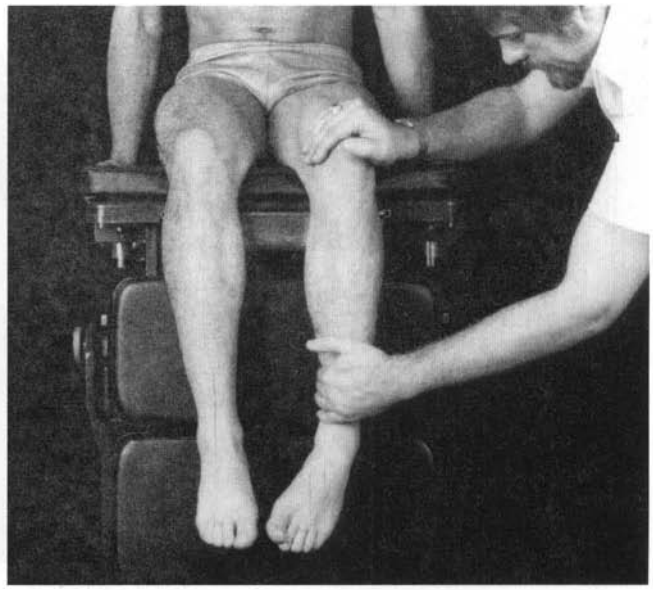
8—7. Posterior view of piriformis.

8—8. Anterior view of piriformis.





8—9. Prone piriformis test.



8—10. Seated piriformis test.

Hamstrings

Semitendinosus — Medial Hamstring

Origin: ischial tuberosity with tendon of biceps femoris.
Insertion: proximal portion of medial surface of the tibia and deep fascia of the leg.
Nerve supply: sciatic (tibial branch, which develops two branches), L4, **5, S1, 2**.

Semimembranosus — Medial Hamstring

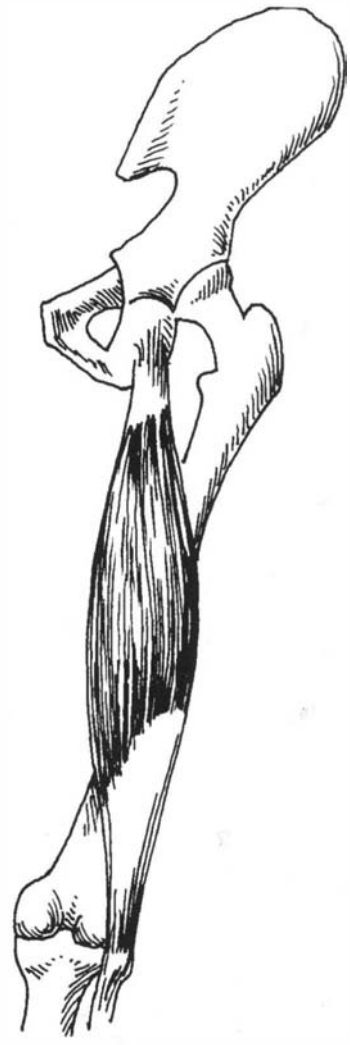
Origin: upper and lateral aspect of ischial tuberosity.
Insertion: posteromedial surface of the medial condyle of the tibia.
Action of medial hamstrings: flexes and internally rotates the knee; extends, adducts, and internally rotates the thigh.
Nerve supply: sciatic (tibial branch), L4, **5, S1, 2**.
Test for medial hamstrings: Pressure is directed against the distal leg in a direction of knee extension, slightly laterally. The examiner should note the direction of pressure that best raises the medial hamstring tendons and minimizes the raising of the lateral hamstring tendon.

Biceps Femoris — Lateral Hamstring

Origin:
Long head: ischial tuberosity and sacrotuberous ligament.
Short head: lateral lip of linea aspera, lateral supracondyle of femur, and lateral intermuscular septum.
Insertion: lateral side of the head of the fibula, lateral condyle of the tibia, deep fascia on the lateral side of the leg.
Action: flexes knee, extends thigh, externally rotates the knee joint, externally rotates and adducts thigh.
Nerve supply:
Long head: sciatic, tibial branch, L5, **S1, 2**, 3.
Short head: sciatic, peroneal branch, **L5, S1, 2**.
Test: During the test, the examiner should observe for the direction of pressure that best puts tension on the tendon of the biceps femoris and less tension on the tendons of the semimembranosus and semitendinosus. Observe for muscular contraction of the biceps femoris and diminished contraction of the semimembranosus and semitendinosus muscles by palpation.

8—11. Semitendinosus

8—12. Semimembranosus



8—13. Biceps femoris

Test of hamstring muscles as a group: With 60° knee flexion, the examiner directs pressure against the distal leg in a direction of knee extension. Stabilizing pressure in the bellies of the muscles will help prevent cramping. Pressure should not be directed against the calcaneus, because it is possible to challenge a calcaneal subluxation while simultaneously performing the hamstring muscle test. Also, pressure against the calcaneus lessens the examiner's ability to evaluate ankle position changes for efforts to increase synergism of the gastrocnemius.

Testing the hamstrings in a weight-bearing position is best accomplished with the patient leaning against a hi-lo table. If one is not available, the patient can be stabilized against a wall. The test is accomplished in a manner similar to that in the prone test.

Neurolymphatic:

Anterior: over the lesser trochanter of the femur.

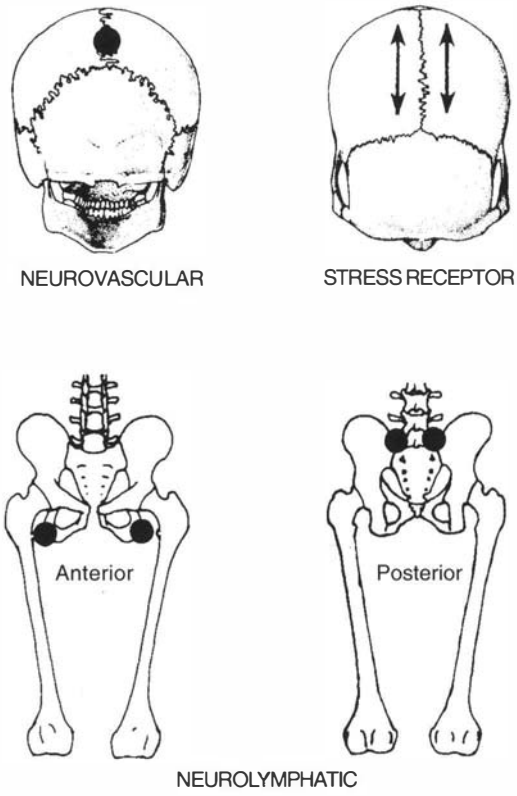
Posterior: upper sacroiliac articulation by the posterior superior iliac spine.

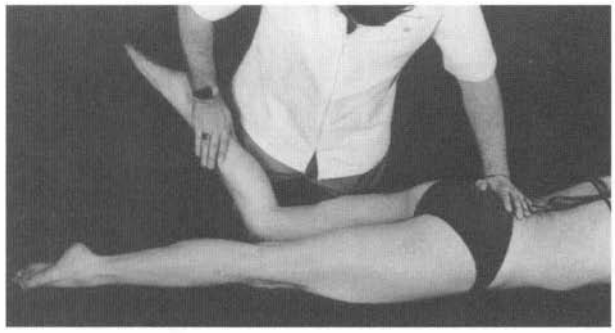
Neurovascular: 1" above the lambda.

Nutrition: vitamin E; consider the possible need for betaine hydrochloride if muscle cramping is present, or other calcium requirements.

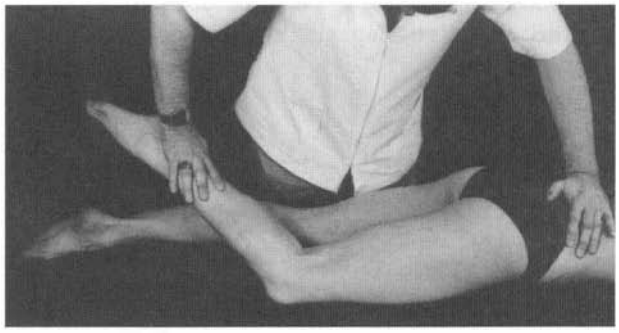
Meridian association: large intestine.

Organ association: rectum.

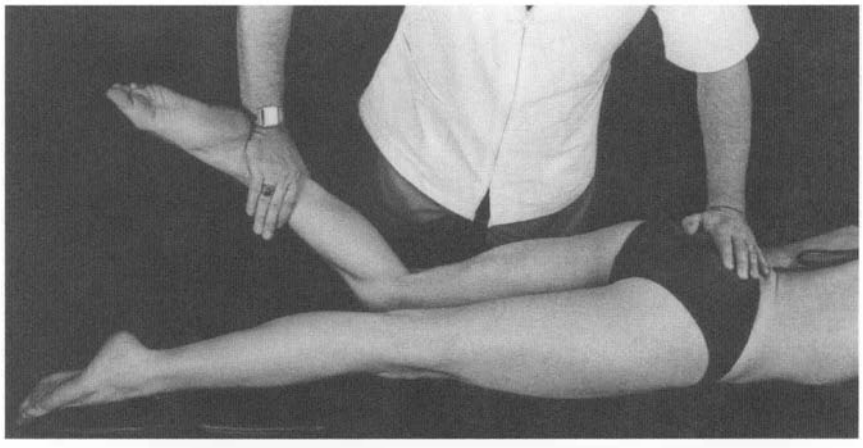




8—14. Medial hamstrings.



8—15. Lateral hamstring.



8—16. Hamstring muscles as a group.

Quadriceps

Rectus Femoris

Origin:

Straight head: from anterior inferior iliac spine.

Reflected head: from groove on upper brim of acetabulum.

Insertion: upper border of patella with the ligamentum patellae extending to tibial tubercle.

Action: extends the leg and flexes the thigh.

Test: The examiner directs force against the anterior thigh just proximal to the knee in a direction toward hip extension, ascertaining that no thigh rotation is present and that the knee stays flexed approximately 90°. A slightly built examiner may need to hold the side of the table to provide added power in this test. The psoas is very active in this test and must be evaluated separately to make a comparison with the rectus femoris. Observation of the patient going into the test position reveals considerable information regarding his hip flexor strength.

Vastus Medialis

Origin: lower half of the intertrochanteric line, linea aspera, medial supracondylar line, medial intermuscular septum, tendons of adductor magnus and adductor longus.

Insertion: medial border of the patella with the ligamentum patellae extending to the tibial tubercle.

Action: extends the leg and draws the patella medially.

Vastus Intermedius

Origin: proximal two-thirds of the anterolateral surface of the femur, lower half of the linea aspera, upper part of the lateral supracondylar line, lateral intermuscular septum.

Insertion: by tendons of the rectus and vastus muscles into the superior border of the patella with the ligamentum patellae extending to the tibial tubercle.

Action: extends the leg.

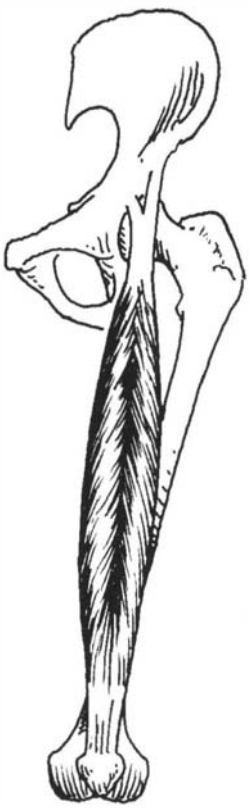
Vastus Lateralis

Origin: intertrochanteric line, greater trochanter, gluteal tuberosity, linea aspera, lateral intermuscular septum, capsule of the hip joint.

Insertion: lateral border of the patella with the ligamentum patellae extending to the tibial tubercle.

Action: extends the leg and draws the patella laterally.

Test (as a group): With the patient seated, the examiner directs pressure against the distal anterior leg just above the ankle in the direction of knee flexion. Care must be taken not to allow the patient to lock the knee in extension. If the table edge is sharp, the examiner should place his hand under the knee to cushion it. The examiner should observe for change of pelvic position during the testing procedure.



8—17. Rectus femoris

8—18. Vastus medialis

8—19. Vastus intermedius

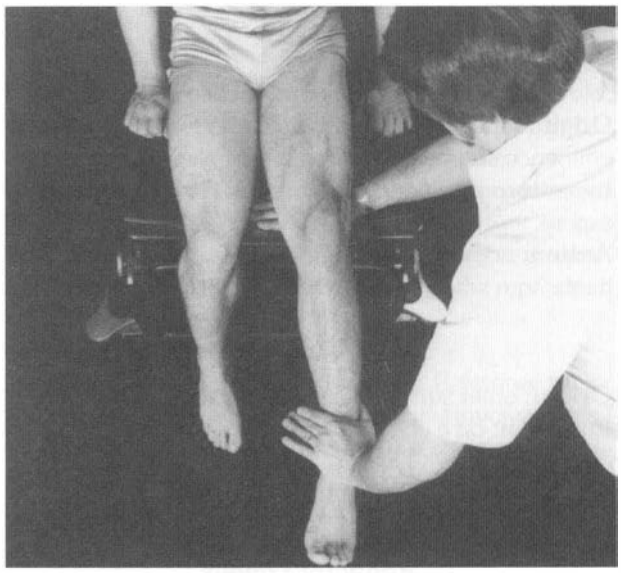
8—20. Vastus lateralis

Nerve supply: femoral, **L2, 3. 4**.
Neurolymphatic:

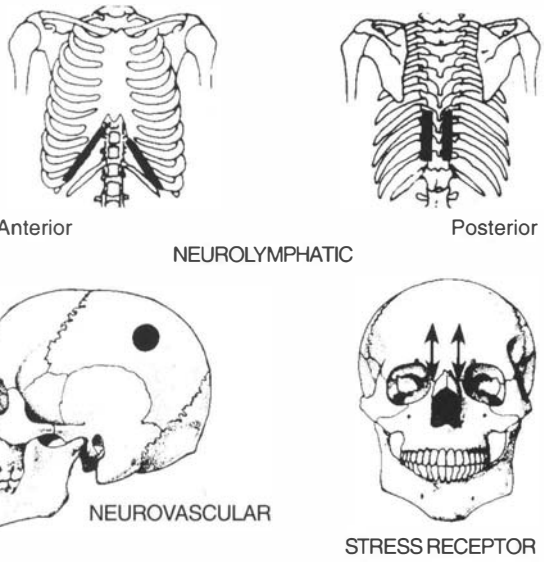
Anterior: along costochondral junction of the 8th-11th ribs. Activity of this linear neurolymphatic is inverse to involvement of the quadriceps muscle divisions. In other words, for the vastus lateralis, neurolymphatic activity will be medial; for the vastus medialis, the activity will be lateral on the reflex area.

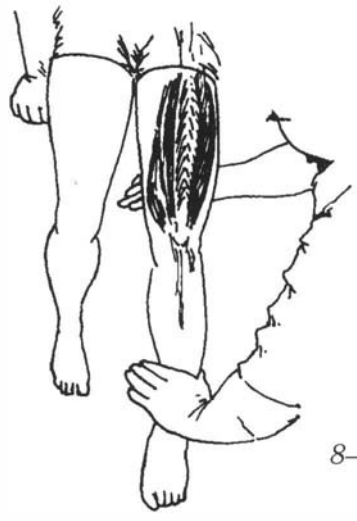
Posterior: T8-11 laminae.

Neurovascular: parietal eminence, posterior aspect.
Nutrition: vitamin D, vitamin B complex, small intestine nucleoprotein extract or concentrate.
Meridian association: small intestine.
Organ association: small intestine.

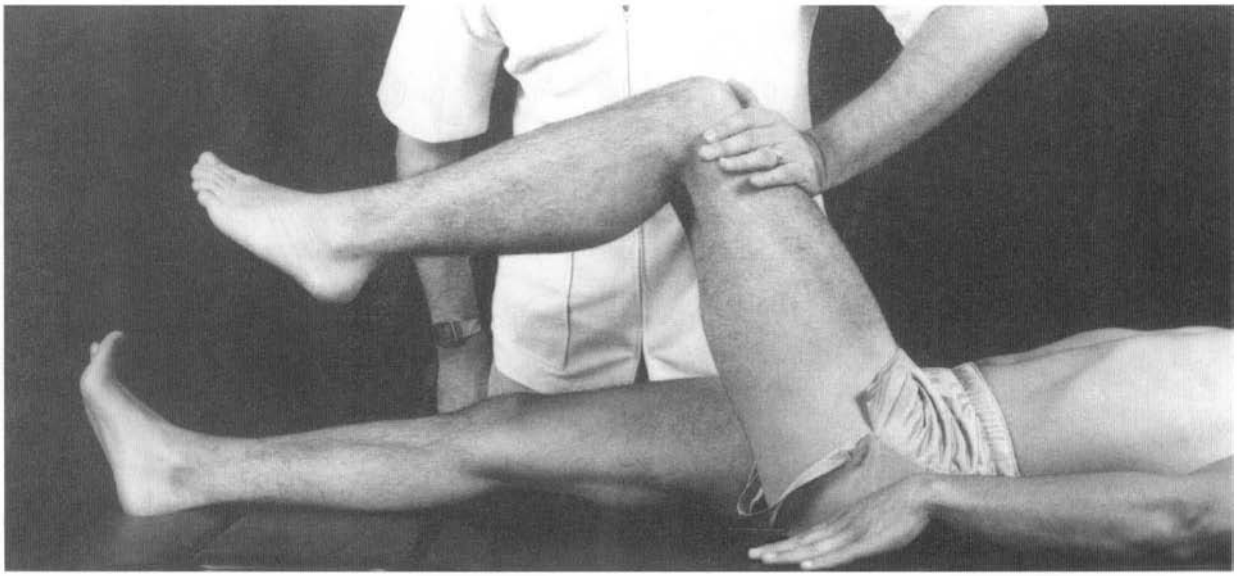


8—21. Group quadriceps test





8—22.



8—23. Care must be taken that no internal or external thigh rotation is allowed during test.

Adductors

Pectineus

Origin: superior surface of the pubis between iliopectineal eminence and pubic tubercle.
Insertion: pectineal line from lesser trochanter to linea aspera.
Action: adduction, flexion, and internal rotation of the thigh.

Adductor Brevis

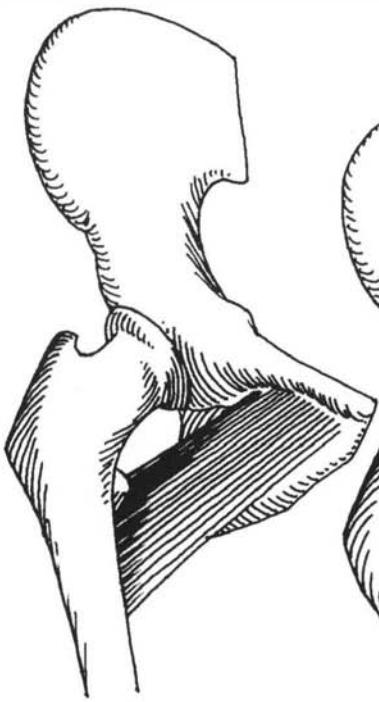
Origin: outer surface of inferior ramus of pubis.
Insertion: on a line extending from lesser trochanter to linea aspera.
Action: hip adduction, with some assistance in hip flexion.

Adductor Longus

Origin: anterior of pubis in angle between crest and symphysis.
Insertion: middle one-third of medial lip of linea aspera.
Action: adducts thigh with some assistance in hip flexion.

Adductor Magnus

Origin:
Posterior fibers: ischial tuberosity.
Anterior fibers: ramus of ischium and pubis.
Insertion: from a line extending from the greater trochanter along linea aspera, medial supracondylar line, and ending at the adductor tubercle of the medial condyle of the femur.
Action: adduction in combination with other hip adductors. Fibers arising from ischium and ramus of ischium primarily insert distally and aid in hip extension. Fibers arising from ramus of pubis insert proximally and aid in hip flexion.



8—24. Pectineus

8—25. Adductor brevis

8—26. Adductor longus

8—27. Adductor magnus

Test (all): With patient side-lying, the examiner abducts the upper non-tested leg. This is simply to move the leg out of the way of the test. The patient adducts the lower tested leg. The examiner applies the testing pressure at the knee in the direction of abduction.

Nerve supply:

Pectineus: femoral and obturator nerves, **L2, 3**, 4.

Adductor magnus: obturator and sciatic, L2, **3, 4**, 5, S1.

Adductor longus: obturator, L2, **3**, 4.

Adductor brevis: obturator, L2, **3, 4**.

Neurolymphatic:

Anterior: behind areola, not in the breast tissue. If the patient is female have her lift her breast out of the way. The patient can stimulate the reflex to avoid any question of sexual impropriety.

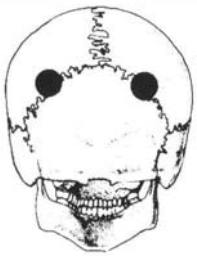
Posterior: below inferior angle of scapula.

Neurovascular: on lambdoidal suture between lambda and asterion.

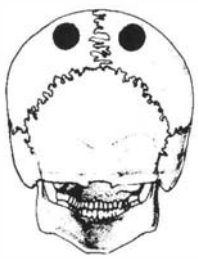
Nutrition: vitamin E, endocrine concentrates or nucleoprotein extracts, usually of some aspect of the reproductive system.

Meridian association: circulation sex.

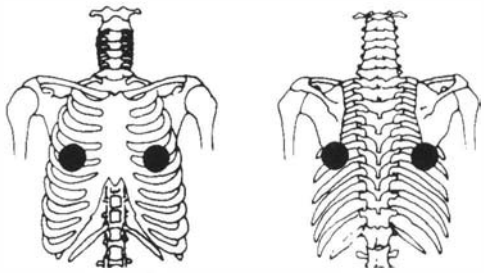
Organ/gland association: climacteric. Reference to climacteric indicates a wide range of potential involvements, including the reproductive organs and glands, adrenal, liver, pituitary, and thyroid. The interplay of the endocrine system is responsible for this wide range of associations.



NEUROVASCULAR

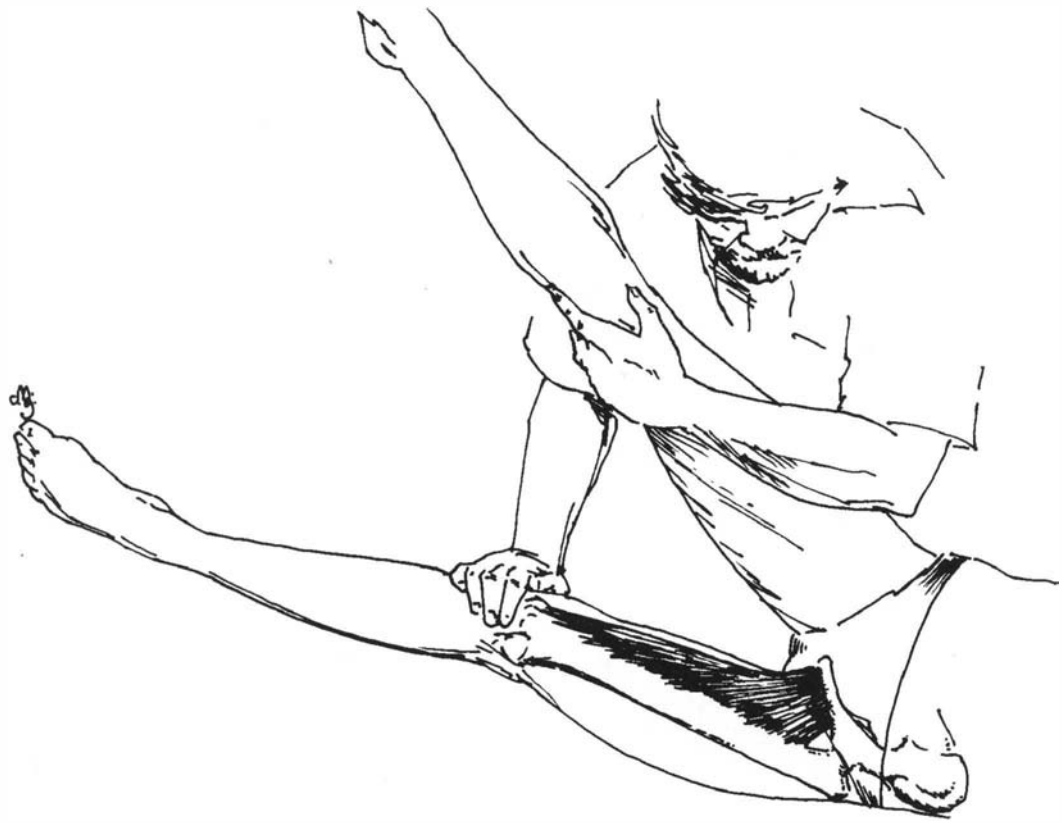


STRESS RECEPTORS



Anterior Posterior

NEUROLYMPHATIC



8—28. Test of adductor muscles as a group. Pressure is directed only to leg next to table.

Chapter 8

Abdominals

Transverse Abdominal

Origin: lateral third inguinal ligament, anterior three-quarters of the internal edge of the iliac crest, lumbodorsal fascia, and from the inner surfaces of the lower six costal cartilages.
Insertion: into the linea alba aponeurosis, which passes behind the rectus abdominis.
Action: constricts abdominal contents. Assists in forced expiration and stabilizes linea alba.

Rectus Abdominis

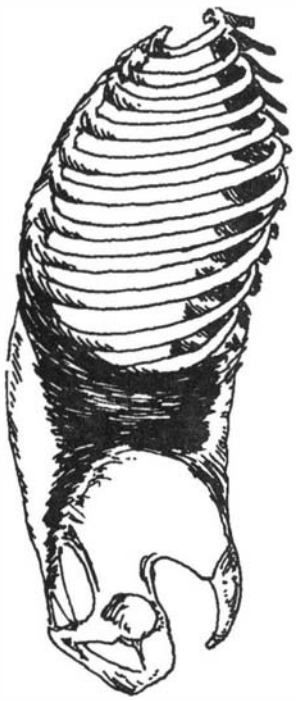
Origin: from the crest of the pubis and the symphysis pubis.
Insertion: into the costal cartilage of the 5th, 6th, and 7th ribs and the side of the xiphoid process.
Action: in standing position, supports organs anteriorly. By way of supporting organs and holding rib cage and pubis together, gives anterior support to the lumbar spine. With aid of gluteus maximus, keeps pelvis from going into anterior tilt.

External Oblique Abdominal

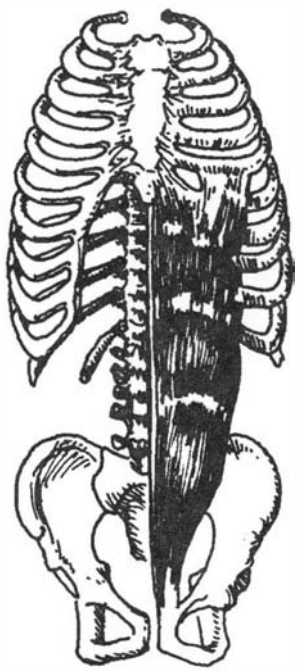
Origin: from the external inferior borders of the lower eight ribs. The five superior attachments interdigitate with the serratus anticus, and the lower three interdigitate with the latissimus dorsi and their attachments.
Insertion: into the anterior half of the outer lip of the iliac crest and a broad aponeurosis, which ultimately inserts into the linea alba.
Action: anterolateral abdominal wall stability, giving support to the organs and anterior support to lumbar spine. Flexes vertebral column when acting together, and draws pubis toward xiphoid process. Assists rectus abdominis in obtaining anterior pelvic stability with the gluteus maximus. Unilateral action assists in lateral bending, or rotates the vertebral column, bringing the shoulder of the same side forward.

Internal Oblique Abdominal

Origin: from lateral half of the inguinal ligament, from the anterior two-thirds of the intermediate line of the iliac crest, and from the lower portion of the lumbar aponeurosis near the crest.
Insertion: inferior borders of the lower three or four costal cartilages and into an aponeurosis that terminates in the linea alba.
Action: compresses the abdominal contents, supporting the viscera and giving anterior stability to the lumbar spine. Aids in bringing pelvis and thorax together as well as flexing the lumbar spine when acting together. Acting unilaterally, laterally flexes the vertebral column and rotates it, bringing the shoulder of the opposite side forward.



8—29. Transverse abdominal



8—30. Rectus abdominis

8—31. External oblique abdominal

8—32. Internal oblique abdominal

Test (all): sitting, patient with spine at approximately 60-75°, with legs extended. Examiner directs force against anterior upper thorax while stabilizing legs against table. This primarily tests the rectus abdominis. The obliques also act bilaterally. The obliques are tested in the same manner with the patient's spine rotated. When the patient is rotated to his right, the left external and the right internal obliques are being tested.

Neurolymphatic:

Anterior: upper one-half of anteromedial thigh.

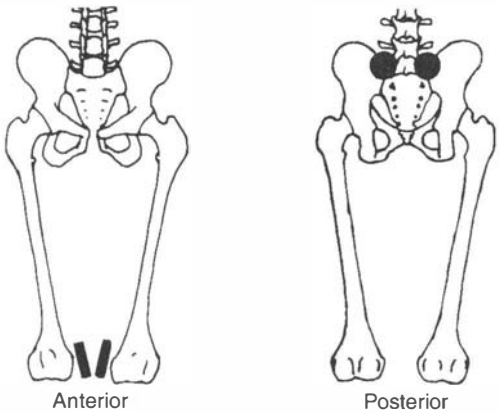
Posterior: between PSIS and L5 transverse process.

Neurovascular: bilaterally on parietal eminence, 2" posterior to frontal parietal suture.

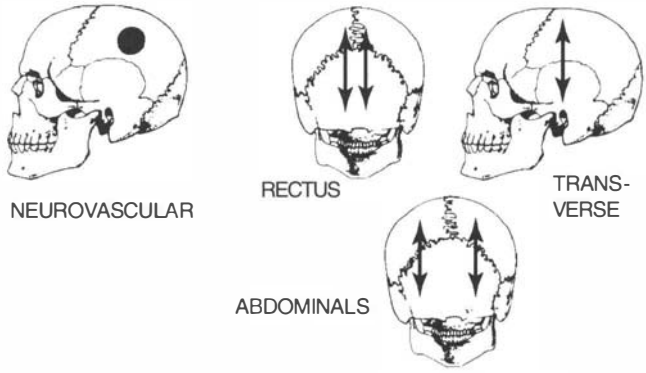
Nutrition: vitamin E, duodenal concentrate or nucleoprotein extract.

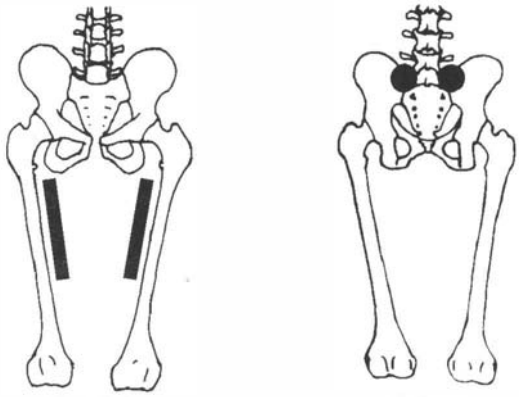
Meridian association: small intestine.

Organ association: small intestine.

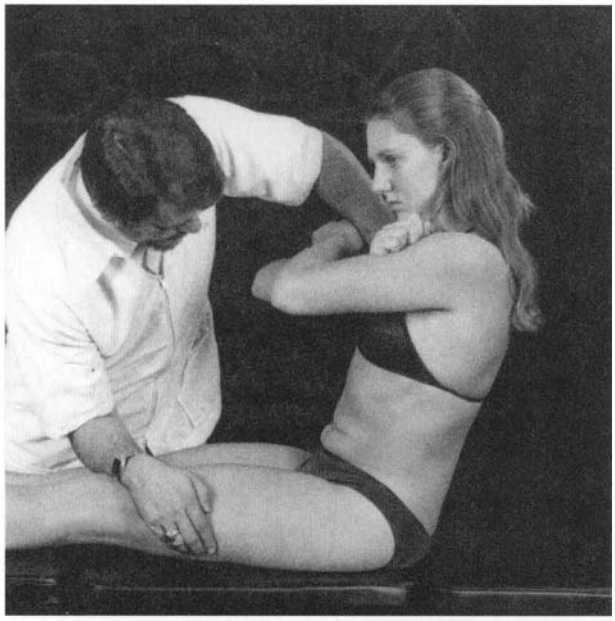


NEUROLYMPHATIC — RECTUS ABDOMINIS

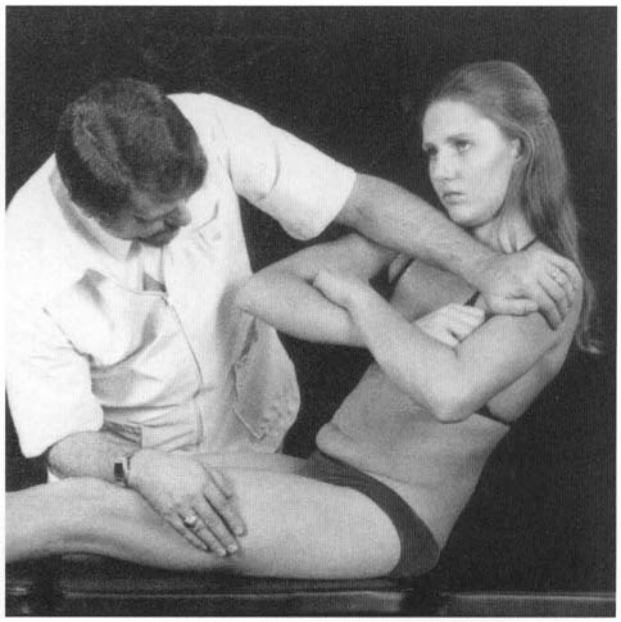




NEUROLYMPHATIC — OBLIQUE ABDOMINALS



8—33. General test for rectus abdominis.



8—34. Right external and left internal abdominal muscle test.

Abdominal Muscle Exercise

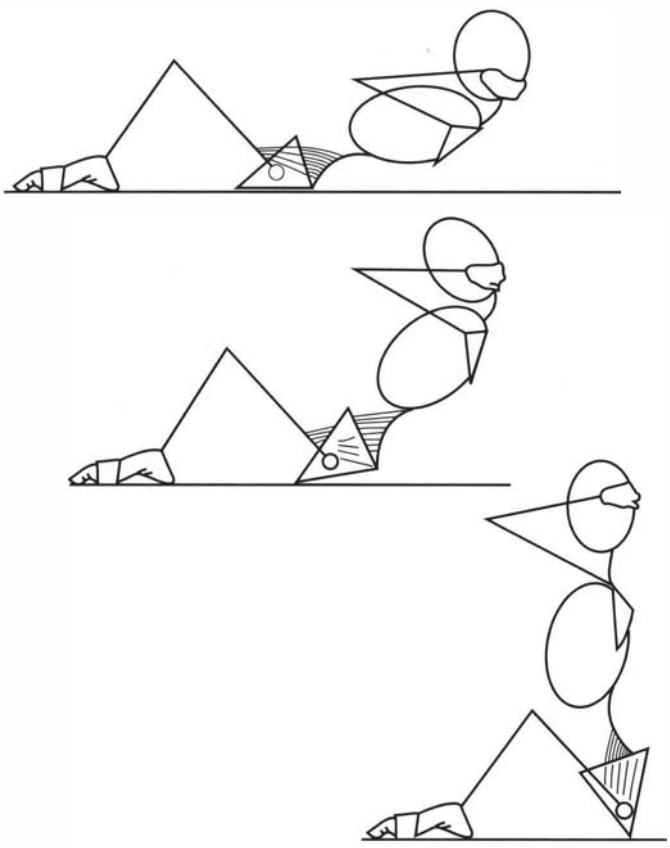
The abdominal muscles require exercise as a follow-up to applied kinesiology treatment more often than any other muscles. They are often found weak in lower back conditions. Clinical experience reveals that abdominal exercises are not very effective if one of the five factors of the IVF is causing weakness. After correcting the applied kinesiology finding, the patient can exercise more effectively with better results.

Weak abdominal muscles are often part of the low back complex causing increased lumbar lordosis and facet jam. The abdominal muscles help support the anterior pelvis superiorly, and the hamstrings and gluteus maximus pull it down posteriorly. Weakness of these muscles causes an anteriorly tilted pelvis, often compounded by the hip flexors and lower portion of the sacrospinalis being hypertonic and/or shortened.

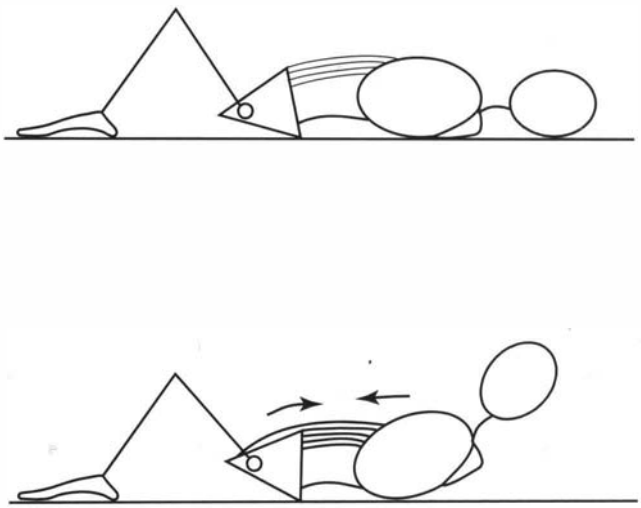
The abdominal muscles are most frequently involved with sagittal suture jamming. Poor posture puts a downward pull on the fascia that continues to the vertex of the head. This fascial pull tends to cause the sagittal suture jam, which in turn is often responsible for weak abdominals; thus a vicious circle develops. Until the posture improves, there is a tendency for recurrence of the sagittal suture jam; until the sagittal suture is corrected, the abdominal weakness tends to recur.

The gluteus maximus weakness is most often associated with an upper cervical fixation. Along with correcting the abdominal and gluteus maximus muscles, one should evaluate the length of the hip flexors (psoas, iliacus, and rectus femoris) and use applied kinesiology techniques to lengthen them, if necessary. The psoas muscle often needs strain/counterstrain technique.

Even if an individual does not have short hip flexors, it is good practice to prescribe abdominal exercises that do not activate them unless there is a specific indication to do so. Halpern and Bleck[20] studied and developed the "shoulder lift, hook-lying sit-up." Activity in this exercise requires only that the scapula be lifted from the table. Using surface electrodes, they electromyographically demonstrated significant activity of the rectus abdominis and external oblique during this exercise. They also point out the extraordinary increase in vertebral disc pressure during a forced sit-up. This activity may be harmful to the intervertebral disc if the annulus fibrosis has poor integrity. Their shoulder lift exercise changes the angle between L1 and L5 by only 3°, thus placing less stress into the intervertebral disc. Since many individuals who have abdominal muscle weakness have a compromised lumbar spine, it is good practice to improve the postural balance and avoid any damage in the therapeutic procedure.



8—35. Many sit-up type exercises strengthen the psoas, which increases the lumbar lordosis.



8—36. Hold the abdominal muscles in isometric contraction with the shoulder lift exercise.

Tensor Fascia Lata

Origin: anterior part of the outer lip of the iliac crest, anterior border of the ilium.
Insertion: middle one-third of the iliotibial tract of the fascia lata.
Action: thigh flexion, abduction, and internal rotation. Tenses fascia lata along with the gluteus maximus, pulling on the iliotibial band and
Test: The supine patient holds the leg in a position of abduction, medial rotation, and hip flexion, with the knee in hyperextension. Testing pressure is directed against the lower leg in a direction of adduction and extension. Observe that the patient does not flex the knee during the test.
Nerve supply: superior gluteal, **L4, 5, S1**.
Neurolymphatic:

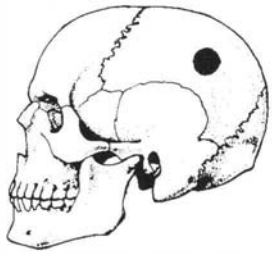
Anterior: anterolateral thigh bilaterally. This neurolymphatic reflex is divided into sections correlating with the sections of the large intestine.

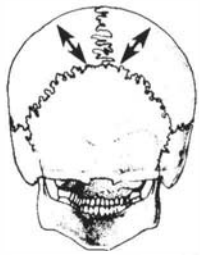
Right thigh: upper portion, cecum; middle three-fifths, ascending colon; lower portion, first portion of transverse colon.

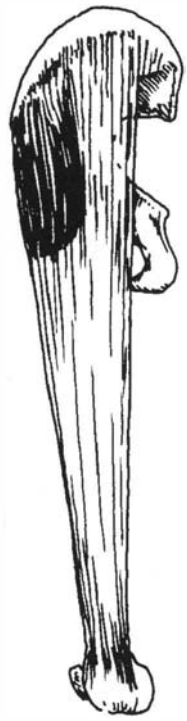
Left thigh: lower portion, last three-fifths of transverse colon; lower middle portion, descending colon; upper middle portion, upper sigmoid colon; upper area, junction of sigmoid colon with rectum.

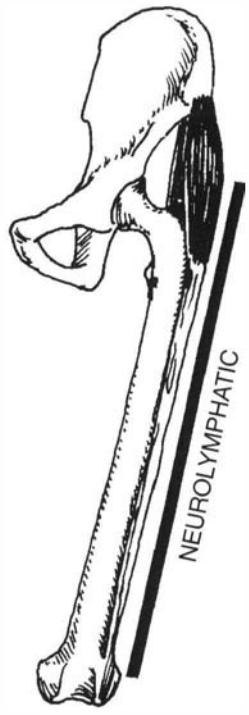
Posterior: triangular area with apexes at L2, L4, and the crest of the ilium.

Neurovascular: parietal eminence at the posterior aspect.
Nutrition: acidophilus, vitamin D. If bilateral, evaluate for iron deficiency anemia.
Meridian association: large intestine.
Organ association: large intestine.

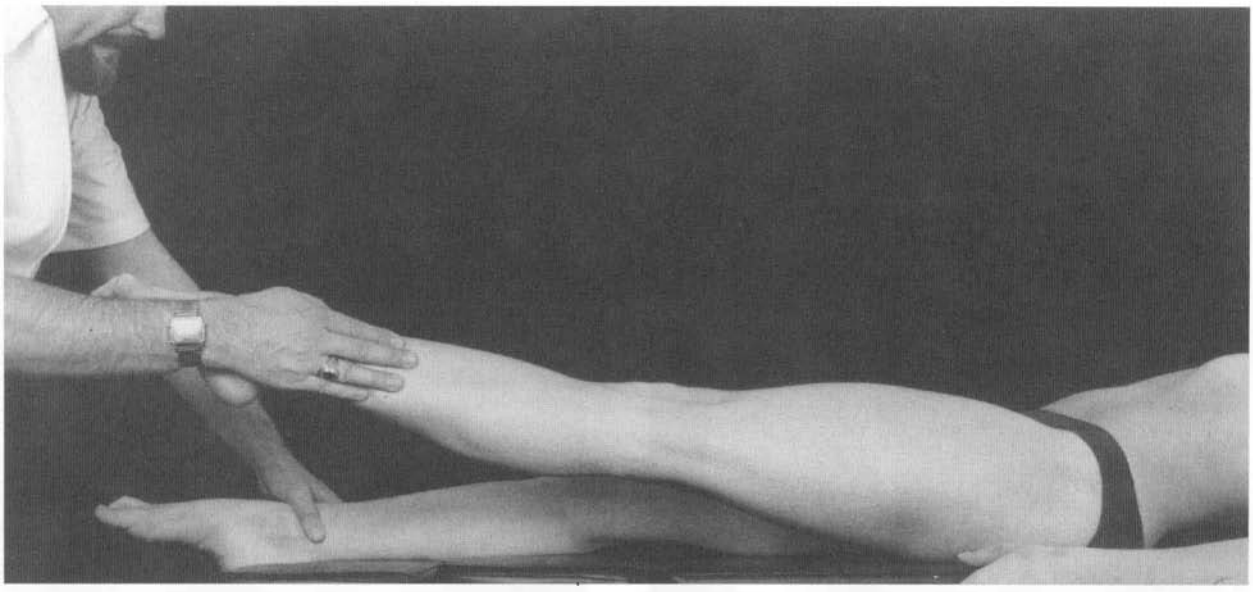

NEUROVASCULAR


STRESS RECEPTOR


8—37.



8—38.


8—39. Knee is maintained in extension and thigh in medial rotation.

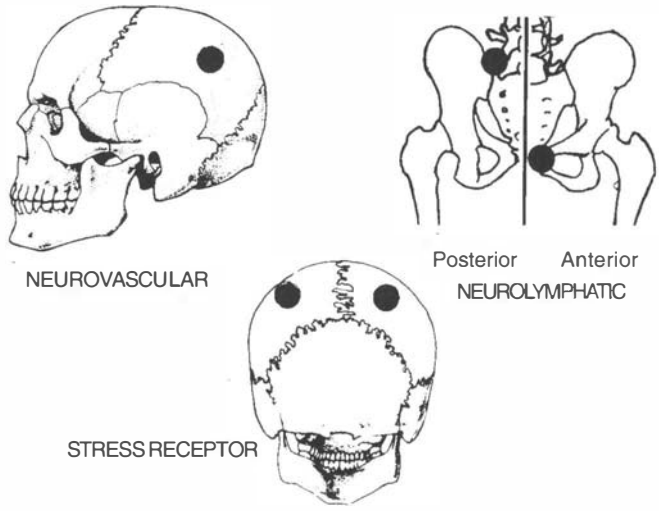
Gluteus Medius/Gluteus Minimus

Gluteus Medius

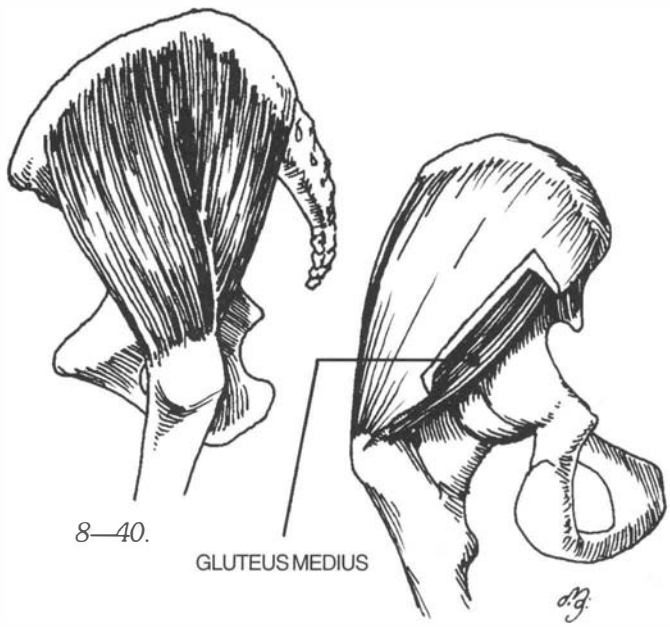
Origin: outer surface of ilium from iliac crest and posterior gluteal line above, to anterior gluteal line below, gluteal aponeurosis.
Insertion: lateral surface of greater trochanter.
Action: abducts thigh and rotates it internally. With gluteus minimus is major lateral pelvic stabilizer. Aids in early activity of hip flexion.

Gluteus Minimus

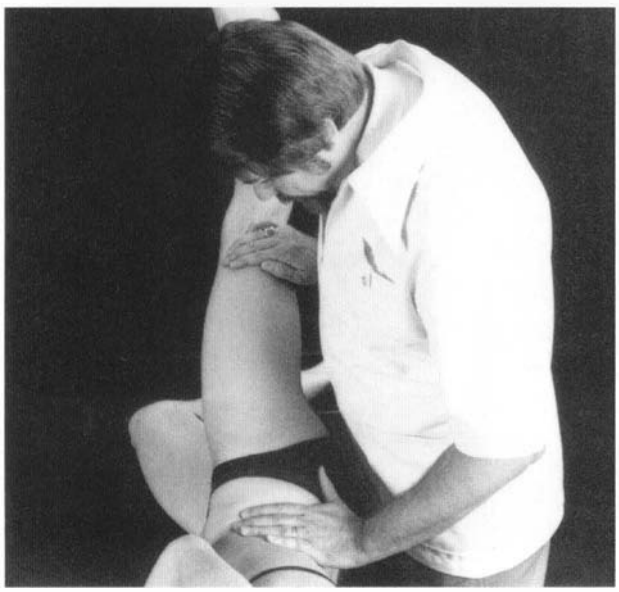
Origin: outer surface of ilium between anterior and inferior gluteal lines and margin of greater sciatic notch.
Insertion: anterior border of greater trochanter.
Action: abducts thigh and rotates it internally; assists gluteus medius in most functions.
Test (both): The side-lying patient flexes the hip and knee of the lower non-tested leg for stability. The examiner stabilizes the pelvis to prevent rotation. The patient abducts the hip with slight extension, keeping the knee in extension. Pressure is directed against the knee or ankle, depending on leverage required, in a direction of adduction and slight flexion. The patient's effort to shift the pelvis indicates substitution of the tensor fascia lata or gluteus maximus muscle.
Nerve supply: superior gluteal, **L4, 5, S1**.
Neurolymphatic:
Anterior: upper symphysis pubis.
Posterior: between posterior superior iliac spine and L5 spinous process.
Neurovascular: on parietal eminence, posterior aspect.
Nutrition: vitamin E, male or female endocrine nucleoprotein extracts or concentrates.
Meridian association: circulation sex.
Organ/gland association: reproductive organs and glands.



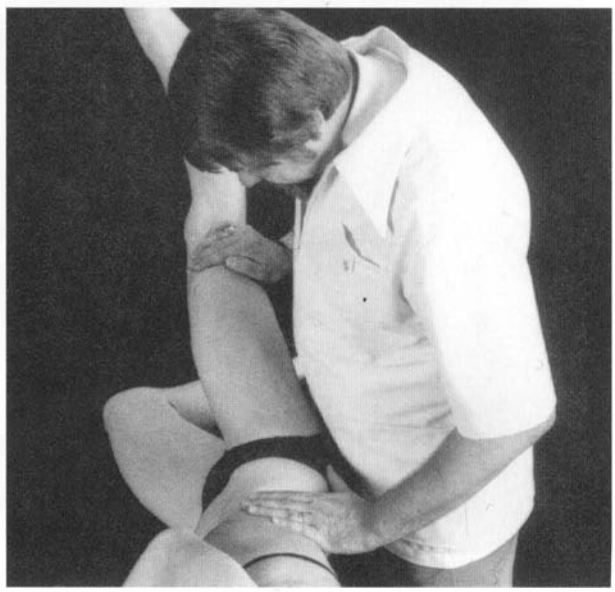
8—40.



8—41.



8—42. *Good alignment for test.*

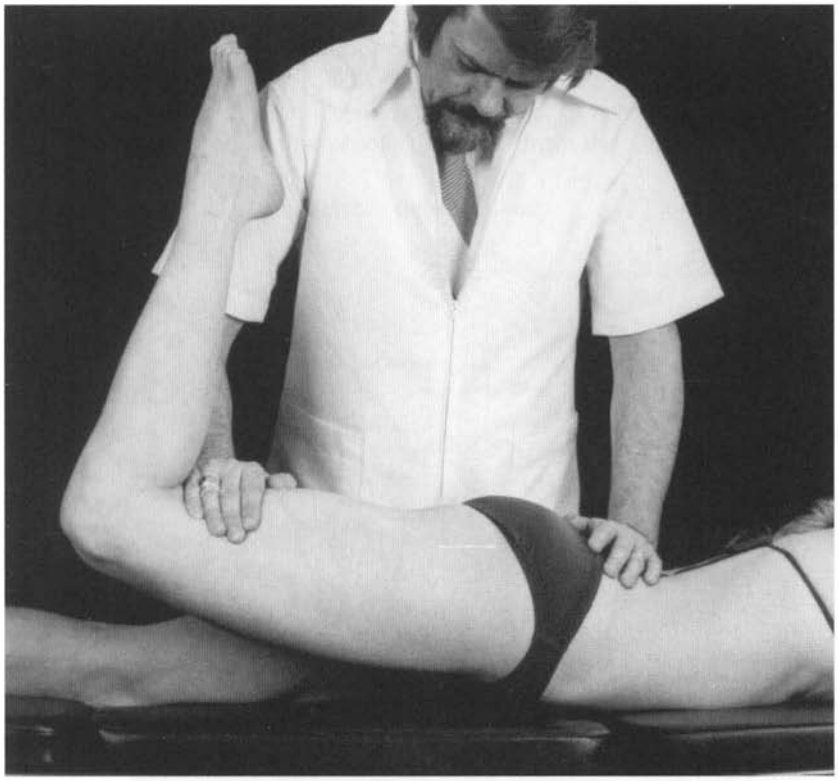


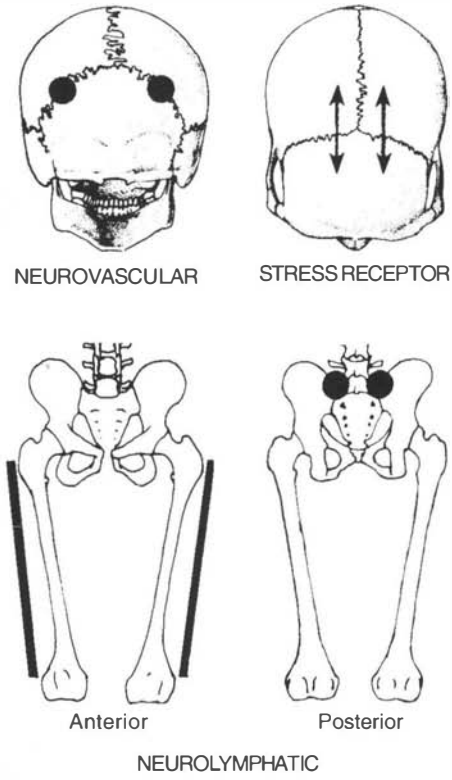
8—43. *Pelvis shifted — poor test.*

Gluteus Maximus

Origin: posterior gluteal line of ilium, tendon of sacrospinalis, dorsal surface of sacrum and coccyx, and sacrotuberus ligament.
Insertion: gluteal tuberosity of femur and iliotibial tract of fascia lata.
Action: extends hip, assists in externally rotating the thigh.
Test: The supine patient flexes the knee and extends the hip. The knee flexion is necessary to help take the hamstrings out of the test. The examiner directs pressure on the distal one-third of the femur in a direction of hip flexion. Observe for adequate pelvic fixation to the trunk by the trunk extensors and oblique abdominal musculature.

Nerve supply: inferior gluteal, L4, **5, S1, 2**.
Neurolymphatic:
Anterior: anterolateral thigh.
Posterior: between posterior superior iliac spine and L5 spinous.
Neurovascular: on the lambdoidal suture midway between lambda and asterion.
Nutrition: vitamin E, male or female endocrine concentrates or nucleoprotein extracts.
Meridian association: circulation sex.
Organ/gland association: reproductive organs or glands.





8—44.

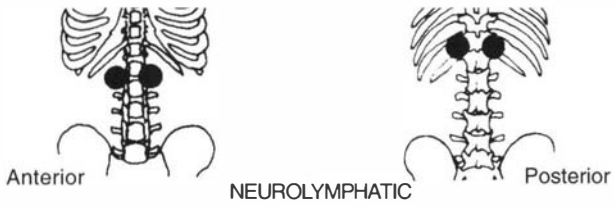
8—45.

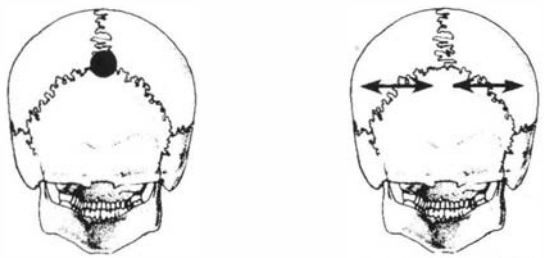
8—46.

Sartorius

Origin: anterior superior iliac spine, upper half of the iliac notch.
Insertion: upper part of medial surface of the tibia, near the anterior border.
Action: flexes knee and hip, rotates the thigh externally. When knee is flexed, rotates tibia internally. Gives medial support to the knee.
Test: The supine patient flexes the hip and knee with abduction of the hip. The examiner directs force against the anterolateral leg, just proximal to the knee, in a direction of hip extension, adduction, and medial rotation. With the other hand he grasps the posterior ankle and extends the knee.
Nerve supply: femoral, **L2, 3**.
Neurolymphatic:

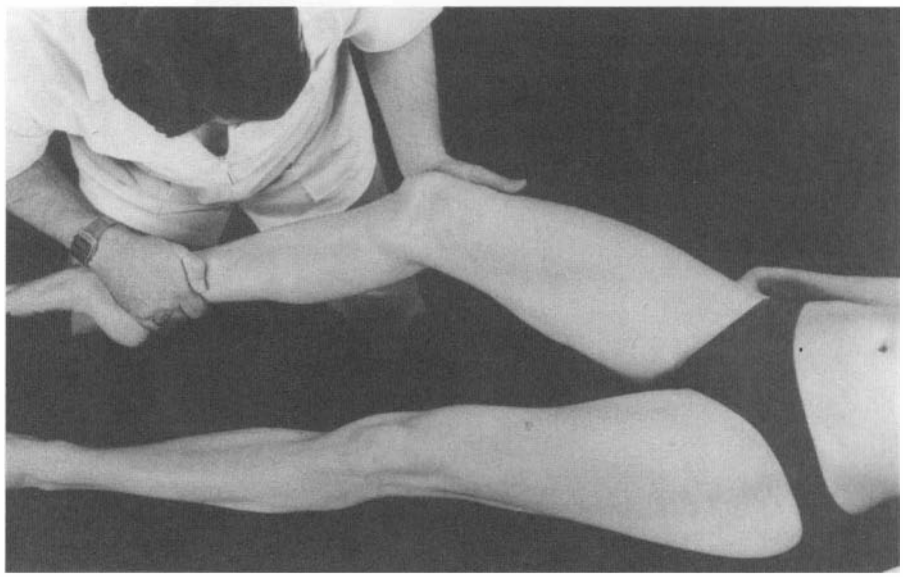
Anterior: 2" above the umbilicus and 1" from the midline.
Posterior: T11, 12 bilaterally near laminae.





Neurovascular: lambda.
Nutrition: adrenal nucleoprotein extract or concentrate, vitamin C, pantothenic acid.
Meridian association: circulation sex (occasionally triple heater).
Gland association: adrenal.

8—47. Sartorius with the femur in external rotation.

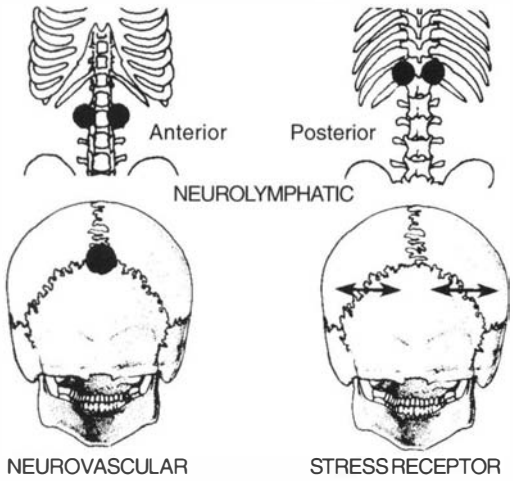


8—48. Sartorius. Extend knee and internally rotate thigh.

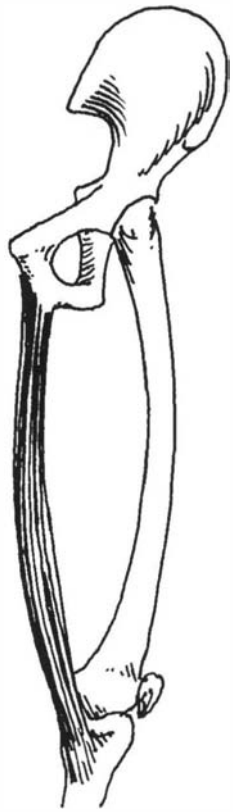
Gracilis

Origin: lower one-half of symphysis pubis and medial margin of inferior ramus of pubic arch.
Insertion: upper part of the medial surface of tibia distal to the condyle.
Action: adducts thigh, flexes knee and hip, and internally rotates the thigh and tibia.
Test: With the patient prone, the knee is flexed to approximately 45° and the thigh internally rotated. The examiner elevates the knee from the table by hip extension. The hip extension shortens the hamstrings and helps take them out of the test. Pressure is directed against the posteromedial aspect of the distal leg in a direction of knee extension and slightly lateral to effect medial thigh rotation. It is important that the examiner maintain the thigh in extension and abduction.
Nerve supply: obturator, L2, **3, 4**.
Neurolymphatic:

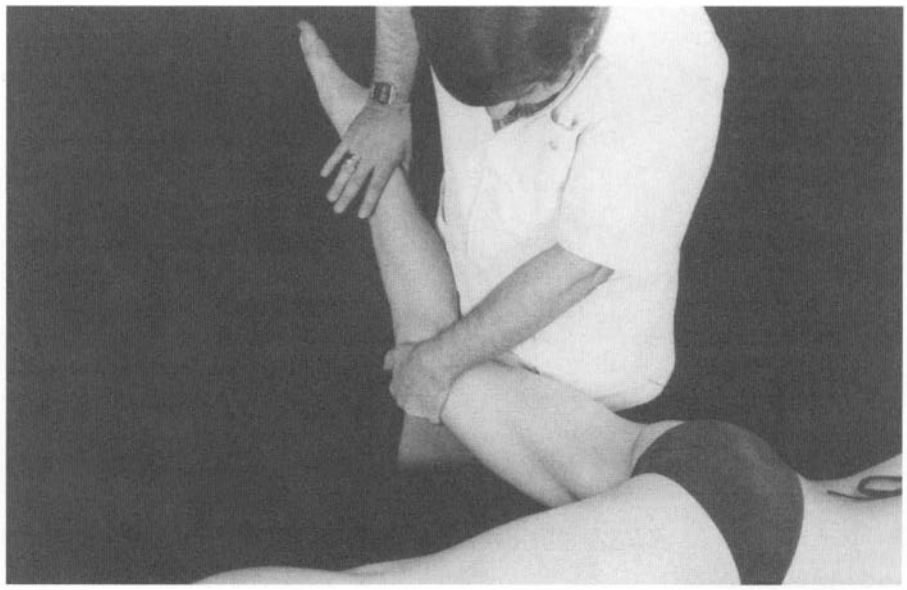
Anterior: 2" above the umbilicus and 1" from the midline.
Posterior: T11, 12 bilaterally near laminae.



Neurovascular: lambda.
Nutrition: adrenal nucleoprotein extract or concentrate.
Meridian association: circulation sex (occasionally triple heater).
Gland association: adrenal.



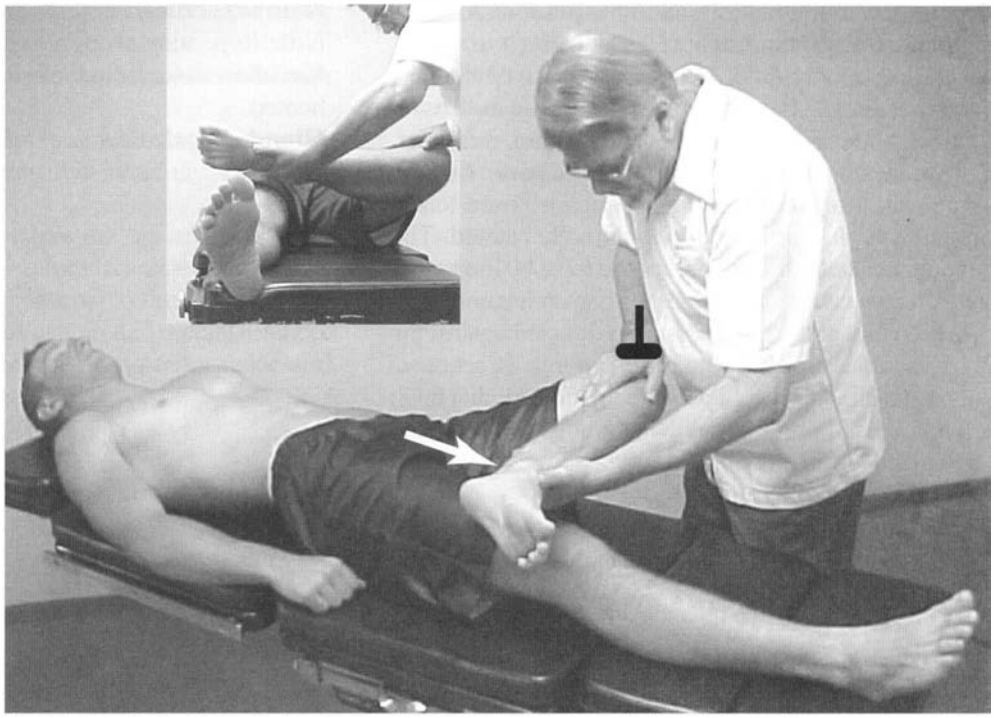
8—49. Gracilis with femur in external rotation.



8—50. Gracilis — starting position: hip extension, abduction, and internal rotation with knee flexion.

Sartorius Alternate Test[4]

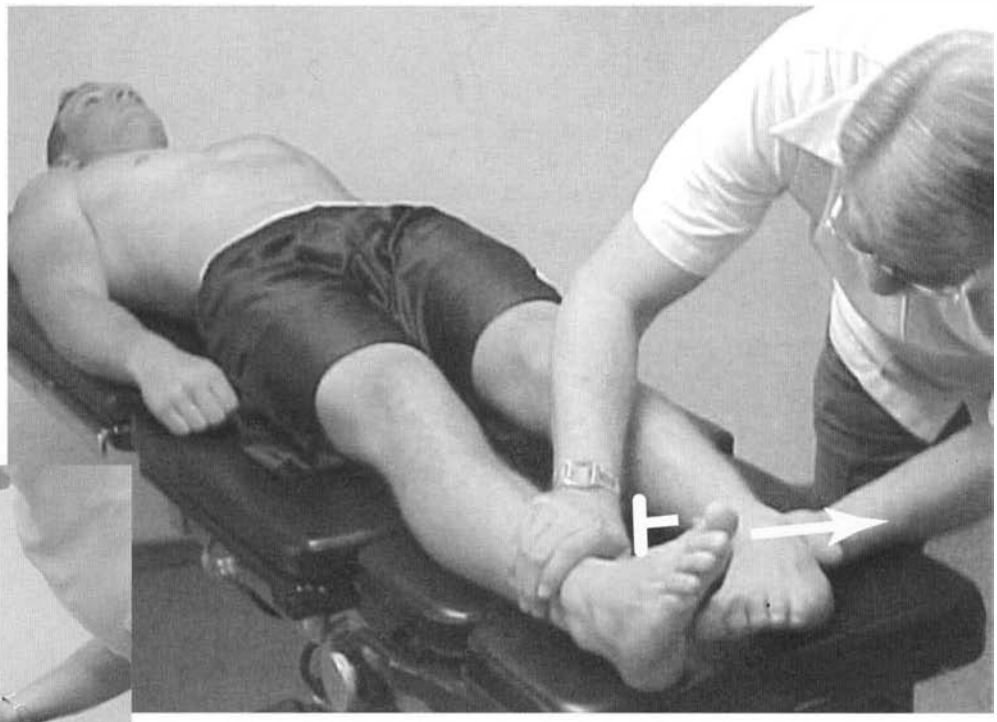
Test: Starting position is with the supine patient's knee flexed to 90° and the hip in flexion, abduction, and externallly rotated to put the foot anterior to the contralateral leg in a figure-4-configuration. The examiner applies stabilization to the knee while holding the patient's ankle a few inches above the contralateral leg. The test pressure is to extend the patient's knee by pulling the ankle through the coronal plane.

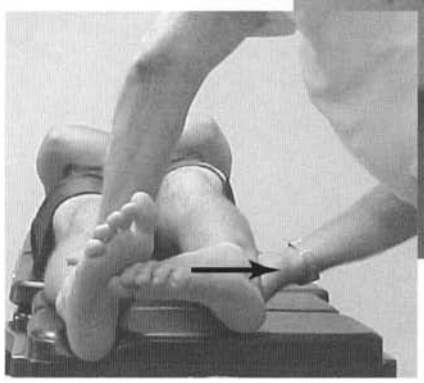


8—51a & b. Sartorius starting position with left thigh in external rotation, abduction, and flexion, and leg in figure-4 position.

Gracilis Alternate Test[4]

Test: Starting position is with the patient's leg straight and the hip in full medial rotation, placing his toes and metatarsal arch inferior to the contralateral arch. The examiner stabilizes the contralateral ankle; test pressure is applied to the ankle to abduct the thigh.

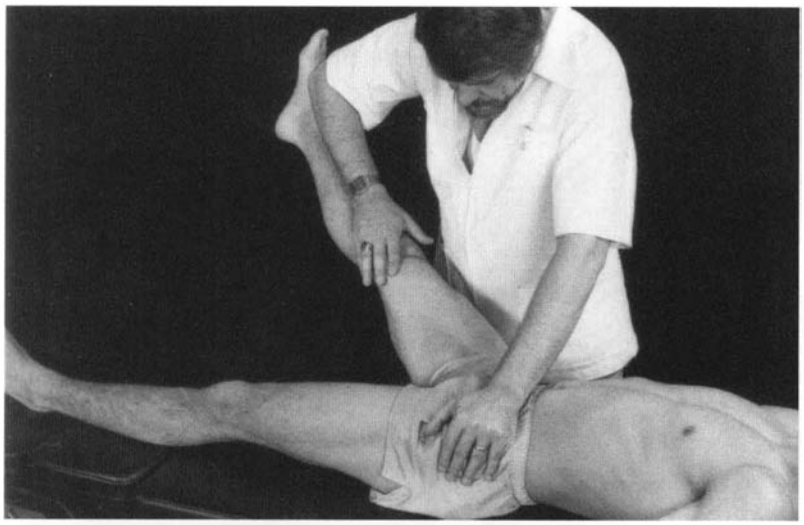




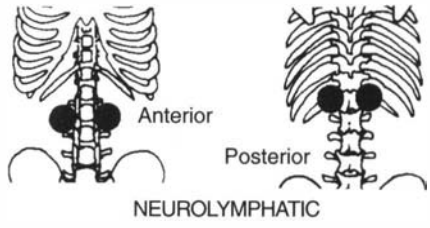
8—52a & b. Gracilis starting position with left leg in internal thigh rotation.

Psoas

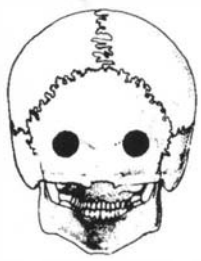
Origin: anterior surface of transverse processes, lateral border of vertebral bodies and corresponding intervertebral discs T12 through L5.
Insertion: lesser trochanter of the femur with the iliacus.
Action: flexes and gives minimal action in external rotation and abduction of the thigh.
Test: The supine patient flexes and abducts the hip with external thigh rotation. Force is directed against the anteromedial aspect of the leg in a direction of extension and slight abduction. The point of the examiner's contact on the leg depends on the amount of leverage required for the test. On most individuals, adequate leverage is achieved by contacting slightly proximal to the knee. On very strong individuals use a longer leverage, contacting at the ankle. The direction of pressure should be vectored between the activity of the rectus femoris and the adductors.
Nerve supply: lumbar plexus, L1, **2, 3**, 4.
Neurolymphatic:
Anterior: 1" above umbilicus and 1" from midline.
Posterior: T12-L1 between spinous and transverse processes.
Neurovascular: l-1/2" lateral to external occipital protuberance.
Nutrition: vitamins A and E, kidney concentrate or nucleoprotein extract.
Meridian association: kidney.
Organ association: kidney.



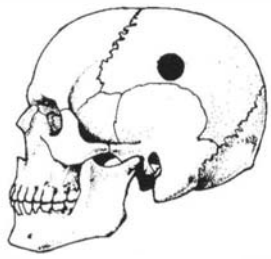
8—53. Testing pressure applied over medial condyle of the tibia. This is adequate for most patients, and helps eliminate possibility of accidentally challenging a knee subluxation.



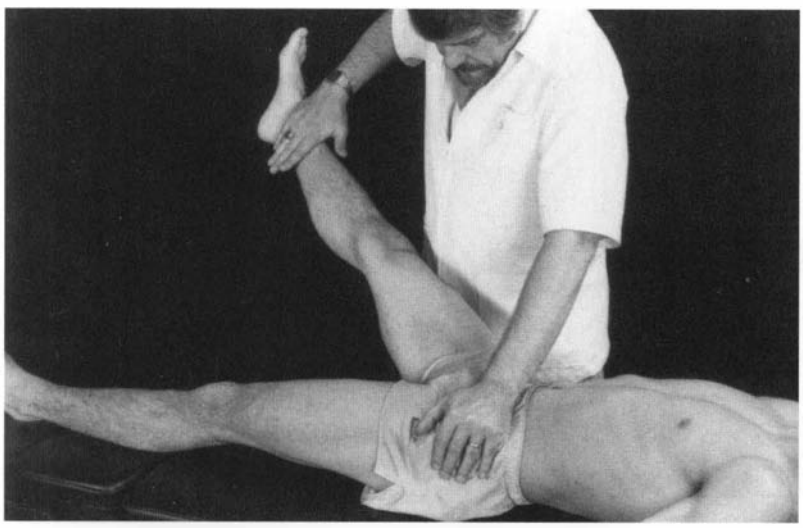
NEUROLYMPHATIC



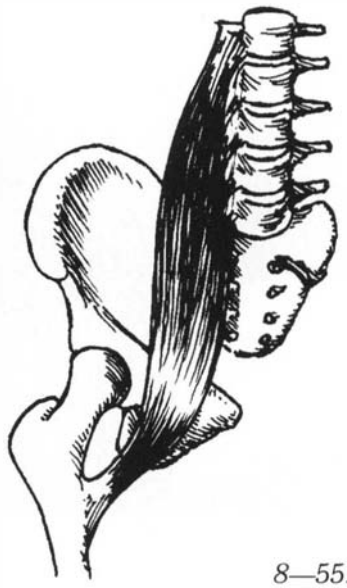
NEUROVASCULAR



STRESS RECEPTOR



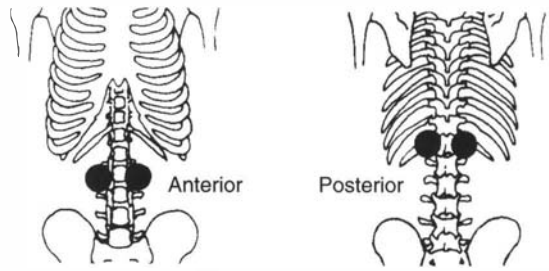
8—54. Greater leverage may be needed on very strong patients.



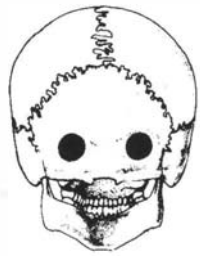
8—55.

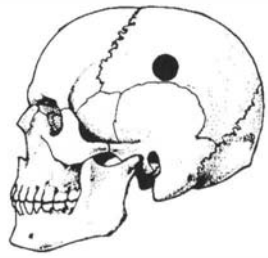
Iliacus

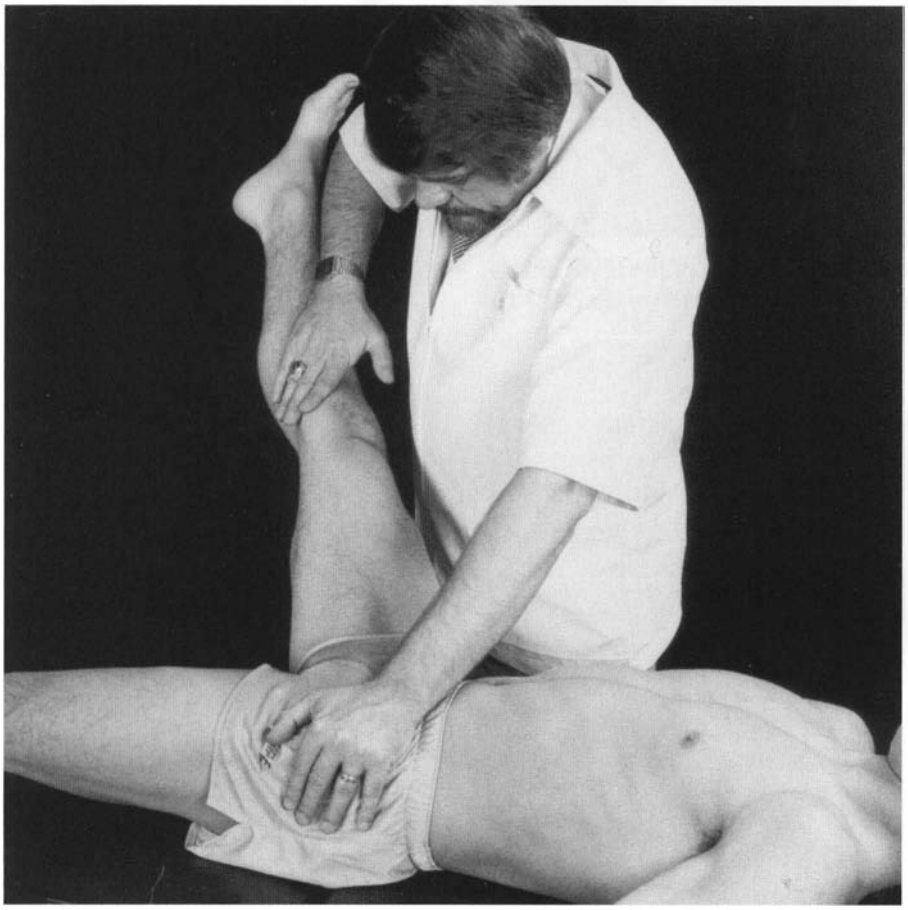
Origin: upper two-thirds of the iliac fossa; internal border iliac crest; anterior sacroiliac, lumbosacral and iliolumbar ligaments; ala of sacrum.
Insertion: lesser trochanter of the femur with psoas major.
Action: with the psoas, flexes thigh; minimal activity on rotation of thigh.
Test: The supine patient places the leg in a position similar to that for the psoas test, only with greater hip flexion and abduction. The examiner makes contact at the anteromedial distal femur or at the ankle, depending on the amount of leverage required. The force is directed toward hip abduction and extension.
Nerve supply: femoral nerve, **L1, 2, 3**.
Neurolymphatic:
Anterior: 1" above umbilicus and 1" from midline.
Posterior: T12, L1 between spinous and transverse processes.
Neurovascular: 1-1/2" lateral to external occipital protuberance.
Nutrition: vitamins A and E, kidney concentrate or nucleoprotein extract.
Meridian association: kidney.
Organ association: kidney.



NEUROLYMPHATIC


NEUROVASCULAR

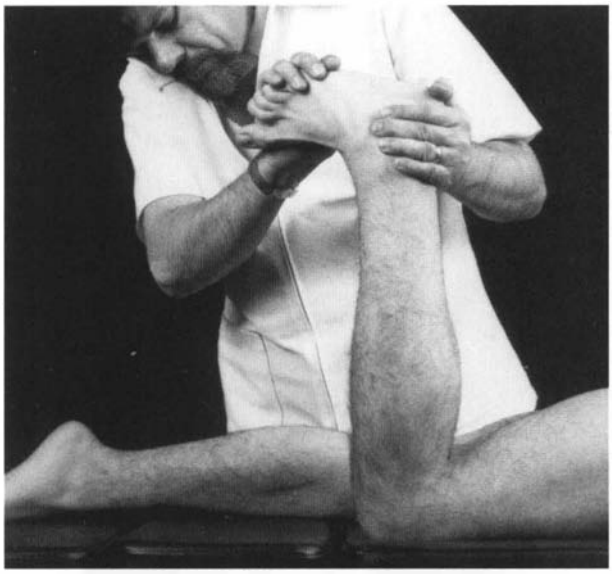

STRESS RECEPTOR


8—56. The iliacus test is the same as the psoas except the starting position is with more hip flexion and abduction.

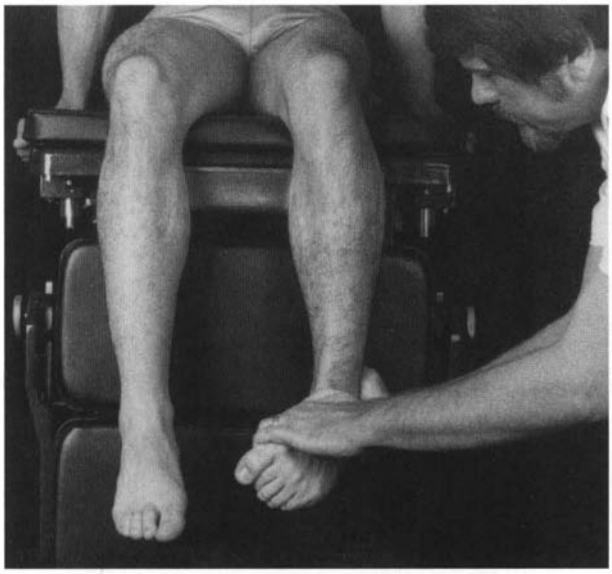

8—57.

Popliteus

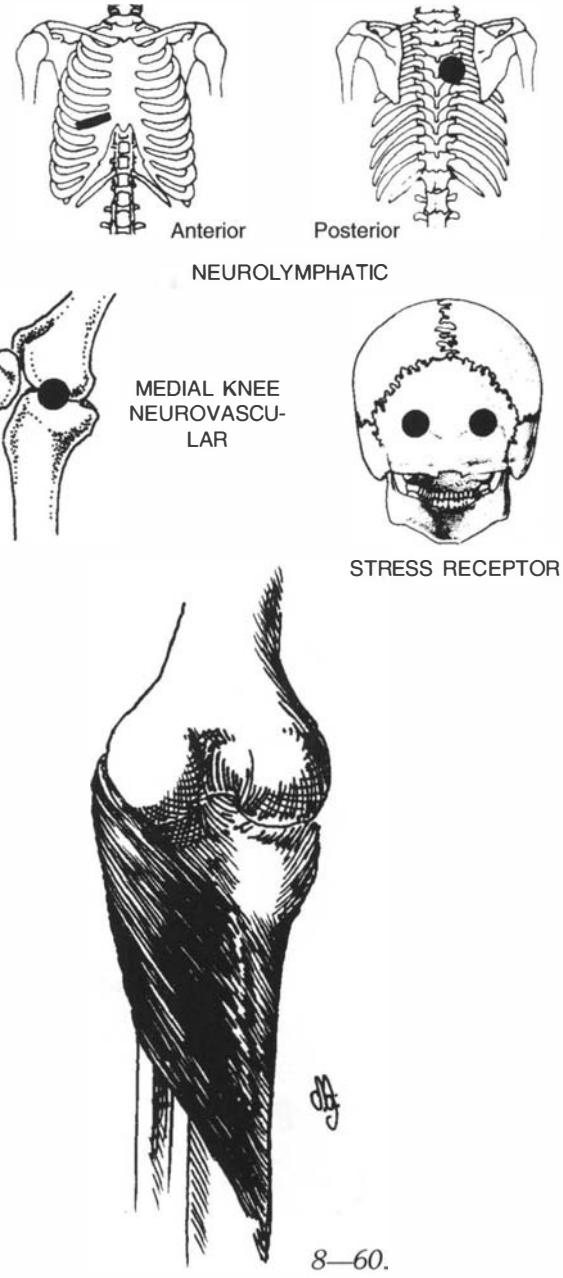
Origin: lateral condyle of femur, posterior horn of lateral meniscus, fibular head.
Insertion: triangular area on posterior surface of tibia above soleal line.
Action: rotates the tibia internally on the femur or the femur externally on the tibia, depending upon the one fixed; withdraws the meniscus during flexion, and provides rotatory stability to the femur on the tibia[2]; brings the knee out of the "screw-home" position of full extension; helps with posterior stability of the knee.
Test: With the patient's knee flexed to 90°, pressure is directed on the distal medial foot, with counter-pressure on the calcaneus to impart lateral rotation of the tibia on the femur. The actual testing motion is slight and can be evaluated only by observing the tibia rotating on the femur and watching for motion of the tibial tubercle. It is quite possible for the examiner to obtain foot rotation, appearing to be a weak popliteus; in fact, it may be a twisting of the tibia and fibula.
Nerve supply: tibial, **L4, 5, S1**.
Neurolymphatic:
Anterior: 5th intercostal space from mid-mamillary line to sternum on the right.
Posterior: between T5-6 laminae on right.
Neurovascular: medial aspect of knee at meniscus.
Nutrition: vitamin A.
Meridian association: gallbladder.
Organ association: gallbladder.



8—58. Examiner observes for tibial tubercle rotation.



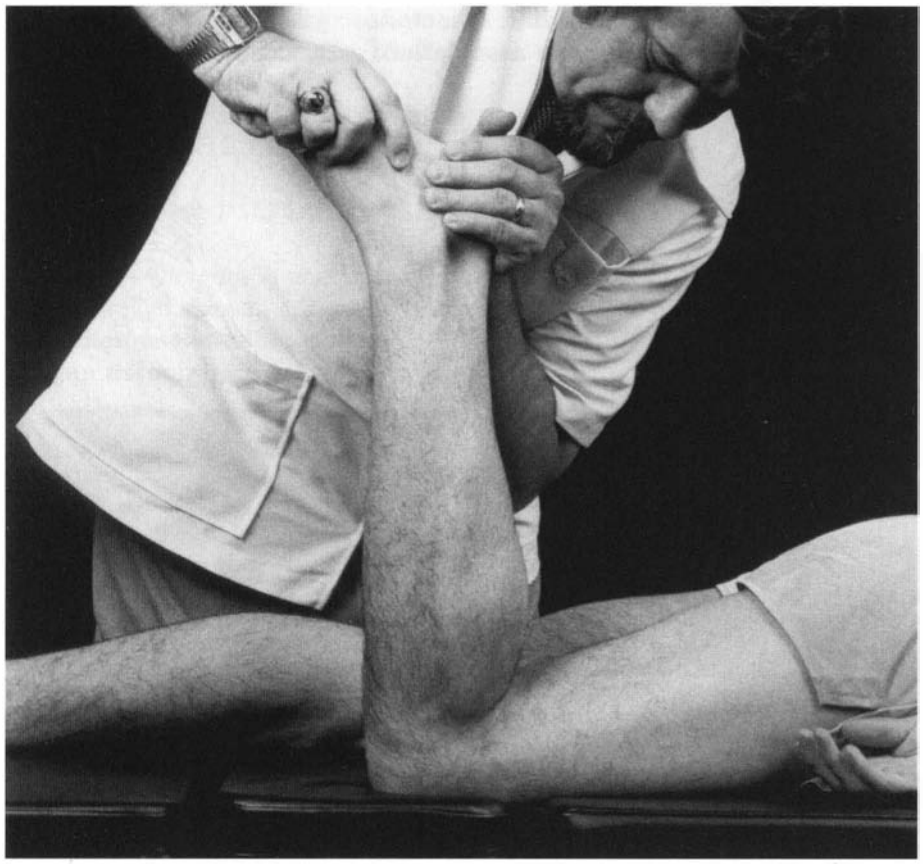
8—59. Seated test with observation for tibial rotation.



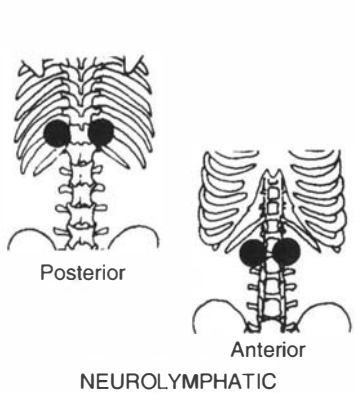
8—60.

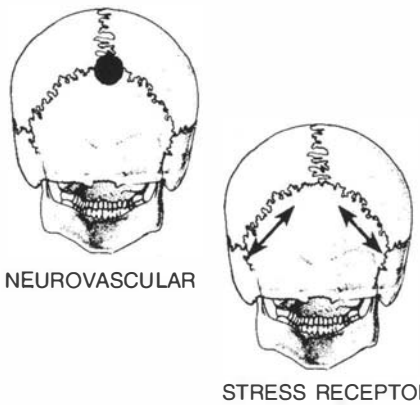
Soleus

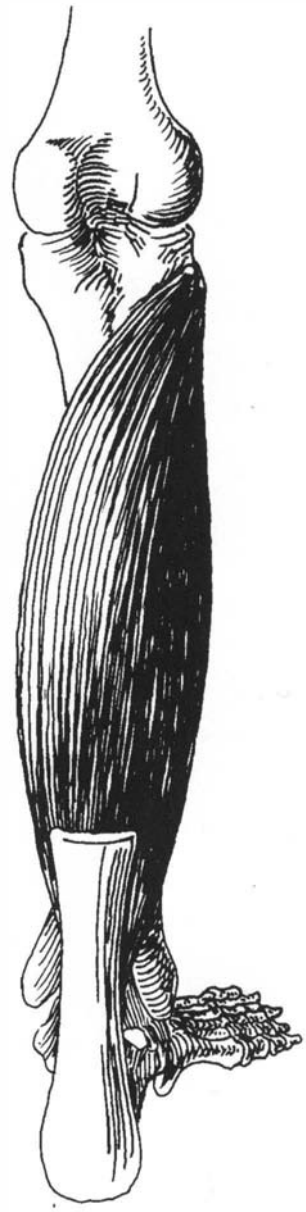
Origin: posterior surface of the head and upper one-third of the shaft of the fibula; middle one-third of the medial border of the tibia; tendinous arch between tibia and fibula.
Insertion: into calcaneus with gastrocnemius by way of the Achilles tendon.
Action: plantar flexes foot.
Test: The prone patient flexes the knee to 90° and plantar flexes the foot. The examiner directs traction on the calcaneus and pressure on the forefoot in a direction of dorsiflexion. The knee flexion helps take the gastrocnemius out of the test. Because of the great strength of the soleus and its limited leverage, this muscle is difficult to evaluate.
Nerve supply: tibial, L4, 5, **S1, 2**.
Neurolymphatic:
Anterior: 2" above umbilicus and 1" from midline.
Posterior: between T11, 12 bilaterally near laminae.
Neurovascular: lambda.
Nutrition: adrenal concentrate or nucleoprotein extract.
Meridian association: circulation sex.
Gland association: adrenal.



8—61. Flexing the knee to 90° helps take the gastrocnemius out of the test.







8—62.

Gastrocnemius

Origin:

Medial head: medial condyle and adjacent part of femur; capsule of knee joint.

Lateral head: lateral condyle and posterior surface of knee joint.

Insertion: into calcaneus by Achilles tendon.

Action: plantar flexes foot.

Test: The medial and lateral heads of the gastrocnemius can be tested as described by Beardall.[5] The test must be correlated with hamstring strength because they are significantly synergistic in the test. For both medial and lateral heads of the gastrocnemius, the supine patient flexes the knee to approximately 110° and maximally plantar flexes the foot. For the medial head, the leg is internally rotated; for the lateral test, it is externally rotated. The examiner stabilizes the knee while extending it by pulling on the calcaneus contact.

Nerve supply: tibial, L4, 5, **S1, 2**.

Neurolymphatic:

Anterior: 2" above umbilicus and 1" from midline.

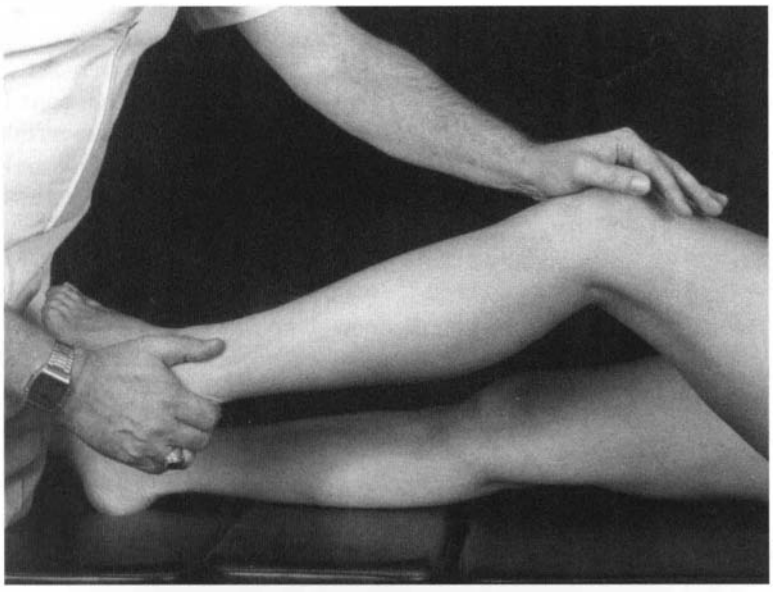
Posterior: between T11, 12 bilaterally near laminae.

Neurovascular: lambda.

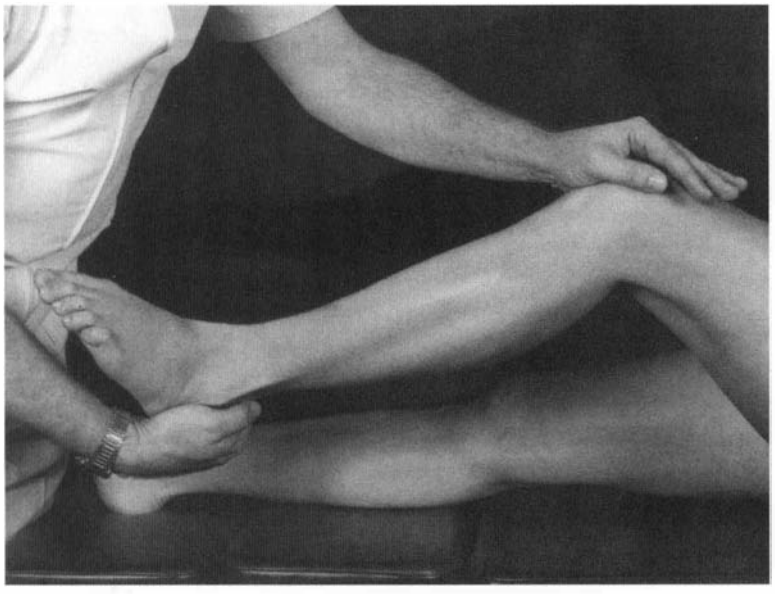
Nutrition: adrenal concentrate or nucleoprotein extract.

Meridian association: circulation sex.

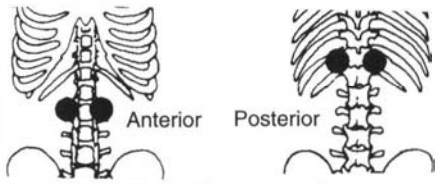
Organ association: adrenal.



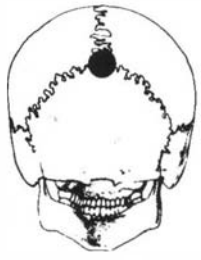
8—63. Internal leg rotation for medial gastrocnemius test.



8—64. External leg rotation for lateral gastrocnemius test.



NEUROLYMPHATIC



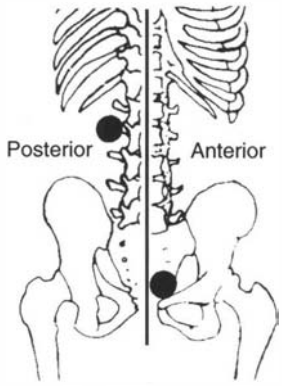
NEUROVASCULAR

STRESS RECEPTOR

8—65.

Tibialis Anterior

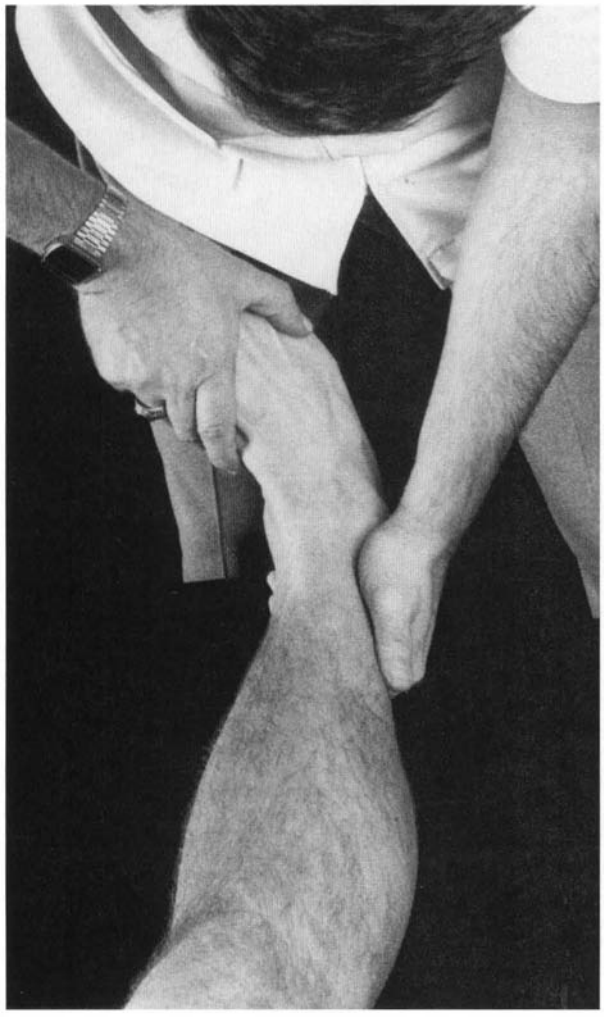
Origin: lateral condyle of tibia, proximal two-thirds of lateral surface of tibia, interosseous membrane, deep fascia and lateral intermuscular septum.
Insertion: medial and plantar surface of medial cuneiform, and base of 1st metatarsal.
Action: dorsiflexes foot and inverts it.
Test: The supine patient inverts and dorsiflexes the foot, with the toes kept in flexion. The examiner applies pressure against the medial dorsal surface of the foot in the direction of plantar flexion and eversion. The examiner should see effective contraction of tibialis anterior as indicated by the tendon elevation during the test.
Nerve supply: peroneal, **L4, 5**, S1.
Neurolymphatic:
Anterior: 3/4" above symphysis pubis bilaterally.
Posterior: L2 transverse process.
Neurovascular: bilateral frontal bone eminences.
Nutrition: vitamin A.
Meridian association: bladder.
Organ association: urinary bladder.



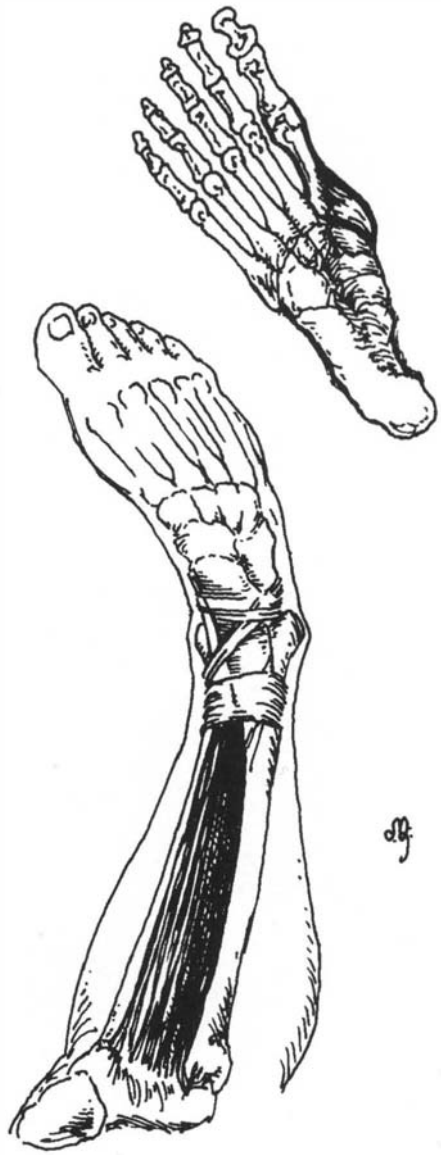
NEUROLYMPHATIC
BILATERAL

NEUROVASCULAR

STRESS RECEPTOR



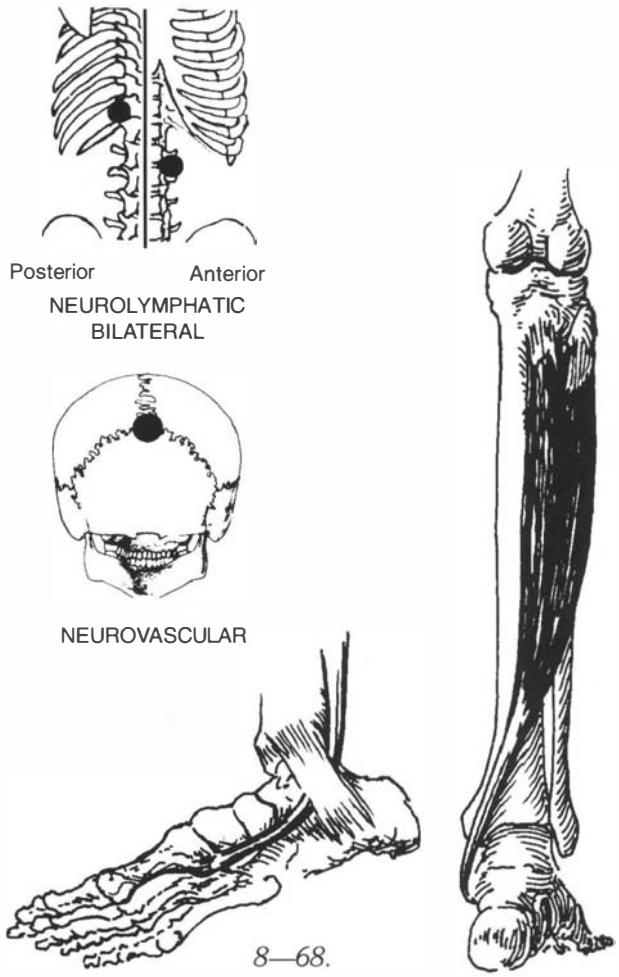
8—66. Observe for the tendon raising during the test.



8—67.

Tibialis Posterior

Origin: lateral part of posterior surface of tibia, medial two-thirds of fibula, interosseous membrane, intermuscular septa, and deep fascia.
Insertion: tuberosity of navicular bone, plantar surface of all cuneiforms, plantar surface of base of 2nd, 3rd, and 4th metatarsal bones, cuboid bone, and sustentaculum tali.
Action: inverts and plantar flexes foot; medial ankle stabilizer.
Test: The supine patient maximally plantar flexes the foot and then inverts it, keeping the toes in a flexed position. The examiner places his hand on the medial side and over the foot. Pressure is directed against the medial side of the foot in the direction of eversion. The examiner should observe for the rising tendon of the tibialis posterior when the muscle contracts. Care should be taken that the patient does not dorsiflex the foot to change the parameters of the test.
Nerve supply: tibial, **L5, S1**.
Neurolymphatic:
Anterior: 2" above the umbilicus and 1" from the midline bilaterally.
Posterior: between T11, 12 bilaterally by laminae.
Neurovascular: lambda.
Nutrition: adrenal concentrate or nucleoprotein extract.
Meridian association: circulation sex.
Organ/gland association: adrenal; possibly urinary bladder.

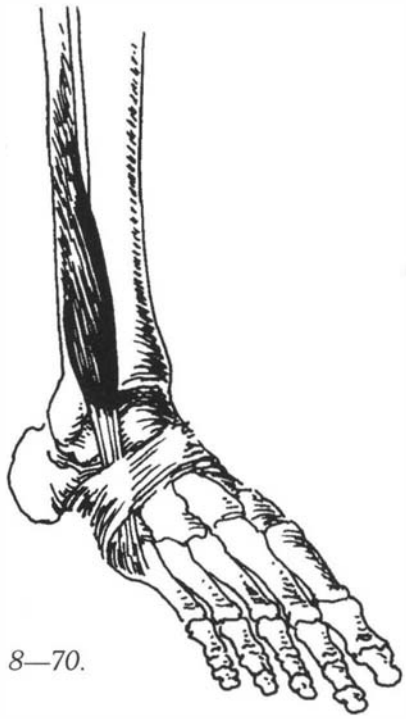


8—68.

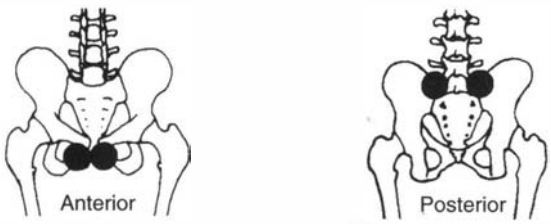
8—69. If patient dorsiflexes the foot, start the test over.

Peroneus Tertius

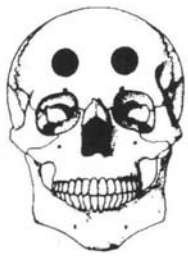
Origin: lower one-third of the anterior surface of the fibula and adjacent intermuscular septum.
Insertion: dorsal surface of the base of the 5th metatarsal.
Action: dorsiflexes and everts the foot.
Test: The supine patient dorsiflexes and everts the foot with the toes kept in neutral position, or toward flexion. Examining pressure is directed against the dorsal lateral surface of the 5th metatarsal in the direction of plantar flexion and inversion. The examiner should evaluate the tendon of the peroneus tertius and the tendons of the extensor digitorum longus for best direction to maximize the effect of the peroneus tertius and minimize that of the toe extensors.
Nerve supply: peroneal, **L4, 5, S1**.
Neurolymphatic:
Anterior: inferior ramus of pubic bones.
Posterior: between L5 transverse and sacrum.
Neurovascular: bilateral frontal bone eminences.
Nutrition: calcium, B complex. Avoid oxalic acid foods, e.g., caffeine, cranberries, plums, and others.
Meridian association: bladder.
Organ association: urinary bladder.



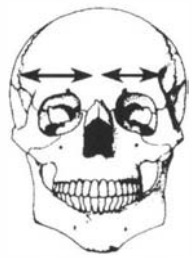
8—70.



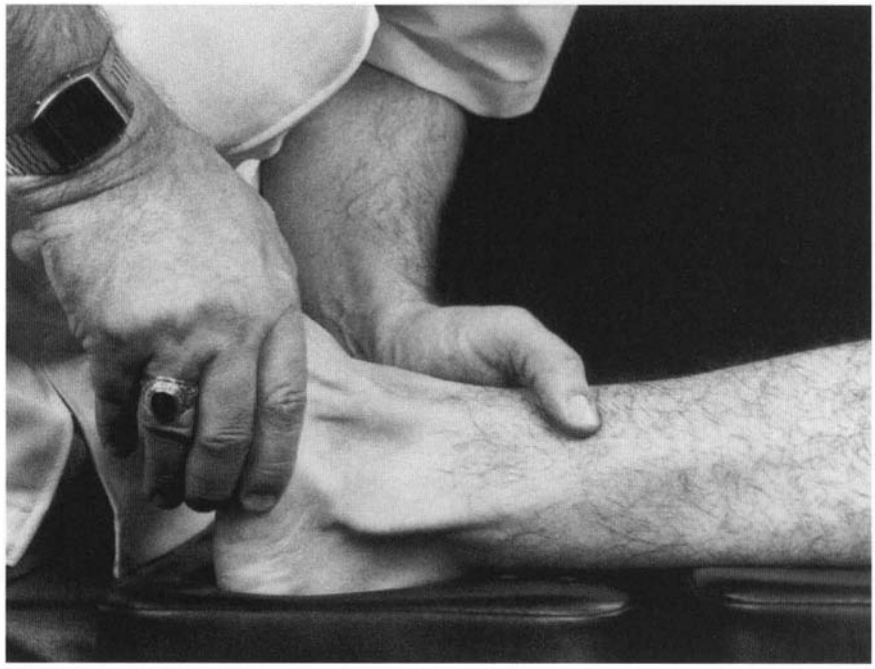
NEUROLYMPHATIC



NEUROVASCULAR



STRESS RECEPTOR



8—71. The patient should not be allowed to extend the toes.

Peroneus Longus and Brevis

Peroneus Brevis

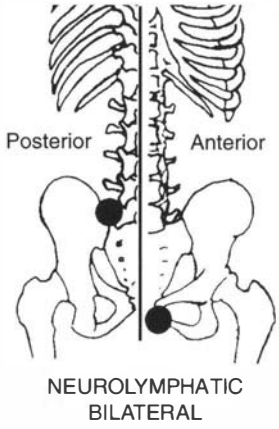
Origin: lower two-thirds of fibula on lateral side and adjacent intermuscular septa.
Insertion: lateral side of proximal end of 5th metatarsal.
Action: plantar flexes foot and everts it; gives lateral stability to the ankle.

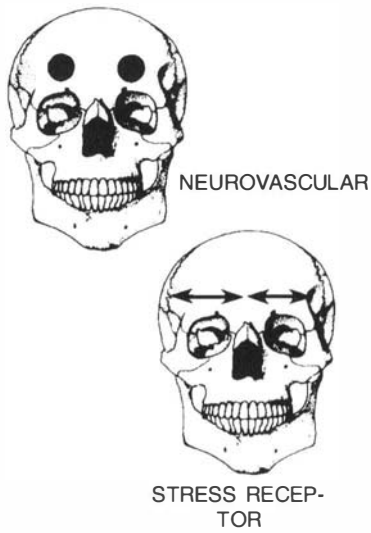
Peroneus Longus

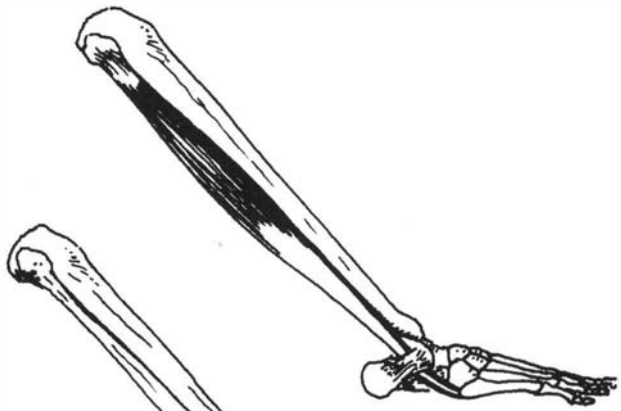
Origin: lateral condyle of tibia, head and upper two-thirds of lateral surface of fibula, intermuscular septa and adjacent fascia.
Insertion: proximal end of the 1st metatarsal and medial cuneiform on their lateral portions.
Action: plantar flexes foot and everts it; gives lateral stability to the ankle.

Peroneus Longus and Brevis

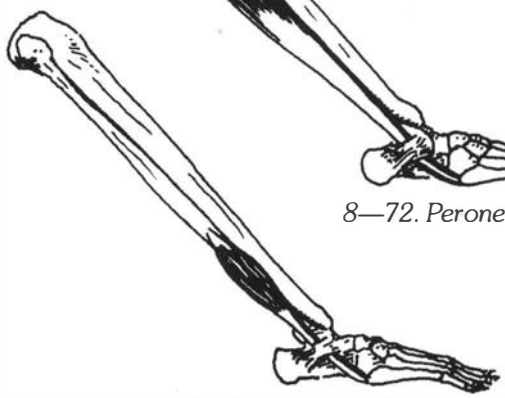
Test: The supine patient maximally plantar flexes the foot and everts it, with the toes kept in flexion or neutral position. Testing pressure is directed to the side of the foot in the direction of inversion. The test must start from the maximum eversion allowed when the foot is in complete plantar flexion. The range of motion in this test is limited. No dorsiflexion of the foot should be allowed, nor should there be any extension of the toes. The examiner should observe the tendon that courses behind the external malleolus as evidence of maximum isolation of the muscles.
Nerve supply: peroneal, L4, **5, S1**.
Neurolymphatic:
Anterior: inferior symphysis pubis, bilaterally.
Posterior: bilaterally between posterior superior iliac spine and L5 spinous process.
Neurovascular: bilateral frontal bone eminences.
Nutrition: calcium, vitamin B complex. Avoid oxalic acid foods.
Meridian association: bladder.
Organ association: urinary bladder.







8—72. Peroneus longus.



8—73. Peroneus brevis.

8—74. Do not allow foot dorsiflexion during the test.

8—75. Insertion of the peroneus longus tendon.

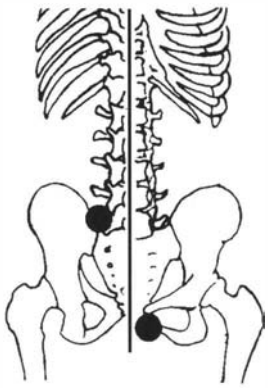
Flexor Hallucis Longus

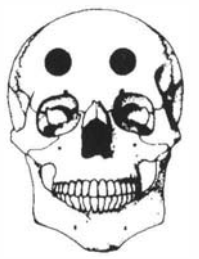
Origin: lower two-thirds of posterior fibula, interosseous membrane and adjacent intermuscular septa and fascia.
Insertion: plantar surface of distal phalanx of great toe.
Action: flexes great toe; continued action aids in plantar flexing the foot; helps give medial ankle stabilization.
Testing position and stabilization: With the patient supine, the examiner stabilizes the metatarsophalangeal articulation in slight extension and holds the foot halfway between dorsal and plantar flexion. The patient flexes the distal phalanx of the great toe. From this testing position of flexion between the proximal and distal phalanx, the examiner directs pressure against the distal phalanx of the great toe in the direction of extension.
Nerve supply: tibial, **L5, S1, 2**.
Neurolymphatic:

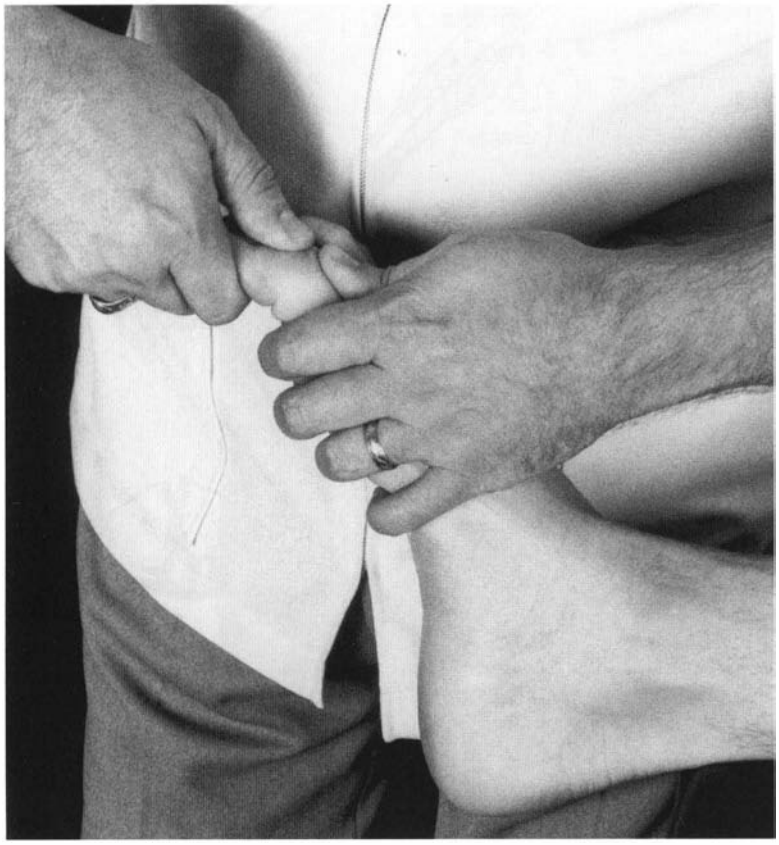
Anterior: inferior to the symphysis pubis at the height of the obturator bilaterally (same as peroneus longus and brevis).

Posterior: bilaterally between PSIS and L5 spinous.

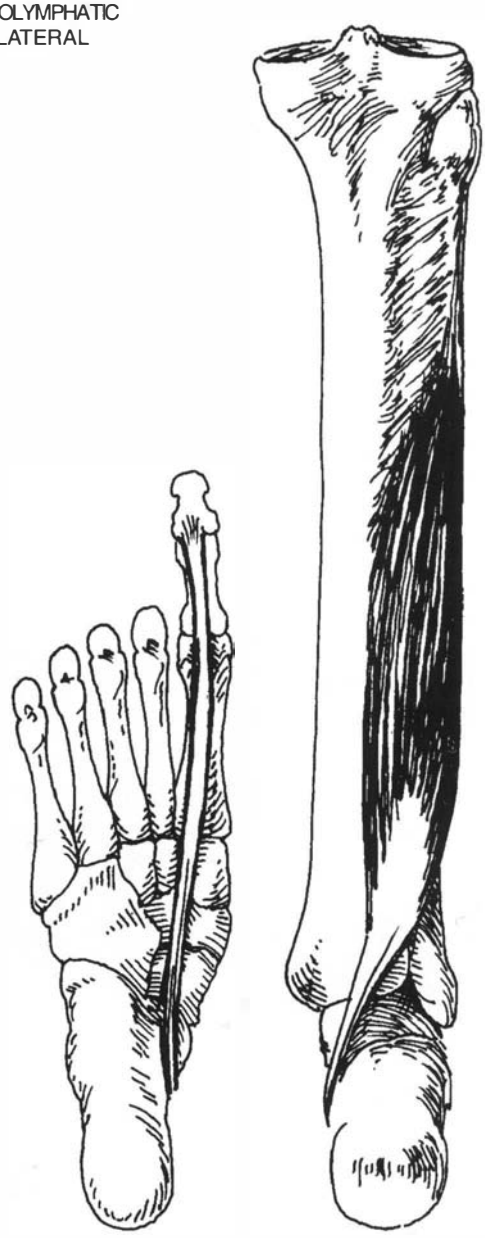
Neurovascular: bilateral frontal bone eminences.
Nutrition: raw bone concentrate correlating with tarsal tunnel syndrome or other subluxations of the foot.
Meridian association: circulation sex.







8—76. Stabilize the proximal phalanx while applying testing pressure to the distal one.



8—77. Insertion of flexor hallucis longus tendon.

8—78.

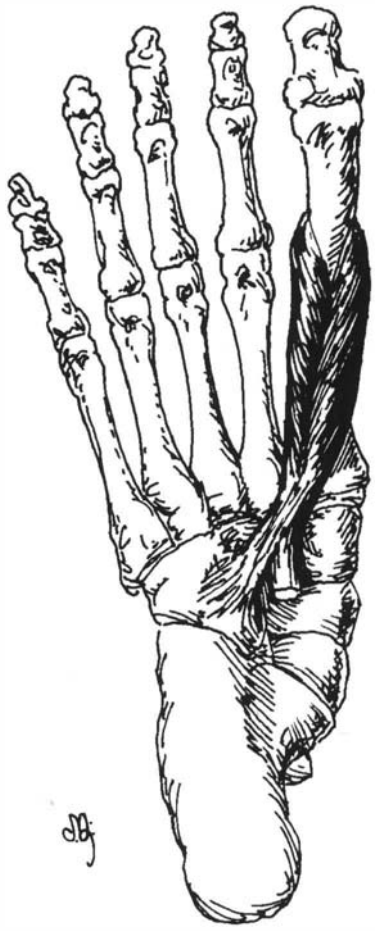
Flexor Hallucis Brevis

Origin: medial portion of the plantar surface of the cuboid bone, adjacent portion of the lateral cuneiform bone, and from prolongation of the tendon of the tibialis posterior.
Insertion: medial and lateral sides of proximal phalanx of the great toe.
Action: flexes metatarsophalangeal articulation of great toe.
Test: The examiner stabilizes the interphalangeal articulation of the great toe and places the metatarsophalangeal articulation in flexion for the starting test position. While maintaining hyperextension of the interphalangeal articulation, the examiner directs pressure against the plantar surface of the proximal phalanx toward extension.
Nerve supply: tibial, L4, **5, S1**, 2.
Neurolymphatic:

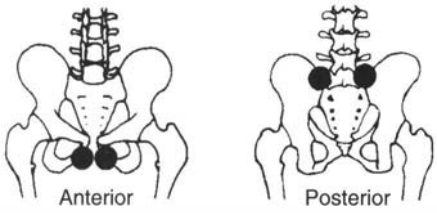
Anterior: inferior to the symphysis pubis at the height of the obturator (same as peroneus longus and brevis).

Posterior: between PSIS and L5 spinous.

Neurovascular: bilateral frontal bone eminences.
Nutrition: raw bone concentrate correlating to tarsal tunnel syndrome or other subluxations of the foot.
Meridian association: circulation sex.

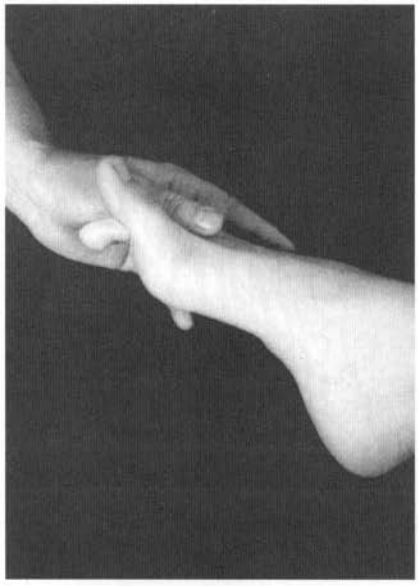


8—79.

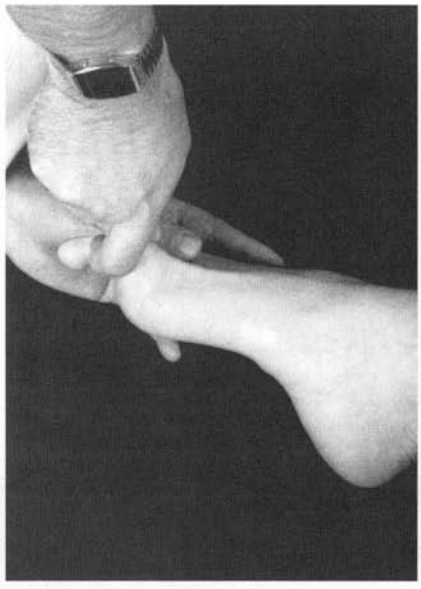


NEUROLYMPHATIC

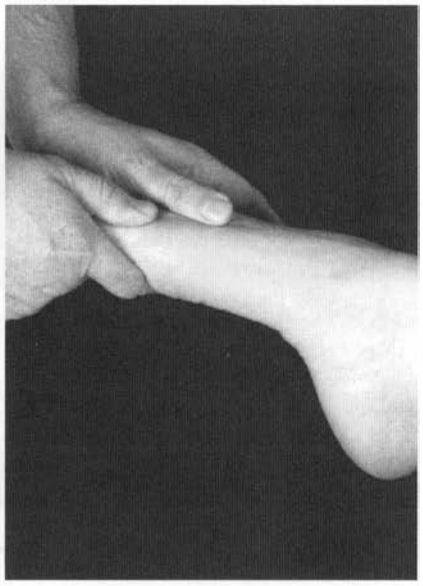
NEUROVASCULAR



8—80. First step — flex 2nd-5th toes.



8—81. Second step — extend great toe and hold extension of interphalangeal articulation.



8—82. Third step — starting position is with flexion at the metatarsophalangeal articulation.

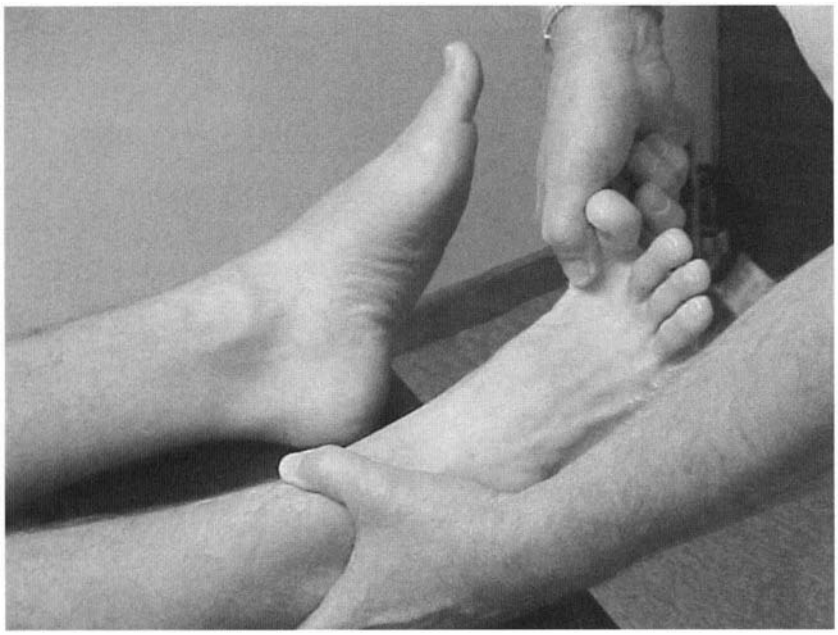
Extensor Hallucis Longus and Brevis

Extensor Hallucis Longus

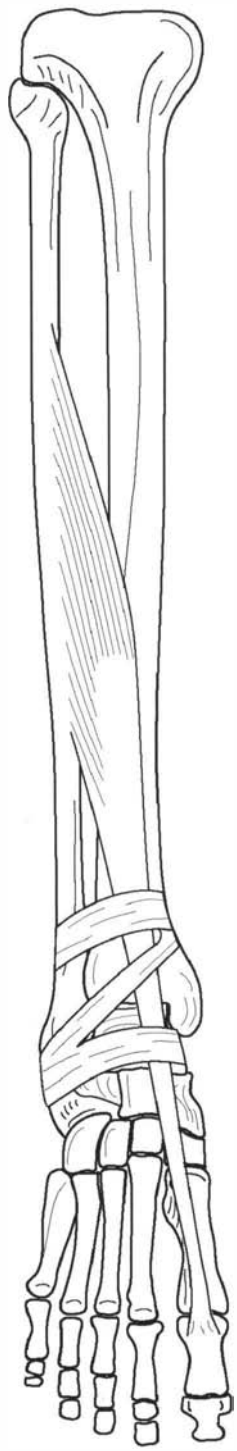
Origin: middle half of anterior tibial surface and adjacent interosseous membrane.
Insertion: base of distal phalanx of great toe.
Action: extends the great toe; continued action assists in dorsiflexion and inversion of the foot and ankle.
Nerve supply: deep peroneal, **L4, 5, S1.**

Extensor Hallucis Brevis

The extensor hallucis brevis is the medial slip of the extensor digitorum brevis (see 8—85).
Origin: anterior portion of the superolateral surface of the calcaneus, lateral talocalcaneal ligament, cruciate crural ligament.
Insertion: dorsal surface of the great toe proximal phalanx base.
Action: extends great toe proximal phalanx.
Nerve supply: deep peroneal, L4, **5, S1.**
Test for longus and brevis: With the patient sitting or standing, his great toe is placed in extension and the examiner stabilizes the foot and grasps the great toe to flex it while the patient resists.



8—83.



8—84. Extensor digitorum longus.

Extensor Digitorum Longus and Brevis

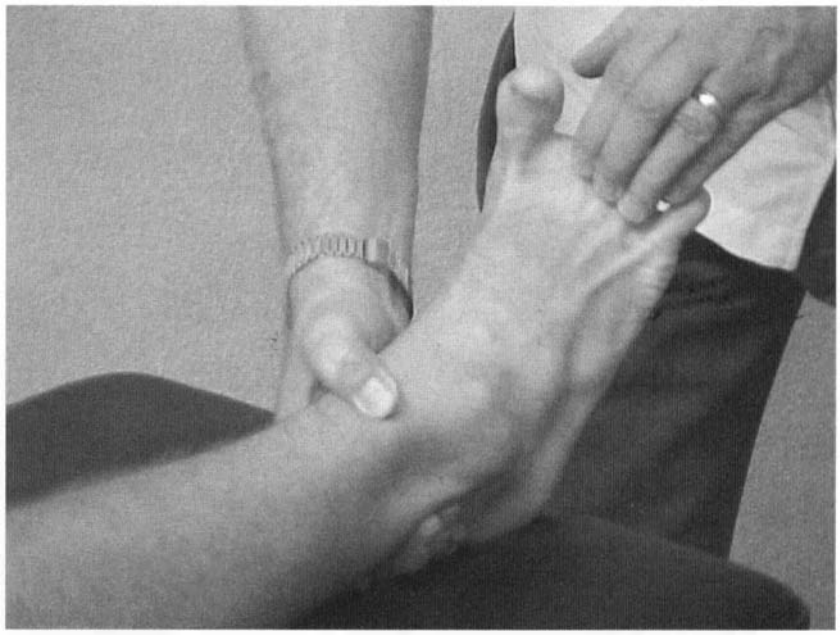
8—85. Extensor digitorum longus and brevis. Extensor hallucis brevis.

Extensor Digitorum Longus

Origin: lateral tibial condyle, proximal three-fourths of anterior fibula body, interosseous membrane, deep fascia, intermuscular septa.
Insertion: lateral four toes, middle and distal phalanges dorsal surface.
Action: extends toes and with continued action assists in foot and ankle dorsiflexion and eversion.
Nerve supply: peroneal, **L4, 5, S1.**

Extensor Digitorum Brevis

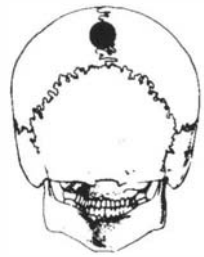
Origin: anterior portion of the superolateral surface of the calcaneus, lateral talocalcaneal ligament, cruciate crural ligament.
Insertion: first tendon to the dorsal surface of the great toe proximal phalanx base. This muscular slip and tendon are often called the extensor hallucis brevis. The other three tendons insert to the lateral sides of the extensor digitorum longus tendons.
Action: extends phalanges of the medial four toes.
Nerve supply: deep peroneal, L4, **5, S1.**
Test of longus and brevis: The examiner stabilizes the foot in slight plantar flexion, and the patient's toes are extended. The test force is to bring the toes into flexion. Usually the middle three toes are tested; the hallux is tested separately.

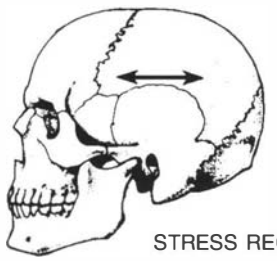


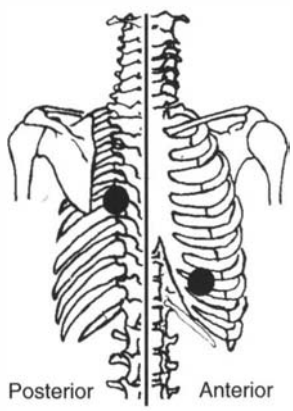
8—86.

Trapezius — Middle Division

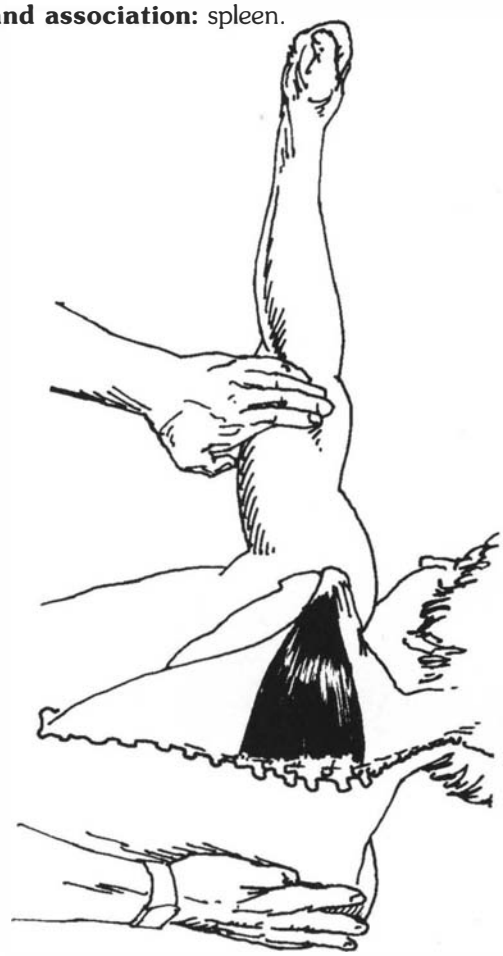
Origin: spinous processes of 1st-5th thoracic vertebrae.
Insertion: superior borders of spine of scapula.
Action: adducts and slightly elevates scapula; draws back acromion process.
Test: The prone patient extends the elbow with the shoulder in 90° abduction and external rotation (thumb toward ceiling). The examiner directs force against the arm toward the floor. Observation should be made for strong glenohumeral fixation. The test is of abduction of the scapula from the spine and must be observed or palpated by the examiner. Many examiners fail to observe for the abduction of the scapula, and list the mid-trapezius as weak simply because the arm fails to resist the testing pressure. The failure may be due to inadequate glenohumeral fixation.
Nerve supply: spinal accessory and ventral ramus, C2, **3, 4**.
Neurolymphatic:
Anterior: 7th intercostal space on the left.
Posterior: between T7, 8 near lamina on left.
Neurovascular: l" above lambda.
Nutrition: spleen concentrate or nucleoprotein extract, vitamin C, calcium.
Meridian association: spleen.
Organ/gland association: spleen.

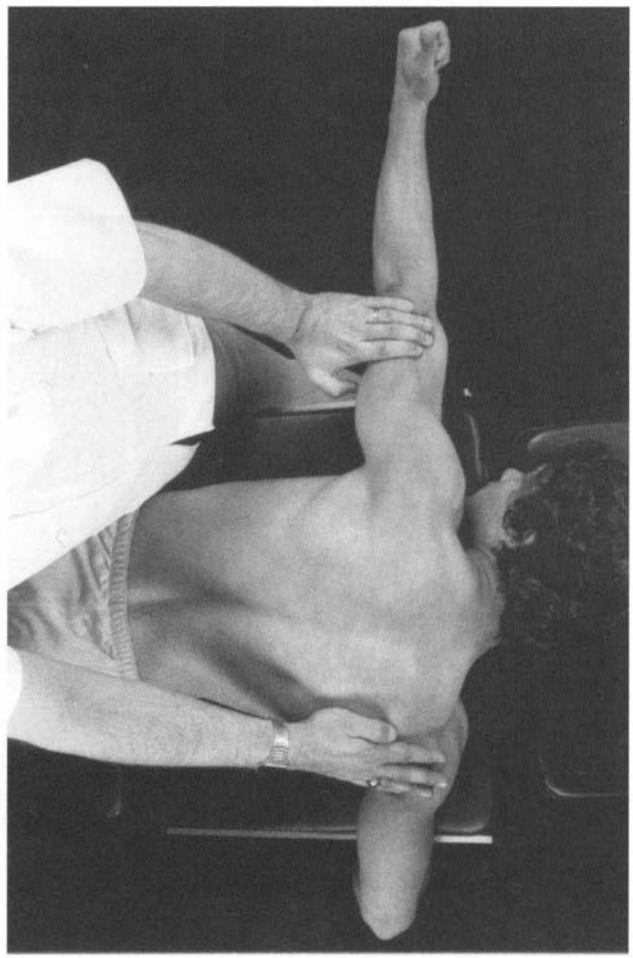

NEUROVASCULAR


STRESS RECEPTOR



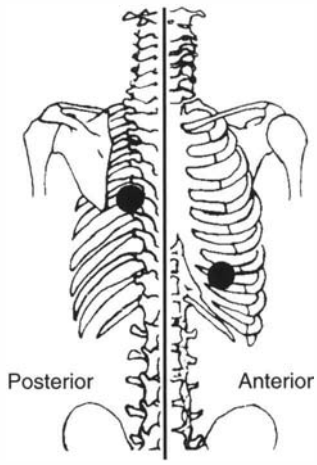
NEUROLYMPHATIC REFLEX USUALLY ON LEFT ONLY


8—87. Observe for scapular separation from the spine.

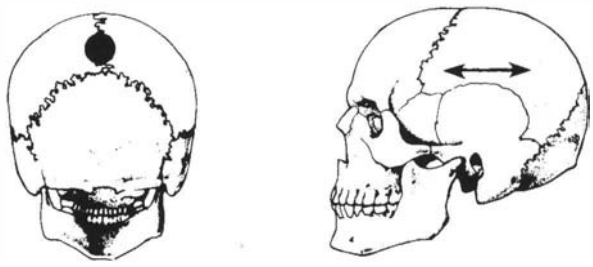

8—88. Patient supine, viewed from above. Observe for scapula separation from the spine.

Trapezius — Lower Division

Origin: spinous processes, 6th-12th thoracic vertebrae.
Insertion: medial one-third of spine of the scapula.
Action: rotation of scapula; gives inferior stabilization to scapula; helps maintain spine in extension; draws back acromion process.
Test: The prone patient extends the elbow and externally rotates the arm (thumb toward ceiling). The arm is abducted to approximately 150° to align the arm with the fibers of the lower trapezius. Examining pressure is directed against the arm toward the floor. The point of contact varies, depending upon the amount of leverage the examiner desires. There should be no motion at the glenohumeral articulation, and the elbow should not flex. The test is abduction and elevation of the scapula from the spine, and must be observed or palpated by the examiner.
Nerve supply: spinal accessory and ventral ramus, C2, **3, 4**.
Neurolymphatic:
Anterior: 7th intercostal space on left.
Posterior: between T7, 8 near lamina on left.
Neurovascular: 1" above lambda.
Nutrition: spleen concentrate or nucleoprotein extract, vitamin C, calcium.
Meridian association: spleen.
Organ/gland association: spleen.

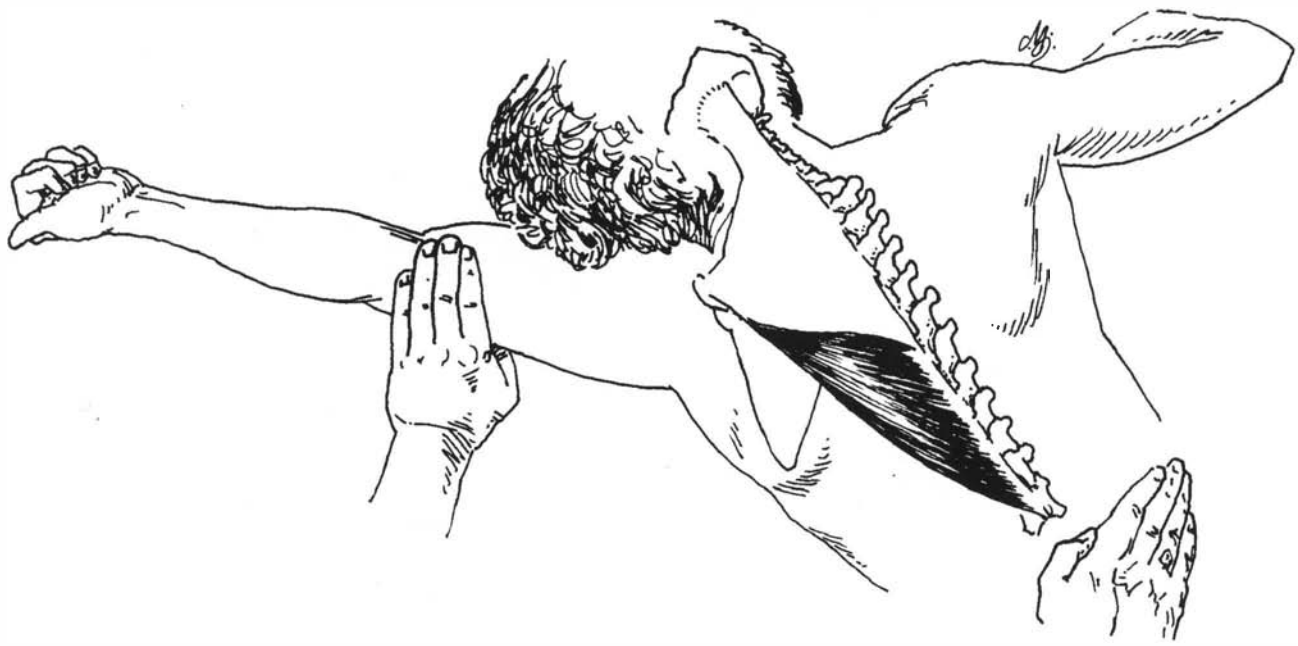


NEUROLYMPHATIC REFLEX
USUALLY ON LEFT ONLY



NEUROVASCULAR

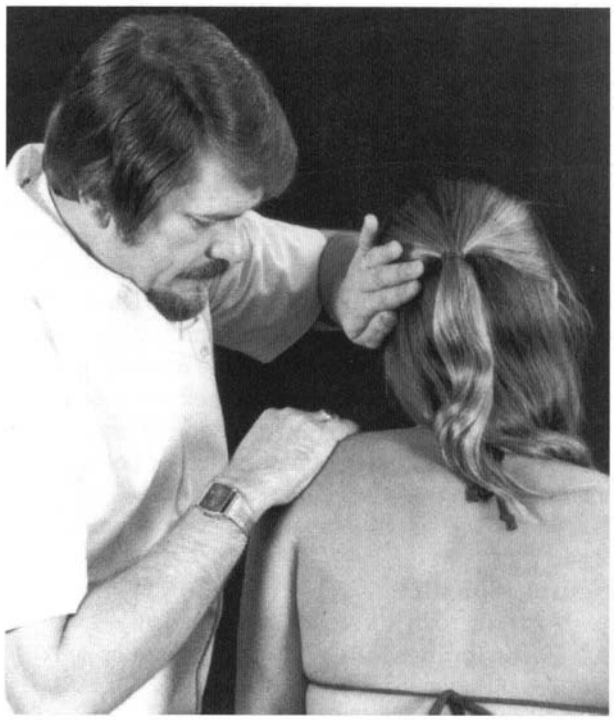
STRESS RECEPTOR



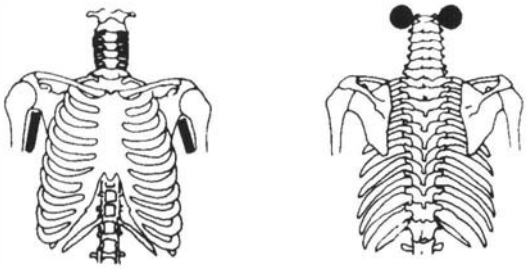
8—89. Observe for scapular movement indicating muscle failure.

Trapezius — Upper Division

Origin: external occipital protuberance, medial one-third of superior nuchal line, ligamentum nuchae and spinous process of C7 vertebra.
Insertion: lateral one-third of clavicle and acromion process.
Action: rotation of scapula so glenoid cavity faces superiorly; adducts the scapula when acting with the other sections of the trapezius.
Test: The seated patient elevates his shoulder and laterally flexes his neck and head, with rotation of the head slightly away from the side being tested. The examiner places one hand on the shoulder and the other on the head, directing force to reduce the approximation of the head and shoulder.
Nerve supply: spinal accessory and ventral ramus of C2, **3, 4**.
Neurolymphatic:
Anterior: 3" of anterior upper arm.
Posterior: posterior arch of atlas to lateral mass.
Neurovascular: on temporal sphenoidal suture just above zygomatic arch.
Nutrition: vitamins A, B, F, G, and calcium.
Meridian association: kidney.
Organ association: eye and ear.

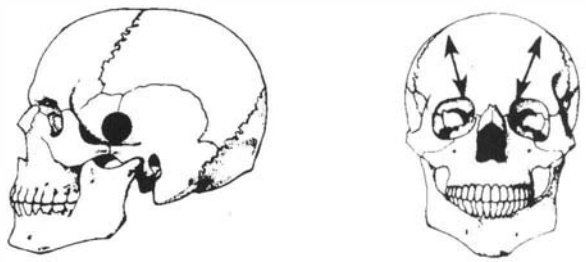


8—91. Head is rotated slightly away from side of test.

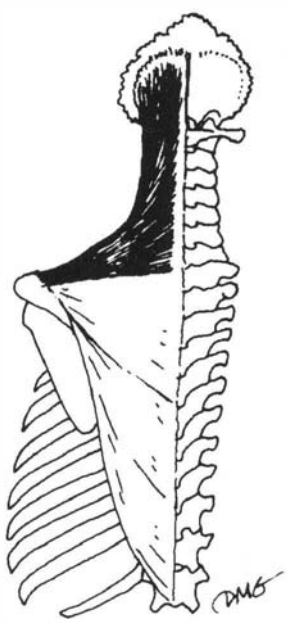


Anterior Posterior

NEUROLYMPHATIC



NEUROVASCULAR STRESS RECEPTOR



8—90

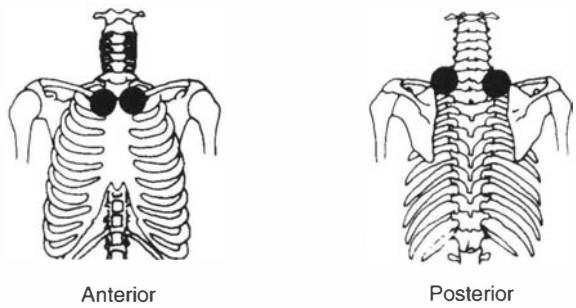
8—92

Subclavius

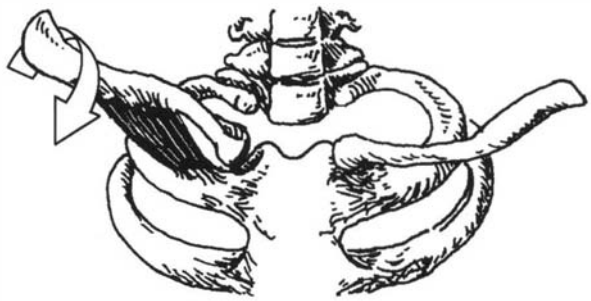
Origin: 1st rib at the junction of the costal cartilage.
Insertion: groove on the inferior surface of the clavicle, between the costoclavicular and conoid ligaments.
Action: draws the clavicle inferiorly and anteriorly; appears to participate in the "crank" action of the clavicle during shoulder abduction.
Test: This muscle cannot be tested by the usual manual muscle testing procedures. It is tested clinically by therapy localization. The patient therapy localizes to the belly of the muscle, and the examiner tests a previously strong indicator muscle. If the subclavius is involved, the strong indicator muscle will weaken. The usual precaution — that therapy localization only tells **where** something is, not **what** it is — must be observed. It is possible to have positive therapy localization over the subclavius muscle when actually the neurolymphatic for the anterior neck flexors or some other factor is involved.
Nerve supply: branch of the brachioplexus, **C5, 6**.
Neurolymphatic:

Anterior: junction of the clavicle, sternum, and 1st rib.

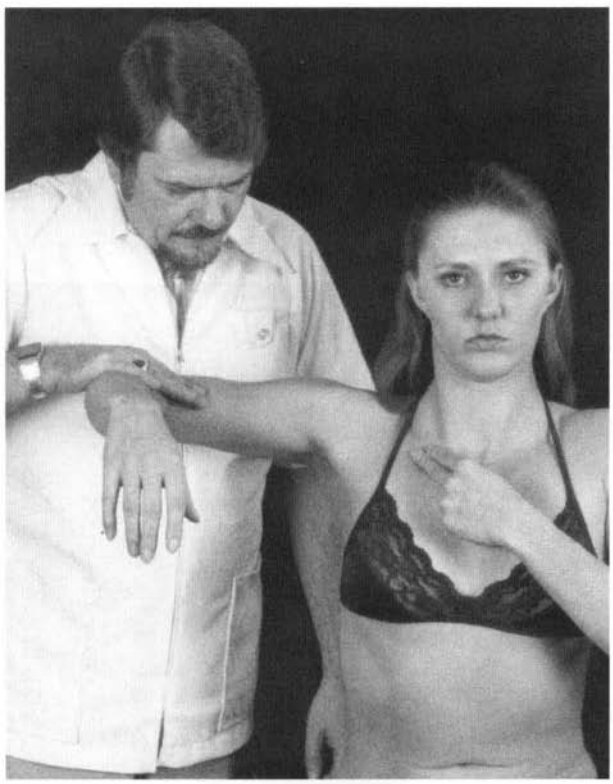
Posterior: laminae of T1.



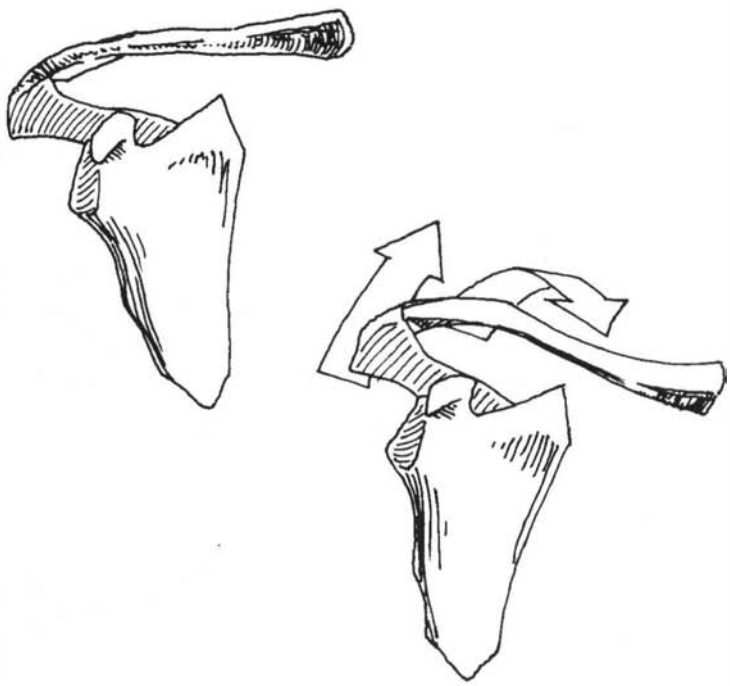
NEUROLYMPHATIC



8—94. Clavicle elevated and rotated to illustrate the subclavius muscle.



8—93. Therapy localization to the subclavius.



8—95. The subclavius rotates the clavicle to elevate its distal end.

Rhomboids — Major and Minor

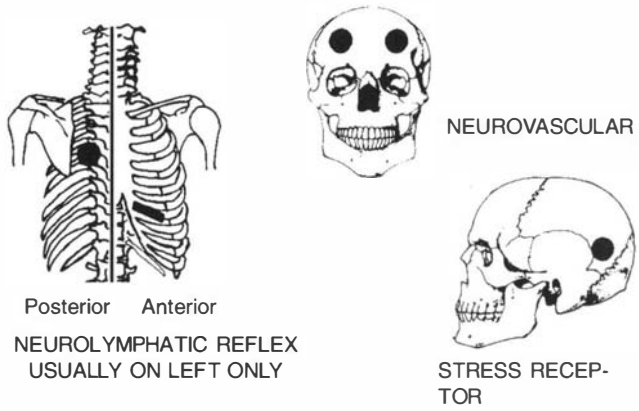
Rhomboid Major

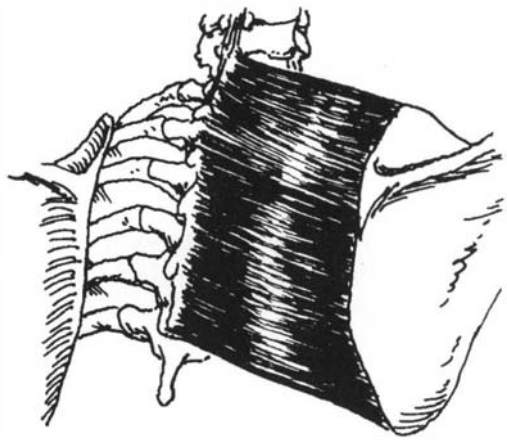
Origin: spinous processes of 2nd-5th thoracic vertebrae.
Insertion: medial border of scapula from spine to inferior angle.
Action: adducts scapula and slightly elevates medial border. The lower fibers of the rhomboid major aid in rotating the glenoid cavity inferiorly. In abduction of the arm, the rhomboids relax to allow scapular abduction, and then contract to stabilize the scapula during scapular rotation with continued abduction.

Rhomboid Minor

Origin: ligamentum nuchae, spinous processes of C7 and T1.
Insertion: medial border of scapula at root of spine of scapula.
Action: adducts and slightly elevates scapula.
Test (both): With the patient seated with his elbow flexed to 90° and the upper arm held in adduction, the examiner contacts the medial elbow and directs force to abduct it from the body. Observation is made for abduction of the scapula from the spine, which indicates rhomboid weakness.
Alternate testing methods: The test can be done with the patient prone or standing in a manner similar to the seated test. A test designed by Goodheart[17] is said to avoid recruitment of the opposite rhomboids. The supine patient rolls onto the scapula on the side opposite the rhomboids being tested. This position seems to immobilize the opposite rhomboids and scapula. The test is done in a manner similar to the sitting test. The examiner must watch carefully for scapular abduction and inferior displacement.
Nerve supply:
Rhomboid major: dorsal scapular, C4, **5**.
Rhomboid minor: dorsal scapular, **C4, 5**.

Neurolymphatic:
Anterior: 6th intercostal space from mamillary line to sternum on left.
Posterior: between T6, 7 by lamina on left.
Neurovascular: bilateral frontal bone eminences.
Nutrition: vitamin A.
Meridian association: liver.
Organ association: liver.





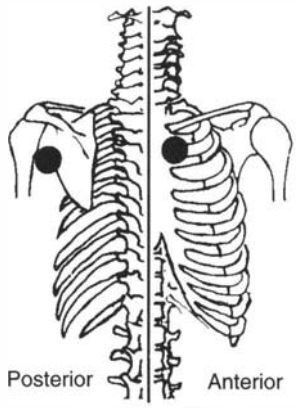
8—97. Rhomboid major and minor.

8—96. Rhomboid test starting position.

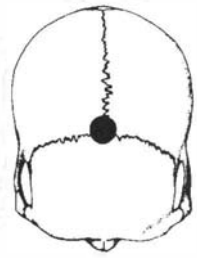
8—98. Observe for scapular abduction.

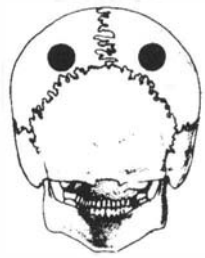
Levator Scapula

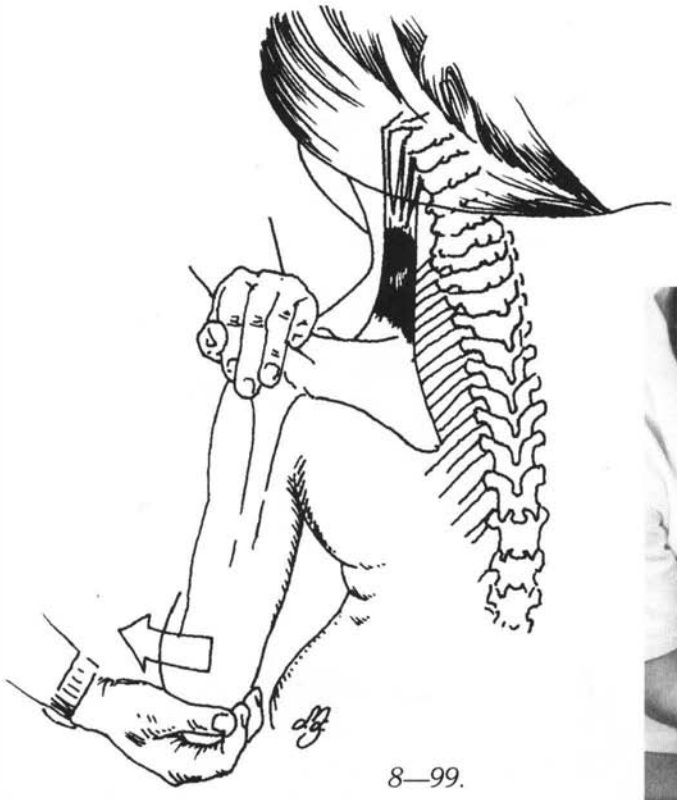
Origin: transverse processes of upper four cervical vertebrae.
Insertion: vertebral border of scapula between superior angle and root of scapular spine.
Action: raises scapula to inferiorly rotate glenoid cavity. Working in combination with the upper trapezius, elevates and adducts scapula.
Test: The seated patient flexes the elbow and maximally drops his shoulder toward the table as if to reach the table with the elbow. The test is improved by minimum lateral spinal flexion. The humerus is maintained in adduction and slight extension. The examiner directs force against the medial elbow in a direction of abduction, observing for inferior rotation of the superior angle of the scapula.
Nerve supply: dorsal scapular, **C3, 4, 5**.
Neurolymphatic:
Anterior: 1st rib intercostal space near sternum.
Posterior: belly of teres minor.
Neurovascular: bregma.
Nutrition: parathyroid concentrate or nucleoprotein extract.
Meridian association: lung.
Gland association: parathyroid glands.

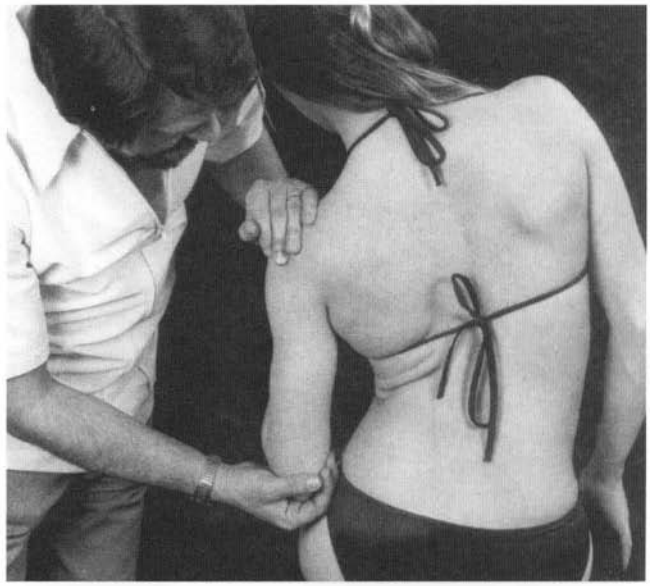


NEUROLYMPHATIC
BILATERAL


NEUROVASCULAR


STRESS RECEPTOR


8—99.


8—100. Observe for scapular movement.

Latissimus Dorsi

Origin: a broad aponeurosis by which it originates from the lower six thoracic vertebrae, spinous processes, lumbar spinous processes, posterior crest of the ilium, lower three or four ribs, and an attachment to the tip of the scapula.

Insertion: twists upon itself to insert into the floor of the intertubercular groove of the humerus.

Action: extends, adducts, and rotates the humerus internally; draws the inferior angle of the scapula inferiorly and medially.

Test: The standing or seated patient holds his arm in adduction, with internal rotation, so the antecubital fossa faces medially. The examiner directs pressure to the patient's wrist in a direction to abduct and slightly flex the shoulder. Care should be taken that the pressure against the wrist does not cause pain to the patient. The examiner must avoid touching the meridian pulse points of the wrist, which are located along the radial artery, accidentally causing therapy localization.

Nerve supply: thoracodorsal from brachial plexus, **C6, 7, 8.**

Neurolymphatic:

Anterior: 7th intercostal space on left at rib cartilage junction.

Posterior: between T7, 8 at lamina on left.

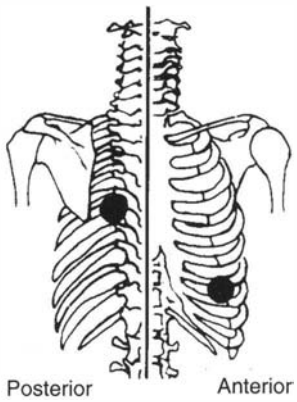
(**Note:** Generally both latissimus dorsi muscles will be affected by the left neurolymphatic reflexes. Occasionally the neurolymphatic reflex may be on the right, influencing the right muscle; if so, evaluate the patient for switching, which may or may not be present.)

Neurovascular: superior to temporal bone on a line slightly posterior to the external auditory meatus.

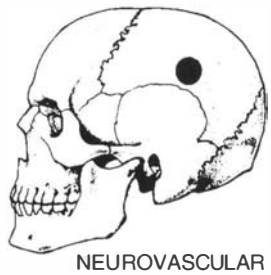
Nutrition: vitamins A, F (unsaturated fatty acids) and betaine; pancreas concentrate or nucleoprotein extract.

Meridian association: spleen.

Gland association: pancreas.

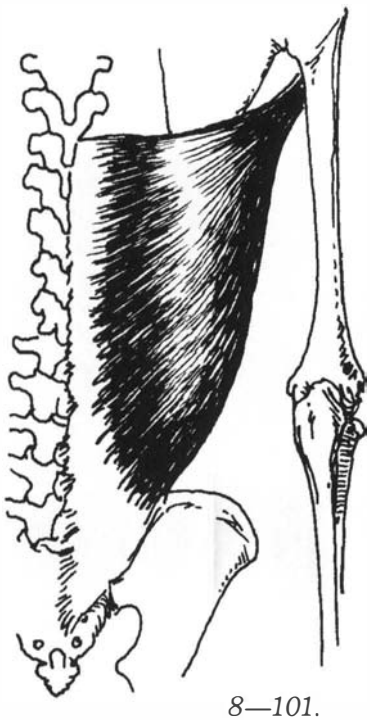


NEUROLYMPHATIC REFLEX USUALLY ON LEFT ONLY

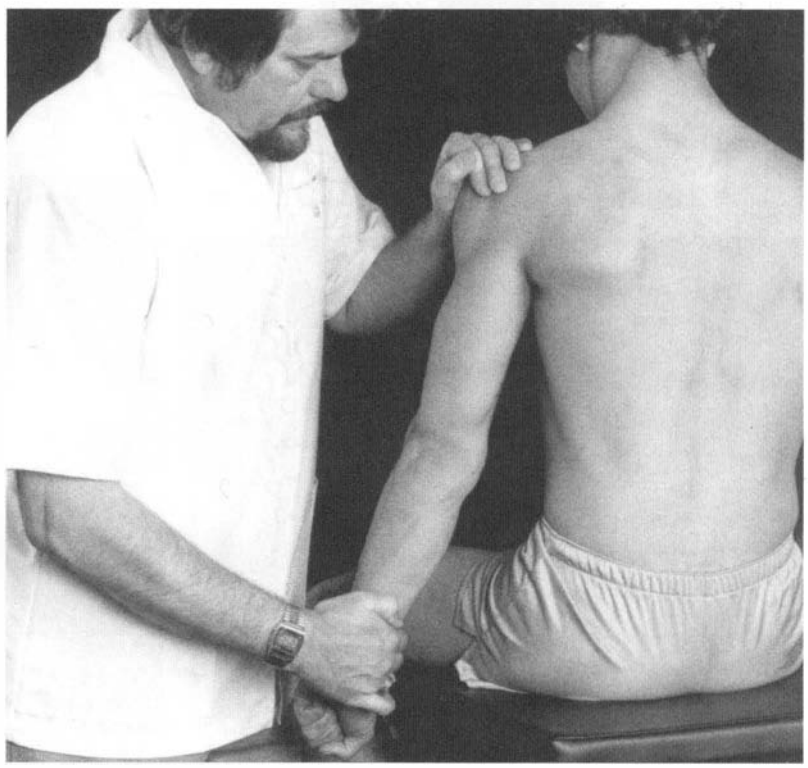


NEUROVASCULAR

STRESS RECEPTOR



8—101.



8—102. Patient's elbow must remain in extension during test.

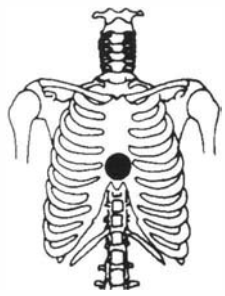
Pectoralis Minor

Origin: 3rd, 4th and 5th ribs near the costal cartilage.
Insertion: coracoid process of the scapula.
Action: pulls coracoid process anteriorly, medially, and inferiorly; an important anterior shoulder stabilizer.
Test: The supine patient lifts his shoulder off the table, drawing the coracoid process anteriorly, medially, and caudally. Pressure is directed against the shoulder to elongate the fibers of the pectoralis minor. Care should be taken not to direct the pressure posteriorly. Make certain the patient does not substitute by using his arm to force the shoulder from the table.
Nerve supply: medial pectoral nerve from brachioplexus, C6, **7, 8,** T1.
Neurolymphatic:

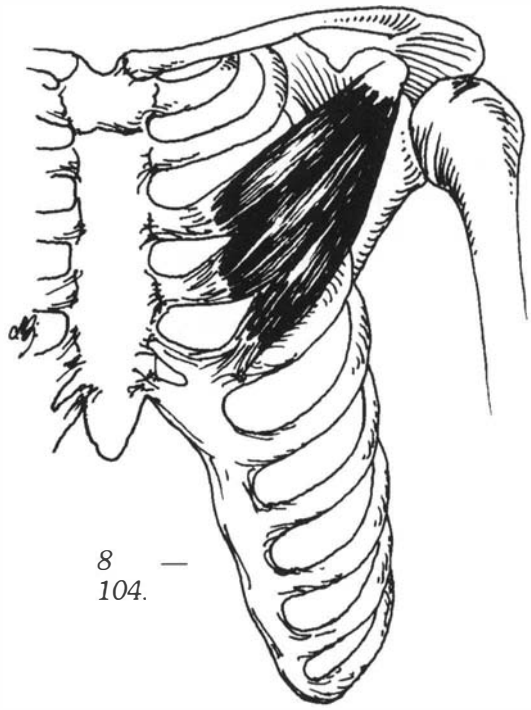
Anterior: immediately above xiphoid process on the sternum. Manipulate until pain on digital pressure ceases.[18]

Posterior: none.

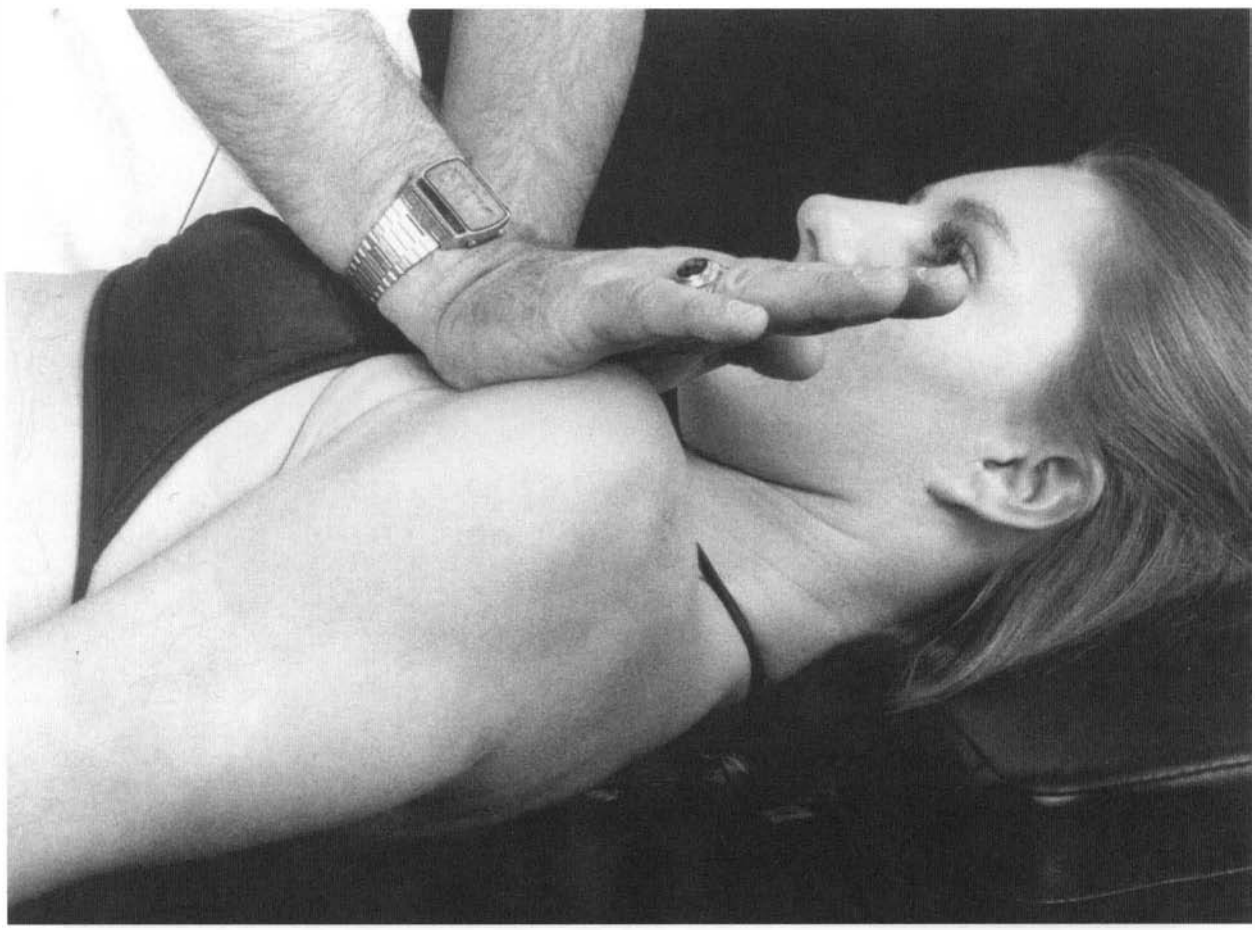
Nutrition: brain concentrate or nucleoprotein extract, ribonucleic acid, niacin or niacinamide, B complex.



NEUROLYMPHATIC



8 — 104.



8—103. Direct testing pressure to elongate the pectoralis minor muscle.

Pectoralis Major — Clavicular Division

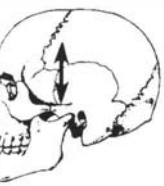
Origin: anterior surface of sternal half of the clavicle.
Insertion: lateral lip of the bicipital groove of the humerus.
Action: flexes the shoulder joint and horizontally adducts the humerus toward the opposite shoulder. Participates in internal rotation in some subjects.
Test: The supine patient extends the elbow and flexes the shoulder to 90°, with internal rotation, so the thumb points toward the feet. Pressure is directed on the distal end of the forearm in the direction of abduction and slight extension of the shoulder. The direction of pressure can best be determined if the examiner visualizes a line from the origin to the insertion, with the direction of pressure extending from that line, giving best alignment of the fibers of the clavicular portion. The vector of testing force will vary somewhat between subjects.

Common testing errors occur when the opposite shoulder is allowed to rise from the table, or when the patient is permitted to recruit additional synergistic action of the biceps by flexing the elbow.
Nerve supply: lateral pectoral, **C5, 6, 7**.
Neurolymphatic:

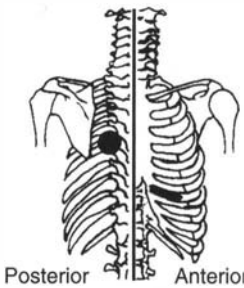
Anterior: 6th intercostal space from mamillary line to sternum on left, which usually affects both right and left muscles. Occasionally found on right, affecting the right pectoralis major (clavicular division). When found on right, always evaluate to determine if the patient is switched.

Posterior: between T6, 7 near lamina on left.
Neurovascular: bilateral frontal bone eminences.
Nutrition: vitamin B, betaine hydrochloride, stomach concentrate or nucleoprotein extract with vitamin B_{12}.
Meridian association: stomach.
Organ association: stomach.

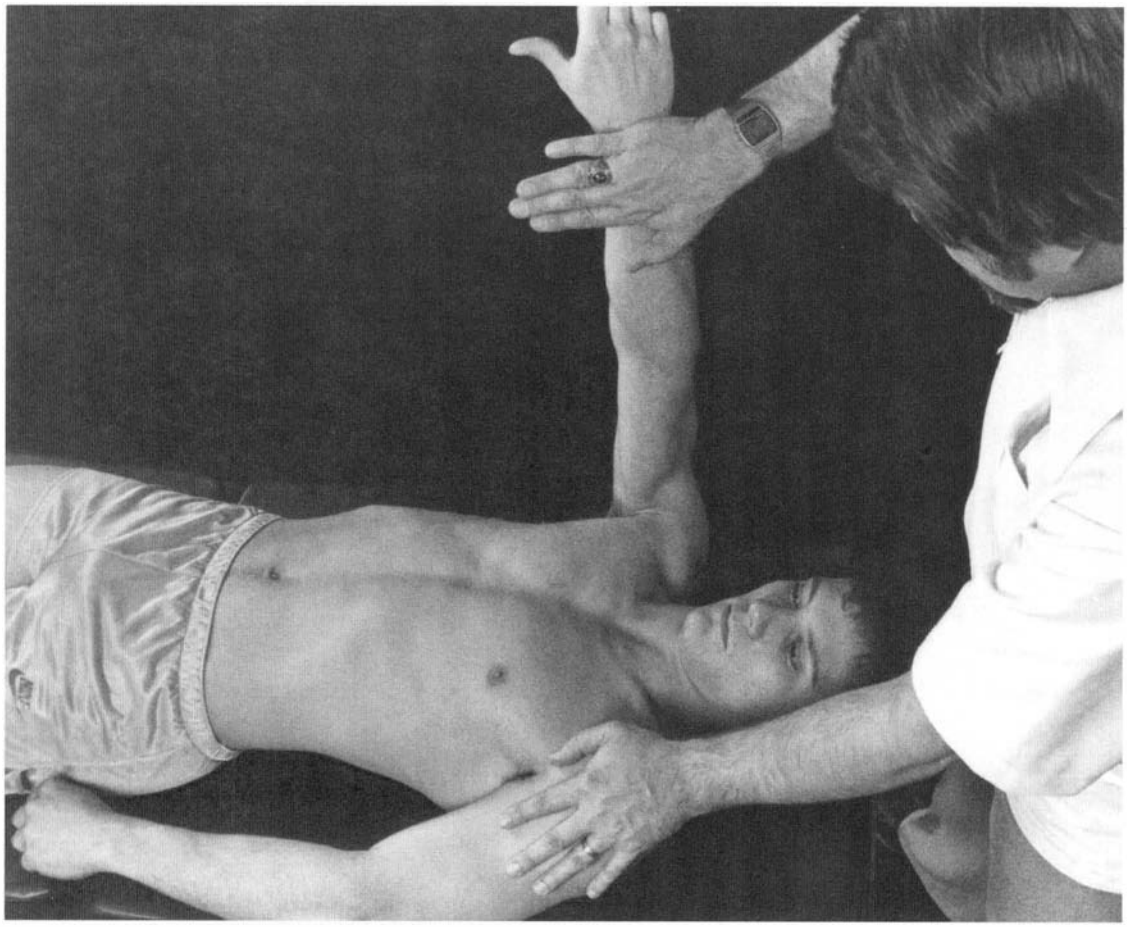
NEUROVASCULAR



STRESS RECEPTOR



NEUROLYMPHATIC REFLEX
USUALLY ON LEFT ONLY



8—105. Patient must maintain his elbow in extension.

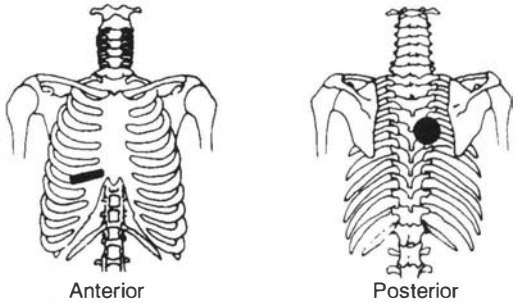
Pectoralis Major — Sternal Division

Origin: sternum to 7th rib, cartilages of true ribs and aponeurosis of external oblique abdominal muscle.
Insertion: lateral lip of the bicipital groove of the humerus.
Action: adducts humerus toward opposite iliac crest; major anterior shoulder stabilizer.
Test: The patient extends the elbow and flexes the shoulder to 90° in internal rotation so the thumb points toward the feet. Pressure is directed on the distal forearm in the direction of abduction and increased shoulder flexion. The best alignment of pressure can be observed by drawing an imaginary line from the middle of the origin through the center of the insertion; the direction of pressure extends from that line.
Nerve supply: lateral and medial pectoral, **C6, 7, 8, T1**.
Neurolymphatic:

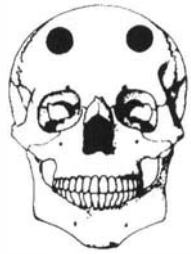
Anterior: 5th intercostal space from mamillary line to sternum on right, which usually affects both right and left muscles. Occasionally the neurolymphatic reflex may be found on the left, especially if there is left pectoralis major (sternal division) weakness. If therapy localization to the left side strengthens the muscle, consider the possibility of switching and treat accordingly.

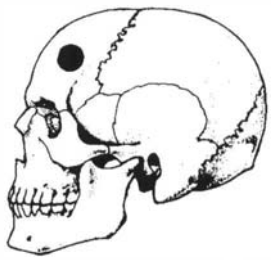
Posterior: between T5, 6 near lamina, usually on right.

Neurovascular: bilaterally 1-1/2" up from prominent bulges on anterior frontal bone, 1-1/2" from midline.
Nutrition: vitamin A, bile salts, liver concentrate or nucleoprotein extract.
Meridian association: liver.
Organ association: liver.



NEUROLYMPHATIC USUALLY ON RIGHT

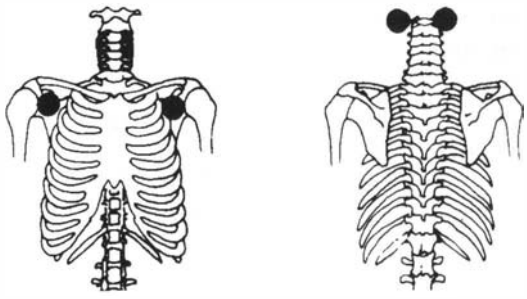

NEUROVASCULAR


STRESS RECEPTOR

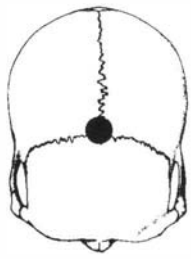
8—106. Direction of pressure is in alignment with muscle fibers.

Supraspinatus

Origin: medial two-thirds of supraspinatus fossa of scapula.
Insertion: superior facet of greater tuberosity of humerus and capsule of shoulder joint.
Action: abducts arm with deltoid. Holds head of humerus in glenoid cavity.
Test: The seated or standing patient abducts his arm approximately 15°, with the antecubital fossa facing anteriorly. The examiner contacts the wrist and directs force toward adduction of the arm with slight extension. The deltoid is synergistic in this test, and must be correlated to make final determination of the supraspinatus muscle function.
Nerve supply: suprascapular, C**5**, 6.
Neurolymphatic:
Anterior: below coracoid process.
Posterior: posterior to transverse process of atlas.
Neurovascular: bregma.
Nutrition: RNA, brain concentrate or nucleoprotein extract.
Meridian association: conception vessel.
Organ association: brain.

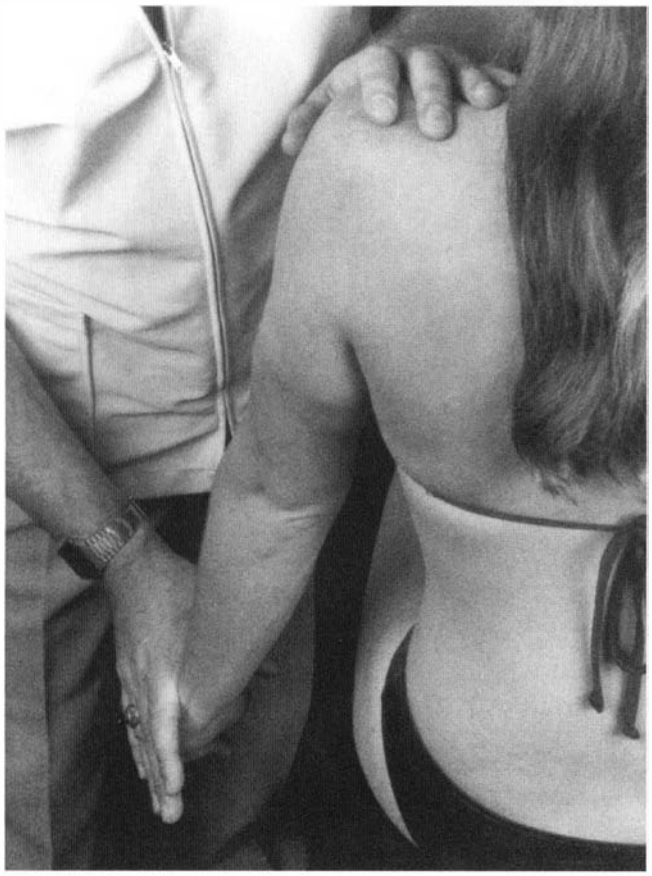


NEUROLYMPHATIC

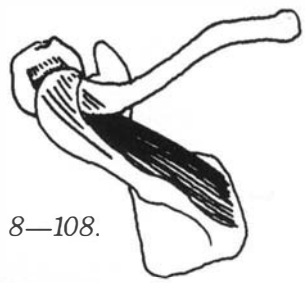


NEUROVASCULAR

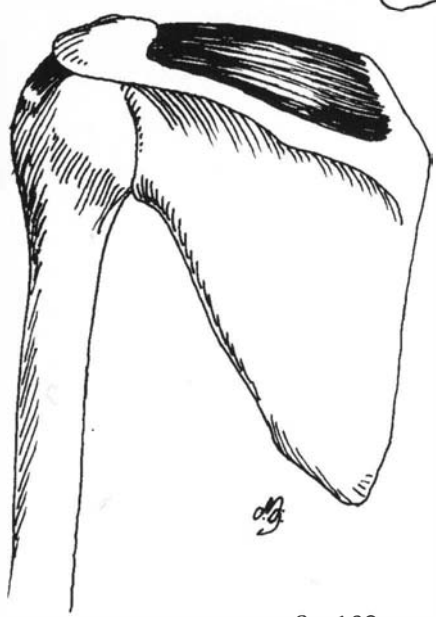
STRESS RECEPTOR



8—107.



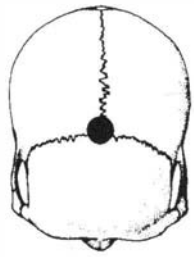
8—108.



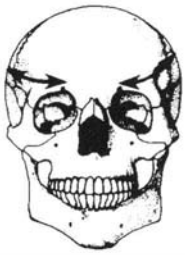
8—109.

Deltoid — Middle Division

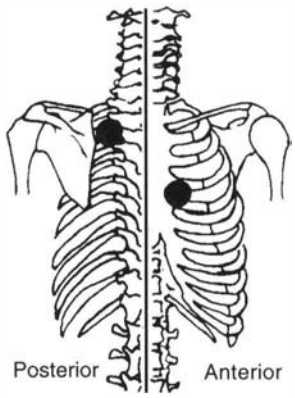
Origin: upper surface of acromion process.
Insertion: deltoid tuberosity of humerus.
Action: abduction of the humerus.
Test: The seated or standing patient flexes the elbow and abducts the shoulder to 90°. The horizontal forearm indicates neutral humerus rotation. Pressure is applied against the distal end of the humerus in straight adduction.
Nerve supply: axillary, **C5, 6**.
Neurolymphatic:
Anterior: 3rd intercostal space near sternum.
Posterior: between T3, 4 near laminae.
Neurovascular: bregma.
Nutrition: lung concentrate or nucleoprotein extract, vitamin C, RNA.
Meridian association: lung.
Organ association: lung.



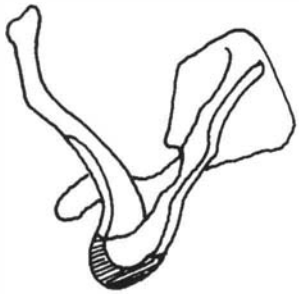
NEUROVASCULAR



STRESS RECEPTOR



NEUROLYMPHATIC
BILATERAL

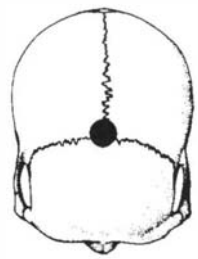


8—110.

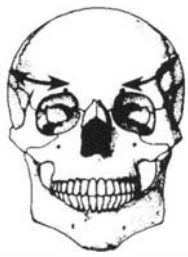
8—111. Pressure is directed toward straight adduction.

Deltoid — Anterior Division

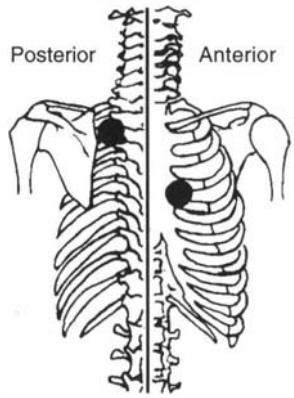
Origin: lateral one-third of clavicle on its anterosuperior border.
Insertion: deltoid tuberosity of humerus.
Action: abduction of humerus in combination with other portions of the deltoid; flexes and internally rotates the humerus.
Test: The seated or standing patient flexes the elbow and shoulder to 90°, with slight external rotation of the humerus. This is indicated by the elevation of the distal forearm. Contact is made at the distal humerus with pressure in a direction of adduction and slight extension.
Nerve supply: axillary, **C5, 6**.
Neurolymphatic:
Anterior: 3rd intercostal space near sternum.
Posterior: between T3, 4 near laminae.
Neurovascular: bregma.
Nutrition: lung concentrate or nucleoprotein extract, vitamin C, RNA.
Meridian association: lung.
Organ association: lung.



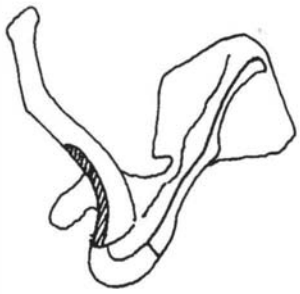
NEUROVASCULAR



STRESS RECEPTOR



NEUROLYMPHATIC
BILATERAL

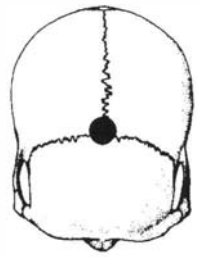


8—112.

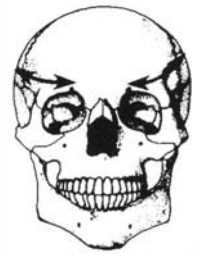
8—113. Pressure is directed toward adduction and slight extension.

Deltoid — Posterior Division

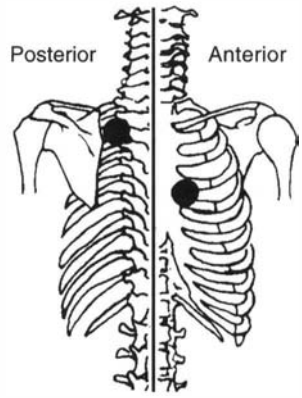
Origin: inferior lip of spine of scapula.
Insertion: deltoid tuberosity of humerus.
Action: abduction of humerus when working with other sections of deltoid. When working by itself, the action is abduction, slight extension, and lateral rotation.
Test: The seated or standing patient flexes the elbow and humerus to 90°, with slight internal rotation. This is observed by the inferior position of the distal forearm. Pressure is directed against the distal end of the humerus in a direction of adduction and slight flexion.
Nerve supply: axillary, **C5, 6**.
Neurolymphatic:
Anterior: 3rd intercostal space near sternum.
Posterior: between T3, 4 near laminae.
Neurovascular: bregma.
Nutrition: lung concentrate or nucleoprotein extract, vitamin C, RNA.
Meridian association: lung.
Organ association: lung.



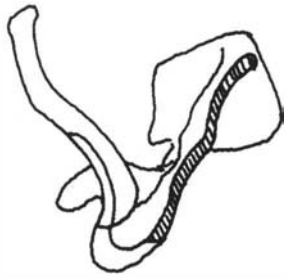
NEUROVASCULAR



STRESS RECEPTOR



NEUROLYMPHATIC
BILATERAL

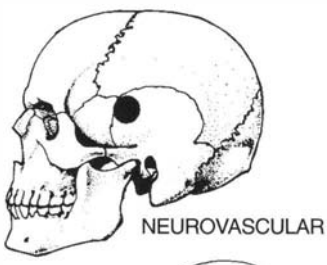


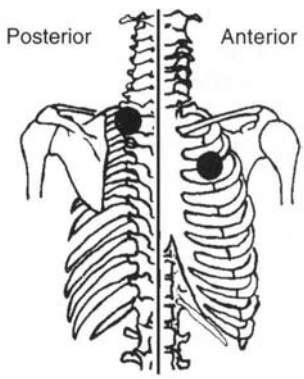
8—114.

8—115. Pressure is directed toward adduction and slight flexion.

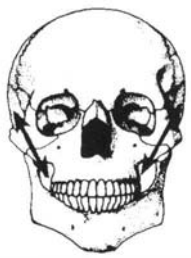
Teres Major

Origin: dorsal surface of inferior angle of scapula and lower one-third of scapular axillary border.
Insertion: medial lip of bicipital groove of humerus.
Action: adducts and internally rotates humerus. Extends shoulder joint. Important muscle to act as couple with deltoid in arm abduction.
Test: The prone patient abducts and extends his shoulder with the elbow flexed at 90°. The wrist rests against the posterior superior iliac crest. The examiner directs pressure against the patient's elbow in a direction of abduction and flexion of the shoulder. Care should be taken not to overpower the patient in this testing position; the shoulder or muscle can be easily injured since the patient is in a position in which he cannot yield, and the examiner has a great deal of leverage advantage.
Nerve supply: lower scapular, C5, **6**, 7.
Neurolymphatic:
Anterior: 2nd intercostal space 2-1/2" from sternum.
Posterior: T3 near laminae.
Neurovascular (provisional): 1" below the pterion and at the junction of the 1st rib, clavicle, and sternum.
Nutrition: Evaluate acid-alkaline balance. When there is excessive perspiration, kelp and/or organic minerals. Zinc, especially when it is difficult to taste food.
Meridian association: governing vessel.
Organ association: generally associated with the spine because of its correlation to thoracic vertebrae fixations.

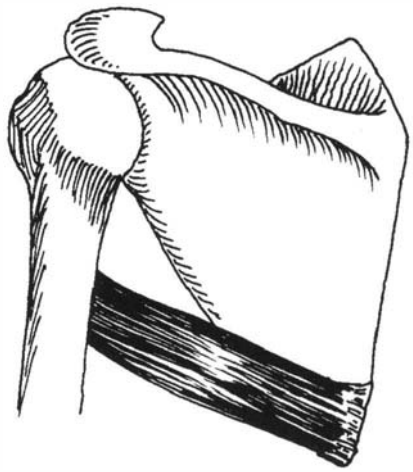




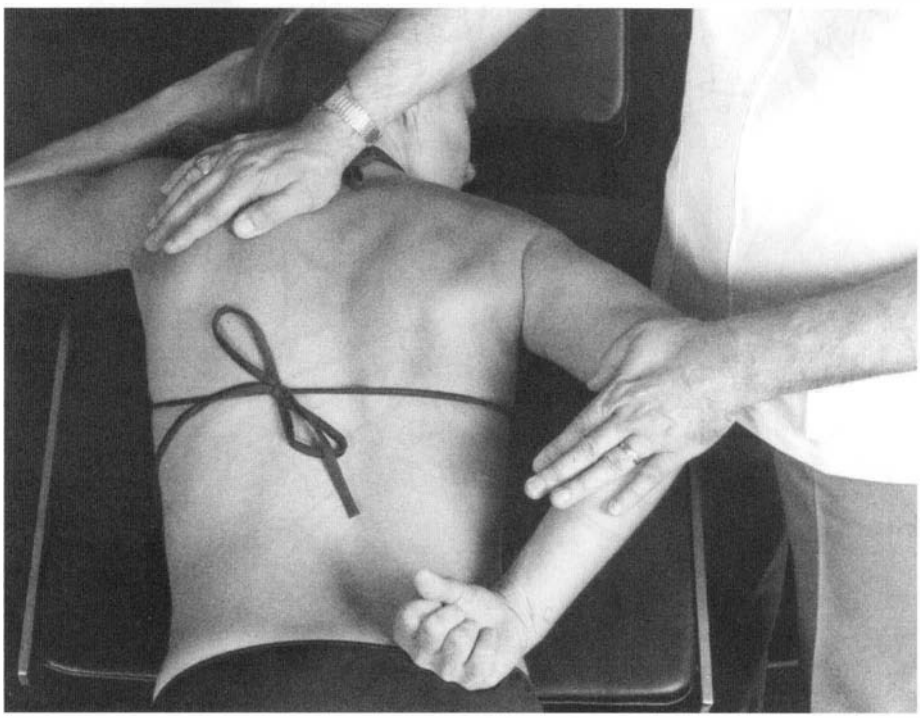
NEUROLYMPHATIC
BILATERAL



STRESS RECEPTOR



8—117.

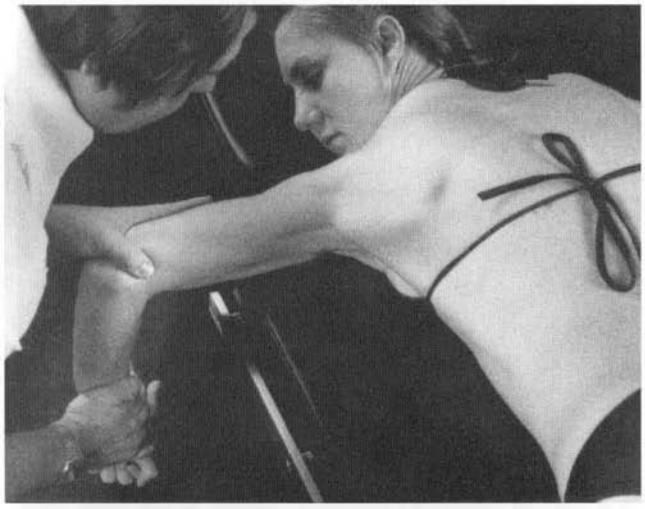


8—116. Observe for rotation of humerus with the scapula.

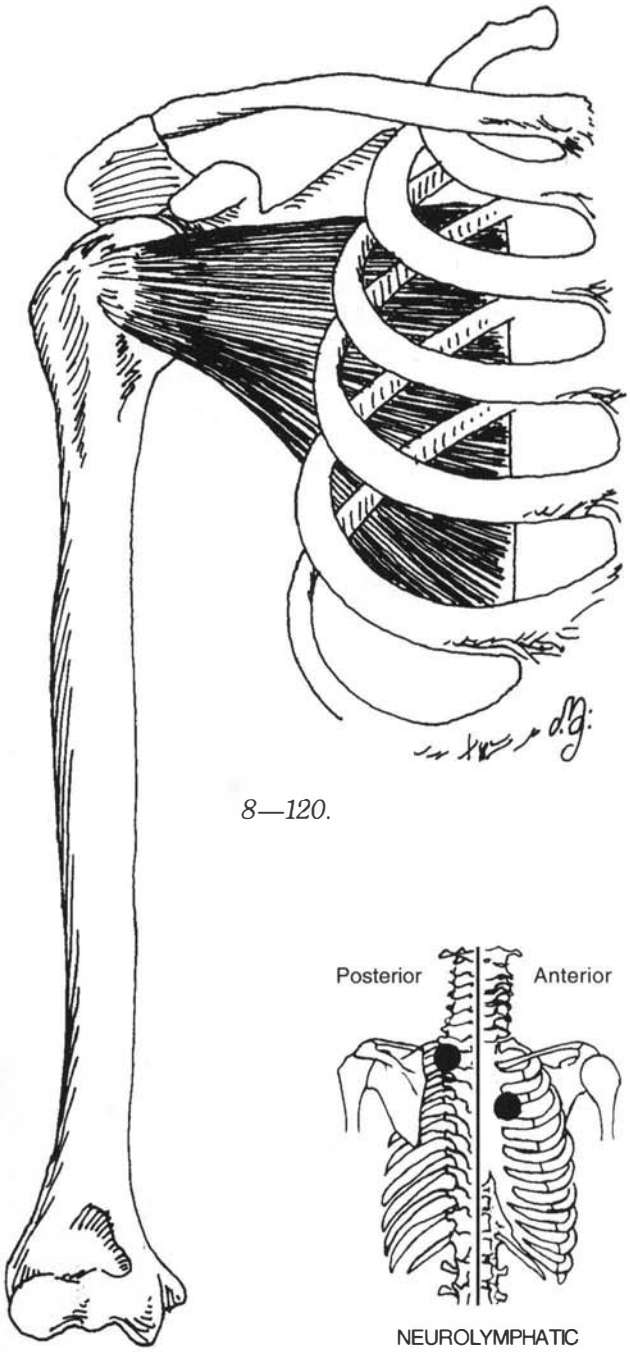
Subscapularis

Origin: subscapular fossa.
Insertion: lesser tuberosity of humerus and capsule of shoulder joint.
Action: internally rotates humerus. Draws head of humerus forward and down when arm is raised, acting as part of the force couple of shoulder abduction.
Test: The seated or prone patient abducts the shoulder to 90°, with the elbow flexed to 90°. The humerus is placed in slight internal rotation. The examiner directs pressure against the patient's wrist to externally rotate the humerus, using the forearm for leverage.
Nerve supply: upper and lower subscapular, **C5, 6**.
Neurolymphatic:
Anterior: 2nd intercostal space near sternum.
Posterior: T2, 3 between transverse processes.
Neurovascular: bregma.
Nutrition: heart concentrate or nucleoprotein extract, vitamin E, B complex, C.
Meridian association: heart.
Organ association: heart.

8—118.

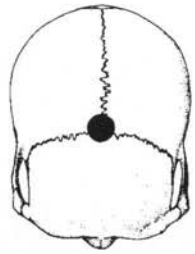


8—119. Failure of scapula to be stabilized by rhomboids and middle trapezius.



8—120.

NEUROLYMPHATIC
BILATERAL

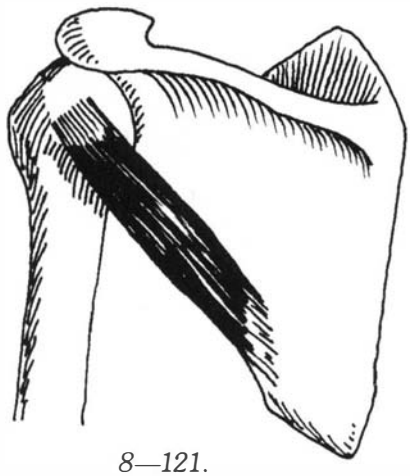


NEUROVASCULAR

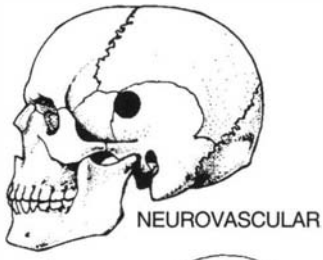
STRESS RECEPTOR

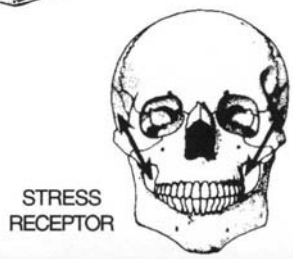
Teres Minor

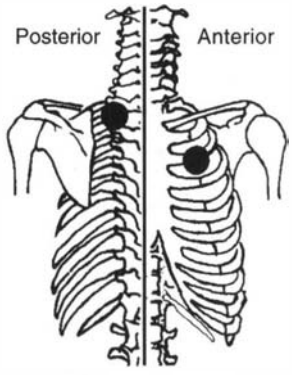
Origin: upper two-thirds of dorsal surface of axillary border of scapula.
Insertion: low on the greater tuberosity of the humerus; capsule of the shoulder joint.
Action: externally rotates the humerus and slightly adducts and extends humerus; stabilizes head of humerus in glenoid cavity during movement, and acts as a couple with the deltoid in arm abduction.
Test: The supine or seated patient flexes his elbow to 90° and externally rotates his humerus. The examiner directs pressure against the wrist, using the patient's forearm for leverage to internally rotate the humerus.
Nerve supply: axillary, C4, **5**, 6.
Neurolymphatic:
Anterior: 2nd intercostal space near sternum.
Posterior: T3 laminae.
Neurovascular: 1" below the pterion and at the junction of the 1st rib, clavicle, and sternum.
Nutrition: thyroid concentrate or nucleoprotein extract, organic iodine.
Meridian association: triple heater.
Gland association: thyroid.

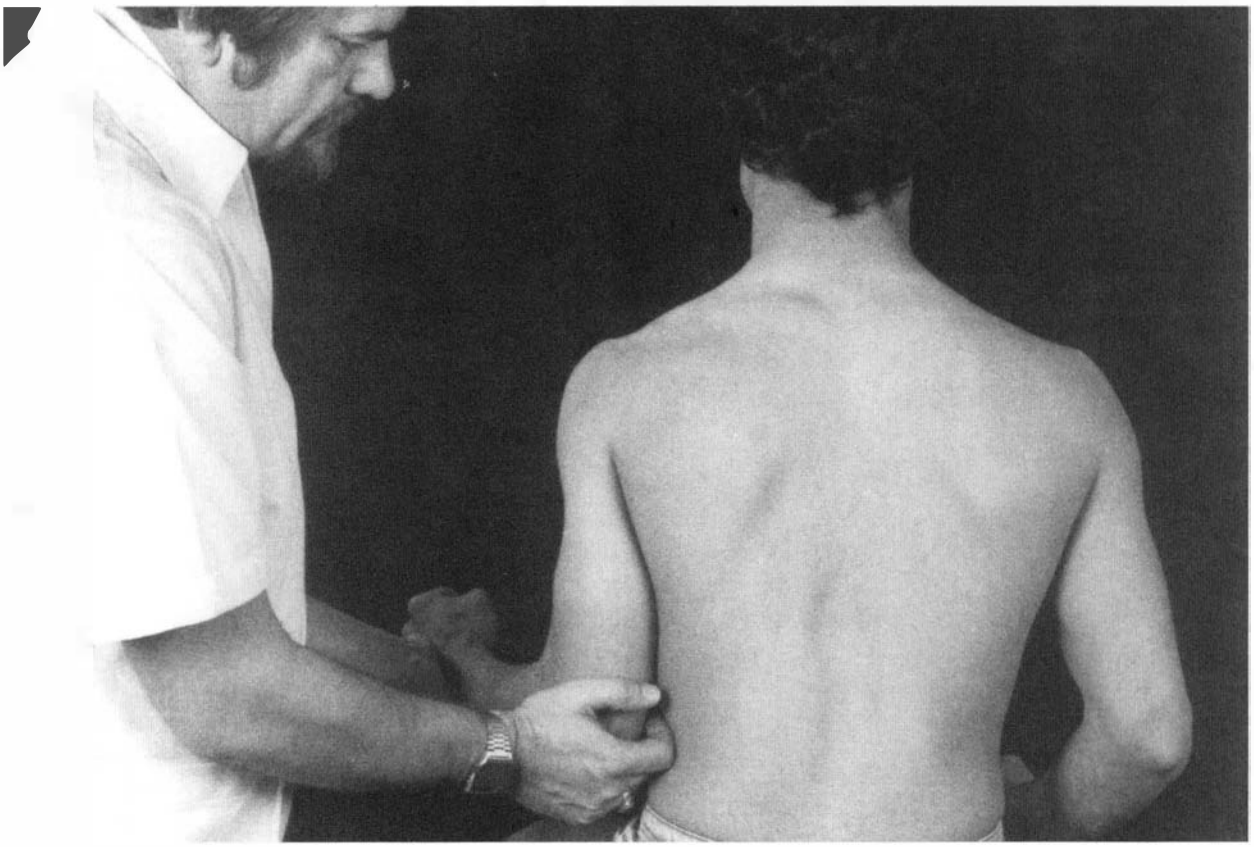


8—121.









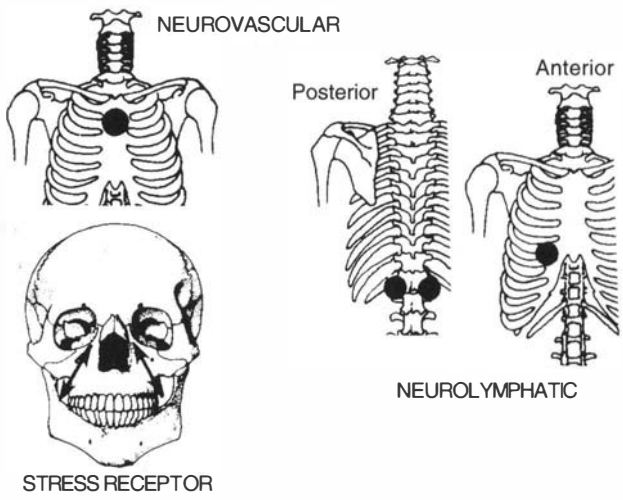
8—122.

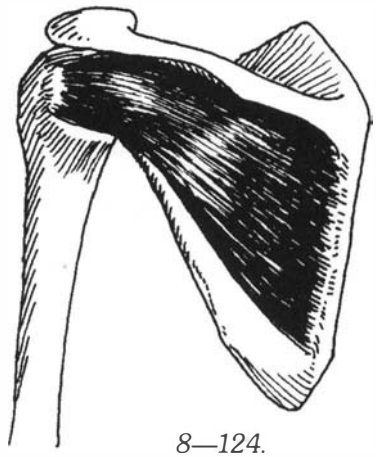
Infraspinatus

Origin: middle two-thirds of infraspinatus fossa of scapula.
Insertion: middle facet of greater tuberosity of humerus, capsule of shoulder joint.
Action: external rotation of humerus with teres minor. Stabilization of the head of the humerus with the glenoid cavity.
Test: The seated or prone patient abducts his humerus to 90°, with 90° elbow flexion and external humeral rotation. The examiner directs pressure to internally rotate the humerus. During the test, the examiner must observe for adequate scapula fixation. When this is not present, an assistant can aid in the test by stabilizing the scapula.
Nerve supply: suprascapular, **C5, 6**.
Neurolymphatic:

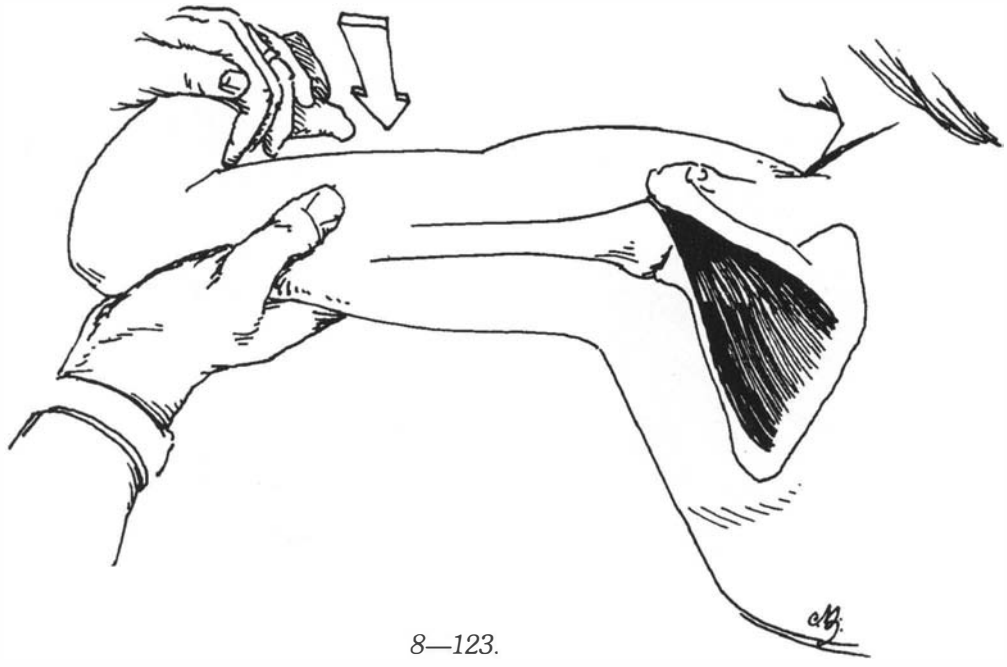
Anterior: 5th intercostal space near sternum on right.
Posterior: T12 laminae, bilateral.

Neurovascular: angle of Louis on the sternum.
Nutrition: thymus concentrate or nucleoprotein extract.
Meridian association: triple heater. The alarm point for involvement of the thymus is CV 18, located on the sternum just above the alarm point for the circulation sex meridian. This is a point that has been determined clinically in applied kinesiology, and is not one of classic acupuncture.[18]
Gland association: thymus.





8—124.

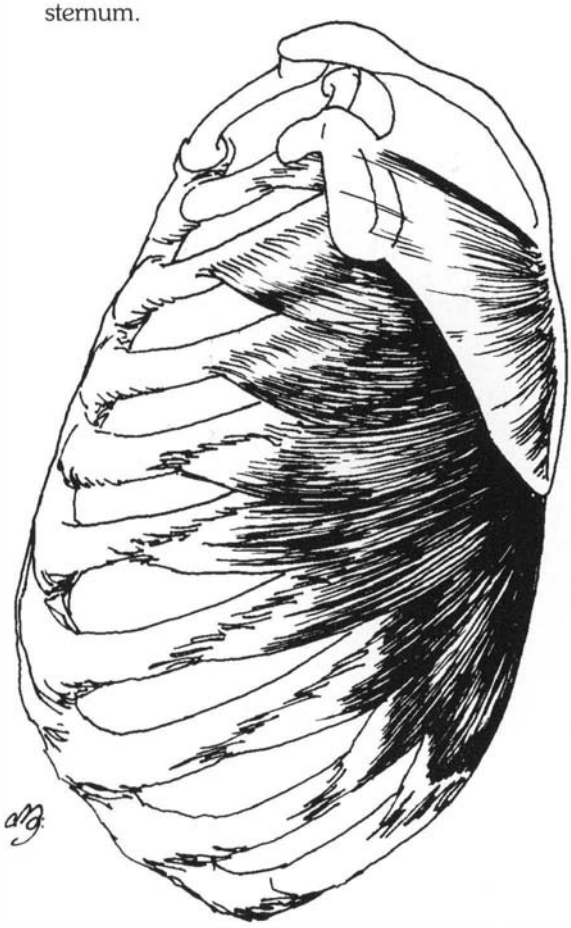


8—123.

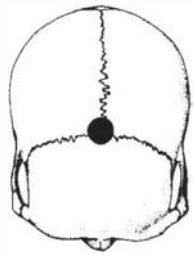
Serratus Anticus

Origin: outer surfaces and superior borders of upper eight or nine ribs.
Insertion: costal surface of the vertebral border of scapula.
Action: abducts scapula and rotates it to point the glenoid cavity more superiorly; holds vertebral border of scapula to thoracic cage, along with rhomboids and middle trapezius.
Test: The seated or standing patient holds his arm to approximately 100-130° flexion, with abduction. This brings the inferior angle of the scapula into abduction and the glenoid cavity into superior rotation. Testing pressure is directed on the humerus or at the wrist, depending upon the amount of leverage the examiner wishes to use. The integrity of the glenohumeral articulation must be taken into consideration, making certain that no movement takes place at that joint. The examiner's other hand contacts the inferior lateral border of the scapula, rotating the inferior angle medially, while applying pressure to bring the arm downward in the direction of extension and adduction. Movement evaluation is of the scapula, not the arm.
Nerve supply: long thoracic, **C5, 6, 7**.
Neurolymphatic:

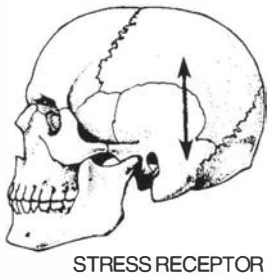
Anterior: 3rd, 4th, and 5th intercostal spaces near sternum.



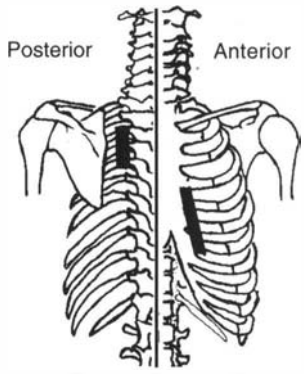
8—125.



NEUROVASCULAR

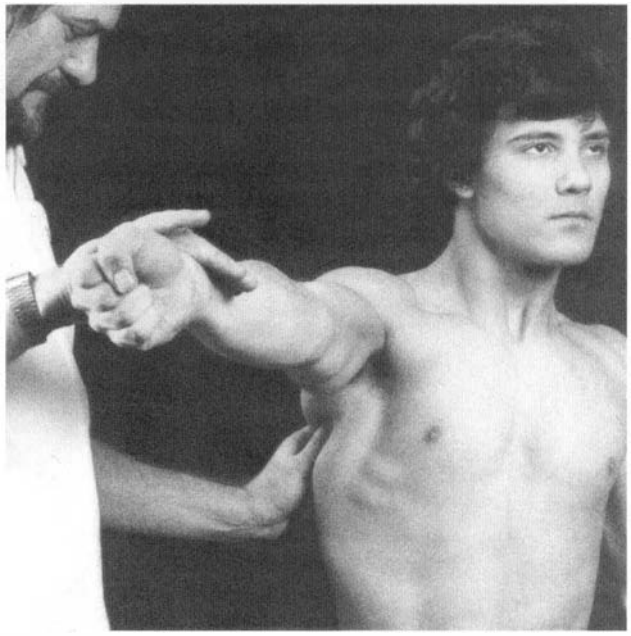


STRESS RECEPTOR



NEUROLYMPHATIC
BILATERAL

Posterior: T3, 4, and 5 at laminae.
Neurovascular: bregma.
Nutrition: lung concentrate or nucleoprotein extract, vitamin C.
Meridian association: lung.
Organ association: lung.

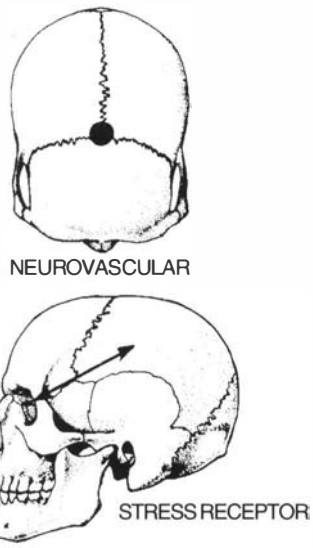


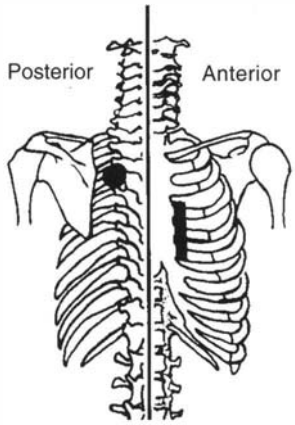
8—126. Evaluate for movement of the scapula, not the arm.

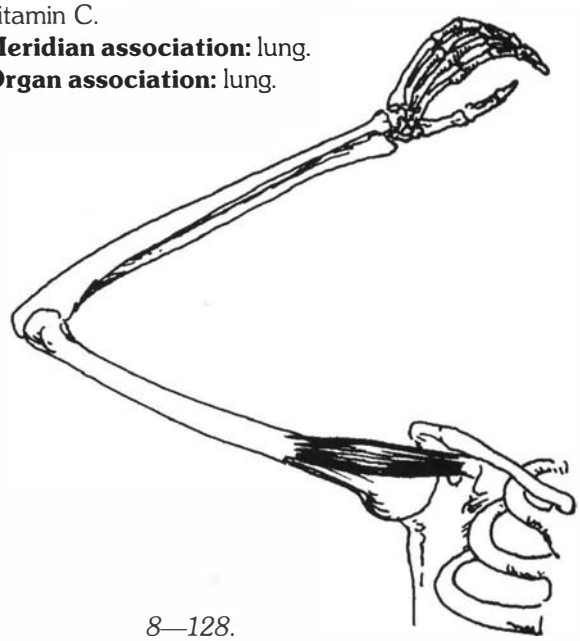
Coracobrachialis

Origin: tip of coracoid process of scapula.
Insertion: middle of medial border of humerus, opposite deltoid tuberosity.
Action: flexion and adduction of arm.
Test: The seated or supine patient flexes and abducts the shoulder with slight lateral rotation. The elbow is held in complete flexion to reduce action of the biceps brachii muscle in the test. Pressure is exerted against the distal part of the humerus in the direction of extension and slight abduction.

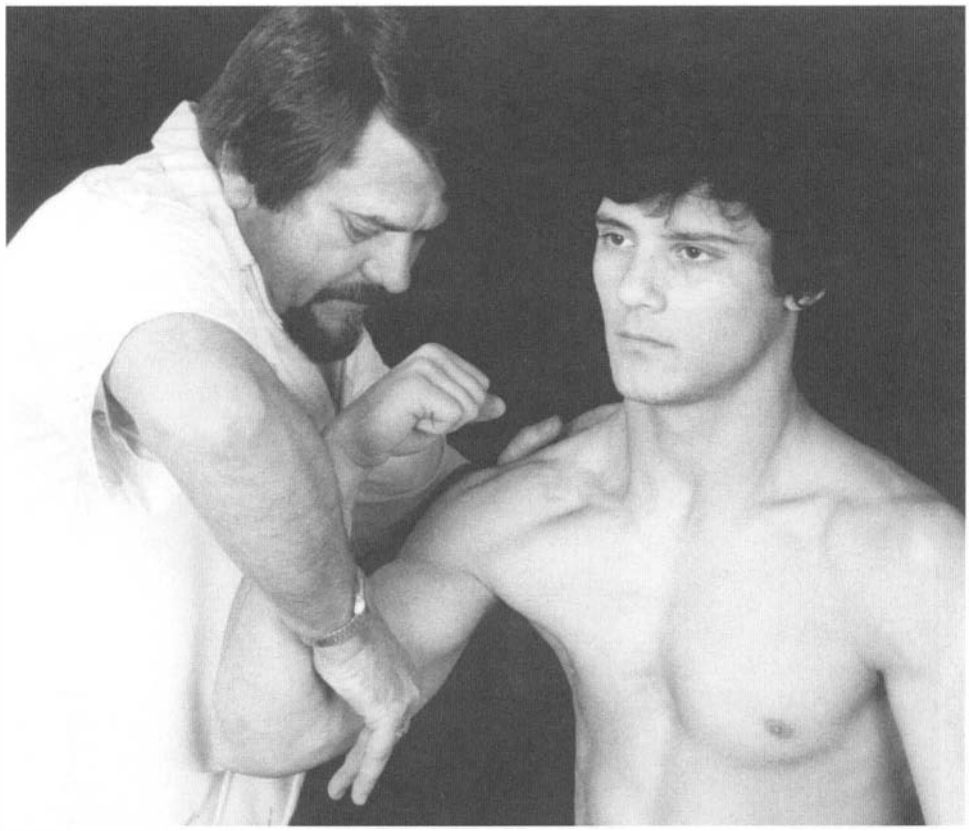
Nerve supply: musculocutaneous, **C6, 7**.
Neurolymphatic:
Anterior: 2nd-4th intercostal spaces near sternum.
Posterior: between T3, 4 near laminae.
Neurovascular: bregma.
Nutrition: lung concentrate or nucleoprotein extract, vitamin C.
Meridian association: lung.
Organ association: lung.







8—128.



8—127.

Biceps Brachii

Origin:

Short head: tip of coracoid process.

Long head: supraglenoid tubercle of the scapula.

Insertion: tuberosity of the radius and lacertus fibrosis, which is a deep aponeurosis continuous with the deep fascia of the flexor muscles of the forearm.

Action: flexes the forearm; supinates the forearm only when the motion is resisted.

Nerve supply: musculocutaneous, **C5, 6**.

Test: The sitting or supine patient flexes the elbow to approximately 75°, with the forearm in supination so that the palm faces upward. The examiner stabilizes at the elbow and directs pressure against the distal forearm to cause elbow extension.

Neurolymphatic:

Anterior: intercostal space 4th-5th ribs, 3" from the sternum.

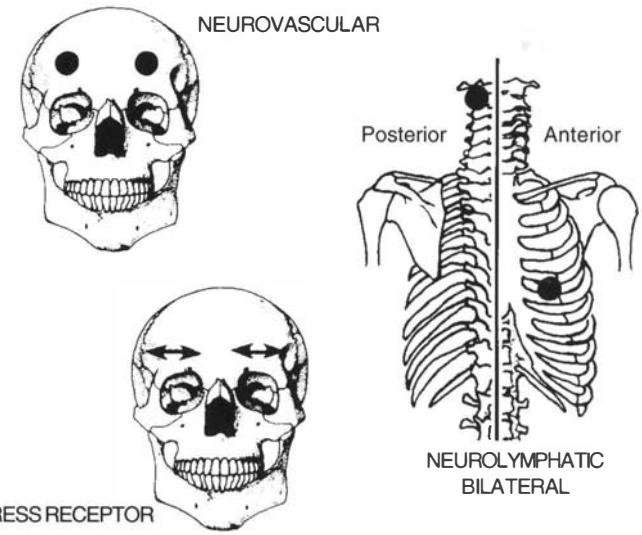
Posterior: C2 laminae.

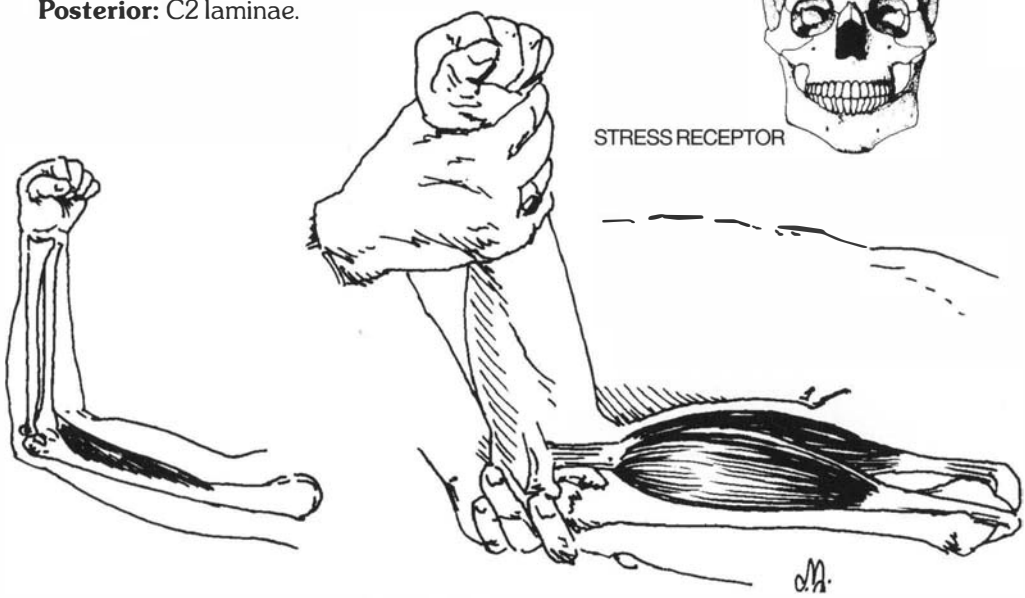
Neurovascular: bilateral frontal bone eminences.

Nutrition: betaine hydrochloride, duodenal concentrate, chlorophyll complex.

Meridian association: stomach.

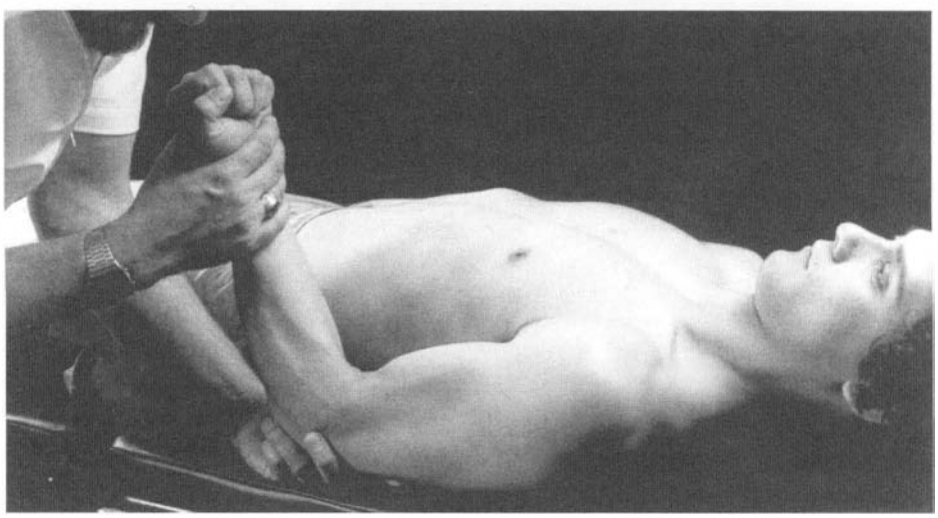
Organ association: stomach.





8—129. Brachialis

8—130. Tests biceps brachii and brachialis.

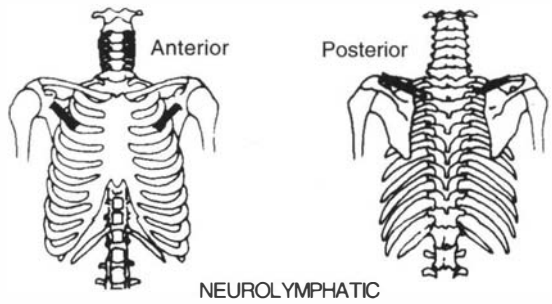


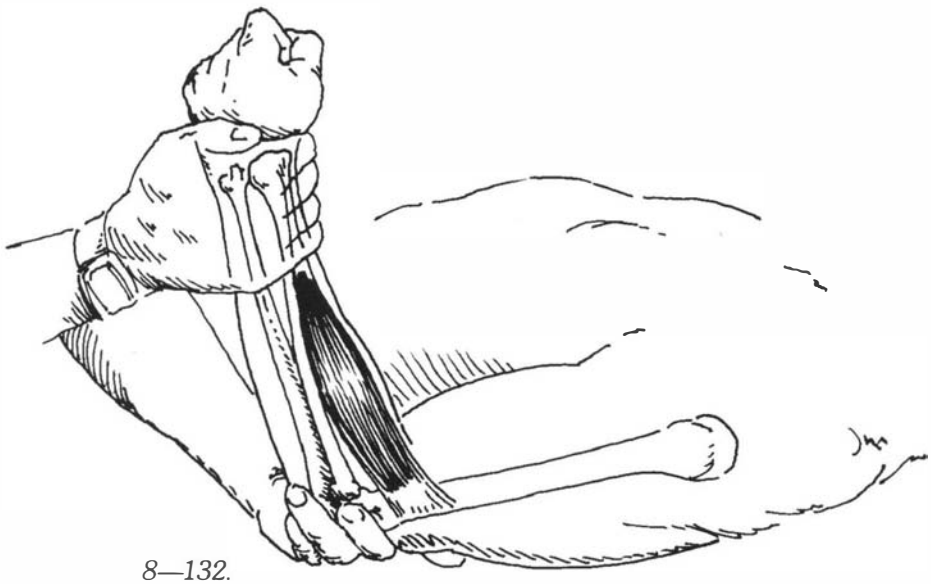
8—131.

Brachioradialis

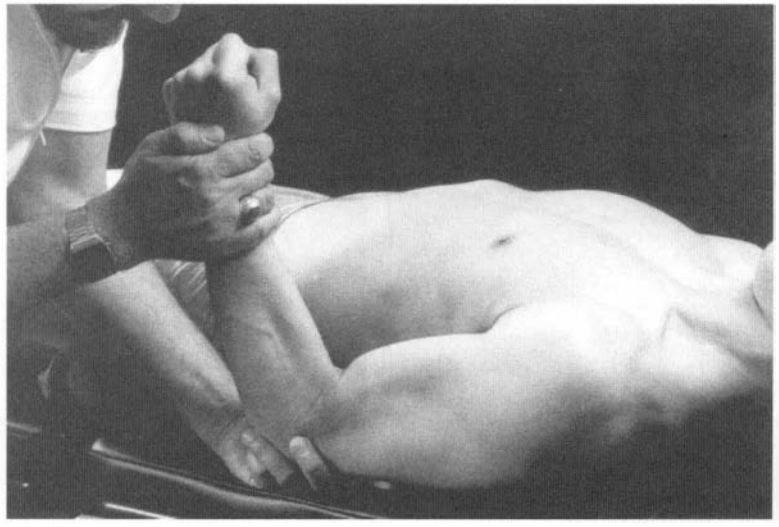
Origin: proximal two-thirds of the lateral supracondylar ridge of humerus and lateral intermuscular septum.
Insertion: lateral side of base of styloid process of radius.
Action: flexes elbow and is exceptionally active on quick bursts of activity. It has been considered that the brachioradialis gives some aid in pronation and supination from the extreme rotated position to the mid-position when acting against resistance.
Test: The seated or supine patient flexes his elbow with the forearm to approximately 75°. The forearm is in neutral rotation, placing the thumb pointing upward. Pressure is directed against the lower forearm in the direction of extension. The brachioradialis belly should be observed for its contraction because of other muscle recruitment.
Nerve supply: radial, **C5, 6**.

Neurolymphatic:
Anterior: over entire pectoralis major, with emphasis over pectoralis minor.
Posterior: over origin of the supraspinatus.
Meridian association: stomach.





8—132.



8—133.

Triceps Brachii

Origin:

Long head: infraglenoid tubercle of scapula.
Lateral head: posterolateral surface of humerus.
Medial head: lower posterior surface of the humerus.

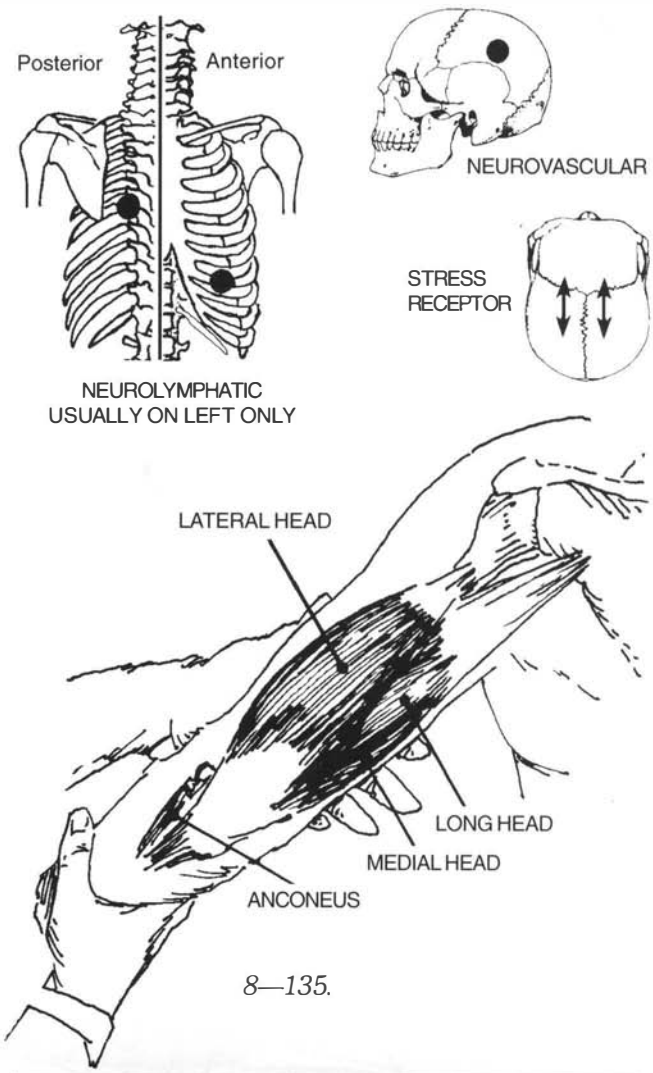
Insertion: upper posterior surface of the olecranon and deep fascia of the forearm.
Action: extends forearm; long head aids in adducting and extending the arm.
Test: The seated, prone, or supine patient abducts and flexes his shoulder and elbow to about 45°. The examiner stabilizes the humerus and directs pressure to the distal forearm to flex the elbow.
Nerve supply: radial, C6, **7, 8**, T1.
Neurolymphatic:

Anterior: 7th intercostal space on left at rib-cartilage junction.
Posterior: between T7, 8 at lamina on left.

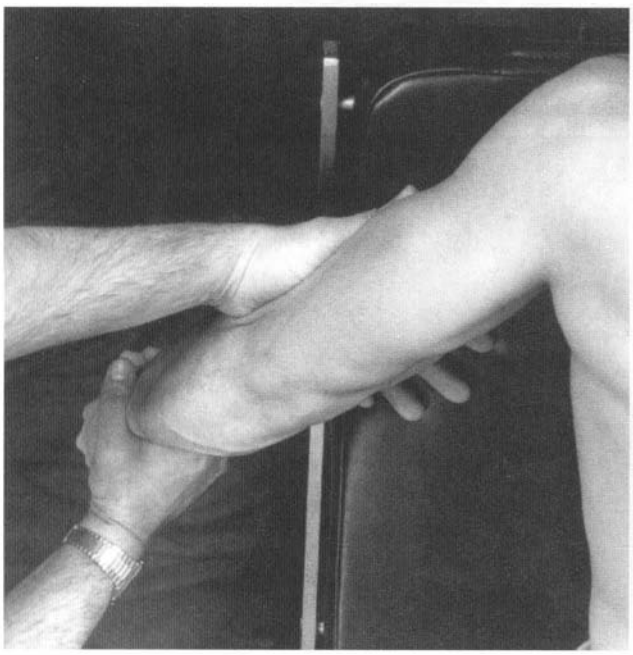
Neurovascular: superior to temporal bone on a vertical line slightly posterior to external auditory meatus.
Nutrition: betaine hydrochloride, pancreas concentrate or nucleoprotein extract, vitamin A.
Meridian association: spleen.
Organ association: pancreas.

Anconeus

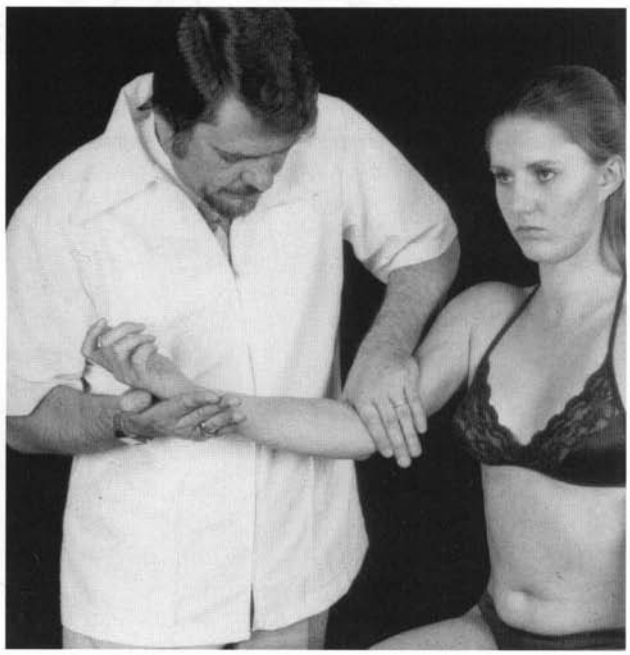
Origin: posterior surface of lateral epicondyle of humerus.
Insertion: lateral side of olecranon and posterior surface of ulna.



8—135.



8—134. Prone triceps test.



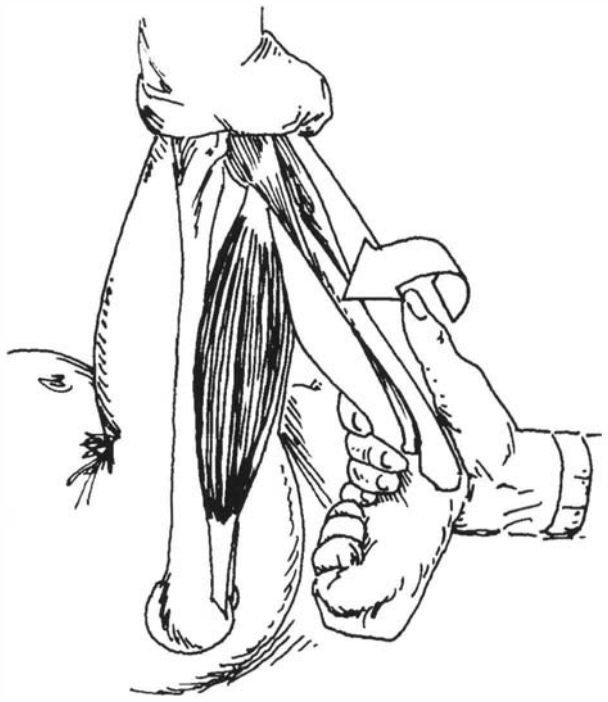
8—136. Seated triceps test.

Supinator

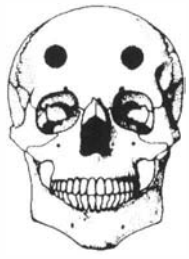
Origin: lateral condyle of humerus, radial collateral ligament of elbow, annular ligament of radius, and supinator crest of ulna.
Insertion: lateral anterior surface of radius on upper one-third.
Action: supinates forearm.
Test: The testing position is designed to place the synergistic biceps brachii at a disadvantage. The patient's elbow is maintained in extension, and the shoulder is extended close to the maximum. Force is directed above the wrist in the direction of pronation. Care should be taken that the examiner's force on the wrist does not cause the patient pain. The examiner must make certain that the patient does not rotate the humerus as the supinator goes from isometric to eccentric contraction.
Nerve supply: radial, C5, **6**.
Neurolymphatic:

Anterior: 6th intercostal space from mamillary line to sternum on left.
Posterior: between T6, 7 near lamina on left. (**Note:** May also be involved with the neurolymphatic for the adductors.)

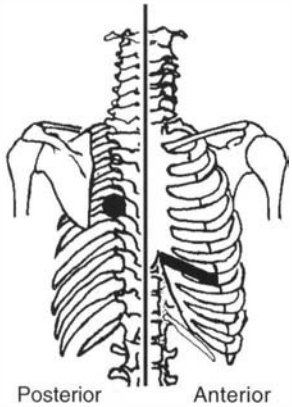
Neurovascular: bilateral frontal bone eminences.
Nutrition: vitamins B, G, and HCl.
Meridian association: stomach.
Organ association: stomach.



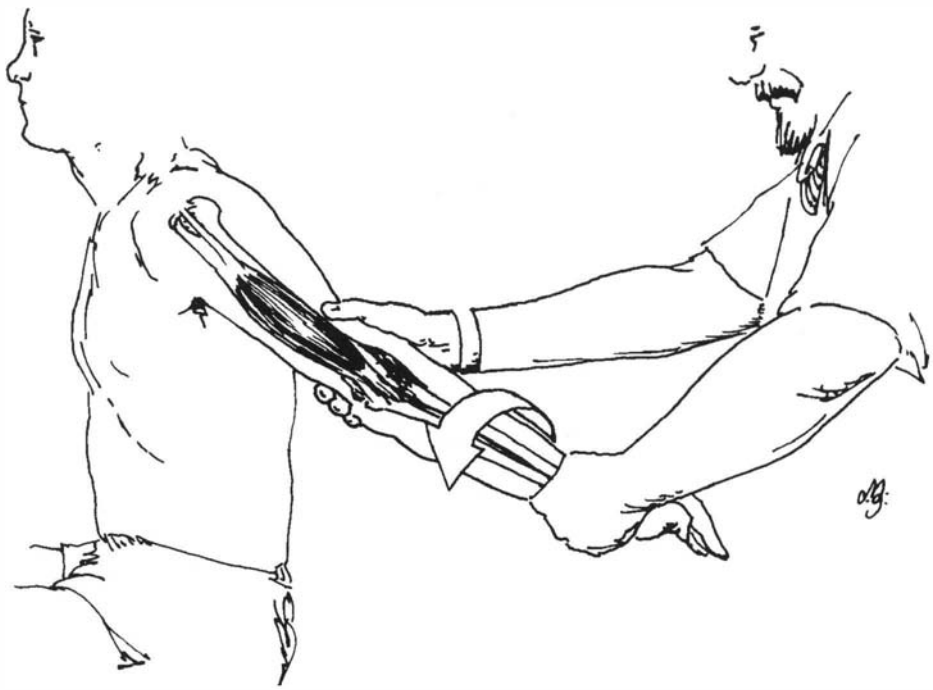
8—138. Alternate test. Full elbow flexion helps take synergistic biceps out of the test.



NEUROVASCULAR



NEUROLYMPHATIC
USUALLY ON LEFT ONLY



8—137. Full elbow extension also helps take the synergistic biceps out of the test.

Pronator Teres

Origin:

Humeral head: from the medial epicondylar ridge and common flexion tendon.

Ulnar head: from medial side of coronoid process of ulna.

Insertion: middle of lateral surface of radius.

Action: pronates forearm and is significant in flexing the elbow.

Test: The supine or seated patient maintains the humerus in adduction, with the elbow flexed to 60° and the forearm in pronation. Force is directed to the distal forearm in a direction of supination. The examiner must take care that the force applied does not cause pain at the contact point.

Nerve supply: median, **C6, 7**.

Neurolymphatic:

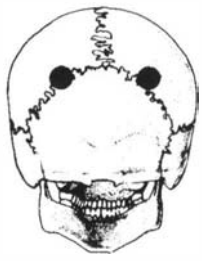
Anterior: behind areola.

Posterior: below inferior angle of scapula.

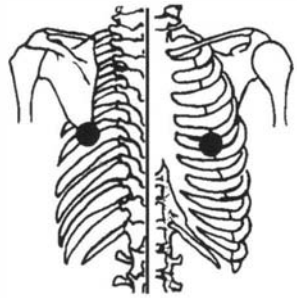
Neurovascular (provisional): on lambdoidal suture midway between lambda and asterion.

Meridian association: stomach.

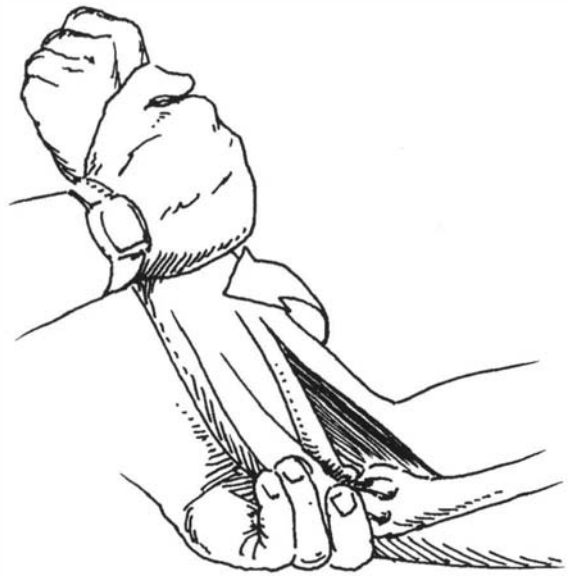
Organ association: stomach.



NEUROVASCULAR



NEUROLYMPHATIC
BILATERAL



8—139. Examiner's force is in the direction of the arrow.

Pronator Quadratus

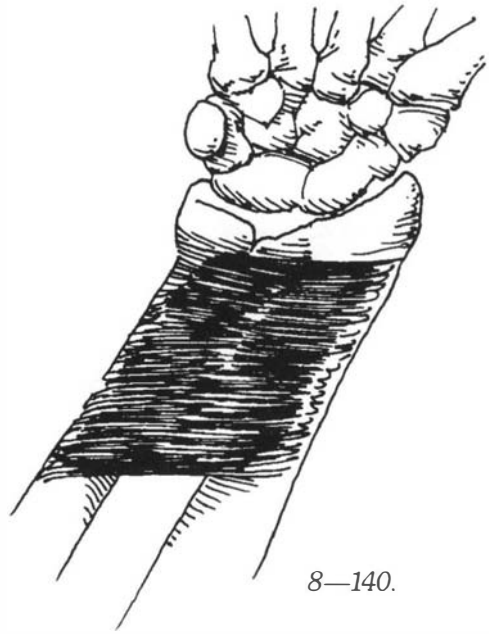
Origin: distal one-fourth of the volar surface of ulna.

Insertion: distal one-fourth of the lateral border, volar surface of the radius.

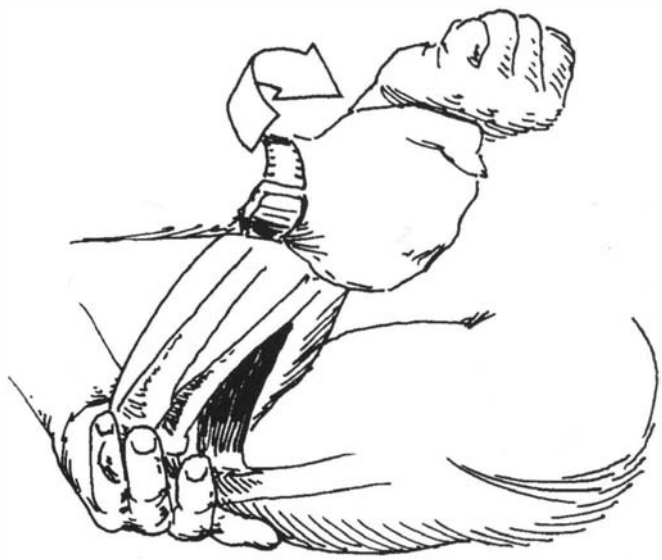
Action: pronates the forearm.

Test: The seated or supine patient maximally flexes his elbow, with the forearm in pronation. The elbow flexion shortens the pronator teres, which somewhat takes out its synergism. A rotational force is directed above the wrist to supinate the forearm. The results of the test must be compared with the pronator teres test.

Nerve supply: median, C7, **8, T1**.



8—140.



8—141. Examiner's force is in the direction of the arrow.

Opponens Pollicis

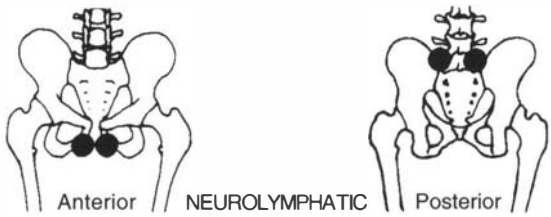
Origin: flexor retinaculum and tubercle of trapezium bone.
Insertion: radial side of entire length of 1st metacarpal bone.
Action: flexes and abducts the 1st metacarpal with slight internal rotation. Abduction means to move the metacarpophalangeal articulation away from the palm.
Test: The patient places the 1st metacarpal into flexion, adduction, and slight internal rotation. The metacarpophalangeal articulation should be kept in extension during the test. Pressure is applied against the distal end of the 1st metacarpal in a direction of extension, abduction, and external rotation.
Nerve supply: median, **C6, 7**.
Neurolymphatic (provisional):

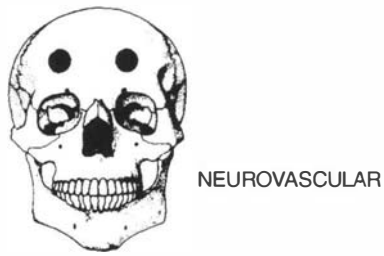
Anterior: inferior to the symphysis pubis at the height of the obturator (same as peroneus longus and brevis).

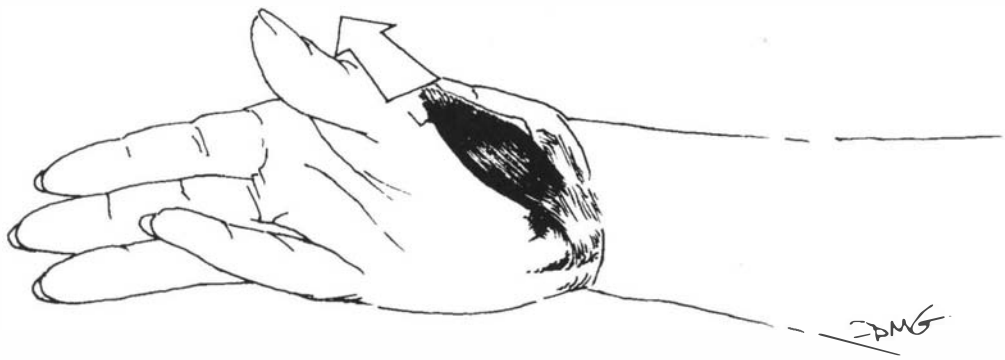
Posterior: between posterior superior iliac spine and L5 spinous process.

Neurovascular (provisional): bilateral frontal bone eminences.

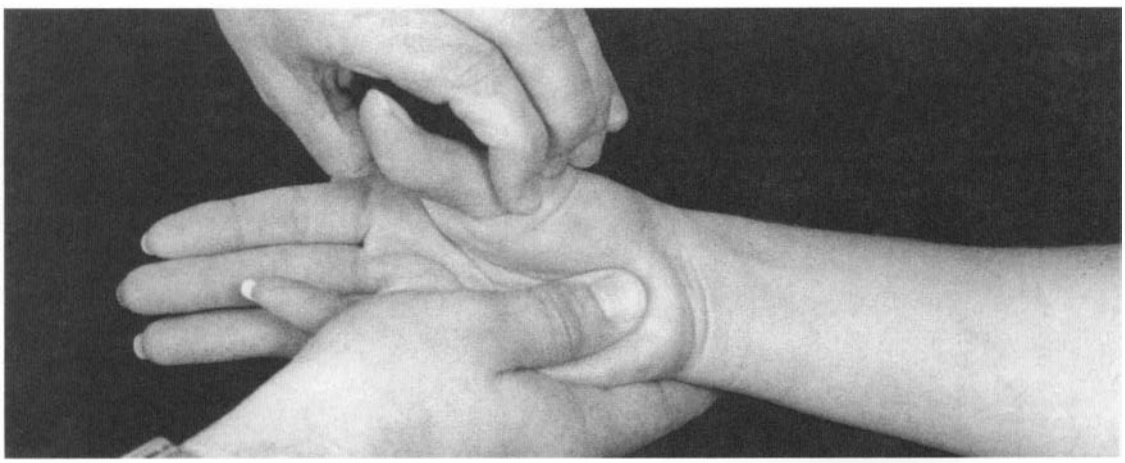
Nutrition: raw bone concentrate or nucleoprotein extract when the origin and insertion or proprioceptors are involved, either directly with the muscle or with a carpal tunnel syndrome.
Meridian association: stomach.
Organ association (provisional): stomach.







8—142.



8—143.

Opponens Digiti Minimi

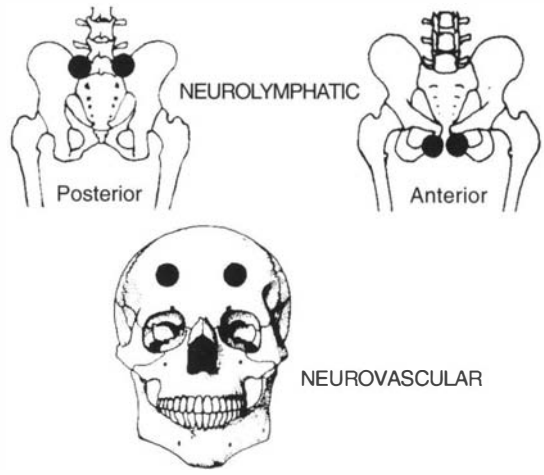
Origin: hamulus of the hamate bone and flexor retinaculum.
Insertion: shaft of the 5th metacarpal on the ulnar side.
Action: flexes and slightly rotates the 5th metacarpal; brings the ulnar portion of the hand into position so that the little finger and thumb can approximate each other; helps cup the palm of the hand.
Test: The patient brings the 5th metacarpal into a position of flexion and slight rotation to cup the palm. Pressure is directed against the palmar surface of the 5th metacarpal head in a direction of extension in an effort to flatten the cupped hand.
Nerve supply: ulnar, C7, **8**, **T1**.
Neurolymphatic (provisional):

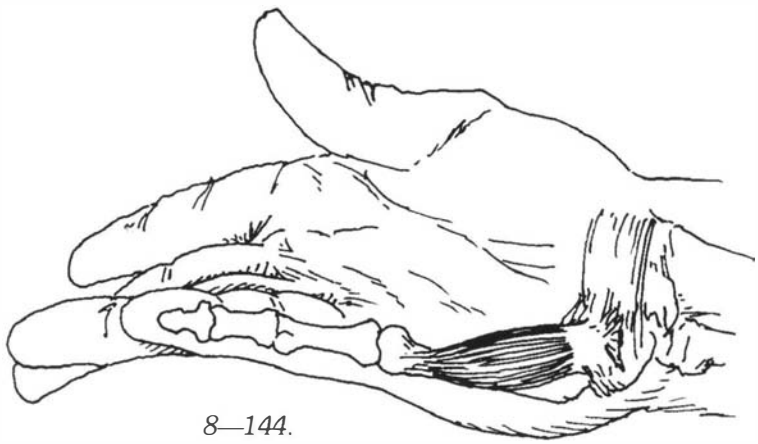
Anterior: inferior to the symphysis pubis at the height of the obturator (same as peroneus longus and brevis).

Posterior: between PSIS and L5 spinous process.

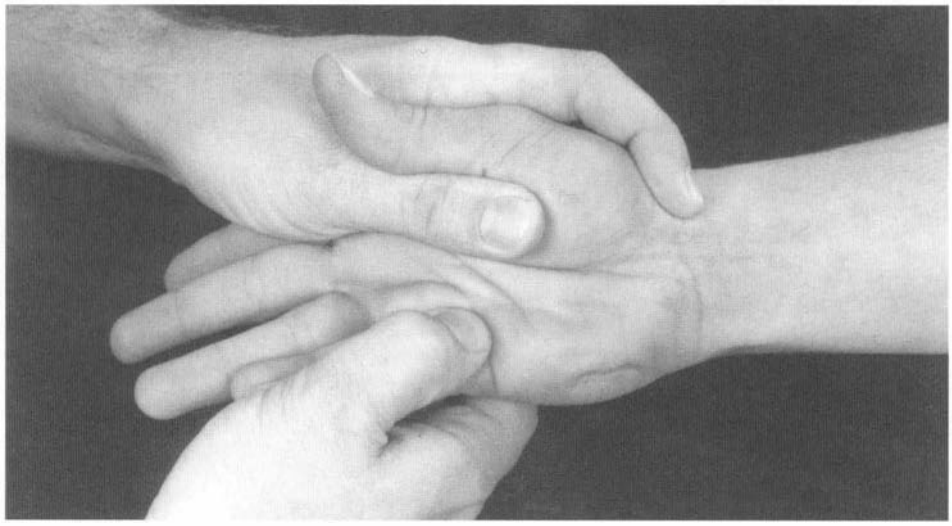
Neurovascular (provisional): bilateral frontal bone eminences.

Nutrition: When weakness is due to pisiform hamate syndrome, raw bone concentrate or nucleoprotein extract may be necessary for ligament involvement.
Meridian association: stomach.





8—144.



8—145. Test pressure is directed to the 5th metacarpal in the direction of extension.

Sternocleidomastoid

Origin:

Sternal head: anterior surface of the manubrium.

Clavicular head: upper surface of the medial half of the clavicle.

Insertion: lateral surface of the mastoid process of the temporal bone, and lateral half of the superior nuchal line of the occiput.

Action: acting unilaterally, draws head toward the ipsilateral shoulder and rotates head to opposite side; acting bilaterally, flexes head.

Test: The supine patient places the hands above the head by shoulder abduction and elbow flexion to avoid pressing against the table during the test. The patient rotates his head away from the muscle to be tested and lifts it from the table. Pressure is applied against the temporal area in a posterolateral direction. The examiner should observe for the patient's attempts to turn his head medially, recruiting more synergistic action of the scalene group and other neck flexors.

Nerve supply: anterior rami of C2, 3; spinal portion of the accessory nerve (cranial XI).

Neurolymphatic:

Anterior: 1st intercostal space, 3-1/2" from the sternum.

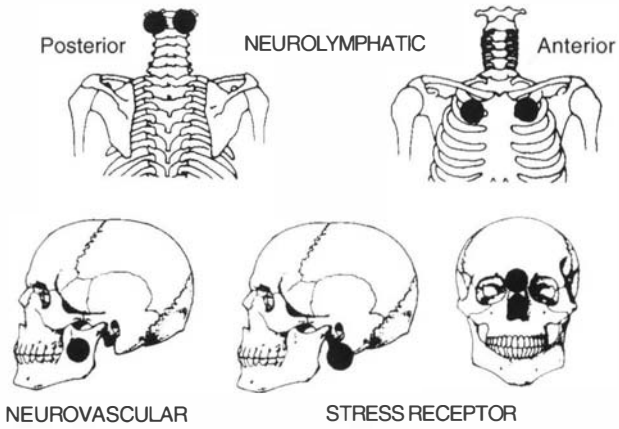
Posterior: laminae of C2.

Neurovascular: ramus of jaw below zygoma.

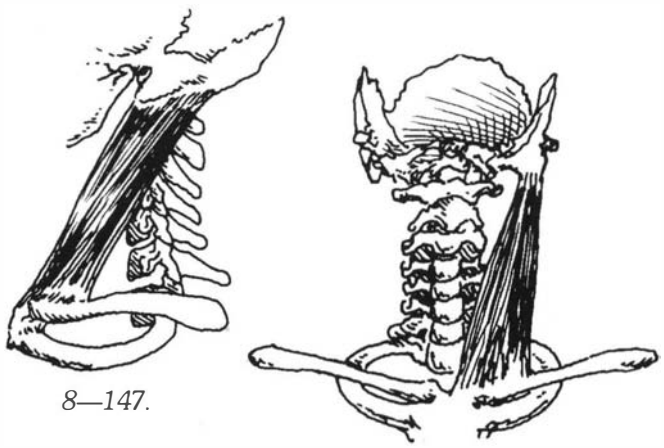
Nutrition: niacinamide or niacin and vitamin B_6. For sinusitis, organic iodine may be needed.

Meridian association: stomach.

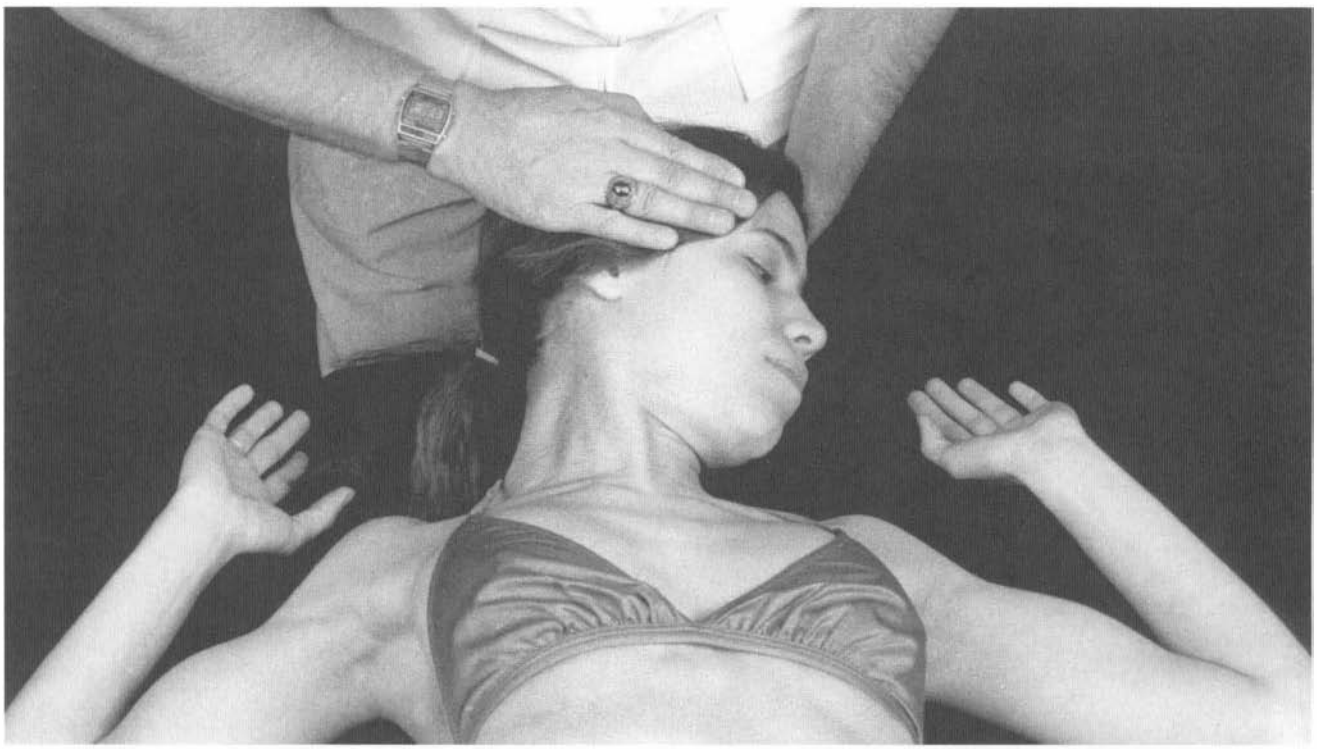
Organ association: sinuses.



8—147.



8—148.

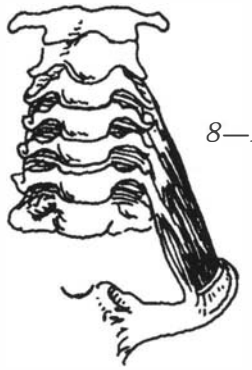


8—146. The patient must maintain maximum head rotation to prevent recruitment of the deep neck flexors.

Neck Flexors (Medial)

Scalenus Anticus

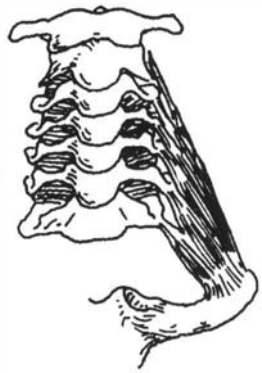
Origin: anterior tubercles of the transverse processes of the 2nd-6th cervical vertebrae.
Insertion: scalene tubercle on upper surface of the 1st rib.
Action: flexes and rotates cervicals; raises 1st rib.
Nerve supply: anterior branches, C5, 6, 7, 8.



8—149. Scalenus anticus

Scalenus Medius

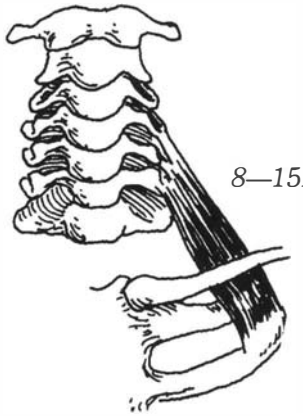
Origin: posterior tubercles of the transverse processes of 2nd-7th cervical vertebrae.
Insertion: upper surface of the 1st rib behind subclavian groove.
Action: flexes and rotates cervical vertebrae; raises 1st rib.
Nerve supply: posterior branches of anterior primary rami of C3, 4; lateral muscular branches of C3, 4.



8—150. Scalenus medius

Scalenus Posticus

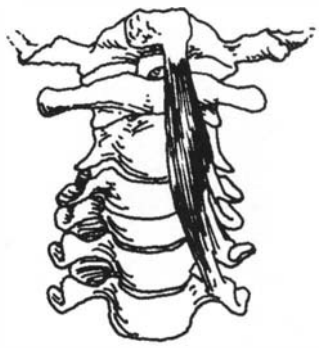
Origin: posterior tubercles of transverse processes of 4th, 5th, and 6th cervicals.
Insertion: outer surface of 2nd rib behind attachment of serratus anticus.
Action: flexes and rotates cervical vertebrae; raises 2nd rib. **Note:** All scalenes, when acting bilaterally, flex the neck.
Nerve supply: posterior branches of C5-8; lateral muscular branches of C3, 4.



8—151. Scalenus posticus

Longus Capitis

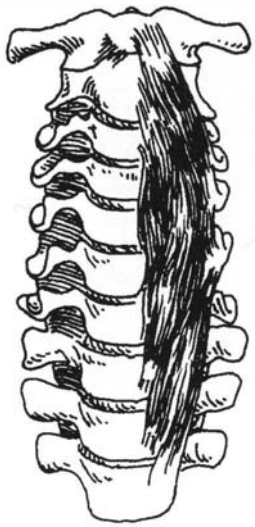
Origin: anterior tubercles of transverse processes of the 3rd-6th cervical vertebrae.
Insertion: inferior surface of the basilar portion of the occiput.
Action: flexes cervical vertebrae and head; unilaterally rotates and flexes cervical vertebrae and head.
Nerve supply: muscular branches of C1-4.



8—152. Longus capitis

Longus Colli

Origin: from bodies of the first three thoracic and last three cervical vertebrae, with slips from other areas.
Insertion: bodies of 2nd, 3rd, and 4th cervicals with slips to other areas.
Action: flexes cervical vertebrae unilaterally; assists in rotation and lateral flexion.
Nerve supply: anterior primary rami of C2-8.



8—153. Longus colli

Neck Flexors (Medial)

Test: The supine patient places his hands above his head by shoulder abduction and elbow flexion. He lifts his head from the table by neck flexion and rotates it 10° away from the side being tested. The examiner uses the ulnar edge of his hand, pressing against the forehead in the direction of neck extension directly toward the table and not in alignment with the 10° rotation of the patient's head. The edge of the hand gives better directional force; it reduces the patient's ability to work rotational factors into the test against the examiner's flat hand. Observation should be made for the patient's attempt to rotate his head, recruiting more activity from synergists. The patient should also be prevented from laterally tilting his head.

Neurolymphatic:

Anterior: 1st intercostal space 3-1/2" from sternum.

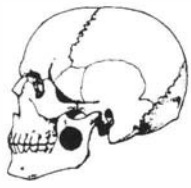
Posterior: laminae C2.

Neurovascular: ramus of jaw below zygoma.

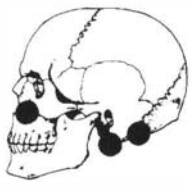
Nutrition: vitamin B_6, niacinamide or niacin.

Meridian association: stomach.

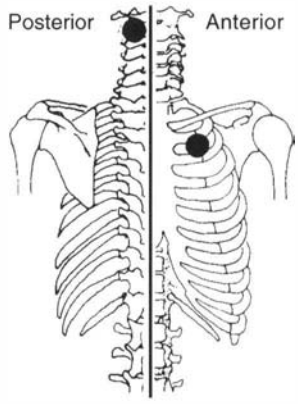
Organ/gland association: sinuses.



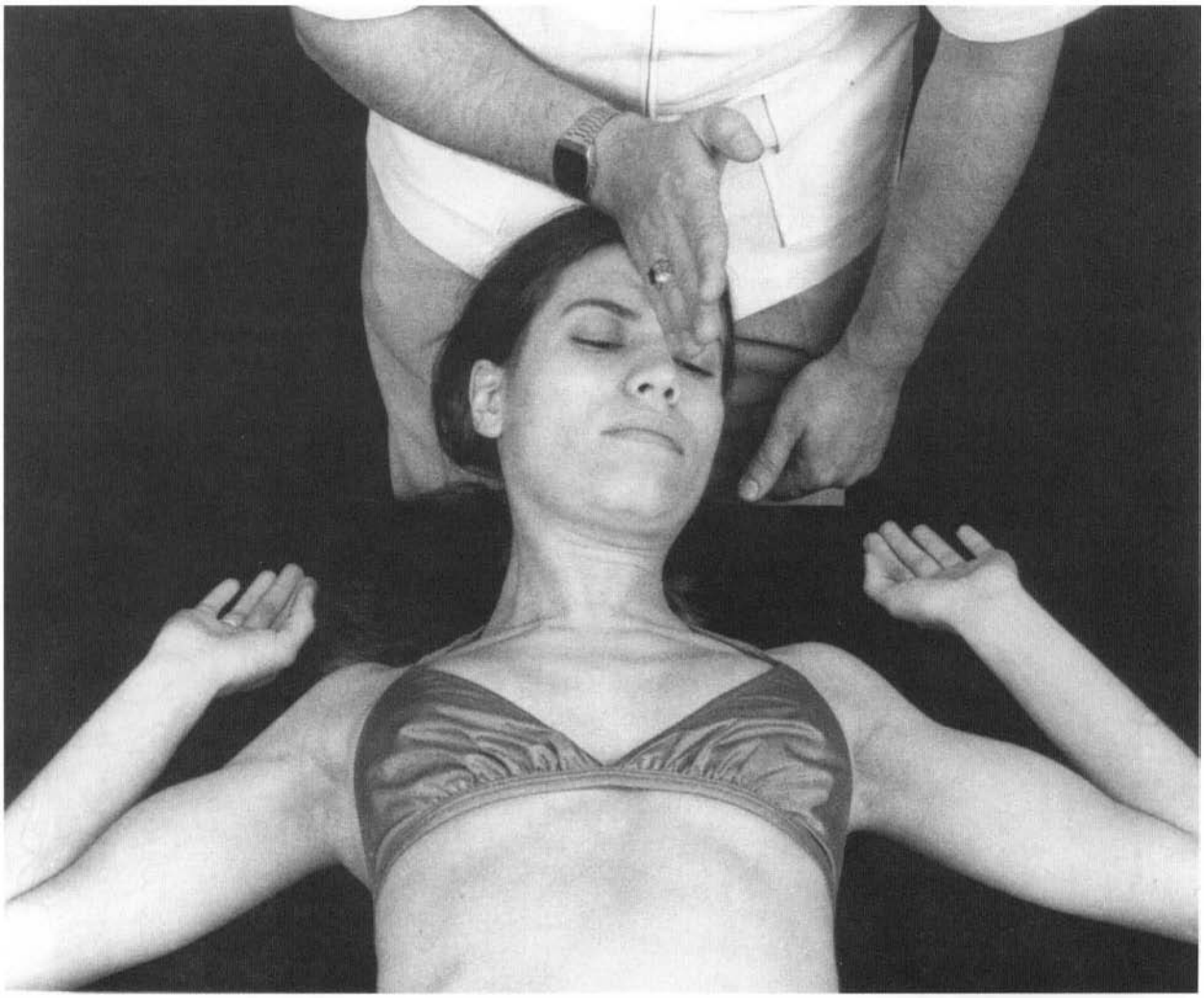
NEUROVASCULAR



STRESS RECEPTOR



NEUROLYMPHATIC
BILATERAL



8—154. Head turned 10° away from the side tested. Do not let patient rotate head.

Neck Extensors (Deep)

Splenius Capitis

Origin: spinous processes of C7-T3, lower half of ligamentum nuchae.
Insertion: mastoid process and lateral portion of superior nuchal line.
Action: extends, laterally flexes, and rotates head and neck.
Nerve supply: middle cervical spinal nerves.

Splenius Cervicis

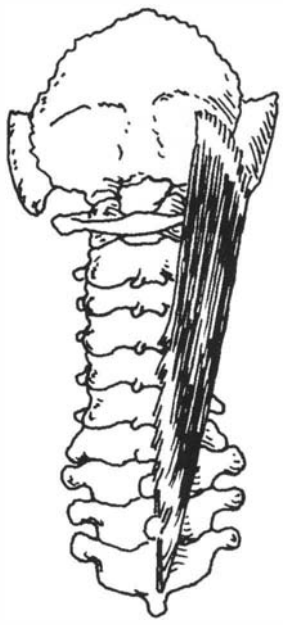
Origin: spinous processes of 3rd-6th thoracic vertebrae.
Insertion: upper three or four cervical vertebrae, transverse processes on the posterior aspect.
Action: extends, laterally flexes, and rotates neck.
Nerve supply: lower cervical spinal nerves.

Semispinalis Capitis

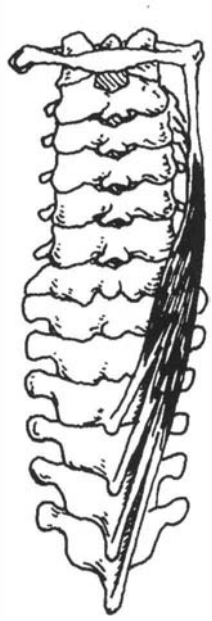
Origin: 7th cervical and 1st-6th thoracic transverse processes and articular processes of 4th, 5th, and 6th cervical vertebrae.
Insertion: between superior and inferior nuchal lines of the occiput.
Action: extension and lateral flexion of the neck and head.
Nerve supply: spinal nerves C1-6.

Semispinalis Cervicis

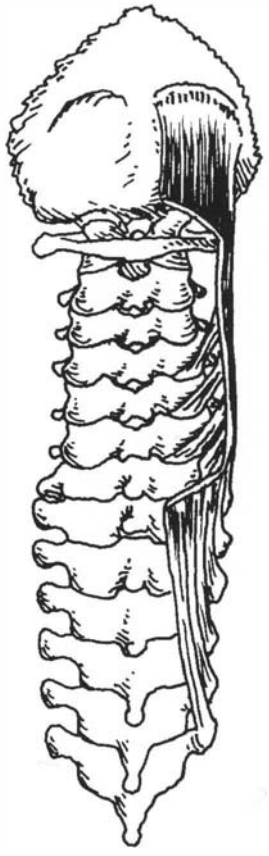
Origin: transverse processes of upper six thoracic vertebrae.
Insertion: spinous processes of 2nd-5th cervical vertebrae.
Action: extension and lateral flexion of neck and head.
Nerve supply: spinal nerves C6, 7, 8.



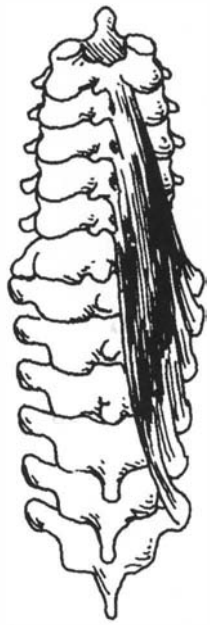
8—155. Splenius capitis



8—156. Splenius cervicis



8—157. Semispinalis capitis



8—158. Semispinalis cervicis

Neck Extensors (Deep)

Test: The prone patient flexes the shoulders and elbows to place the hands above the head so they do not contact the table. The neck and head are rotated to the side of test. Pressure is directed against the posterolateral aspect of the head in a direction toward the table. The bilateral neck extensors can be tested with no head rotation. The bilateral test is used as an indication for functional fixation of the lumbar spine.

Neurolymphatic:

Anterior: 1st intercostal space 3-1/2" from sternum.

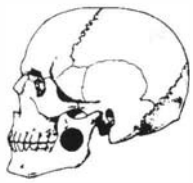
Posterior: C2 laminae.

Neurovascular: ramus of jaw below zygoma.

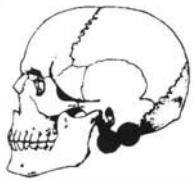
Nutrition: vitamin B_6, niacinamide, organic iodine.

Meridian association: stomach.

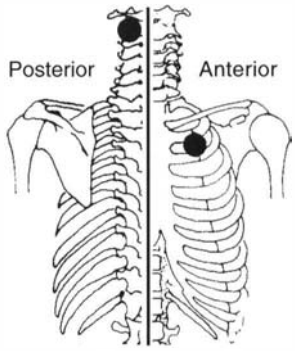
Organ association: sinuses.



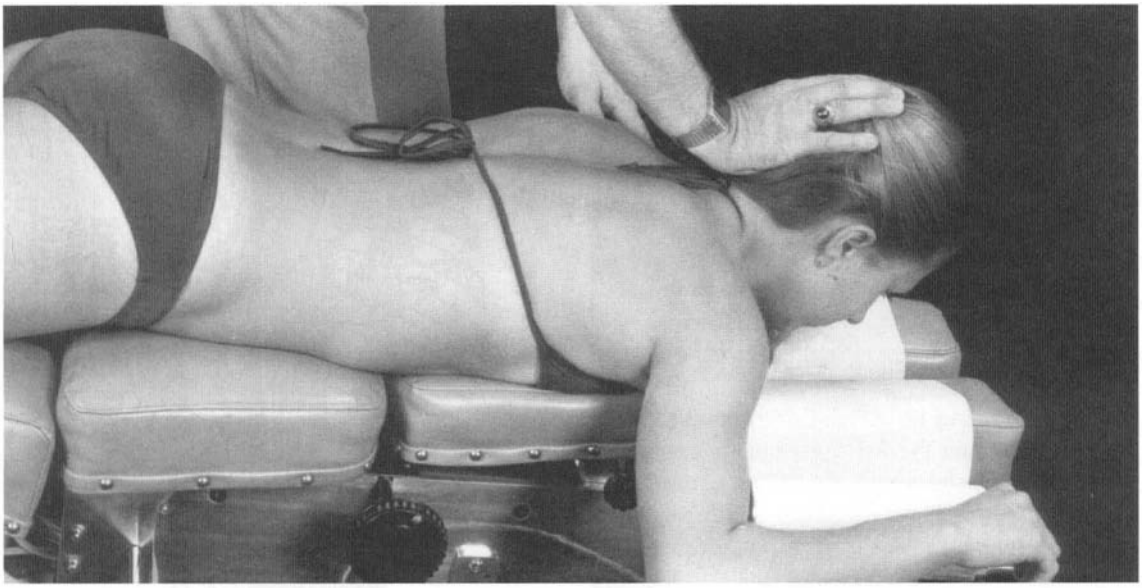
NEUROVASCULAR



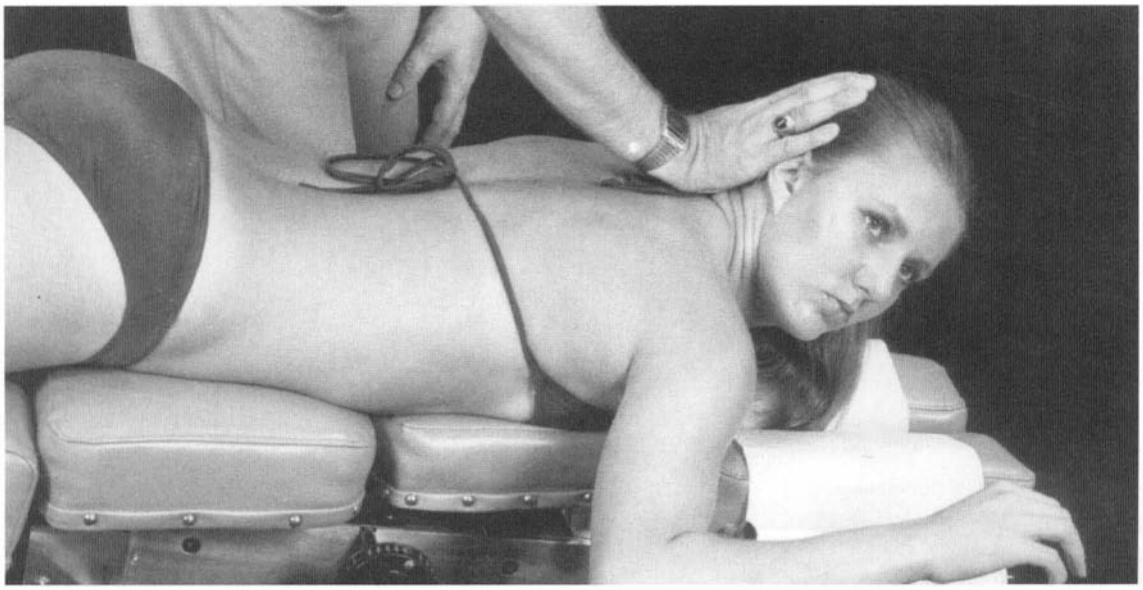
STRESS RECEPTOR



NEUROLYMPHATIC
BILATERAL



8—159. Neck extensors tested bilaterally. Hands are not in contact with the table.



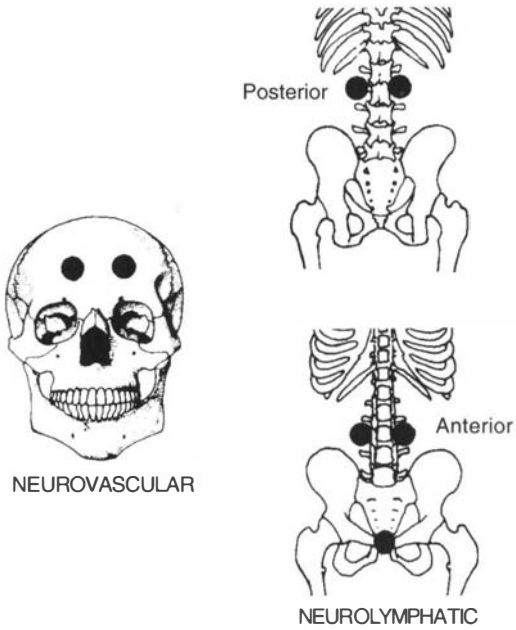
8—160. Neck extensors tested unilaterally.

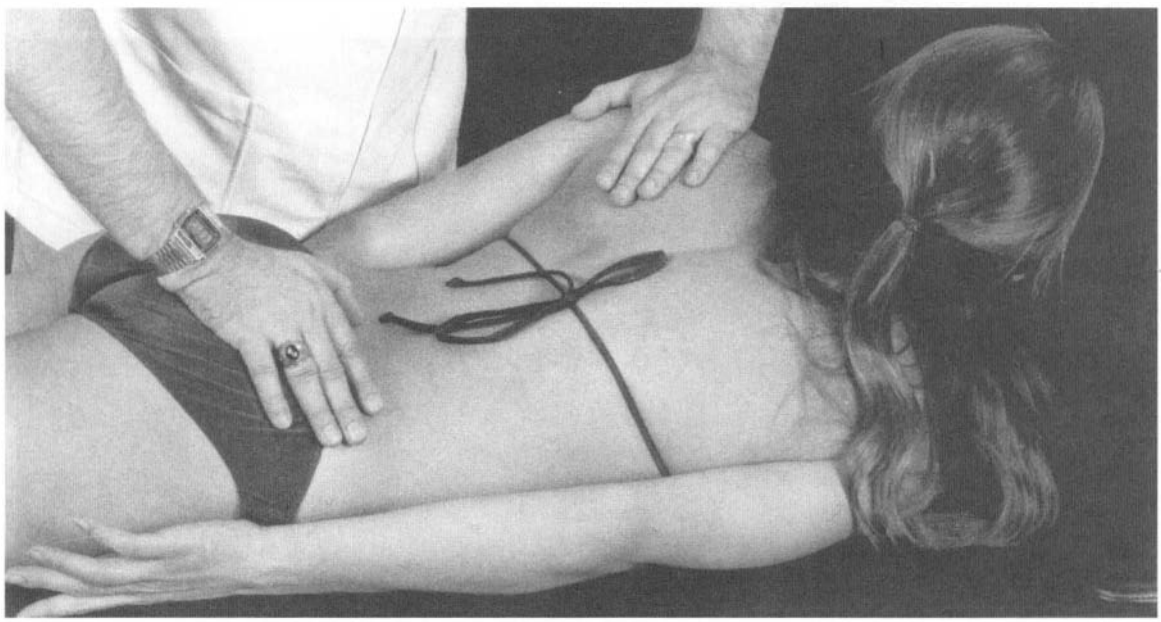
Sacrospinalis (as a group)

Origin: separate slips of muscle arising from the sacrum, crest of the ilium, spinous processes, transverse processes, and ribs.
Insertion: into the ribs, transverse processes, spinous processes, and occiput.
Action: extension, lateral flexion, and rotation of vertebral column; lateral movement of pelvis.
Testing and evaluation: Observation of the patient in Adam's position and while sitting, standing, or prone gives the examiner some indication of the muscles' function by bilaterally comparing for apparent tone or flaccidity. Specific sections of the sacrospinalis can be tested directly with manual muscle testing.[3,57] In all cases, there is considerable synergism and overlap of muscle function. A general test is for the patient to laterally flex the spinal column while the examiner observes for range of motion bilaterally.

The patient laterally flexes the trunk, reaching down the leg as far as possible to indicate lateral flexion ability. The examiner observes how far the hand will travel down the leg, and repeats the test on the opposite side for comparison. The patient will be able to flex laterally farther on the side opposite general sacrospinalis weakness. Care must be taken to evaluate other muscles that restrict lateral flexion capability, such as the quadratus lumborum, psoas, abdominals, and latissimus dorsi. Also, spinal curvatures that may influence lateral flexion should be considered.

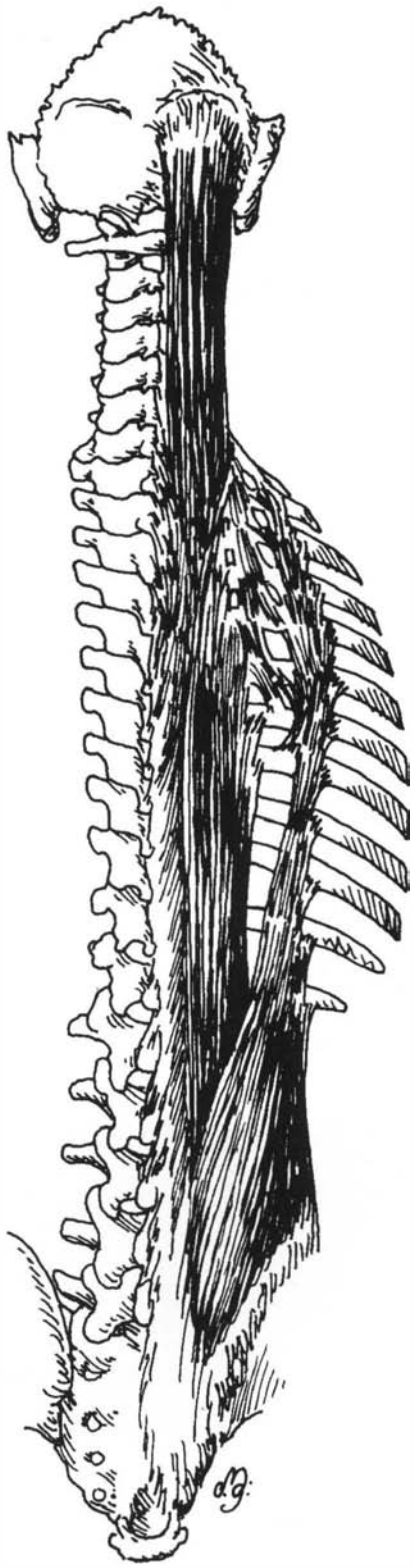
Alternate testing methods: The prone patient hyperextends and rotates the spine without the aid of the arms. The examiner, stabilizing the pelvis against the table, directs pressure against the thorax on the side of rotation toward flexing the spine. The patient must be capable of adequately fixing the pelvis to the legs. This is a general test for the sacrospinalis, and synergistic muscles must be taken into consideration.
Neurolymphatic:
Anterior: over the symphysis pubis and lateral to the umbilicus.
Posterior: L2 transverse process.
Neurovascular: bilateral frontal bone eminences.
Nutrition: vitamins A, C, P, E, and calcium.
Meridian association: bladder.
Organ association: urinary bladder.



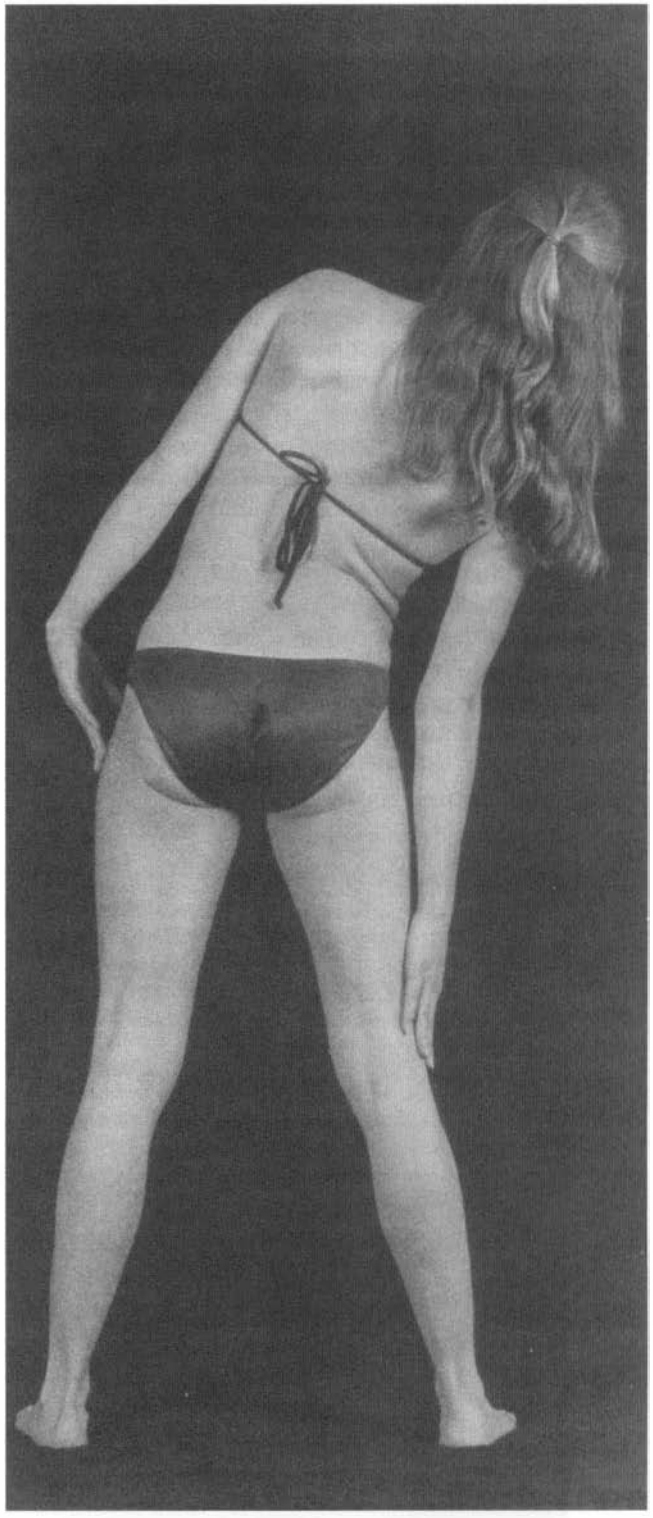


8—161. Prone sacrospinalis general test.

Sacrospinalis



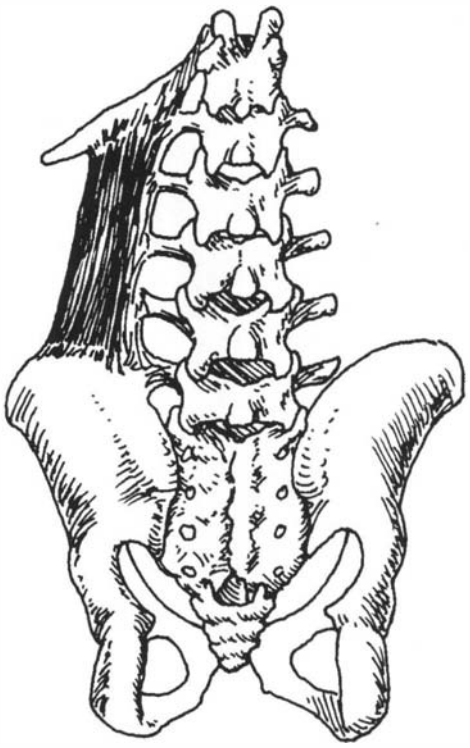
8—162. Portions of the sacrospinalis are hidden by overlying muscles.



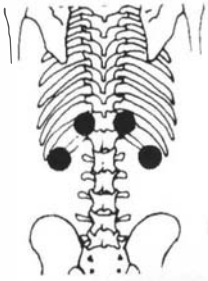
8—163. The patient will laterally flex farther toward the side opposite general sacrospinalis weakness. A similar test can be done in the seated position.

Quadratus Lumborum

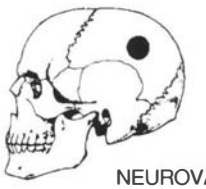
Origin: iliolumbar ligament, posterior part of the iliac crest.
Insertion: inferior border of the last rib and transverse processes of the upper four lumbar vertebrae.
Action: laterally flexes lumbar vertebral column; depresses last rib; helps action of the diaphragm in inspiration.
Test: The supine patient laterally flexes the pelvis in relation to the trunk. The legs stay in alignment with the pelvis to be 10° from the center line of the patient's trunk. The legs are used as levers to impart motion to the pelvis. The examiner contacts the patient's ankles by reaching under the legs. The test pressure is directed to bring the legs to the center line of the table. The examiner must observe for separation of the crest of the ilium and the thoracic cage, indicating a failure of the quadratus lumborum to hold the pelvis in a laterally flexed position with the lumbar spine.
Nerve supply: lumbar plexus T12, **L1, 2, 3**.
Neurolymphatic:
Posterior (two): at end and upper edge of the 12th rib, laminae of T11.
Neurovascular: on parietal eminence, posterior aspect.
Nutrition: vitamins E, C, and A.
Meridian association: large intestine.
Organ association: appendix.



8—165. Quadratus lumborum.

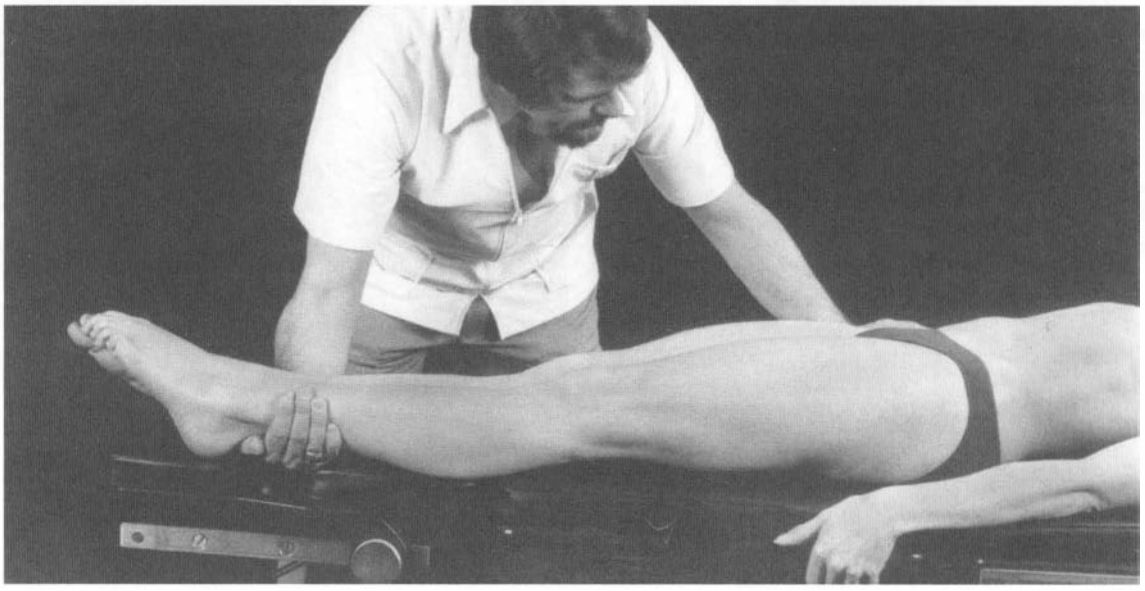


NEUROLYMPHATIC



NEUROVASCULAR

STRESS RECEPTOR



8—164. Quadratus lumborum.

CHAPTER 8 — REFERENCES

1. AMA, *Guides to the Evaluation of Permanent Impairment*, 4th ed (Chicago: AMA, 1993).
2. Basmajian, J.V., & J.F. Lovejoy, "Functions of the popliteus muscle in man," *J Bone Joint Surg*, Vol 53A, No 3 (Apr 1971).
3. Beardall, A.G., *Clinical Kinesiology, Vol I: Low Back and Abdomen*, ed T.W. Brown (Lake Grove, OR: privately published, 1980).
4. Beardall, A.G., *Clinical Kinesiology, Vol II: Pelvis and Thigh*, ed T.W. Brown (Lake Grove, OR: privately published, 1981).
5. Beardall, A.G., *Clinical Kinesiology, Vol V: Muscles of the Lower Extremities — Calf and Foot* (Lake Oswego, OR: privately published, 1985).
6. Beasley, W.C., "Instrumentation and equipment for quantitative clinical muscle testing," *Arch Phys Med Rehabil*, Vol 37 (Oct 1956).
7. Bigland, B., & O.C.J. Lippold, "The relation between force, velocity and integrated electrical activity in human muscles," *J Physiol*, Vol 123 (1954).
8. Blaich, R.M., "Manual and machine muscle testing before and after correction of 'respiratory faults,'" in *Selected Papers of the International College of Applied Kinesiology*, ed B.C. Markham (Detroit, MI: ICAK, 1981).
9. Blaich, R.M., & E.I. Mendenhall, "Manual muscle testing and Cybex machine muscle testing, a search for a correlation," in *1984 Selected Papers of the International College of Applied Kinesiology*, ed W.H. Schmitt, Jr. (Park City, UT: ICAK, 1984).
10. Bohannon, R.W., "Test-retest reliability of hand-held dynamometry during a single session of strength assessment," *Phys Ther*, Vol 66, No 2 (Feb 1986).
11. Brunnstrom, S., "Muscle group testing," *Physiother Rev*, Vol 21, No 1 (Jan/Feb 1941).
12. Calderon, L., & A.P. Lawson, "Interexaminer reliability of applied kinesiology manual muscle testing," *Chiro Sports Med*, Vol 8, No 4 (Nov 1994).
13. Chapman, A.E., & A.Y. Belanger, "Electromyographic methods of evaluating strength training," *Electromyogr Clin Neurophysiol*, Vol 17 (1977).
14. Conable, K.M., & B.T. Hanicke, "Inter-examiner agreement in applied kinesiology manual muscle testing," in *1987 Selected Papers of the International College of Applied Kinesiology*, ed D.H. Schusterman (Shawnee Mission, KS: ICAK, 1987).
15. Cybex, a division of Lumex, Inc., 2100 Smithtown Ave., Ronkonkoma, NY 11779-0903.
16. Elkus, R., & J.V. Basmajian, "Endurance in hanging by the hands. Why do people hanging by their hands let go?" *Am J Phys Med*, Vol 52, No 3 (1973).
17. Goodheart, G.J., Jr., *Applied Kinesiology 1975 Workshop Procedure Manual*, 11th ed (Detroit: privately published, 1975).
18. Goodheart, G.J., Jr., *Applied Kinesiology 1979 Workshop Procedure Manual*, 15th ed (Detroit: privately published, 1979).
19. Gowitzke, B.A., & M. Milner, *Scientific Bases of Human Movement*, 3rd ed (Baltimore: Williams & Wilkins, 1988).
20. Halpern, A.A., & E.E. Bleck, "Sit-up exercises: An electromyographic study," *Clin Orthop*, No 143 (Nov/Dec 1979).
21. Hoggan Health Industries, Inc, 111 East 12300 South, P.O. Box 957, Draper, UT 84020.
22. Hoppenfeld, S., *Physical Examination of the Spine and Extremities* (New York: Appleton-Century-Crofts, 1976).
23. Hoppenfeld, S., *Orthopaedic Neurology — A Diagnostic Guide to Neurologic Levels* (Philadelphia: J.B. Lippincott Co, 1977).
24. Jacobs, G.E., "Applied kinesiology: An experimental evaluation by double blind methodology," *J Manip Physiol Ther*, Vol 4, No 3 (Sep 1981).
25. Kendall, F.P., & E.K. McCreary, *Muscles — Testing and Function*, 3rd ed (Baltimore: Williams & Wilkins, 1983).
26. Kendall, H., F. Kendall, & D. Boynton, *Posture and Pain* (Baltimore: Williams & Wilkins, 1952).
27. Lawson, A., & L. Calderon, "Interexaminer agreement for applied kinesiology manual muscle testing," *Perceptual and Motor Skills*, Vol 84 (1997).
28. Leisman, G. et al., "Electromyographic effects of fatigue and task repetition on the validity of estimates of strong and weak muscles in applied kinesiology," *Perceptual and Motor Skills*, Vol 80 (1995).
29. Leisman, G., P. Shambaugh, & A.H. Ferentz, "Somatosensory evoked potential changes during muscle testing," *Intern J Neuroscience*, Vol 45 (1989).
30. Lippold, O.C.J., "The relation between integrated action potentials in a human muscle and its isometric tension," *J Physiol*, Vol 117 (1952).
31. Lovett, R.W., & E.G. Martin, "Certain aspects of infantile paralysis with a description of a method of muscle testing," *JAMA*, Vol 66, No 10 (Mar 4, 1916).
32. Maitland, K.H., M. Davids, & L. Ruggiero, "Inter-practitioner reliability study. Agreement between examiners on muscle strength and weakness." Proceedings of Summer Meeting, ICAK, Washington, DC, 1987.
33. Marino, M., "Current concepts on rehabilitation in sports medicine," in *The Lower Extremity and Spine in Sports Medicine*, Vol 1, ed J.A. Nicholas & E.B. Hershman (St. Louis: The C.V. Mosby Co, 1986).
34. Marino, M. et al., "The efficacy of manual assessment of muscle strength using a new device," *Am J Sports Med*, Vol 10, No 6 (1982).
35. Martin, E.G., & R.W. Lovett, "A method of testing muscular strength in infantile paralysis," *JAMA* (Oct 1915).
36. Mayo Clinic, Members of Neurology/Physiology sections, *Clinical Examinations in Neurology*, 2nd ed (Philadelphia: W.B. Saunders Co, 1963).
37. Milner-Brown, H.S. et al., "Mechanisms for increased force during voluntary contractions," *J Physiol* (Lond), Vol 226 (Oct 1972).
38. Myotech Corp., Comparative Muscle Tester, 1383-30 Veterans Memorial Hwy, Hauppauge, NY 11788-3048.
39. Newman, L.B., "A new device for measuring muscle strength," *Arch Phys Med*, Vol 30 (1949).
40. Nicholas, J.A. et al., "Factors influencing manual muscle tests in physical therapy — the magnitude and duration of force applied," *J Bone Joint Surg*, Vol 60A, No 2 (Mar 1978).
41. Nicholas, J.A., A.M. Strizak, & G. Veras, "A study of thigh muscle weakness in different pathological states of the lower extremity," *Sports Med*, Vol 4, No 6 (Nov/Dec 1976).
42. Nicholas, J.A., M. Melvin, & A.J. Saraniti, "Neurophysiologic inhibition of strength following tactile stimulation of the skin," *Am J Sports Med*, Vol 8, No 3 (1980).
43. Omura, Y., "Applied kinesiology using the acupuncture meridian concept: Critical evaluation of its potential as the simplest non-invasive means of diagnosis and compatibility test of food and drugs — Part I," *Acupunc Electro-Therapeut Res Int J*, Vol 4 (1979).
44. Perry, J.P. et al., "Predictive value of manual muscle testing and gait analysis in normal ankles by dynamic electromyography," *Foot & Ankle*, Vol 6, No 5 (Apr 1986).
45. Prestigeline Health Products, Inc., 5 Inez Dr., Brentwood, NY 11717.
46. Ryan, A.H., & J.H. Agnew, "Studies in muscular power and fatigue," *Am J Physiol*, Vol 42 (1917).
47. Rybeck, C.H., & R. Swenson, "The effect of oral administration of refined sugar on muscle strength," *J Manip Physiol Ther*, Vol 3, No 3 (Sep 1980).
48. Sabella, M.A., B. Decker, & T. Krawchuk, "An inter-examiner reliability study of manual muscle testing." Proceedings of Summer Meeting, ICAK, Washington, DC, 1987.
49. Schmier, A.A., "Research work on a more precise method of

determining muscle strength in poliomyelitis patients," *J Bone Joint Surg*, Vol 27, No 2 (Apr 1945).
50. Schmitt, W.H., Jr., "Using functional neurology to identify patterns of facilitation and inhibition arising from tonic neck reflexes and tonic labyrinthine reflexes." Proceedings of Winter Meeting, ICAK, Maui, HI, 1985.
51. Schmitt, W.H., Jr., "Muscle testing as functional neurology differentiating functional upper motorneuron and functional lower motorneuron problems," in *1986 Selected Papers of the International College of Applied Kinesiology*, ed W.H. Schmitt, Jr. (Shawnee Mission, KS: ICAK, 1986).
52. Schmitt, W.H., Jr., "Three variations of manual muscle testing." Proceedings of Summer Meeting, ICAK, Boston, 1995.
53. Schmitt, W.H., Jr., "A neurological model for the three types of muscle testing." Proceedings of the Annual Meeting, ICAK-USA, Dearborn, MI 1996.
54. Schmitt, W.H., Jr., & S.F. Yanuck, "Expanding the neurological examination using functional neurologic assessment. Part II: Neurologic basis of applied kinesiology," *Int Jn Neurosci*, Vol 97, Nos 1-2 (1998).
55. Scopp, A.L., "An experimental evaluation of kinesiology in allergy and deficiency disease diagnosis," *J Orthomolec Psychiatry*, Vol 7, No 2 (1978).
56. Unger, J.F., "The effects of a pelvic blocking procedure upon muscle strength: A pilot study," *Chiropractic Technique*, Vol 10, No 4 (Nov 1998).
57. Walther, D.S., *Applied Kinesiology, Volume I — Basic Procedures and Muscle Testing* (Pueblo, CO: Systems DC, 1981).

Chapter 9

Stomatognathic System

Introduction

The term "stomatognathic" refers to the mouth and jaw. The discussion in this chapter is of the stomatognathic **system**, which is the complex interaction of structures and function about the head and neck. Shore[63] states that the stomatognathic system contains components of "...the bones of the skull, the mandible, the hyoid, the clavicle and the sternum; the muscles and the ligaments; the dentoalveolar and the temporomandibular joints; the vascular, the lymphatic and the nerve supply systems; and the soft tissues of the head; the teeth." To Shore's comprehensive list, the connection to the sacrum and coccyx by way of the dura mater must be added. A further step includes the innominate bones, completing the pelvis. This system works in an organized manner that depends on normal function of all its parts.

To put the system's function in proper perspective with total body function, we must take one more step. Because of the connection of the stomatognathic system with the pelvis by the dura mater, the total spinal column is included. One should easily recognize that structural distortions and dysfunction throughout the body can disturb pelvic position and motion and spinal function. Now the entire body is included, and that is as it should be. Disturbance in the stomatognathic system can cause health problems almost anywhere in the body, and of almost any type; conversely, disturbance remote from the stomatognathic system, as defined by Shore, can result in dysfunction of that system.

Before progressing to the study of the stomatognathic system, we should consider whether its function is important to health. Cranial nerve entrapment neuropathy is a common problem resulting from cranial faults. Entrapment neuropathy is considered a localized injury and inflammation in a peripheral nerve that is caused by a mechanical irritation from some impinging anatomical neighbor.[39] Disturbed nerve function can occur from impingement by bony ridges, bony foramina, and/or soft tissues. The type of nerve entrapment with cranial faults is neurapraxia, which is the least involved type. There is no wallerian degeneration, and recovery is rapid when the pressure is relieved. A classic experiment by Granit et al.[31] reveals how minimum pressure on a nerve changes its function. They demonstrated the creation of fiber interaction within a nerve from compression. The artificial synapse was produced by a pressure so gentle that it did not impair conduction of the original impulse. An important aspect of this investigation relates with therapeutic efforts; the nerve returned to normal after being decompressed and irrigated with a saline solution.

Disturbed nerve function from cranial faults can easily be recognized using applied kinesiology examination and treatment procedures. The sternocleidomastoid and upper trapezius muscles receive dual supply from cranial nerve XI and spinal nerves. Cranial nerve XI exits the skull through the jugular foramen. When the sternocleidomastoid and upper trapezius test weak due to a cranial fault, they return to normal function immediately after cranial correction. The exact mechanism causing cranial nerve XI's failure to properly control the muscle is unknown. The margins of the jugular foramen are made up by the temporal and occipital bones. The temporal bone's contribution to the jugular foramen is by its petrous portion, which is a highly mobile structure in the cranial primary respiratory mechanism. There may be actual distortion of the jugular foramen, or there may be torsion of the dura mater that ensheaths cranial nerves IX, X, and XI as they emerge from the skull through the jugular foramen.

Often weakness of the sternocleidomastoid and upper trapezius muscles causes lateral head tilt, which may contribute to an occipital or upper cervical subluxation. There may be improper stimulation to the head-on-neck reflexes, an important part of the equilibrium mechanism. Similar nerve dysfunction may be present in cranial nerve X (vagus nerve) at the jugular foramen; however, it is more difficult to directly assess.

Muscle dysfunction throughout the body is often changed by cranial fault corrections. It may be by improved nervous system function, or from other mechanisms such as circulation and control of cerebrospinal fluid. Sutherland[67] is given most of the credit for discovering the cranial-sacral primary respiratory mechanism. He attributed many of the health problems resulting from cranial disturbance to improper control and movement of cerebrospinal fluid.

Magoun[44,47] cites Sutherland as indicating that "...the inactivity of the pituitary body in the sella turcica through mechanical membranous articular restriction is the primary cause of pituitary secretory disturbances." The coupling of improper pituitary secretion with poor distribution of the pituitary hormones by reduced cerebrospinal fluid flow helps to understand how endocrine function is often influenced by correcting cranial faults. Goodheart has also described clinical evidence indicating the direct influence of cranial dysfunction on the pineal gland.[26,27] In addition, the endocrine system may be indirectly influenced by cranial nerve entrapment as a result of cranial faults. Because of the great amount of integration among the glands of the endocrine system, when one gland functions improperly many that depend upon inhibition or stimulation by that gland's secretions contribute to further endocrine failure.

Applied kinesiology is highly successful in treating certain types of learning disabilities. A major cause of this type of disability is dysfunction in the stomatognathic system. Often there is a failure of proper visual function. It may be due to peripheral entrapment of cranial nerves III, IV, and/or VI, which control the extraocular muscles. When ocular lock (discussed on page 172) is present, it is

often corrected by treatment to the stomatognathic system.

Visual acuity may change immediately after correction of cranial faults. This may result from improved cranial nerve II function or a change in the shape of the bony orbit, which is made up of seven bones with a great amount of flexibility. The intraocular pressure of glaucoma may also improve with cranial fault correction.[15]

One does not know what improvements may develop in addition to those problems specifically being treated when cranial faults are corrected. While teaching an applied kinesiology seminar, Duffy[15] returned central vision to an octogenarian; in addition, the patient experienced the full return of her hearing. Besides influencing hearing, change in cranial nerve VIII may also influence the vestibular apparatus. The visual righting, labyrinthine, and neck righting reflexes all contribute to report the head's position. If the head is level and the visual and neck righting reflexes report this, but the labyrinthine reflexes report head tilt due to irritation on cranial nerve VIII from a cranial fault, the central nervous system's interpretation of the information will be confused. This is a common source of neurologic disorganization, which may cause dysfunction throughout the body. Disturbance in the cranial-sacral primary respiratory mechanism is a common finding in adolescent idiopathic scoliosis.[45]

Numerous health problems improve with correction of the cranial-sacral primary respiratory mechanism, which is one of the five major treatment methods in applied kinesiology. It is the CSF factor for the cerebrospinal fluid function of the cranial-sacral primary respiratory system. All patients should routinely be screened for dysfunction in the stomatognathic system; the cranial primary respiratory mechanism is an important part of that system.

Cranial Motion

One who works with the cranial-sacral primary respiratory mechanism is often confronted with the question, "Do the bones of the skull really move?" Although the question becomes an irritant to those knowledgeable about cranial function, it is a reasonable one to ask since most persons trained in the healing arts are taught that the primary purpose of the skull is to protect the brain and provide a place to hang the face. In addition, students are exposed to the skull as a dried osteologic specimen, or in a cadaver wherein little study is usually made of the skull and its mechanisms.

This writer has spent hundreds of hours studying the disarticulated bones of the skull for initial education, and hundreds more while doing photography and reviewing the motion and sutures with an artist for a textbook.[75] I cannot understand how anyone can study the pivot points, angles of articulation, lines from bones sliding on each other, and gear-train mechanisms of a disarticulated skull and not conclude that it is designed for motion. The ossification of sutures that occurs in some skulls in later life is a pathologic process, not a natural one.

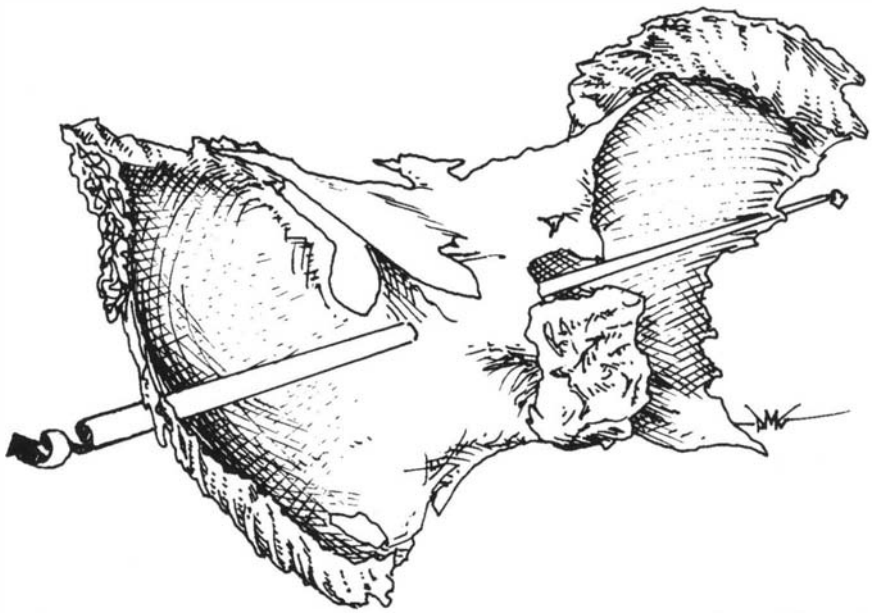
In addition to the joints of the skull, one must recognize the flexibility of living bone. What is not considered in most doctors' education is that the skull *in vivo* is a dynamic, plastic structure that has organized activity. Studying the skull from a dried specimen or in a cadaver is similar to studying a telephone pole as if it were a living tree. The telephone pole is rigid, the sap is dried, there is no living flow, and an effort to bend it could cause it to break. A living tree, on the other hand, has water and nutrients in its "veins." It bends and sways with the breeze and progresses through its life cycle from year to year.

The plastic nature of bone *in vivo* is illustrated in a study by Stowe et al.[65] Orthogonal x-ray beams were used to measure the absolute and relative movements and consequent torsion of the adult human forearm *in vivo* during its rotation from maximum voluntary supination to maximum voluntary pronation. A torsion of 69° was observed in the ends of the radius; 34° was seen between the ends of the ulna.

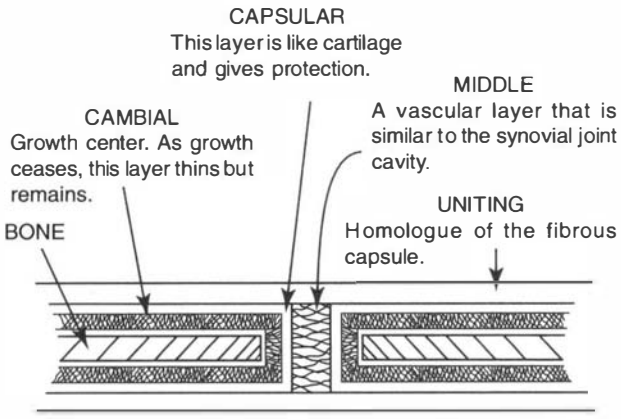
The histology of a suture is that of a joint designed for motion. Pritchard et al.[56] found five distinct layers of cells and fibers between the edges of the adjoining bones in human specimens. They concluded that "...histology of the sutures suggests that it has two main functions, viz. that it is a site of active bone growth and that it is at the same time a firm bond of union between the neighboring bones, which nevertheless allows a little movement." The first of five layers making up the suture is bone. The cambial layer is the growth center that thins as growth ceases, and the capsular layer is light cartilage that provides protection. The middle aspect in the suture is a vascular layer similar to the synovial joint cavity. Finally, covering the suture is the uniting layer that is the homologue of the fibrous capsule.

Upledger et al.[72] studied specimens taken from living adult skulls at the time of neurosurgical craniotomies. Along with connective tissue, the sutures were shown to have the presence of viable myelinated and unmyelinated nerve fibers and nerve receptor endings.[58,59] One method of examining the cranium for faults is for the applied kinesiologist to apply pressure to various portions of the skull and observe for a manual muscle test change. It appears that stimulation of the neurostructures in the suture is responsible for some of the remote muscle changes observed.

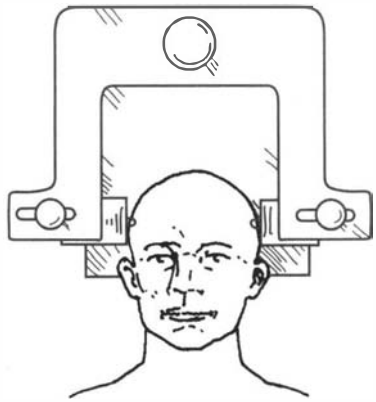
Motion of the skull bones has been objectively measured. The first study was made by Frymann.[18] When she applied force transducers to a subject's head, she found a rhythmic autonomous motion supporting Sutherland's[67] original observation of 10-14 cycles per minute.



9—1. The sphenoid rotates about the sphenosquamous pivot in the transverse plane. Above the pivot the suture is squamous, with a sliding action. Below, it is serrate and the motion is of compaction and separation.



9—2. Suture histology, after Pritchard et al.[56]



9—3. Study of motion with fixed transducers. After Frymann.[18]

Further motion studies have been done to eliminate effects of intervening tissue between the transducer probe and the skull. Michael and Retzlaff[50] attached force transducers to a screw eye placed in the parietal bone of anesthetized monkeys. A cyclic cranial bone displacement of 5-7 cycles per minute was observed that could not be attributed to either respiration or heart rate.

The position of cranial bones in relation to each other can be observed on accurately positioned x-rays.[33] Cranial nerve V angles over the petrous apex of the temporal bone. Gardner[21] measured the height of the petrous apex by x-ray and found that trigeminal neuralgia occurred three times more often on the side of the high petrous apex than on the low side.

Cranial distortion may change the dental occlusion by mandibular movement change or by disrelation of the cranial bones. Malocclusion is a common complaint following an auto accident with whiplash dynamics to the neck and head. Many doctors fail to appreciate this because the patient often does not discuss it with the orthopedically inclined physician. If the patient discusses the matter with a dentist who is not knowledgeable about cranial motion, the teeth may be equilibrated to match the distorted skull; this locks in the cranial faults and makes correction difficult or impossible without further dental attention. Baker[3] found a 0.0276" increase in the distance between the second molars following cranial manipulation.

It is important to understand all of the aspects that can be involved in dysfunction of the stomatognathic system. A patient may come to a chiropractor for a whiplash-type injury to the cervical spine. This condition often relates with cranial faults that may in turn cause malocclusion. Correcting the cranial faults may correct the malocclusion; however, if the condition has been present for a considerable time the teeth may have changed position by the natural process of remodelling, thus locking in the cranial faults. In this case it may be necessary to consult a dentist for a bite

plane, and perhaps eventual equilibration of the teeth.

The pelvis and sacrum are intricately involved in the cranial primary respiratory mechanism, and may require chiropractic correction to eliminate temporomandibular joint or occlusal problems. A short leg has been indicted as a cause of malocclusion. Strachan and Robinson[66] of the Chicago College of Osteopathy were the first to observe a short leg's influence on malocclusion. Evaluating the pattern of masticatory muscles with electromyography, they removed a 3/8" heel lift from a standing subject's shoe and found an altered firing sequence of the muscles of mastication during chewing. When the lift was worn, the muscles showed the firing pattern of normal occlusion; with it removed, the firing pattern was one of a severe malocclusion. In applied kinesiology, an apparent short leg is quite often found to be a result of pelvic distortion. The leg usually balances with pelvic correction. In any event, their study reveals the importance of remote postural imbalance on the stomatognathic system.

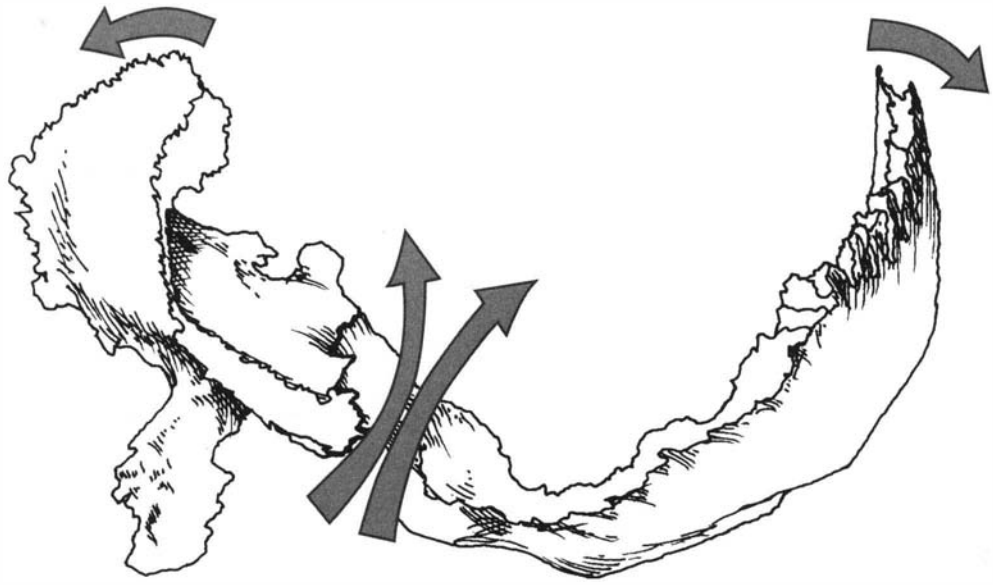
The stomatognathic system is very active in the dynamic motions of the body. During walking the sternocleidomastoid and upper trapezius muscles are alternately inhibited and facilitated, providing a rhythmic pull on cranial bones. Because of the stomatognathic system's muscle interaction with the rest of the body, improper gait and other dysfunction may be the perpetuating cause of recurrent cranial faults.

Integration of the Stomatognathic System

With normal function there is predictable movement of the cranial bones. It continues throughout life, cycling 10-14 times per minute, and is called "the primary respiratory mechanism."[67] The motion is separate from the heart or breathing rate; however, cranial motion is enhanced by thoracic respiration. Although this influence is always present, relaxed breathing and primary respiration do not always parallel

Cranial motion is a combination of bending bone and suture motion. The motion between the sphenoid and the occiput is in the sagittal plane. There is flexion and extension of the sphenobasilar junction. Prior to the approximate age of twenty-five, the motion is at the synchondrosis between the bones. After ossification, the motion is flexion and extension of the cancellous bone. Sphenobasilar flexion consists of raising the sphenobasilar junction and separating the superior portions of the occipital squama and greater wings of the sphenoid. Sphenobasilar extension is dropping of the sphenobasilar junction and approximation of the superior portion of the occipital squama and greater wings of the sphenoid. Sphenobasilar flexion is enhanced by a deep phase of inspiration; extension is enhanced by expiration. Often sphenobasilar movement is called sphenobasilar inspiration or expiration.

The general axis of temporal bone rotation is through the petrous portion, which is at approximately a 60° angle with the temporal bone's squamous. The apex of the petrous portion rises on inspiration with the sphenobasilar junction, and the petrous ridge rotates anterolaterally, causing the squamous of the temporal bone to rotate externally. The mastoid process moves posteriorly and medially with inspiration.

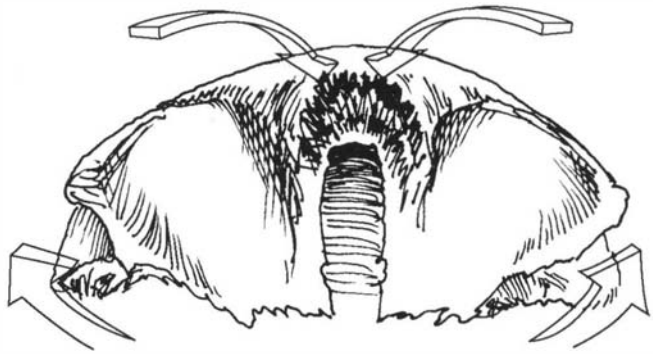


9—4. General motion of sphenoid and occiput on inspiration (flexion).

9—5. Lateral view of temporal bone on inspiration.

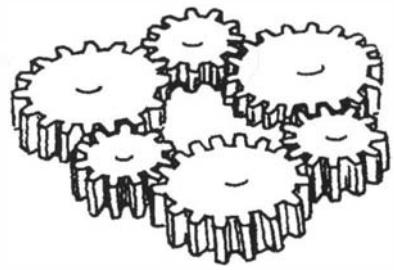
9—6. General axis of temporal bone showing general axis of rotation through petrous portion.

The frontal bone, in most subjects, ossifies at the metopic suture. In some cases a remnant or the entire suture persists throughout life. Motion on sphenobasilar flexion (inspiration) spreads the frontal bone's squamous portion. In applied kinesiology this is called internal frontal motion, as if the metopic suture were moving internally. On sphenobasilar extension (expiration), the squamous portions of the frontal bone move medially. In applied kinesiology this is called external frontal rotation, as if the metopic suture area were moving externally. This is confusing, because an external frontal in DeJarnette's sacro occipital technique[11,12] is called an internal frontal in applied kinesiology, and vice versa.



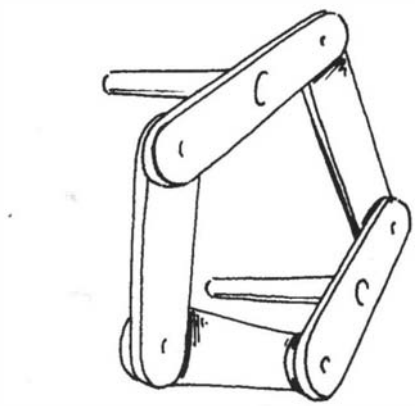
9—7. Inferior view of frontal bone showing internal rotation.

The rest of the bones of the skull and face have specific movement in relation to sphenobasilar flexion and extension. There is continuity in the movement by way of pivot points, gear trains, sliding action, and lever mechanisms; this interaction constitutes a closed kinematic chain. In any closed kinematic chain, when one portion moves the entire chain must move. This can be demonstrated by a system of levers or gears, both of which are present in the cranial closed kinematic chain. The occipitomastoid suture acts like teeth on a gear. The vomer is a lever mechanism between the rostrum of the sphenoid and the intermaxillary and palatine sutures of the maxillary and palatine bones.



9—8. Closed kinematic chain of gears.

There is a synchronous movement between the sphenobasilar junction and the sacrum. The connection is the dura mater, which has a firm attachment at the foramen magnum and 2nd and 3rd cervical vertebrae. Attachment along the rest of the spinal column is loose, until the dura and arachnoid firmly attach at the 2nd sacral segment. The general axis of sacral rotation is at the level of the 2nd sacral segment about its transverse axis. Motion of the sacrum consists of lifting the anterior portion of the 2nd sacral segment as the sphenobasilar junction lifts in inspiration; thus, the apex of the sacrum moves anteriorly and the base posteriorly with inspiration, and opposite with expiration.



9—9. Closed kinematic chain of levers.

The innominate bones also have a primary movement organized with cranial function. If one compares the innominate with the temporal bone, the comparative parts move in the same direction with inspiration and expiration (see illustrations 9—67, 9—70). The ilium is compared with the squama of the temporal bone; it moves anterolaterally with inspiration. The ischium, compared with the mastoid process, moves posteromedially. Pressure from the abdominal contents and the abdominal muscles contributes to this motion. On inspiration the abdominal contents move downward, putting pressure on the ilium to move the anterior iliac spine laterally, anteriorly, and inferiorly. Simultaneously with inspiration, the abdominal muscles relax to permit this motion. During expiration, the abdominal muscles contract, reversing the motion of the ilia.

Organization between the closed kinematic chain of the cranium, with its movement of the sacrum by the dura mater and innominate movement by abdominal muscle contribution, emphasizes how integrated the motion of the stomatognathic system is with the rest of the body.

Within the stomatognathic system as Shore[63] defines it — from the shoulder girdle up — there is further integration and another closed kinematic chain. This is a muscular closed kinematic chain made up of the cervical flexors and extensors, hyoid muscles, and muscles of mastication. The original presentation of the muscular closed kinematic chain of the stomatognathic system was described in a program of the University of Illinois[10] in 1949. Mintz[52] discusses the interaction of this closed kinematic chain and its correlation with the rest of the body. Disturbance here can cause a chain reaction throughout the musculoskeletal system. On the other hand, remote musculoskeletal imbalance or dysfunction can disrupt the harmony within the stomatognathic closed kinematic chain.

In an article directed toward the legal profession, Moses and Skoog[53] draw attention to the similarity of symptoms between cervical whiplash and temporomandibular joint conditions, indicating that either may be caused by an auto accident. Recognizing the interplay within the stomatognathic system, it is entirely reasonable that there are similar symptoms. Often when the cervical spine is injured, the temporomandibular joint ultimately becomes involved; if the jaw is injured, affecting TMJ dysfunction, the cervical spine may ultimately become involved. They also point out the potential hazard of the conventional treatment of cervical bracing and traction on the TMJ, which is not designed to be a pressure-bearing joint.[60]

Indications to Evaluate the Stomatognathic System

There are specific conditions and types of trauma that indicate probable involvement with the stomatognathic system; one should keep these constantly in mind throughout a patient's examination and treatment. The initial examination of all new patients should include screening of the stomatognathic system for disturbed function. Although this is a good procedure for initial evaluation, negative findings are inadequate to rule out the system as a part of the patient's health problem. Sometimes cranial faults are difficult to find, but when they are corrected the results obtained are very rewarding.

One of the common indicators of stomatognathic system involvement is any condition relating with cranial nerves. Such a condition will very probably require cranial or other treatment to the stomatognathic system. One should be very familiar with cranial nerve anatomy and examination. Many of the tests for cranial nerve function are designed to determine nervous system pathology. Negative findings do not rule out subtle impingement on the nerve with resulting functional problems of the organs, glands, and/or structure.

When considering nerve control, one should include balance between the parasympathetic and sympathetic systems. In addition to cranial-sacral outflow, this includes the endocrine system. The apparent relationship of the pituitary gland with normal cranial movement can be an initiating problem in imbalance of the endocrine system. The endocrine glands are also controlled by the nervous system, which can be adversely influenced by cranial faults. The symptomatic pattern may be remote from what one would think might be caused by dysfunction in the stomatognathic system.

One of the most frequent causes of neurologic disorganization is disturbance in the stomatognathic system. The predictability of muscle tests throughout the body may be disturbed by poor organization of the equilibrium proprioceptors, such as the visual righting, labyrinthine, and head-on-neck reflexes. If there is a disturbance in eye function, such as ocular lock, the visual righting reflexes may be telling the central nervous system that the head is tilted; actually it is not, and the labyrinthine and head-on-neck reflexes are sending information that the head is balanced. The central nervous system can only act on this conflicting afferent information with unpredictable results.

Etiology of Cranial Faults

A major factor to consider when examining a patient is what seemed to initiate the health problem. Of-

ten there is specific evidence of trauma to the stomatognathic system. One not familiar with the mechanism may fail to elicit this information from the patient, ignore it if presented, or improperly consider it.

Trauma. Direct trauma to the skull is often misinterpreted by chiropractors. Many chiropractors fail to recognize cranial trauma from a blow to the head or a difficult birth. They may recognize an upper cervical subluxation that actually may be secondary in nature because of the cervical muscular imbalance caused by disturbed cranial nerve XI function. The upper cervical area can be adjusted successfully, but the subluxation will probably return if the muscles are not balanced.

Birth trauma is a common cause of cranial faults. Often a baby's flexible skull corrects itself. As will be pointed out later, the cranial-sacral primary respiratory mechanism is a self-correcting, self-maintaining mechanism. Actions such as nursing at a mother's breast activate the cranium by tongue and masticatory muscle action. When a baby is bottle-fed with a large nipple, this proper action is lost. For any of many reasons, birth trauma may not be overcome by natural action and cranial faults may persist, possibly causing a lifetime of health problems.

Frymann[17] studied the cranial-sacral motion of 1,250 unselected infants and correlated it with symptoms of the nervous system, such as vomiting, hyperactive peristalsis, tremor, hypertonicity, and irritability. Her study suggests a significant relationship between various forms of cranial-sacral primary respiratory dysfunction and the children's symptomatology.

Upledger[71] studied 203 grade school children for proper cranial-sacral motion. There was a statistically significant correlation between poor motion and children classified by the school authority as learning disabled or having behavioral problems. There was also a statistically significant correlation of poor cranial-sacral motion with a history of a complicated delivery. The correlation of poor cranial-sacral motion was statistically highest for those children presenting numerous problems.

General trauma to the head, such as raising up beneath a kitchen cabinet or striking the head on a car door, may cause cranial faults. Sometimes violent blows to the skull create no fault, whereas a relatively mild blow does; the sum of the circumstances yields the ultimate result. Is the trauma in precisely the correct direction to lock bony movement, and is the individual in the phase of respiration that allows it? Symptoms from cranial faults may not develop for several days, or even months or longer after the actual fault has developed.

Hyperflexion-hyperextension cervical trauma, often seen in automobile accidents and loosely termed "whiplash," is a potent force that may directly or indirectly cause cranial faults.[43] The entire stomatognathic system may be involved in this type of injury. A patient may obtain good results from chiropractic care of the cervical spine, yet he reaches a plateau with residual problems that do not respond to treatment. Unfortunately, these problems are often rated as a permanent disability because of the doctor's lack of knowledge about the total stomatognathic system. In many instances, the lack of knowledge of these functional conditions is even greater in professions other than chiropractic.

Dental malocclusion, for whatever reason, can cause cranial faults.[41] When an individual clenches his teeth in normal occlusion, the muscles of mastication pull on levers of the cranial mechanism to activate it. This is part of the self-correcting, self-maintaining mechanism of the stomatognathic system. If there is malocclusion in which one tooth hits before the others, imbalanced force is put into the cranium. This can readily be observed by having a normally functioning subject bite on a toothpick placed between the molars. A previously strong indicator muscle, such as the sternocleidomastoid, will test weak in nearly all cases. Henningsen,[34,35] a dentist knowledgeable about the cranial-sacral primary respiratory system, cautions his profession that "...techniques should be completed with the greatest of care." Furthermore, "...the use of elevators, forceps and mouth props can be powerful fulcra." Holding the mouth open for prolonged dental procedures can cause cranial faults, as can tooth extraction. A crown can be responsible for cranial dysfunction as a result of pressures when seating it or failure to correctly equilibrate it, causing an occlusal problem. Malocclusion causes strain in the cranial primary respiratory mechanism every time an individual chews, swallows, or bites forcefully.[42,46]

There are many other causes of cranial faults and imbalance within the total stomatognathic system. Remote structural imbalance, disturbance in gait, and improper modular interaction can all cause recurrent cranial faults and strain in the stomatognathic system. Patients may have habit patterns that create cranial faults, such as propping one's chin on a hand while reading at a desk, poor sleeping posture, or lying in bed on one side while propping the head on a hand to read. Examining the patient with equilibrium synchronization, PRYT, and dural tension techniques often leads to correction of the cause of disturbed stomatognathic function. The sternocleidomastoid and upper trapezius may have an improper temporal pattern of facilitation and inhibition that pulls on the mastoid process and occiput to create or perpetuate cranial faults when the patient walks. Examine the patient for walking gait and foot subluxations.

The bottom line is that the stomatognathic system has great integration within itself and with total body function. The wide range of examination procedures available in applied kinesiology will generally find the cause of recurrent problems in the stomatognathic system.

Cranial Examination and Treatment

Diaphragmatic and Cranial Respiratory Correlation

A deep phase of thoracic inspiration or expiration influences cranial bone movement. This can be used as an asset in applied kinesiology examination. With a deep inspiration the sphenobasilar junction flexes; that is, it moves superiorly. If a cranial fault is such that the sphenobasilar junction is in an extended position (inferior), a deep phase of inspiration improves the cranial bone position. A muscle that tests weak as a result of excessive sphenobasilar extension will test strong when a deep phase of inspiration is taken, because the cranial bone position is temporarily improved. Likewise, a muscle that tests weak because of excessive sphenobasilar flexion will test strong when a deep phase of expiration is held.

Many of the cranial faults listed in applied kinesiology have a specific phase of respiration that improves position of the bones. When a patient holds this phase of respiration a weak associated muscle becomes strong, sometimes indicating the type of cranial fault that may be present.

When cranial faults are present, patients will often subconsciously take and hold a phase of respiration when muscle tests are done. A knowledgeable physician recognizes this as body language that the cranial-sacral primary respiratory mechanism should be examined. Be constantly aware of a patient taking a phase of respiration during an examination. There may not be an obvious inhalation or exhalation; rather, there is a forced holding of the breath during muscle tests. Likewise, changing facial expression during a muscle test indicates probable cranial faults. Often a patient can contract facial muscles that will improve the position of the cranium for improved muscular performance during a muscle test.

In the earlier general description of how the cranial bones move, it was explained that there is disagreement among some authorities regarding the direction of movement of certain portions of bones. Specifically, there is disagreement about the movement of the mastoid process with sphenobasilar flexion (inspiration). Some believe that it moves anteriorly, while in applied kinesiology it is described as moving posteromedially.

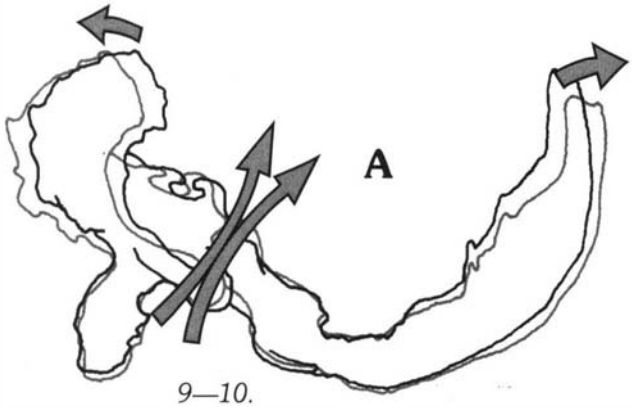
An experiment can be done to evaluate cranial bone motion with respiration. The experiment is to resist cranial bone movement in different directions on an individual with a normally functioning cranium. All who describe cranial bone motion indicate that the squamous portion of the temporal bone flares laterally on sphenobasilar flexion (inspiration). To test this or other cranial motion, the motion can be restricted on a person with a normally functioning skull. The examiner contacts the squamous portion of the temporal bones bilaterally and applies light pressure while the individual takes a deep inspiration. After the deep inspiration the subject resumes normal breathing, and the examiner releases his pressure on the temporal bones. A previously strong indicator muscle will test weak because of the restriction of normal temporal bone movement. The experiment may or may not work on an individual with cranial faults, depending upon the type of faults present.

The same principle can be applied to most of the cranial bones. For example, when the mastoid processes are kept from moving posteromedially during inspiration, a previously strong indicator muscle will weaken after the individual resumes normal breathing and the examiner releases pressure on the mastoid process tips. Likewise, if the mastoid processes are prevented from moving anterolaterally during a deep expiration, a previously strong indicator muscle will weaken.

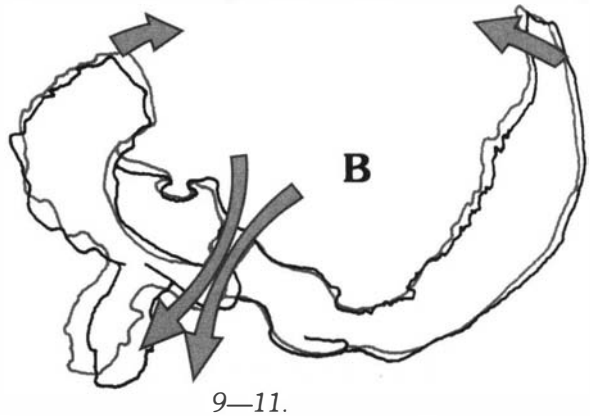
Most cranial bone movement has been evaluated in applied kinesiology using this method. The bones appear to move in the direction originally described by Sutherland,[67] and further described by Magoun[47] and Upledger and Vredevoogd.[73]

Muscle Association with Cranial Faults

There is specific muscle dysfunction with some cranial faults. For example, the frontal faults are often associated with weak neck flexors. When weak neck flexors are found, one should examine for the cranial faults; however, the muscle dysfunction is not always due to a



9—10.



9—11.

Black lines represent the normal neutral position of the sphenoid and occipital bones. The gray lines in A represent exaggerated position on inspiration, and in B on expiration.

cranial fault. One must use differential diagnosis to determine the therapeutic approach. All cranial faults can relate with muscle dysfunction anywhere within the body. Most faults have no specific muscle dysfunction associated with them.

Therapy Localization

All cranial faults have positive therapy localization; the problem is that it will not always be at the same location. Positive therapy localization occurs along sutures that are apparently in strain. Certain cranial faults will have consistent therapy localization. These are generally the sutural faults, such as the sagittal suture. Positive therapy localization will be along the suture; however, the entire suture may not show positive therapy localization.

In the past specific locations have been described for therapy localization of different cranial faults; many of these areas will not be described in this text. It has been found that therapy localization may be negative at the location but the cranial fault, as examined by challenge, is nevertheless present. As mentioned, there will always be positive therapy localization with a cranial fault, but it may not be at a specifically designated site. The use of therapy localization as a single entity to determine if there is a cranial fault will miss many faults; it is not recommended.

Therapy localization can be an aid in determining if a cranial fault has been adequately corrected. When there is positive therapy localization and it is believed to be associated with a cranial fault, one can retherapy localize the area after a corrective attempt. If the positive therapy localization is abolished, it gives added information — along with negative challenge — that the fault was corrected.

When therapy localizing for cranial faults, remember that there are many other factors — such as stress receptors, muscle proprioceptors, and neurovascular reflexes — that may be involved in the therapy localization. In addition, pathology such as sinus infection may be responsible for positive therapy localization. When positive therapy localization is abolished by a phase of respiration, there is evidence that cranial bone dysfunction is involved. This is not a conclusive test that positive therapy localization is associated with a cranial fault.

Challenge

The best method to determine if a cranial fault is present is challenge, i.e., putting a vector of force into the cranium and testing for muscle function change. There are two types of challenge: rebound and static. In a rebound challenge, a vector of force is put into the cranium and released. With static challenge, a vector of force is put into the cranium and held while a muscle is tested.

There has been some confusion about the challenge mechanism in applied kinesiology, primarily because previously written material did not delineate whether reference was to a static or a rebound challenge; it was simply called challenge.[64,74] Since those writings, it appears that the better method is usually rebound challenge. It has become the standard approach in applied kinesiology for the spinal column and skull, with few exceptions. When the word "challenge" is used in conjunction with the cranium or pelvis, it refers to the rebound challenge unless a static challenge is specifically indicated.

Applied kinesiology challenge to the cranium consists simply of pressure applied to a cranial bone(s) in a direction expected to obtain correction. The pressure may introduce a force into the cranium that influences many bones through the closed kinematic chain, or it may be a force that is limited to influencing the local area. Challenge for an inspiration assist cranial fault is the first one discussed. It is an example of a single force influencing many bones by way of the closed kinematic chain. Pressure is applied to the mastoid process, which acts as a lever to move the temporal bone. By way of a gear-train action at the occipitomastoid suture, the posterior occiput is moved. The basilar portion of the occiput is moved, along with the petrous portion of the temporal bone. There is a sliding action between the squama and greater wing of the sphenoid, and movement of the zygomatic bone by the zygomatic arch of the temporal bone.

Localized dysfunction of the cranial mechanism is demonstrated by the sutural faults; the lambdoidal suture is an example of this. Force is applied to the occipital and parietal bones to separate or approximate the suture, as indicated by challenge. This has a more limited influence on the cranium; however, one often sees other cranial faults influenced by correction of the lambdoidal suture.

The method of challenge will be described with each cranial fault. Precise challenge evaluation is important, because it indicates the presence of a cranial fault and provides information for the optimal correction. In most cases, challenge is applied from numerous vectors until one is found that creates the maximum indicator muscle weakness. The optimal vector is usually quite limited; it indicates the best vector of force to influence the cranial closed kinematic chain.

The use of challenge vector analysis for cranial faults is strongly recommended. It is possible to apply forces to the cranium that create iatrogenic cranial faults. No iatrogenic problems have been observed by this writer when vector challenge is used as a basis for treatment.

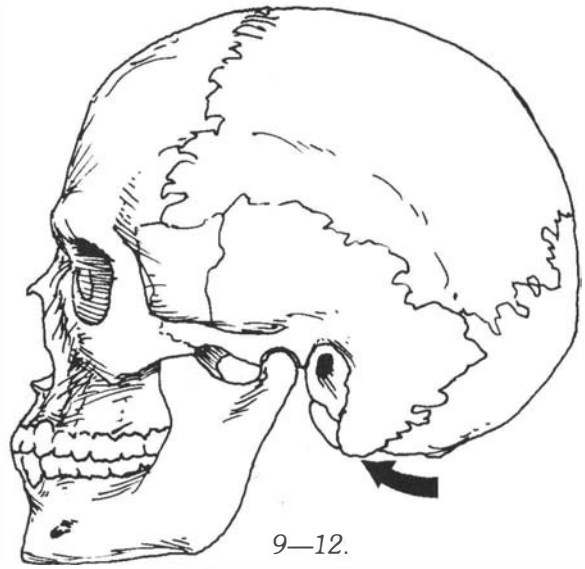
Correction

Correction of cranial faults is obtained by applying pressure in the direction of optimal challenge on the phase of respiration that correlates with the cranial fault. The pressure should be gentle and continuous throughout the associated phase of respiration, rather than jabbing or stabbing. Strong forces should be avoided

because they produce body resistance. The treatment pressure should work with the body, not attempt to force it. The pressure is usually repeated through several phases of respiration. With experience, a physician will begin to feel the cranium unlock; a subtle movement will be detected. With the initial few applications of pressure, the skull will often feel rigid and non-yielding. As the correction is obtained, "life" is felt. This feeling is analogous to pushing on the dead wood of a telephone pole versus pushing on the trunk of a live tree.

Inspiration Assist Cranial Fault

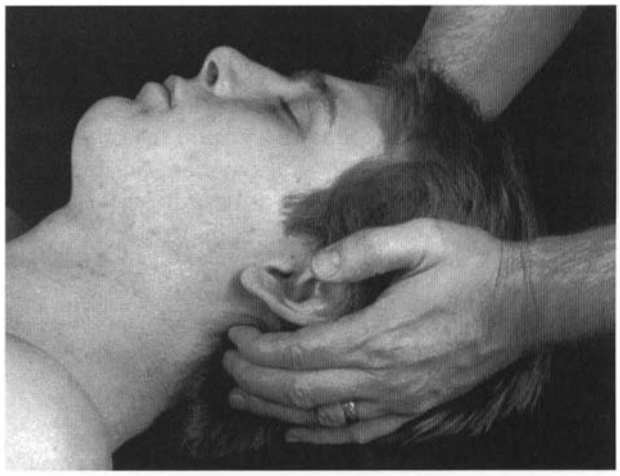
The inspiration and expiration assist cranial faults were the first described in applied kinesiology. As other faults were added, many began to consider the inspiration and expiration assist cranial faults "simple" faults. This caused more attention to be directed toward other faults with more complicated contact points. When one understands the cranial closed kinematic chain, it becomes obvious that treatment of the inspiration or expiration assist cranial faults can influence the entire cranium. In a high percentage of cases, there is an inspiration assist cranial fault on one side and a compensating expiration assist cranial fault on the opposite side. In this situation, it is best to treat them first because many other cranial faults are often corrected in the process. If one wants to document the presence of cranial faults in a personal injury case or for some other reason, one should examine the cranium for all faults and record the results before any are treated. It is good practice to examine for all cranial faults before treatment when one is learning this therapeutic approach. As expertise is acquired, the ability to eliminate additional faults with correction of the inspiration and expiration assist faults improves.



9—12.

Breathing Pattern. A muscle that tests weak as a result of the cranial fault will become strong when the patient holds deep inspiration. A strong muscle that can be influenced by the cranial fault will become weak when the patient holds deep expiration.

Challenge. Challenge is a vector of force generally applied to the mastoid process posterior tip directed anteriorly. A mild force of 1-2 pounds is applied and released, and a previously strong indicator muscle is tested for weakening. Several challenges should be applied, combining a medial or a lateral vector with the posterior-to-anterior force to challenge the three-dimensional aspects of the cranial fault. The optimal vector of correction is the one causing greatest weakening of the indicator muscle. Quite often it will be primarily posterior-to-anterior, but occasionally there will be a great amount of lateral or medial component to the optimal vector.



9—13. Pressure is applied for inspiration assist in the direction indicated by maximum weakening of an indicator muscle during challenge.

Therapy Localization. The area that most frequently therapy localizes with the inspiration assist cranial fault is the occipitomastoid suture.

Correction. Correction is obtained by having the patient take a deep phase of inspiration while pressure in the vector that caused maximum weakening of the indicator muscle is applied to the mastoid process. Force is applied through the full phase of inspiration. Make certain that the patient takes a slow, even inspiration from full exhalation to full inspiration. Patients have a tendency to breathe too rapidly during this process. A slow, even pressure applied through an inspiration of 6-8 seconds is optimal. Usually 4-6 repetitions of the application are adequate for correction. As one gains expertise in making this correction, movement of the cranium will be felt, indicating effective treatment. On rare occasions, movement is not felt until 20-40 applications of force have been applied. Re-examine with various vectors of force to determine if the corrective attempt was effective.

The inspiration assist cranial fault may be unilateral or bilateral. As mentioned, there is often an inspiration assist on one side and a compensatory expiration assist cranial fault on the other.

Expiration Assist Cranial Fault

The expiration assist cranial fault is exactly opposite the inspiration assist fault.

Breathing Pattern. A muscle that tests weak as a result of an expiration assist cranial fault will become strong when the patient holds a full phase of expiration. A strong muscle affected by this cranial fault will become weak when a full phase of inspiration is held.

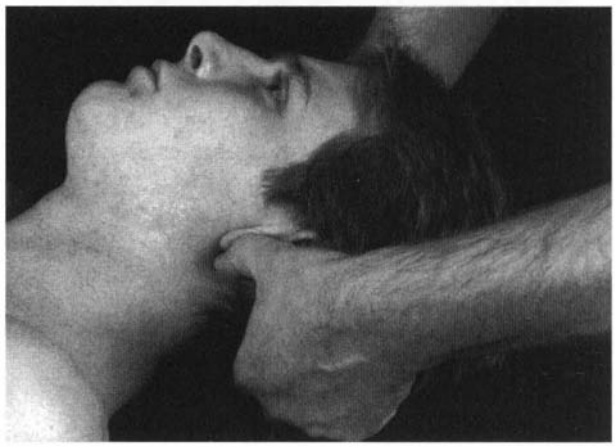
Challenge. The challenge is a vector of force applied to the anterior aspect of the mastoid process in a posterior direction. Several vectors of challenge are applied to include lateral or medial components with the anterior-to-posterior vector. Quite often the greatest component of the vector will be anterior-to-posterior, but sometimes the medial or lateral component is greater.

Therapy Localization. The area that most frequently therapy localizes in an expiration assist cranial fault is along the occipitomastoid suture.

Correction. Pressure is applied to the mastoid tip in the direction of optimal challenge in a manner similar to the inspiration assist correction.

Combined Inspiration and Expiration Assist Cranial Faults. Before treating an inspiration or expiration assist cranial fault, challenge the mastoid process bilaterally to determine the type of correction(s) necessary. When both sides have an inspiration or expiration assist fault, they are treated simultaneously. In this case, contact both mastoid processes and simultaneously apply the proper vector of force during inspiration or expiration. When the patient has an inspiration assist fault on one side and an expiration assist on the other, treat the inspiration assist fault as the patient inhales, then treat the expiration assist fault as he exhales.

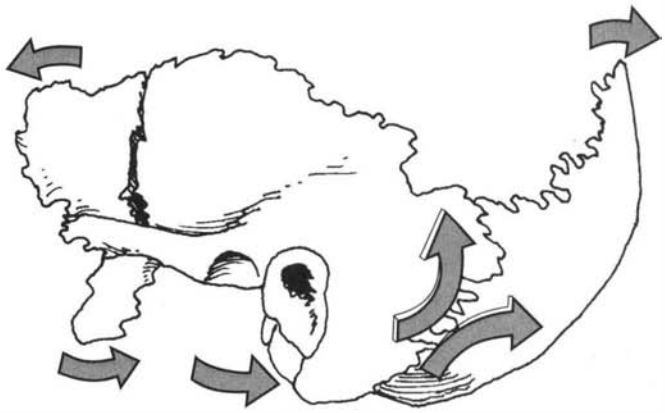
The patient will often feel a sensation through the facial bones as these corrections are made. The more accurate the vector of force, the more frequently there will be a sensation throughout the cranium. Occasionally the patient will even feel an articular release in the cranium.



9—15. Contact for expiration assist challenge and correction.

Most cranial faults are corrected on a rebound basis. The pressure applied to the skull increases the cranial fault, tightening the dural reciprocal tension membrane. It is postulated that when the treatment pressure is released, the reciprocal tension membrane makes the correction.

9—14. Movement of bones in inspiration assist correction.



9—16. Movement of bones in expiration assist correction.

Sphenobasilar Inspiration Assist Cranial Fault

The sphenobasilar inspiration assist cranial fault is similar to the inspiration assist cranial fault. The sphenoid and occiput are in excessive extension. The major difference between the two faults is that the correction of the sphenobasilar inspiration assist requires two hands; thus the cranial closed kinematic chain is treated from two points. This is often advantageous in correcting a severely locked cranium.

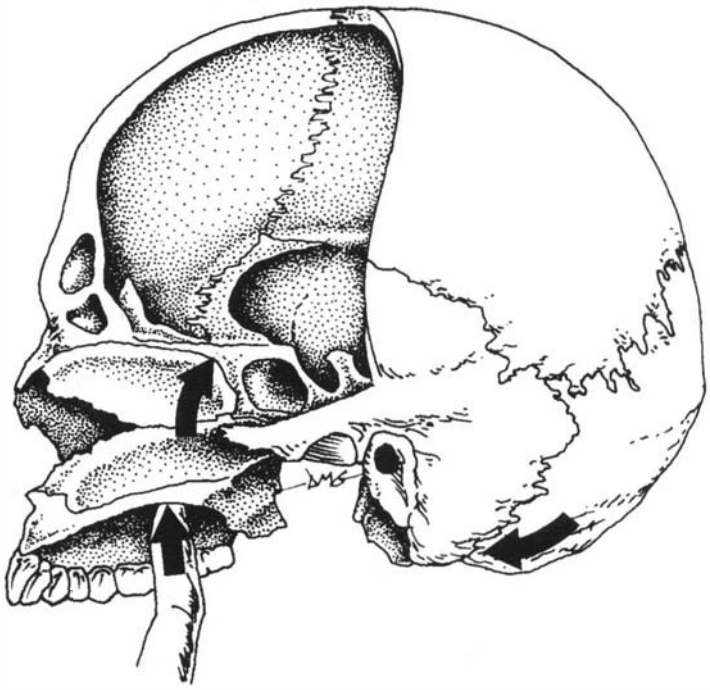
Breathing Pattern. A weak muscle involved with the sphenobasilar inspiration assist cranial fault will test strong when the patient holds "forced inspiration." Forced inspiration means that the patient takes in as much breath as possible and then attempts to increase the inspiration even more. Forced expiration causes a muscle strong in the clear to weaken if it is influenced by the cranial fault.

Challenge. The sphenobasilar inspiration assist cranial fault will show a positive challenge on the mastoid process similar to that previously described for the inspiration assist cranial fault. In addition, there will be a positive challenge with a superior vector of force to the hard palate. Force is directed to the hard palate in the general area of the palatomaxillary suture on the side of the fault in a generally superior direction. This force transfers through the vomer to the rostrum of the sphenoid, generally moving the anterior portion of the sphenoid superiorly and causing the clivus near the sphenobasilar junction to move inferiorly. Challenge with various vectors to find the one that causes maximum weakening of a previously strong indicator muscle.

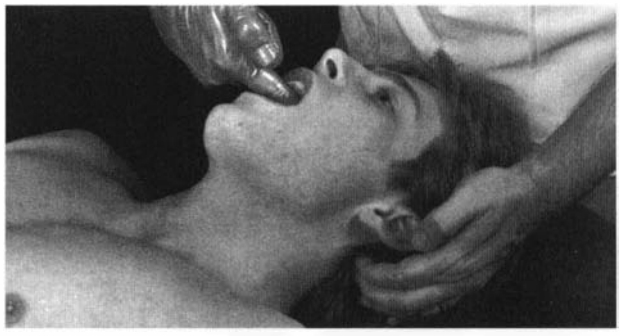
Generally, one can independently challenge the mastoid process and the palate. Just prior to treatment, challenge the two areas simultaneously to determine that there is maximum weakening of the indicator muscle.

Therapy Localization. The posterior component of the sphenobasilar inspiration assist cranial fault is usually at the occipitomastoid suture. The anterior portion of the fault will usually therapy localize at the cruciate suture of the palate, and at the pterygoid process of the sphenoid. This therapy localization point is probably due to strain between the pterygoid process and the pyramidal process of the palatine bone.

Correction. Pressure is applied simultaneously to the tip of the mastoid process and to the palatomaxillary suture area as the patient goes from full expiration to full forced inspiration. Repeat with four or five inspirations or until cranial movement is felt. Re-therapy localize and challenge to determine if correction was obtained.



9—17. Force applied at the intermaxillary or interpalatine suture is transmitted through the vomer to the rostrum of the sphenoid, increasing sphenobasilar extension. Simultaneous pressure is applied on the posterior mastoid tip in an anterior direction determined by challenge.



9—18. Pressure is applied simultaneously on the two areas in the direction of maximum challenge, which was determined together or independently for each area.

Sphenobasilar Expiration Assist Cranial Fault

The sphenobasilar expiration assist cranial fault is similar to the expiration assist cranial fault; however, it is locked more securely, as is the sphenobasilar inspiration assist. In the sphenobasilar expiration assist cranial fault, the sphenobasilar junction is in excessive flexion; therefore, it is identical to what has been called a "sphenobasilar flexion fault."

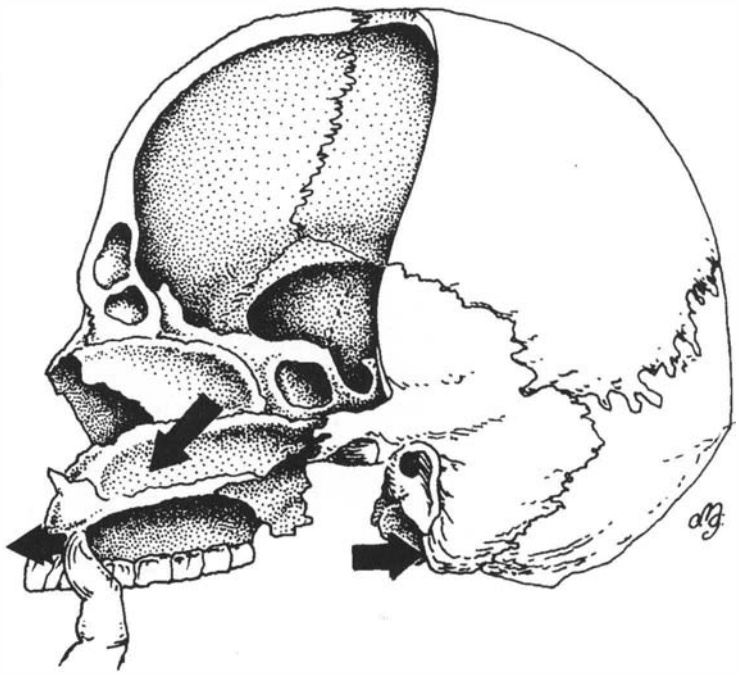
Breathing Pattern. A muscle weak because of a sphenobasilar expiration assist cranial fault will test strong on "forced expiration," i.e., the patient exhales to the maximum and then attempts to exhale more. A previously strong muscle associated with this fault will test weak on forced inspiration.

Challenge. Challenge is the same at the mastoid process tip as for the expiration assist cranial fault. The maxilla is challenged by contacting the central incisor on the side being evaluated, or immediately above it on the maxillary bone. A vector of force in a generally anterior direction is applied, and a previously strong indicator muscle is tested for weakening. Apply the force in various vectors until one is found that causes maximum indicator muscle weakness. One must take care that there is not a "neurologic tooth" (described later) causing the positive challenge. When the optimal positive challenge has been found at the tip of the mastoid process and the maxillary bone, challenge them simultaneously to determine that maximum weakening of the indicator muscle occurs.

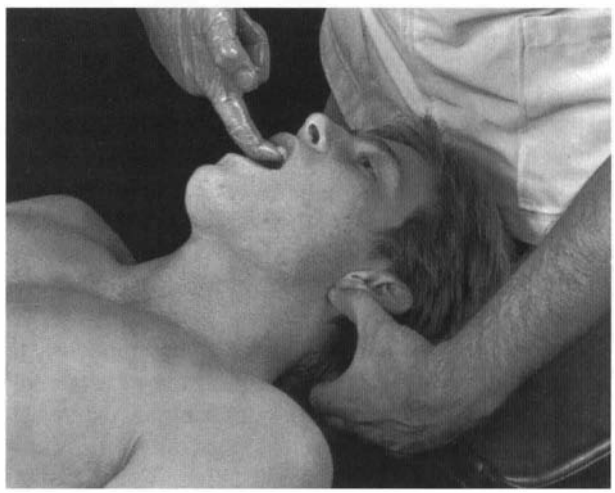
Therapy Localization. The most common areas for positive therapy localization are at the occipitomastoid and cruciate sutures, and at the pterygoid process junction with the pyramidal process of the palatine bone.

Correction. Correction is applied simultaneously at the two contact points while the patient goes through forced expiration. This is repeated with four or five expirations, or until the physician feels movement in the cranium. Re-evaluate with challenge and therapy localization to determine the effectiveness of the corrective attempt.

Combined Sphenobasilar Inspiration and Expiration Assist Cranial Faults. If the cranium is first treated for inspiration and/or expiration assist cranial faults and additional correction is still needed, there will usually be a sphenobasilar assist fault on only one or the other side. If there are bilateral faults the same inspiration or expiration assist will usually be present bilaterally. It is possible that further treatment of sphenobasilar faults following correction by inspiration and/or expiration faults will not correlate with the previous treatment. Always challenge and treat accordingly.



9—19. Direction of pressure on the mastoid process is determined by challenge the same as for an expiration assist correction. The anterior portion of the sphenoid is pulled anteriorly and inferiorly by anterior pressure on incisors to increase sphenobasilar flexion for a rebound into extension. Connection from the maxillae to the sphenoid is by way of the vomer.



9—20. Vectors are applied in the exact direction of challenge which caused the maximum weakening of a strong indicator muscle. Challenge may be accomplished one area at a time, or simultaneously.

Glabella Cranial Fault

The glabella cranial fault is characterized by a previously strong indicator muscle weakening as a result of the patient breathing, either through his mouth or nose. The breathing pattern of other cranial faults seems to correlate with positioning the bone into a more optimal state of function. It is unknown why the glabella cranial fault reacts as it does to oral or nasal inspiration. It may relate to the respiration influencing the bones making up the nasal cavity.

Breathing Pattern. To test for a glabella cranial fault, the patient takes a deep phase of inspiration through either the nose or mouth and holds it while a previously strong indicator muscle is tested for weakening. There will not be weakening on both oral and nasal inspiration; it is more common for the weakening to develop with oral inspiration. It is best for the examiner to pinch the patient's nose closed during oral inspiration to ensure that all air is coming through the mouth. When nasal inspiration is positive, there is usually history of trauma to the nose.

Challenge. Challenging with various vectors of force is not as important in the glabella cranial fault. Generally there is a positive challenge when pressure is applied to the glabella and the external occipital protuberance in a direction to approximate the two. If there is lack of significant weakening of the indicator muscle, challenge with a vector of force slightly away from the center line.

Therapy Localization. Therapy localization will be positive when the patient touches the glabella with the fingers of one hand and the external occipital protuberance with the fingers of the other hand.

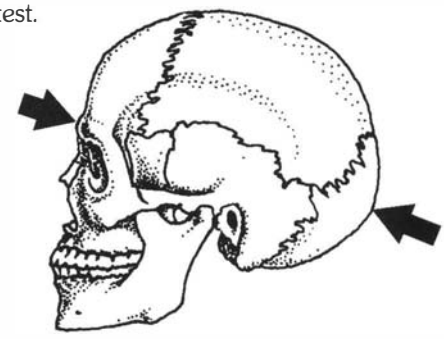
Correction. Correction requires two steps.

Step 1. Pressure is applied at the glabella and external occipital protuberance in the direction of optimal challenge. The two hands are pressed toward each other on either the oral or nasal inspiration that did not cause a strong indicator muscle to weaken. This is done four or five times, or until the physician feels skull movement.

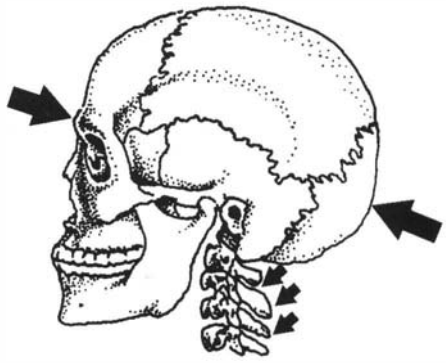
Step 2. The physician continues to contact the external occipital protuberance with the 5th digit of his hand, with the index, middle, and ring fingers contacting the spinous processes of the 3rd and 2nd cervical vertebrae and the posterior arch of the 1st cervical vertebra, respectively. In addition to applying pressure on the external occipital protuberance and the glabella as in the first step, simultaneous caudal pressure is applied to the cervical vertebrae spinous processes and posterior arch with inspiration. Again repeat four or five times. Re-evaluate with therapy localization, challenge, and oral or nasal inspiration to determine if the corrective attempt was effective.

The glabella cranial fault has been associated with hypertension. There is often a drop in diastolic blood pressure of 10-20 mm Hg in idiopathic hypertension immediately following correction.

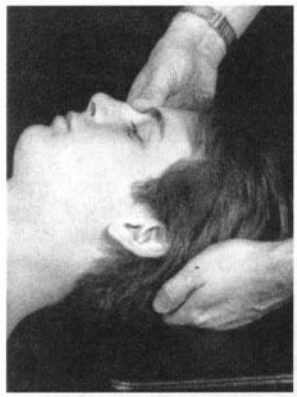
Correlating Sacral Fault. Approximately 60% of the time there will be a sacral fault with the same respiratory pattern. With the patient prone, test the hamstrings on both nasal and oral inspiration. If a sacral fault is present, it should be on the same oral or nasal inspiration as the cranial fault. Correct with anterior pressure on the sacral apex on the oral or nasal inspiration that did not weaken the hamstrings. Repeat four or five times and re-test.



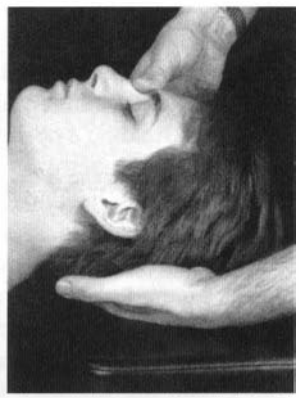
9—21. Step 1 approximates the glabella and external occipital protuberance.



9—22. Step 2 adds inferior movement of the three upper cervical vertebrae.



9—23. Step 1.



9—24. Step 2.

Temporal Bulge Cranial Fault

The temporal bulge cranial fault is an external rotation of the squamous portion of the temporal bone, with the parietal and other bones on that side of the skull following suit and causing the general appearance of a bulge. Early in applied kinesiology, the temporal bulge cranial fault was referred to as a "banana head." The skull generally appears to have a convexity on the side of the temporal bulge and a concavity on the opposite side where there is often a parietal descent cranial fault (described next), giving the appearance of a curve like a banana.

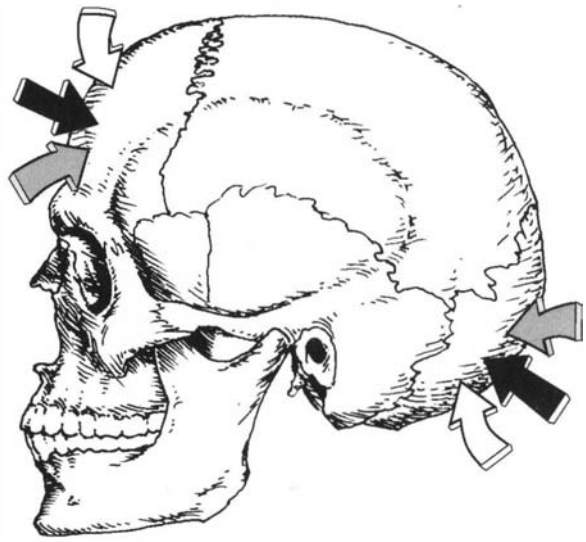
Muscle Association. The pectoralis major (clavicular division) muscles will often test weak when tested together; there may or may not be individual muscle weakness. It is unknown why this muscular pattern tends to exist with a temporal bulge cranial fault. It has been associated with hypochlorhydria in applied kinesiology, but this is not a 100% correlation.

Breathing Pattern. The bilateral pectoralis major (clavicular division) muscle weakness will strengthen when the patient holds approximately one-half inspiration. This is accomplished by having the patient exhale, take a portion of a breath in, and stop breathing while the muscles are tested. Sometimes the phase of inspiration held must be very specific. If there is failure of the bilateral muscles to strengthen, have the patient take a small amount of inspiration and hold it while the muscles are tested. If there is no strengthening, have the patient take in a little more breath and hold it, and re-test the muscles. Continue doing this until the exact phase of respiration is found that strengthens the muscles.

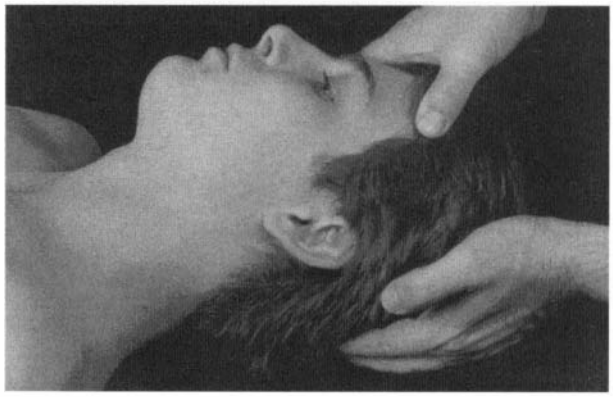
Challenge. Challenge is directed to the frontal and occipital bones in a direction to accentuate the temporal bulge. Pressure is applied to the frontal and occipital bones simultaneously toward the center of the temporal bone, with some lateral vector. The first challenge is usually with the examiner's thumbs moving toward each other. Three-dimensional challenge is done by adding an inferior component to one thumb and a superior one to the other. Reverse this challenge to apply the opposite rotation. One vector of force will provide the greatest weakening of a previously strong indicator muscle.

Therapy Localization. Therapy localization is very generalized in this cranial fault. It is best to identify the fault by holding a portion of inspiration to strengthen weak bilateral pectoralis major (clavicular division) muscles, and by challenge to weaken a previously strong muscle.

Correction. The two-handed vector of force that causes the greatest weakening of a previously strong indicator muscle is the direction of pressure to apply for correction. Corrective force is applied as the patient inspires, with the strongest force applied at the point of inspiration causing the bilateral pectoralis major (clavicular division) muscles to strengthen. As with other cranial faults, pressure should be mild and work with the cranium, not attempting to force it. After correction, re-test the pectoralis major (clavicular division) muscles, which should be strong; there should be no positive challenge.



9—25. The various arrow combinations indicate vectors of challenge for the temporal bulge cranial fault. The black arrows are challenged together for one torque into the cranium, the gray arrows for another, and the clear arrows for yet another.



9—26. Pressure is exerted in the direction of optimal challenge. Maximum pressure is exerted in the half-breath phase of inspiration.

Parietal Descent Cranial Fault

The parietal descent cranial fault is most often present when there is a temporal bulge on the opposite side. First correct the temporal bulge and re-evaluate for a parietal descent. In some instances the parietal descent will be corrected along with the temporal bulge.

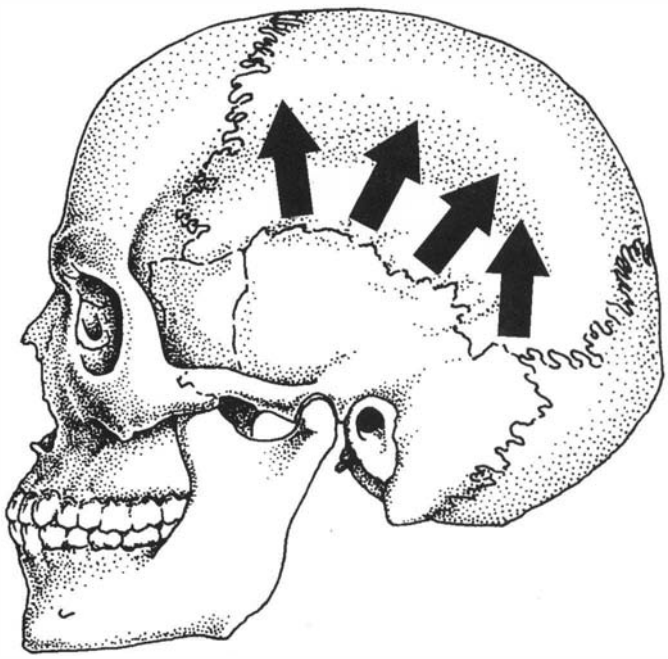
Muscle Association. Any muscle can be involved with the parietal descent fault. The muscles most often associated are the deep neck flexors, principally the scalene muscles.

Breathing Pattern. A muscle that tests weak as a result of a parietal descent cranial fault will strengthen on approximately one-half held expiration. As with the temporal bulge, the phase of held expiration may need to be quite specific to strengthen the associated muscle. If the muscle does not strengthen with an approximate one-half held expiration, have the patient exhale in small increments and hold his breath while the muscle is tested, until the phase is found that strengthens the muscle.

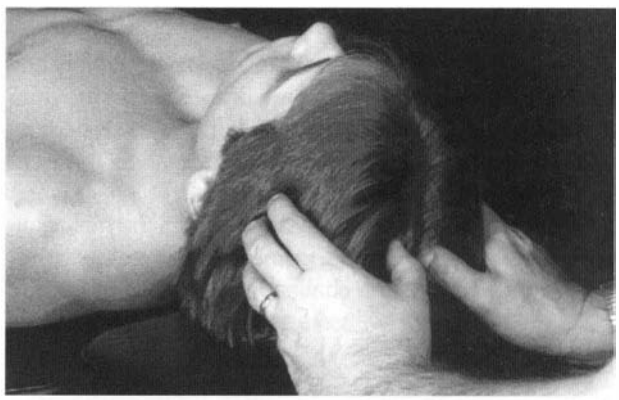
Challenge. Challenge for this cranial fault does not have the vectoring requirement of other cranial faults to determine the direction of correction. The usual challenge is to lift the temporal border of the parietal bone and observe for weakening of a previously strong indicator muscle. Sometimes a positive challenge is elicited by pushing the temporal border of the parietal bone inferiorly.

Therapy Localization. Several areas can show positive therapy localization with this fault. The best methods of fault identification are challenge and the breathing pattern strengthening an associated muscle.

Correction. The parietal descent cranial fault is corrected by contacting the parietal bone just superior to the temporoparietal suture. The parietal bone is lifted away from the temporal bone as the patient exhales. The greatest lifting is at the point of exhalation that caused the most strengthening of the weak associated muscle. A good precaution is to place the thumbs of both hands on opposite sides of the sagittal suture and spread it while lifting the parietal bone with the fingertips; this avoids jamming the sagittal suture. After correction, re-evaluate the cranium with challenge and the weak associated muscle for strengthening to determine if correction was obtained.



9—27. Because of the primarily sliding nature of the suture involved with this cranial fault, both a separating and an approximating challenge may be positive. The correction is always made by lifting the parietal away from the temporal bone.



9—28. Strongest lift of the parietal bone is generally done as the patient passes through one-half expiration. The sagittal suture is protected from jamming by the physician's thumbs separating it at the same time the parietal bone is lifted.

Internal Frontal Cranial Fault

The frontal bone is considered as if the metopic suture persists, thus making a right and left frontal bone. When the metopic suture ossifies, the movement remaining is that of living, flexible bone. An internal frontal fault refers to the metopic suture area rotating internally, with the squamous portion of the frontal bone moving laterally. This terminology is opposite that of DeJarnette's sacro occipital technique.[11,12] In the frontal faults, as in most cranial faults, numerous skull bones are involved.

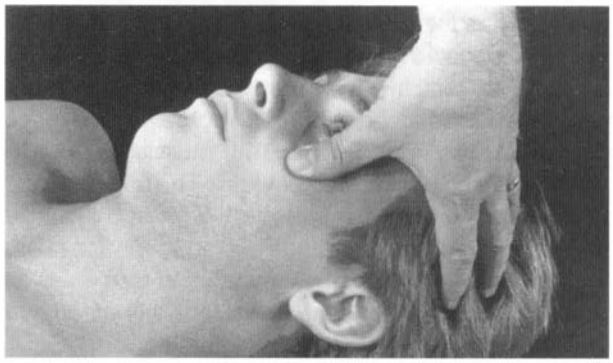
9—29. Internal frontal bone rotation as defined in applied kinesiology.

Muscle Association. The deep neck flexors and sternocleidomastoid muscles are often weak in the presence of an internal frontal cranial fault. There is no consistent breathing pattern that will strengthen the neck flexors when frontal faults are present.

Challenge. Challenge is directed to the malar surface of the zygomatic bone, primarily in a medial, slightly posterior direction. The challenge pressure may need to be redirected in several vectors to obtain weakening of a previously strong indicator muscle.

Headaches characterized by "pain behind the eye" are often associated with a frontal bone cranial fault. The patient will frequently have considerable tenderness when digital pressure is applied to the eye with the eyelid closed. Relieving this tenderness by applying pressure to the general area of the cruciate suture is a modified type of challenge to find the optimal vector of correction.

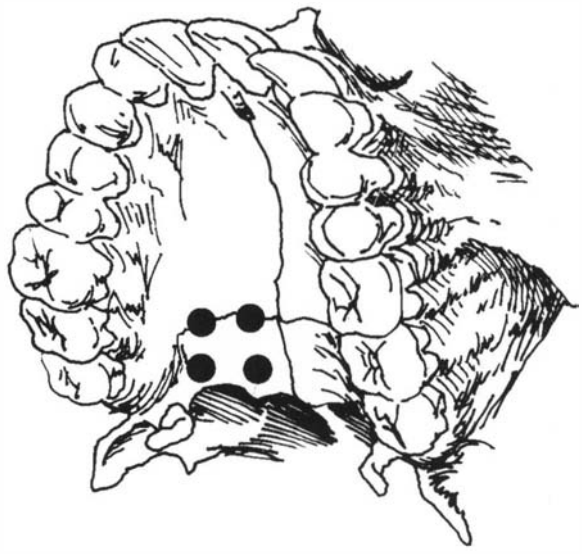
Therapy Localization. The most common area for positive therapy localization is at the pterygoid process. The therapy localization appears to evaluate strain between the pterygoid process of the sphenoid bone and pyramidal process of the palatine bone.



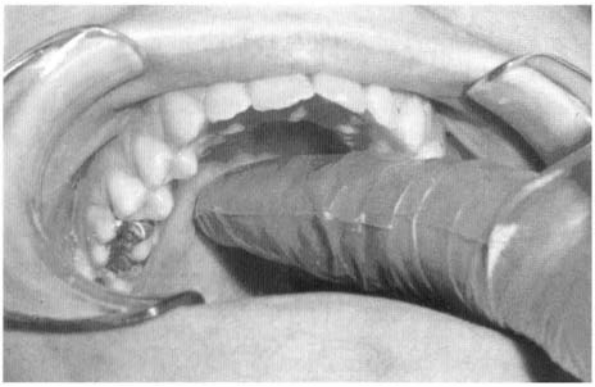
9—30. Challenge to malar surface of zygomatic bone is best method for determining whether an internal frontal fault is present.

Correction. Correction is made in three steps.

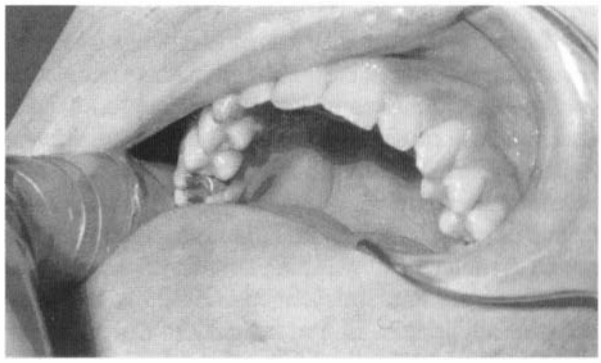
Step 1. Find the maximum tenderness on one of the eyes with digital pressure. The physician applies digital pressure with the index finger of his other hand to the posterior aspect of the palate on the side of internal frontal rotation. Pressure is held while eye tenderness is re-evaluated. A specific point and vector of force will relieve the eye tenderness, usually almost entirely. Generally the applicable pressure on the palate is mostly superior, but it can have a major aspect of lateral, medial, posterior, or anterior direction. When the maximum relief of eye pain has been obtained, hold the pressure



9—31. Optimal contact to relieve eye pain may be at any of the dots. Vary the vector of force at various locations until maximum relief of eye pain is obtained.



9—32. Illustrations are for a right internal frontal fault. Step 1: Hold pressure in direction that relieved eye pain.



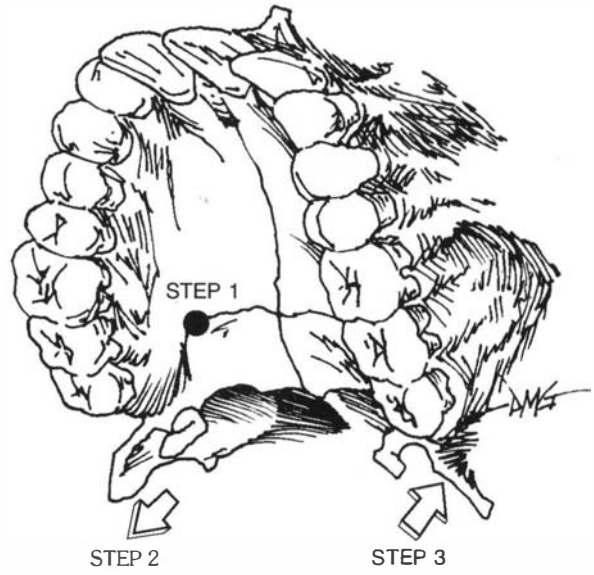
9—33. Step 2: Buccal approach to lateral pterygoid plate. It may sometimes be better to contact the medial plate with a lingual approach.

(3-4 pounds) on the palate for 20-40 seconds, or until the skull yields to the pressure. Sometimes a pulsation of the palate tissue is felt, which is also an indication of effective release. It is important to obtain relief of the eye pain by a specific vector of force. It is speculated that the relief is due to released strain of the seven bones making up the bony eye orbit. If there is no pain on digital pressure to the eye, apply the force to the palate in a generally superior direction.

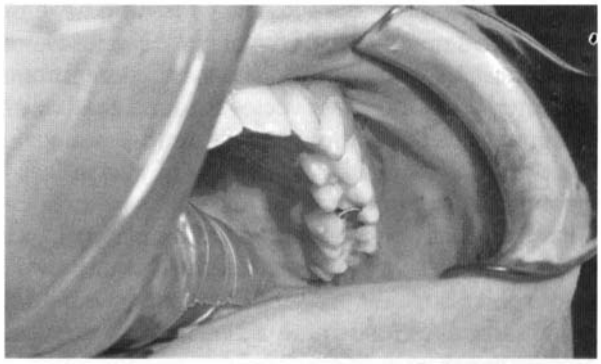
Step 2. Contact the pterygoid process on the side of internal rotation and pull it inferiorly. Contact of the pterygoid process is usually easier on the buccal side, with the physician's finger slipping between the cheek and the pterygoid process to hook on the lateral pterygoid plate. It may be necessary to contact the pterygoid process from the lingual side when the patient's pterygoid process is positioned medially. In some instances the physician can obtain the best contact with two fingers, one placed buccally and the other lingually. Hold the caudal pressure on the pterygoid process for 10-20 seconds, or until movement of the bone is felt. Considerable experience is necessary to feel this motion. This contact point is often very tender to the patient, so only gentle contacts should be made.

Step 3. The pterygoid process on the side opposite internal frontal rotation is contacted on the distal aspect of the pterygoid process, and pressure is applied in a superior, slightly posterior direction. Again, hold the pressure for 10-20 seconds, or until motion of the sphenoid bone is felt.

Re-test the associated weak muscles for strengthening and challenge the zygomatic bone to determine if correction was obtained.



9—34.

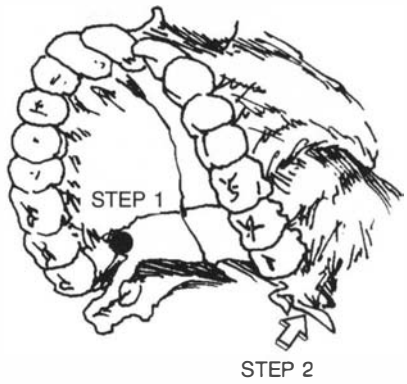


9—35. Step 3: Lingual approach to inferior pterygoid plates.

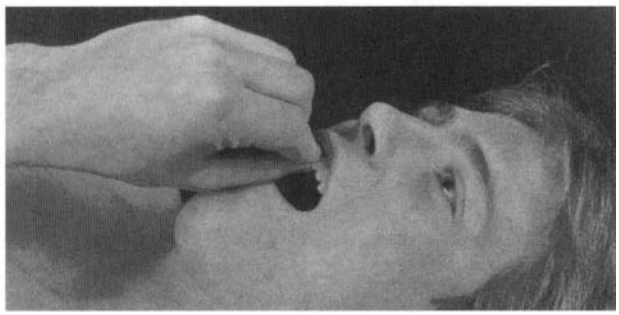
External Frontal Cranial Fault

Movement causing an external frontal cranial fault is based on the frontal bone hinging at the metopic suture area as if there were two frontal bones. After the metopic suture ossifies, movement is that of living, flexible bone. The metopic suture area moves externally, and the lateral border of the frontal squama moves medially and posteriorly. The ethmoid notch narrows slightly. As with the internal frontal cranial fault, this movement is exactly opposite that described in DeJarnette's sacro occipital technique.[11,12]

Challenge. To challenge for an external frontal fault, the patient grasps the central incisor on the side being tested and pulls it caudally. A previously strong indicator muscle weakens when the challenge is positive. One must rule out a positive neurologic tooth (described later).



9—37.



9—36. Patient or examiner places an inferior challenge on the central incisor and releases it; then an indicator muscle is tested for weakening.

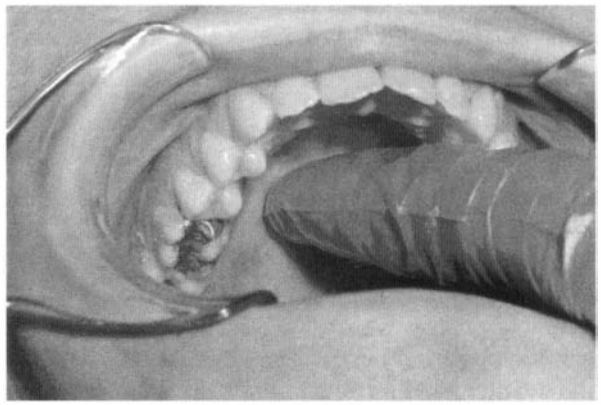
Therapy Localization. The most common area for positive therapy localization is at the pterygoid process. The therapy localization appears to evaluate strain between the pterygoid process of the sphenoid bone and pyramidal process of the palatine bone.

Correction. Correction is accomplished in two steps.

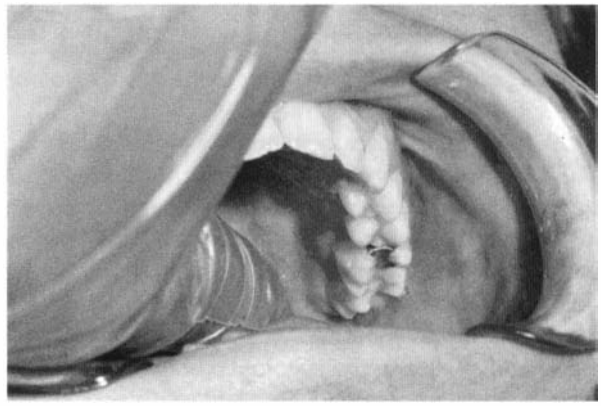
Step 1. As with the internal frontal fault, there will usually be eye pain on digital pressure. The physician continues to monitor the eye pain while his other hand applies pressure to the hard palate in the general area of the cruciate suture on the side opposite external frontal rotation. When the exact vector is found that causes maximum relief of eye pain, pressure (3-4 pounds) is held for 20-40 seconds, or until the physician feels a slight movement or pulsation.

Step 2. The inferior portion of the pterygoid process is contacted on the side of external frontal bone rotation, and a superior pressure (3-4 pounds) is held for 10-20 seconds or until the physician feels the pterygoid process move.

Re-test the associated weak muscles for strengthening and re-challenge at the central incisor to determine if correction was obtained.



9—38. Illustrations are for a left external frontal fault. Step 1: Apply pressure that relieved eye pain.



9—39. Step 2: Pressure applied to the inferior pterygoid plates.

Nasosphenoid Cranial Fault

Treatment of the nasosphenoid cranial fault indirectly influences sphenoid bone motion in the cranial closed kinematic chain. Although challenge and treatment pressure are applied in the general area of the nasal bone, the force is primarily being applied to the frontal or maxillary bone or both to influence the sphenoid. Effective correction of the nasosphenoid cranial fault removes or reduces the tenderness at the sphenoid sutures with other bones.

The nasosphenoid cranial fault is associated with overt or subtle endocrine problems that may be associated with dysfunction of the pituitary.[28] There is often sacral and/or coccygeal pain associated with this fault. Since sphenoid motion is a key in proper cranial function it is good policy to examine for this fault whenever there is dysfunction in the cranial primary respiration, and it is quick and easy to do.

Therapy Localization. As with most other cranial faults there is no consistent area of positive therapy localization that is pathognomonic for a nasosphenoid fault. Therapy localization to the pterygoid process and palatine pyramidal process articulation is most often positive with any fault of the sphenoid, but that does not indicate the best method of correction. Challenge remains the best diagnostic method for both determining the fault is present and the best method of correction.

Palpation and Observation. When this fault is present the sphenoid is tilted in the transverse plane, i.e., it is elevated on one side and depressed on the other. On the side of elevation there will be tenderness at the lower portion of the sphenoid greater wing along the TS line. On the side of depression the tenderness is at the sphenoid greater wing upper suture, i.e., at the TS line. The pterygoid pocket feels tight to palpation on the sphenoid high side.

Generally, the eye on the sphenoid high side will be more prominent, appearing to bulge slightly. The best way to observe this is to observe from close to the patient's abdomen toward his eyes.

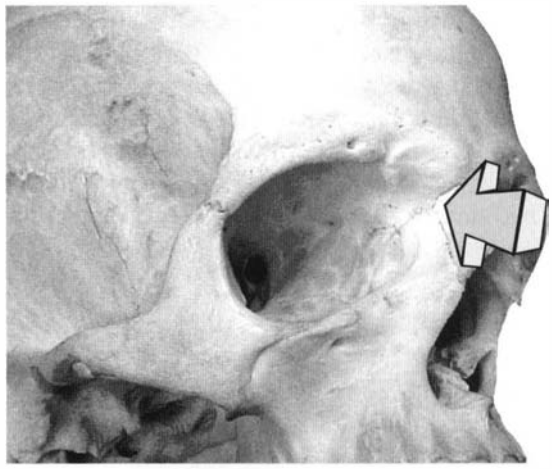
Challenge. Light thumb or index finger pressure is applied on the high sphenoid side to the nasal area generally toward the top of the opposite TS line. Weakening of a strong indicator muscle is a positive test. The vector of pressure is varied until maximal weakening of the indicator muscle is found, which will be the best vector of pressure for optimal treatment.

Observe in illustration 9—40 that your vector of force is probably going mostly into the frontal or maxillary bone. Changing the force vector directs pressure more into one bone or the other. It is for this reason that the optimal vector of correction varies considerably from patient to patient. When the optimal challenge is found, have the patient take a deep inspiration and re-test. Usually inspiration will immediately abolish the positive challenge; if not, test for expiration abolishing the positive challenge.

Correction. Treatment pressure is applied in the direction of positive challenge on the phase of respiration that strengthened the weakened indicator muscle. As with other cranial faults the pressure is applied gently. After several applications of treatment pressure with respiration one should sense a subtle release, indicating correction effectiveness.

Sometimes it is effective to use a two-hand contact to correct this fault.[14] In addition to the nasal contact the sphenoid greater wing can be contacted on the low sphenoid side and lifted simultaneously with the nasal pressure. An alternate method is to lift superiorly on the inferior pterygoid process. Both of these contacts may be very tender and should be approached cautiously.

Finally, re-test the patient with various vectors of challenge which should now be negative. If not re-evaluate and re-treat, possibly with the two-hand technique.



9—40. Rebound challenge is directed to the nasal area on the high sphenoid side. The vector of pressure is varied until there is maximum weakening of the strong indicator muscle. Determine phase of respiration that abolishes the positive challenge. Apply treatment pressure on phase of respiration that abolished the positive challenge.

Sacral–Coccyx Association. There is often a sacrococcygeal fault associated with the nasosphenoid fault. There will be positive therapy localization at the sacrococcygeal junction that is abolished with the same phase of respiration as the nasosphenoid fault. In the case of inspiration assist, treatment is directed toward the sacral apex and coccyx base from posterior to anterior with inspiration. If an expiration assist, lift the sacral apex posteriorly and move the coccyx base anteriorly on expiration.

Universal Cranial Fault

The universal cranial fault was originally called the "interosseous cranial fault" in applied kinesiology. The universal cranial fault is counterrotation of the sphenoid and occipital bones about the sagittal axis. When this occurs, there will be associated rotation of the temporal bones on both sides.

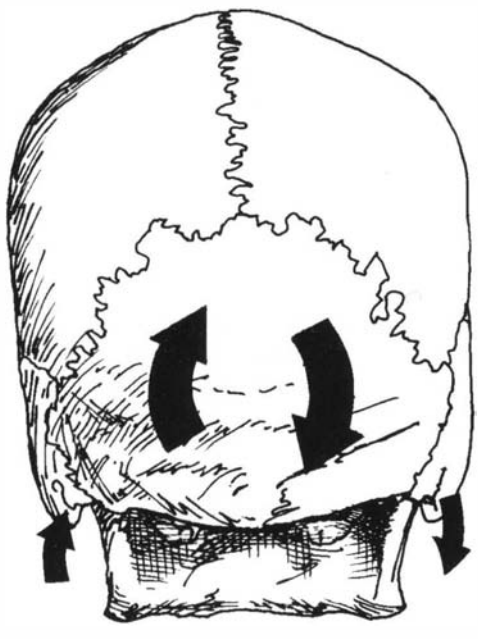
Breathing Pattern. The universal cranial fault is characterized by a previously strong indicator muscle weakening when the patient breathes through only one nostril. The examiner blocks one nostril and the patient takes a deep breath through the open side; a previously strong indicator muscle is tested for weakening. A muscle weak as a result of a universal cranial fault will strengthen when a patient breathes through one nostril and holds the inspiration while the muscle is tested. This will be the same nostril that caused a previously strong indicator muscle to weaken. This cranial fault must be differentiated from an ionization problem, which is discussed briefly on page 588. Challenge accomplishes the differentiation and provides information for correction of the fault.

Challenge. With the patient prone, one mastoid is pressed in a caudal direction and the other in a cephalad or superior direction. This is accomplished with the thumb and index finger of one hand. The physician's other hand contacts the occipital bone, challenging it with the same rotation as that applied to the mastoid processes. The cranium is challenged first with one rotation and then the other. Positive challenge is when a previously strong indicator muscle (usually the hamstring group) weakens; it will only be in one direction.

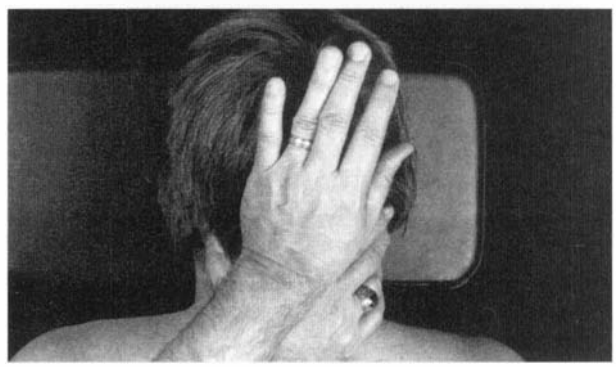
Therapy Localization. Therapy localization for the universal cranial fault was originally described as touching one side of the occipital bone with both hands. This will usually be positive with the universal cranial fault, but several other areas also often have positive therapy localization, such as along the lambdoidal and occipitomastoid sutures and the asterion. The best method of examination is challenge.

Correction. Unlike correction of other cranial faults, this correction is obtained opposite the direction that caused a previously strong indicator muscle to weaken. If one is able to find a muscle that tests weak due to the universal cranial fault, it will strengthen with the challenge opposite that which causes a strong indicator muscle to weaken. This indicates that this cranial fault is corrected directly rather than on a rebound basis, as are other faults.

Contact is made with the thumb and index finger on the mastoid processes, moving one superiorly and one caudally in the direction opposite that which caused a previously strong indicator muscle to weaken. The physician's other hand contacts the occiput and rotates it in the same direction as the mastoid processes. The torque to the cranium is applied with patient inspiration.



9—41. Pressure for challenge or treatment is applied on the occiput and mastoid process in the direction of the arrows, and then opposite the arrows. Contact on the occiput is kept low to avoid contact with the parietal bones.



9—42. The physician's left hand contacts only the occiput.

After the corrective attempt, re-challenge and test the patient for the breathing pattern to determine if correction was obtained.

Generally this correction is made with the patient's face in the split headpiece of an adjusting table, which may cause pressure to the facial bones. Avoid heavy pressure of the patient's face into the headpiece so that additional cranial faults are not created.

Sutural Cranial Faults

The sutural cranial faults are limited to specific sutures. Generally they are examined for after the previously discussed cranial faults have been corrected, since sutural faults are often corrected simultaneously with other faults.

When a sutural cranial fault is present, it is because of either jamming or separation. There is a positive rebound challenge and phase of respiration that will abolish the positive challenge. The only exception to this is the sagittal suture cranial fault.

Only a few of the sutural cranial faults are considered here: the sagittal, squamosal, zygomatic, and lambdoidal. The same principles discussed here are applicable to other sutures, such as the coronal suture and smaller facial bone sutures.

Sagittal Suture Cranial Fault

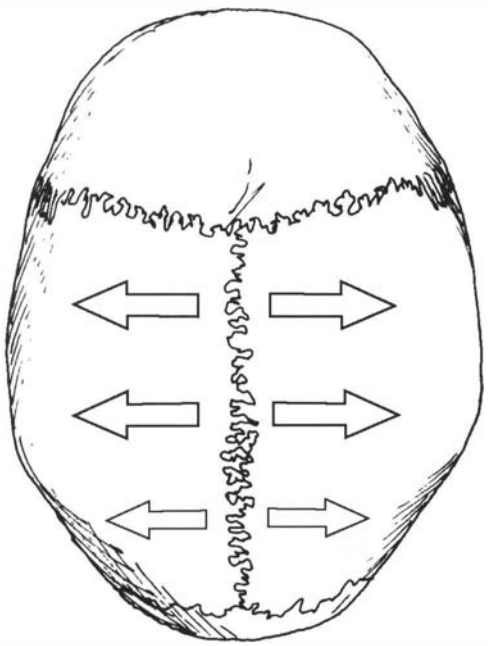
When there is dysfunction of the sagittal suture it is nearly always jammed, needing separation, and rarely ever separated. There is continuity of the fascia throughout the body,[61] and when there is postural distortion, the last pulling point of the fascia is at the vertex of the head. There will usually be tenderness at this area when there is a jammed sagittal suture.

Muscle Association. Weak abdominal muscles are often associated with the sagittal suture fault. This seems to be a vicious circle. When the abdominal muscles are weak, there is usually an increased lumbar lordosis contributing to pull on the fascia, culminating at the crown of the head to perpetuate the jamming of the sagittal suture. In addition to strengthening the abdominal muscles by correcting the sagittal suture, one should evaluate all other factors that may be causing the abdominal muscle weakness.

Challenge. Pressing the sagittal suture together will cause a general indicator muscle to weaken. Challenge is generally not necessary to determine if a sagittal suture cranial fault is present. One is usually aware of its possibility from observing weak abdominal muscles or tenderness across the crown of the head.

Therapy Localization. When the abdominal muscles are weak, therapy localization to the sagittal suture will cause them to test strong.

Correction. The physician uses the fingertips of both hands on each parietal bone along the sagittal suture. Pressure is applied to separate the suture. There is no specific breathing pattern associated with this fault to strengthen weak associated muscles; however, inspiration as pressure is applied appears to improve the correction.



9—43. Contact points for separation of sagittal suture. Care must be taken that contact is over area of involvement.

9—44. Sagittal suture separation.

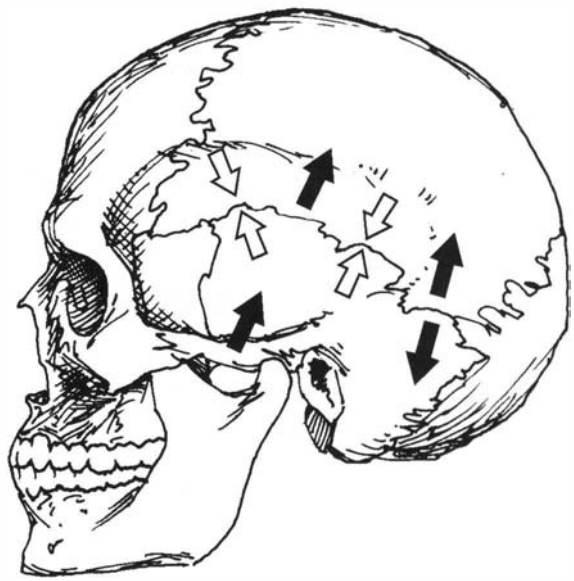
Squamosal Suture Cranial Fault

The squamosal suture cranial fault is either a jamming or a separation of the squamosal suture.

Challenge. The squamosal suture is challenged by pressing on the temporal and parietal bones adjacent to the suture in a direction of separation or approximation of the suture. A positive challenge is weakening of a previously strong indicator muscle. Immediately have the patient take an inspiration and re-test the muscle for strengthening. If it does not strengthen, have the patient exhale after obtaining a positive challenge and re-test for strengthening. One phase of respiration will abolish the positive challenge, usually inspiration.

Therapy Localization. When there is a squamosal suture cranial fault, therapy localization will be positive somewhere along the length of the suture; it may not be positive the entire length. Note the site of positive therapy localization for further evaluation after corrective attempts have been made.

Correction. Correction is obtained by pressing on the parietal and temporal bones in the direction of positive challenge while the patient takes the phase of respiration that abolished the positive challenge. Repeat this three or four times and re-test to determine if correction was obtained. Therapy localization is also a good method to determine if correction was obtained.



9—45. Challenge to separate or approximate the suture. Correct in the direction which weakens an indicator muscle, on the phase of respiration that abolishes the weakness.

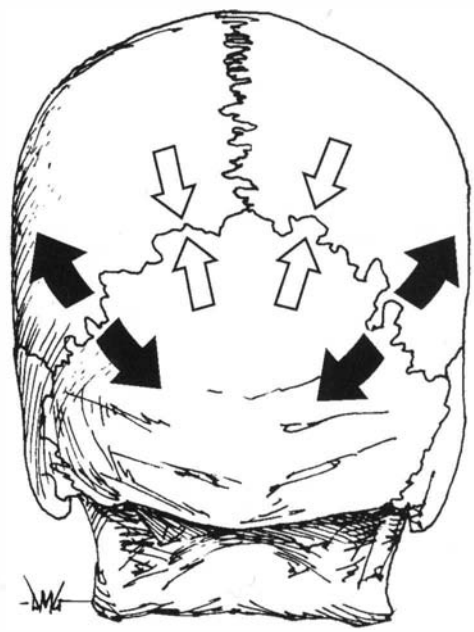
Lambdoidal Suture Cranial Fault

The lambdoidal suture cranial fault is often associated with a closed ileocecal valve syndrome. It is often corrected secondarily when one of the other, more complex faults is effectively treated.

Challenge. When the fault is present, a previously strong indicator muscle will weaken when the suture is separated or approximated. There may not be a fault along the entire suture. It may be necessary to challenge at several different areas before a positive challenge is found. A positive challenge will be negated when the patient holds inspiration or expiration.

Therapy Localization. There will always be therapy localization at some point along the lambdoidal suture when the fault is present. Note the area of positive therapy localization to re-evaluate after the corrective attempt is made.

Correction. Correction is obtained by separating or approximating the lambdoidal suture in the direction that caused a previously strong indicator muscle to weaken on challenge. Pressure is applied on the phase of respiration that abolished the positive challenge. Re-evaluate with challenge and therapy localization to determine if correction was obtained.



9—46. Challenge to separate or approximate the suture. Correct in the direction which weakens an indicator muscle on the phase of respiration that abolishes the weakness.

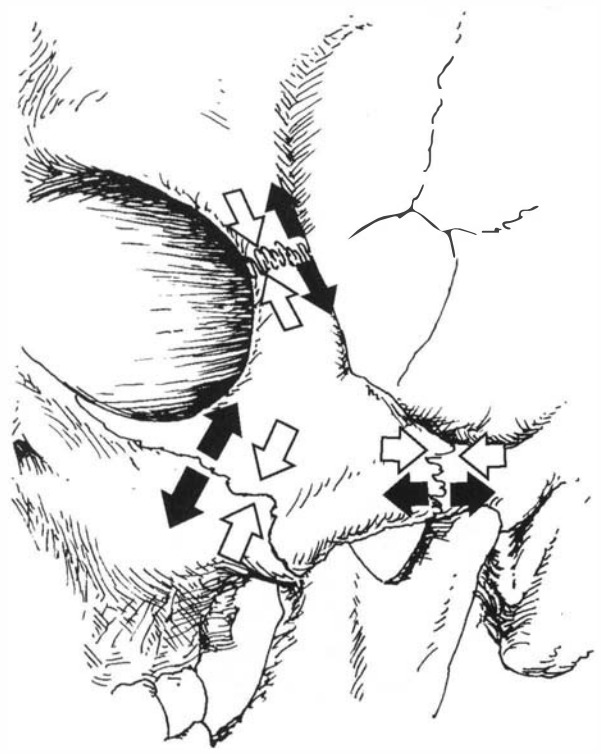
Zygomatic Suture Cranial Faults

There are three zygomatic cranial faults located at the temporozygomatic, zygomaticomaxillary, and frontozygomatic sutures. One, two, or all three of the zygomatic sutures may be involved. They are often associated with an open ileocecal valve syndrome.

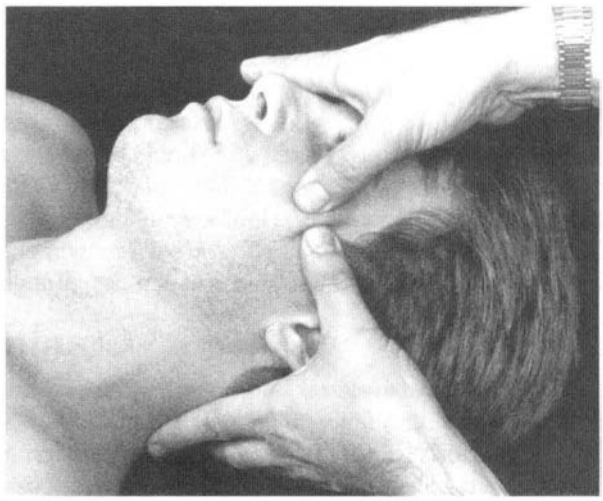
Challenge. The suture being evaluated is challenged for approximation or separation. A positive challenge occurs when a previously strong indicator muscle weakens. The muscle will regain strength when the patient inhales or exhales.

Therapy Localization. There will always be positive therapy localization over the suture when it is involved.

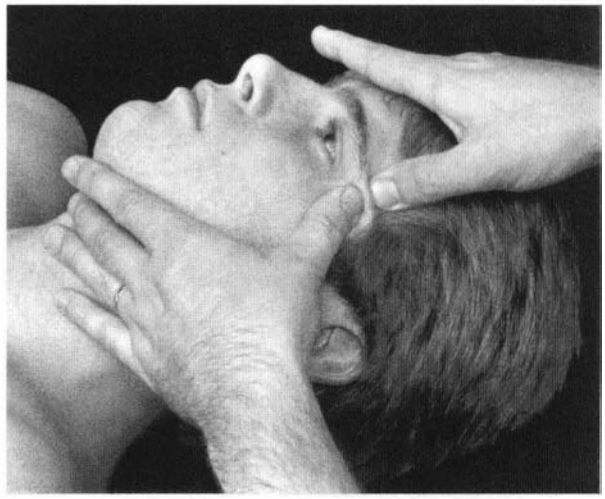
Correction. Correction is obtained by separating or approximating the suture as indicated by challenge, on the phase of respiration that abolished the positive challenge. When more than one zygomatic suture is involved, treat first the one that caused greatest weakening of the indicator muscle on challenge. Re-challenge additional sutures before treating, as correction of the first one sometimes corrects additional zygomatic suture faults. Re-therapy localize and challenge to determine that correction was obtained.



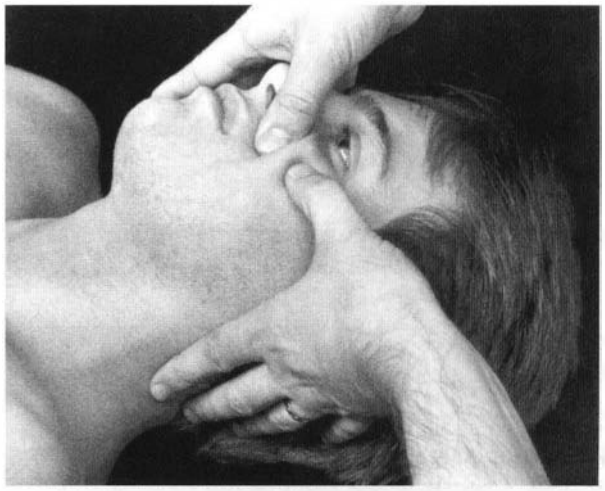
9—47. Challenge the zygomatic suture that shows positive therapy localization. Challenge for separation and approximation to find which causes an indicator muscle to weaken. Correct in the direction of positive challenge on the phase of respiration which abolished the weakness.



9—48. Correction of temporozygomatic suture.



9—49. Correction of frontozygomatic suture.



9—50. Correction of zygomaticomaxillary suture.

Custom Cranial Faults

The presentation here of fifteen cranial faults provides a basic background for treating the cranium with applied kinesiology principles. Each skull is unique, and there may be variation of the cranial faults from those discussed here. To further one's knowledge about cranial movement, a disarticulated skull should be obtained to study the types of sutures and their relationship with one another. This enables one to understand the role of the sutures and flexibility of bone in cranial movement. With experience, modifications of the fifteen cranial faults presented here can be made to effectively correct almost all cranial dysfunction.

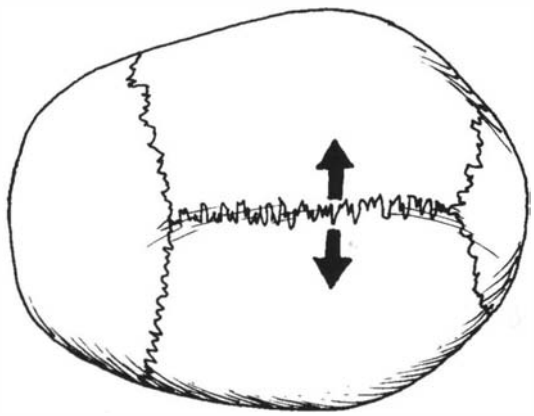
In addition to understanding cranial movement, one must correlate the integration of the rest of the stomatognathic system, the pelvis, and general structural function throughout the body. The cranial primary respiratory mechanism is only a portion of the total complex. When other areas dysfunction, effectively corrected cranial faults will probably return.

Cranial Summary

Simple Suture
Sagittal suture fault
Lambdoidal suture fault
Squamosal suture fault
Zygomatic suture fault

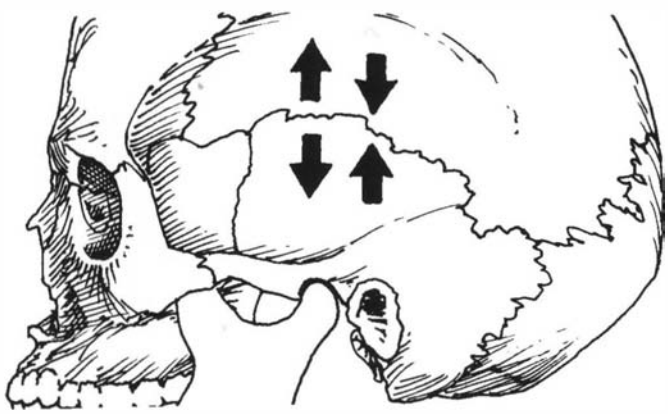
Flexion-Extension
Inspiration assist fault
Expiration assist fault
Sphenobasilar extension fault
Sphenobasilar flexion fault
Glabella fault

Rotation
Temporal bulge fault
Parietal suture fault
Internal frontal fault
External frontal fault
Nasosphenoid cranial fault
Universal cranial fault



Sagittal suture fault
9—51. No specific breathing pattern necessary, but helpful if done on inspiration.

Lambdoidal suture fault
9—52. Correct in direction of positive challenge on phase of respiration that abolished the weakness.

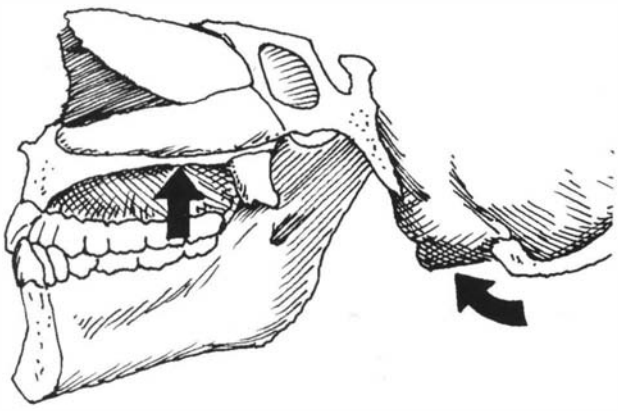


Squamosal suture fault
9—53. Correct in direction of positive challenge on phase of respiration that abolished the weakness.

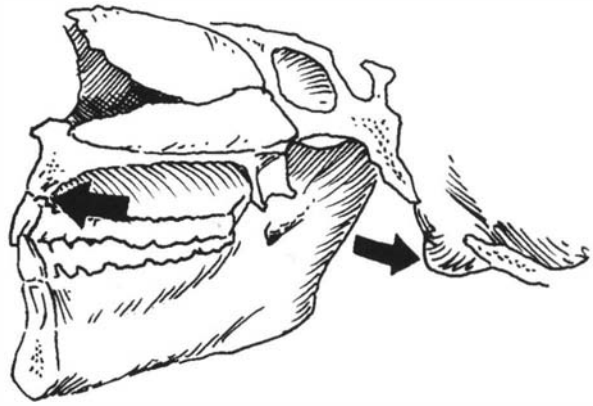
Zygomatic suture fault
9—54. Correct any zygomatic suture fault in direction of positive challenge on phase of respiration that abolished the weakness.

9—55. **Inspiration assist fault.** *Correct on inspiration.*

9—56. **Expiration assist fault.** *Correct on expiration.*



9—57. **Sphenobasilar inspiration assist fault.** *Correct on forced inspiration.*



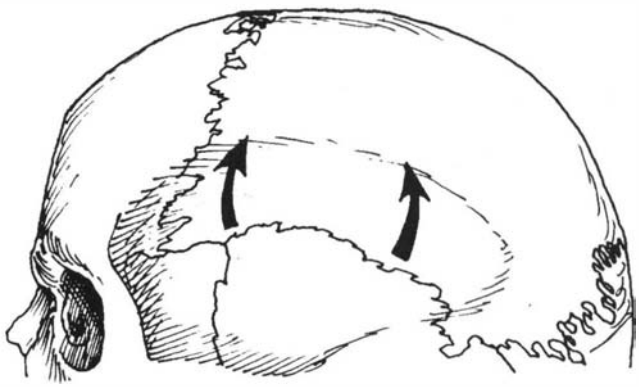
9—58. **Sphenobasilar expiration assist fault.** *Correct on forced expiration.*

Glabella fault

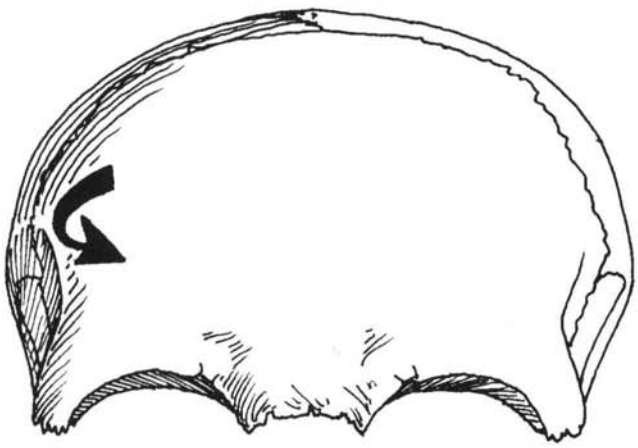
9—59. Step 1: Pressure on glabella and external occipital protuberance on oral or nasal inspiration which did not weaken the indicator muscle.

9—60. Step 2: Add inferior movement to atlas, axis, and 3rd cervical while continuing the first step.

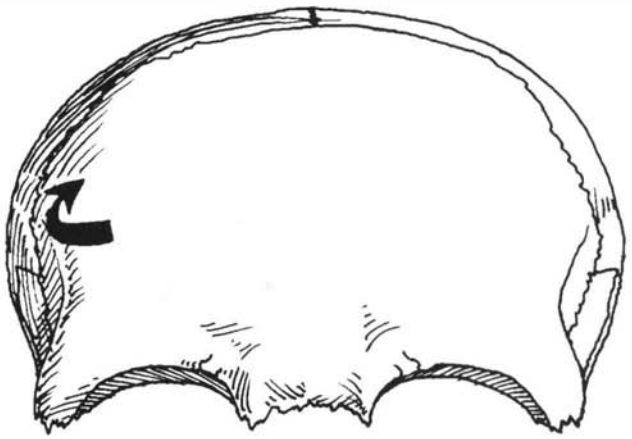
9—61. **Temporal bulge cranial fault.** *Correct on one-half inspiration in direction of positive challenge.*



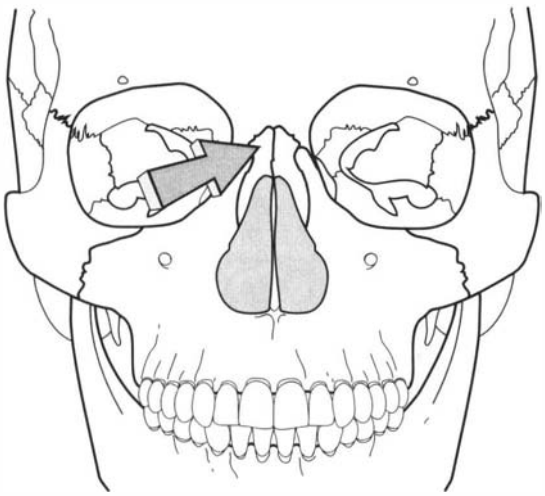
9—62. **Parietal descent cranial fault.** *Correct on one-half expiration while stabilizing sagittal suture.*



9—63. **Internal frontal rotation fault.** *Correct in three steps. Consult page 392.*



9—64. **External frontal rotation fault.** *Correct in two steps. Consult page 394.*



9—65. **Nasosphenoid cranial fault.** *Correct in direction of positive challenge, generally toward opposite TS line, on phase of respiration that abolishes the positive challenge.*

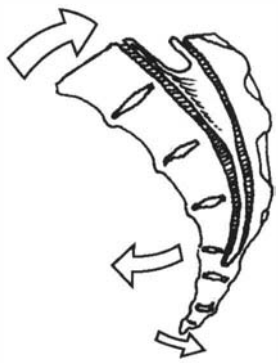
9—66. **Universal cranial fault.** *Correct in direction of challenge which made a weak muscle strong or opposite direction that made a strong muscle weak. Correct with 4 or 5 normal respirations.*

Sacral Respiratory Function

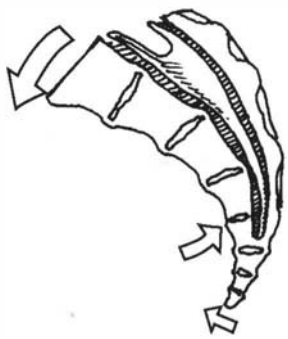
When cranial faults are corrected, there should be a routine evaluation of the pelvis because there will often be a category I or II. Examine and correct as discussed in Chapter 3.

The sacrum has a respiratory movement of flexion and extension in the sagittal plane. The sacrum's axis of rotation is in the general area of the 2nd sacral segment about the transverse axis. Organization between the cranium and the sacrum is via the dura mater. The dura mater attaches firmly at the foramen magnum and 2nd and 3rd cervicals, with only loose attachment until the dura mater attaches to the posterior portion of the 2nd sacral body. As the sphenobasilar junction flexes with inspiration the dura tightens, pulling on the anterior portion of the 2nd sacral segment to rotate the sacrum apex anteriorly and its base posteriorly. If there is failure of proper sacral motion, corrected cranial faults will often recur, even as quickly as when the patient walks.

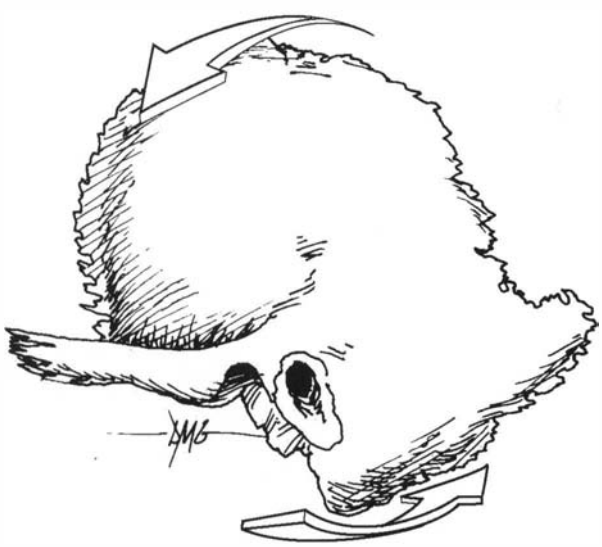
When there is sacral dysfunction, the piriformis muscle is often involved because it is an important sacral stabilizer. The psoas muscle may also be involved because it crosses the sacroiliac articulation. Evaluate these muscles for weakness or hypertonicity and make necessary corrections to help stabilize the sacral correction.



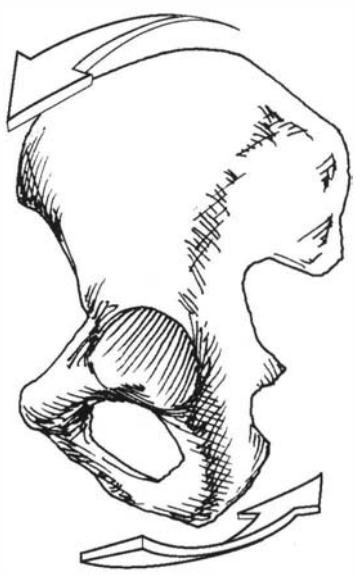
9—68. Inspiration of sacrum and coccyx.



9—69. Expiration of sacrum and coccyx.



9—67.



9—70.

There is a correlation of movement between the temporal and innominate bones. The arrows indicate movement on inspiration (sphenobasilar flexion).

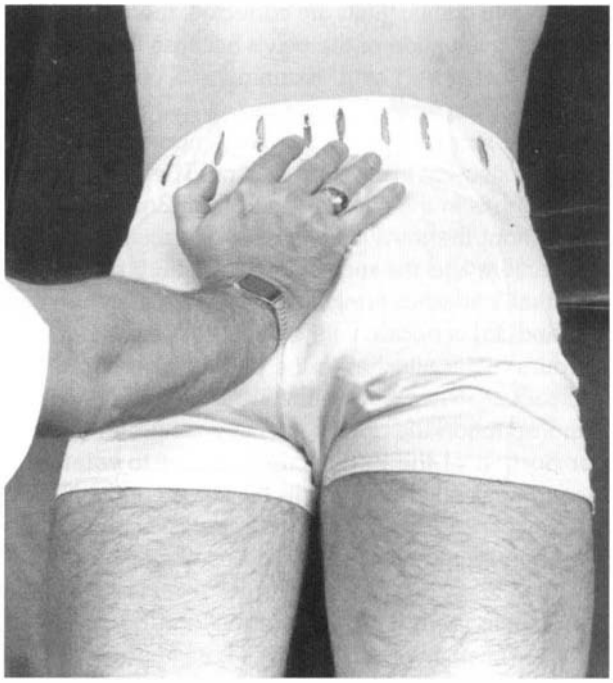
Sacral Inspiration Assist Fault

Breathing Pattern. If the hamstring muscles are weak due to a sacral inspiration assist fault, they will strengthen when the patient holds a deep inspiration. If the hamstrings are strong, they will weaken when a deep expiration is held.

Challenge. As with cranial faults, challenge provides information concerning optimal correction. On an inspiration assist, pressure is applied to the sacral apex to move it anteriorly. Apply several challenges with various lateral and medial vectors until maximum weakening of the indicator muscle is observed. If the hamstring muscles are weak, the piriformis may be used as an indicator muscle. If all muscles convenient to test are weak, treat them with some other aspect of the five factors of the IVF to gain at least temporary strength.

Therapy Localization. Therapy localization over the sacrum will generally be positive. Challenge is the best method of evaluation.

Correction. Correct in the direction of challenge that produced maximum weakness of the indicator muscle as the patient slowly takes a full phase of inspiration. Repeat four or five times, or until improved movement is felt. Re-evaluate with challenge and therapy localization to determine that correction was obtained.



9—71. Inspiration assist correction. First challenge for optimal vector, then correct with patient's inspiration until freedom of motion develops.

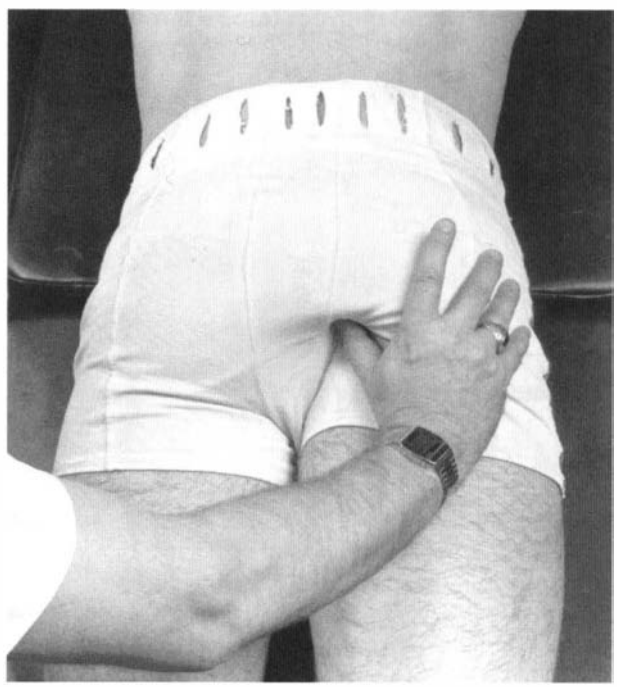
Sacral Expiration Assist Fault

Breathing Pattern. If the hamstring muscles are weak due to the sacral expiration assist fault, they will strengthen when the patient holds a deep exhalation. If the hamstrings are strong, they will weaken when an inhalation is held.

Challenge. The physician's thumb contacts the anterior portion of the sacral apex on one side. This contact is more easily made when the pelvic portion of a chiropractic adjusting table is elevated. If the expiration assist is bilateral, the physician contacts both sides of the sacral apex with his thumbs to lift the sacrum posteriorly. Apply several vectors of challenge until maximum weakening of the indicator muscle is observed. Sometimes a better challenge can be obtained by lifting the sacral apex posteriorly with one hand while the other hand pushes the superior aspect of the sacral base anteriorly.

Therapy Localization. There will generally be positive therapy localization over the sacrum, as in the sacral inspiration assist. Challenge and the respiratory correlation differentiate the faults.

Correction. Correct by applying the pressure that produced maximum weakening of the indicator muscle as the patient slowly goes through a full phase of expiration. Apply the force in the same way as optimal challenge, whether it is one- or two-handed. Re-evaluate with challenge and therapy localization to determine that correction was obtained.



9—72. Expiration assist. Challenge first for optimal vector of correction.

Sacral Wobble

This sacral involvement appears similar to the inspiration and expiration sacral faults, but it is more significantly locked and has more than the usual rotatory aspect. The sacral wobble obtained its name because the sacrum appears to wobble during walking when the fault is present. The sacral wobble fault will sometimes be corrected by category I or sacral inspiration or expiration treatment.

Breathing Pattern. Examine for sacral wobble when held inspiration or expiration strengthens the hamstring muscles, but challenge for a sacral fault is not positive.

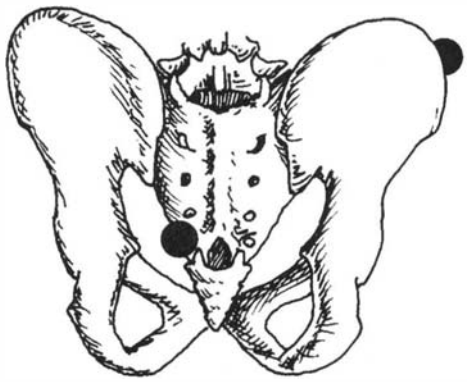
Challenge. For an inspiration assist sacral wobble, the sacrum is contacted at the apex on the side on which the associated hamstring muscle becomes strong with inspiration. The opposite anterior superior iliac spine (ASIS) is contacted with the physician's other hand; the challenge is to move the sacral apex and opposite ASIS toward each other. The challenge is repeated with various vectors to find the maximum positive challenge, which is a weakening of a previously strong indicator muscle.

To challenge an expiration assist sacral wobble, the anterior aspect of the sacral apex is contacted with the physician's thumb on the side on which the hamstring group strengthens with held expiration. The physician contacts the contralateral sacral base with the pisiform of his other hand. The two hands move somewhat toward each other, and the sacral apex is moved posteriorly while the base is moved anteriorly. This imparts a rotation to the sacrum for optimal challenge. Various vectors are tested until the challenge that causes the greatest weakening of an indicator muscle is found.

Both the inspiration and expiration assist sacral wobble faults are corrected in the direction of maximum challenge on the phase of respiration that caused strengthening of the hamstring group.

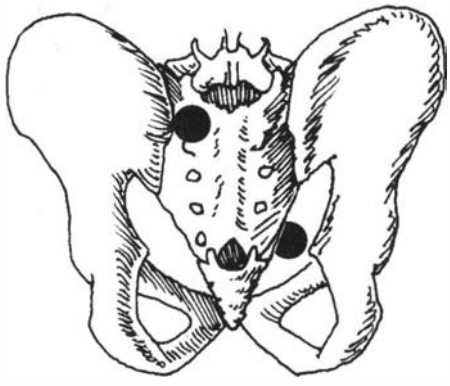
After the corrective attempt is made, re-evaluate with therapy localization and challenge to confirm the correction. The hamstring group should test strong.

The sacral wobble fault is often associated with basic postural distortions. One should re-evaluate posture after this correction is made and treat accordingly.

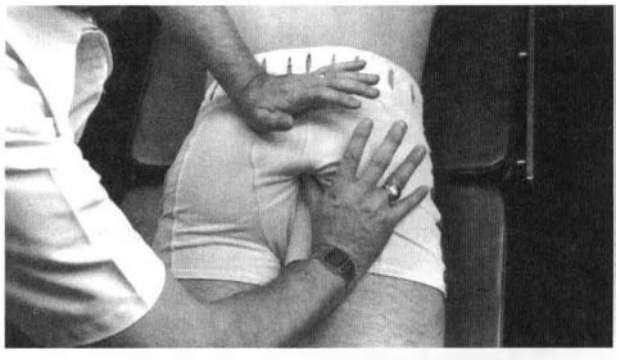


9 —73. Contact points for inspiration assist.

9—74. Sacral wobble inspiration assist.



9—75. Contact points for expiration assist.



9—76. Sacral wobble expiration assist.

Atlanto-Occipital Countertorque

The atlanto-occipital countertorque is often associated with cranial-sacral primary respiratory system dysfunction. It appears to relate with torsion of the dura mater between the cranium and upper cervical region. It is frequently associated with the sacral wobble and other sacral and pelvic faults.

Therapy Localization. Therapy localization is best done by simultaneously contacting numerous points. The patient uses four fingers, placing one each on the occipital bone, mastoid process, and the atlas and axis. Positive therapy localization in this manner indicates probability of atlanto-occipital countertorque, especially when the individual areas do not show positive therapy localization.

9—77. One finger is on the occipital bone, one on the mastoid process, and one each on the atlas and axis for therapy localization of atlanto-occipital countertorque.

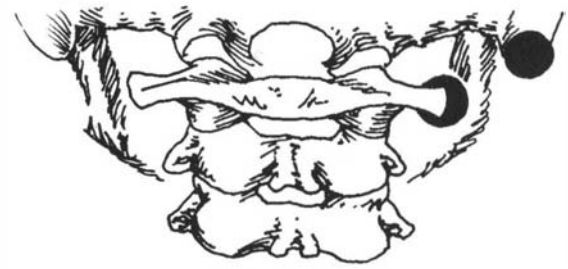
Breathing Pattern. The breathing pattern of atlanto-occipital countertorque is observed when therapy localization is positive. Either held inspiration or held expiration will eliminate the positive therapy localization.

Challenge. Contact points for challenge are the mastoid and transverse processes of the temporal bone and atlas vertebra. When positive therapy localization is abolished by inspiration, the physician presses anteriorly on the tip of the mastoid process and posteriorly on the anterior portion of the atlas transverse process, then releases the pressure. A previously strong indicator muscle will weaken.

When positive therapy localization is abolished by held expiration, the physician contacts the anterior tip of the mastoid process and presses it posteriorly, while the posterior aspect of the atlas transverse process is pressed anteriorly. A positive challenge is weakening of a strong indicator muscle.

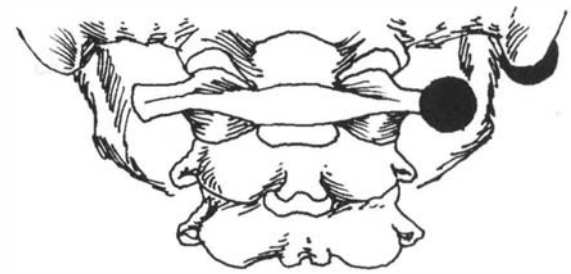
Either inspiration or expiration challenge should be evaluated with various vectors of force to determine which causes maximum weakening of the indicator muscle.

Correction. Correction is done on the phase of respiration that abolished the positive therapy localization, in the direction of optimal positive challenge. Pressure of 3-4 pounds is applied to the mastoid and atlas transverse process as the patient slowly takes the phase of respiration that abolished the positive therapy localization. Re-therapy localize and challenge to determine that the correction was effective.

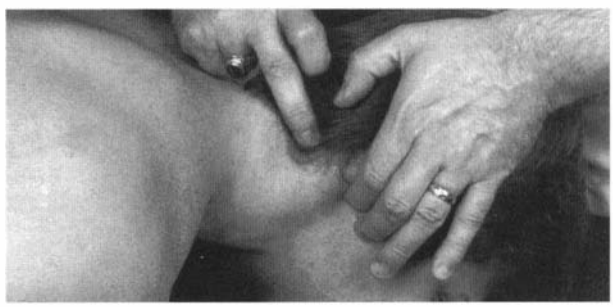


9—78.

9—79. Inspiration assist.



9—80.



9—81. Expiration assist.

Integration Within the Stomatognathic System

As previously discussed, the stomatognathic system integrates with function of the pelvis and spine, and those three divisions integrate with the rest of the body's actions. The integration can be narrowed down to specific functions, such as neurologic positive support action and gait proprioceptors of the feet. The positive support action maintains balance between the flexors and extensors of the body when upright, and the gait proprioceptors are important in maintaining proper temporal patterns of the sternocleidomastoid and upper trapezius muscles during walking. There is similar neurologic organization in the stomatognathic system when one considers it as being from the shoulder girdle up.

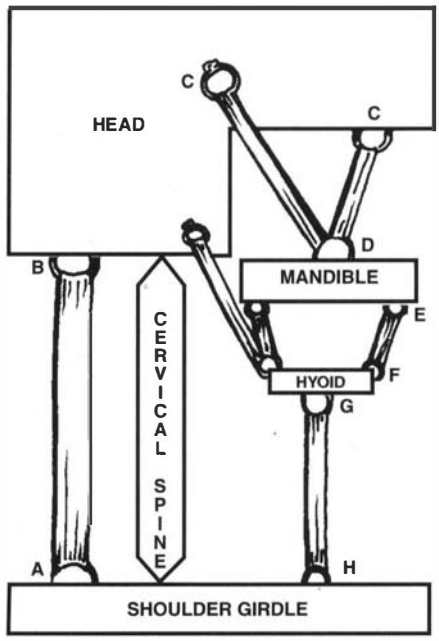
Stomatognathic System Closed Kinematic Chain

Within the stomatognathic system there is a closed kinematic muscular chain comprising the neck flexors and extensors, muscles of mastication, and hyoid muscles. Obviously when one flexes the neck the extensor muscles must relax, but only enough to allow movement so that control is maintained. Likewise, when an individual chews the mandible moves up and down; there must be relaxation and contraction of the hyoid muscles at the appropriate time during mandibular movement.

The closed kinematic chain is illustrated by a diagram originally distributed in 1949 through the University of Illinois.[10] It is amazing that more interest and attention have not been given to improving the balance of these structures and muscles since that time.

The activity of the posterior cervical group (A-B) must equal the activity of the anterior cervical group (C-H) for equilibrium to be present. Inhibition of the posterior group, with no change in the anterior group, will cause the head to tilt forward. Increased activity of the posterior group, with no change in the anterior group, will cause the head to tilt backward. Change in any section of the anterior group — whether it be the mandibular elevators (C-D), suprahyoid (E-F), or infrahyoid (G-H) — will influence the entire group. To maintain head equilibrium and have mandibular elevation, there must be contraction of the mandibular elevators concurrent with inhibition of the suprahyoid muscles; otherwise, the head would bob up and down when a person chews or talks because the anterior portion of the closed kinematic chain fails to equalize the posterior division.[77]

This integration takes place in many of the stomatognathic system's activities. During swallowing the hyoid bone must elevate, which is accomplished by the suprahyoid muscles pulling down on the mandible. Counteracting the swallowing activity is contraction of the muscles of mastication to bring the teeth together, making a solid base from which the suprahyoid muscles function. A similar action takes place during mastication. The mandible has wide, excursive movements, yet the hyoid bone is relatively stable. Thompson and Brodie[69] state, "This coordination permits of mastication, deglutition, and speech without any accompanying nodding of the head. We have been thinking of the mandible too much as a bone concerned with a single function, rather than as a connecting link in this anterior muscle chain. The muscle tension acting on the mandible is balanced; that is, there is just as much downward pull as there is upward pull on this bone."



9—82. Block diagram of the closed kinematic chain of the stomatognathic system. The sternocleidomastoid muscle has been left out for clarity. Redrawn and modified from the University of Illinois Telephone Extension Program, Current Advances in Dentistry, *1949.*

As we progress, the importance of proper neurologic control and balance of the muscles comprising the closed kinematic chain of the stomatognathic system will become apparent. Applied kinesiology provides the examination tool to determine the area of dysfunction so

that proper treatment can be applied. Primary areas in which dysfunction may occur include the cranial-sacral primary respiratory system, muscles of mastication, occlusion, hyoid muscles, cervical spine, and cervical muscles. Added to this are remote factors such as gait and postural balance, including the interaction of body modules.

An in-depth study of the stomatognathic system reveals that all three sides of the triad of health can cause dysfunction. Here we will discuss primarily the structural aspect; however, one should not forget that clenching the teeth and other mental reactions and chemical imbalances can cause disharmony within the stomatognathic system.

Examination of the stomatognathic system should include a screening of the cranial-sacral primary respiratory system and the divisions included in the rest of this chapter. The screening gives the physician an overview of dysfunction. Often when one begins to treat the stomatognathic system, corrections are easily obtained but rapidly lost. Knowledge of the interaction helps find the areas responsible for the loss of correction.

It is usually most effective to correct the cranial-sacral primary respiratory system first. When there is dysfunction in the stomatognathic system, the cranial-sacral primary respiratory mechanism is nearly always involved. It may be primary or develop secondarily as the result of some other dysfunction creating cranial faults. Correcting cranial faults and related dysfunction in the pelvis and/or spine is extremely productive because cranial nerves control the muscles of mastication, the hyoid and facial muscles, and some of the neck muscles.

Muscles of Mastication and Mandibular Movement

The mandible's position depends on balance of the sling-type arrangement of the muscles of mastication. Movement at the temporomandibular joint is by rotation of the condyle at the inferior aspect of the articular disc and translation of the disc in the mandibular fossa above, giving a great range of motion to the TMJ. The position of the condyle and disc in the mandibular fossa depends on muscle balance. Much of applied kinesiology's examination and treatment of temporomandibular joint dysfunction is related to examining the muscles for intrinsic function and interaction between the muscles. Disturbance of the muscle itself may be due to dysfunctioning neuromuscular spindle cells or Golgi tendon organs, active reflexes, meridian imbalance, or the need for fascial release or trigger point techniques.

The neurolymphatic and neurovascular reflexes, stress receptor, and meridian association are the same for all muscles of mastication.

Neurolymphatic:

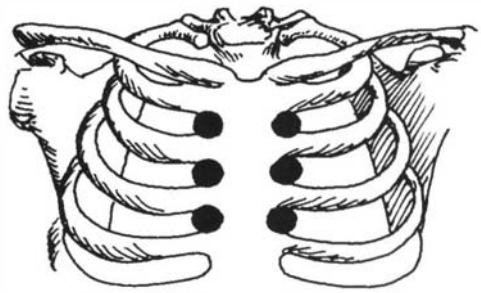
Anterior: 2nd, 3rd, and 4th intercostal spaces adjacent to the sternum.

Posterior: at T2, 3, and 4 near the laminae.

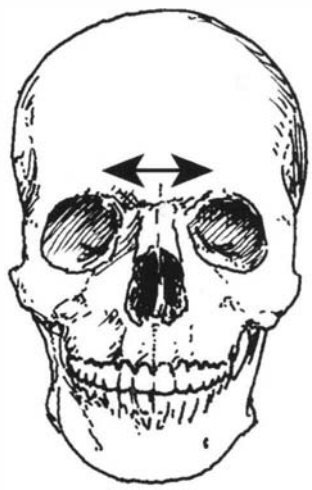
Neurovascular: ramus of the mandible below the zygoma.

Stress receptor: in the transverse plane, approximately 1" above the glabella.

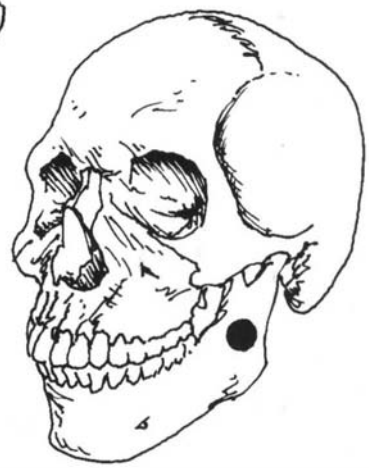
Meridian: stomach.



9—83. Neurolymphatic reflexes.



9—84. Stress receptor.

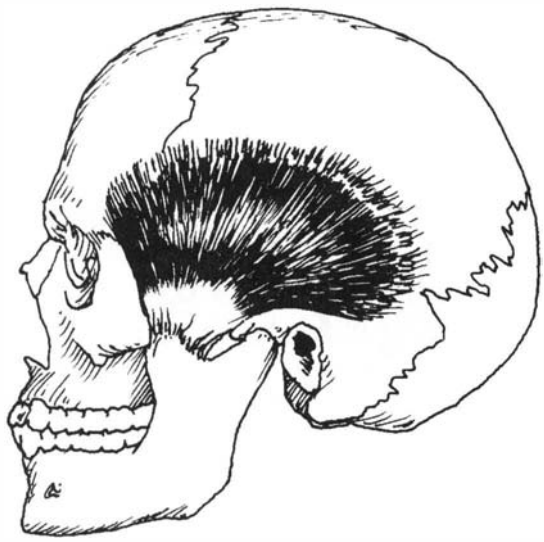


9—85. Neurovascular.

Temporalis

Origin: from the entire temporal fossa (excluding the part formed by the zygomatic bone) and the deep surface of the temporal fascia.
Insertion: by a tendon to the medial surface, apex, anterior and posterior borders of the coronoid process, and the anterior border of the ramus of the mandible, nearly as far as the last molar tooth.
Nerve supply: deep temporal branches of the anterior trunk of the mandibular nerve.
Description: The temporalis muscle is covered by the temporal fascia, a strong fibrous sheet aponeurotic in appearance. Because of its thickness, it is difficult to palpate subtle changes in the muscle, such as a dysfunctioning neuromuscular spindle cell.

There are three divisions of the temporalis: the anterior, middle, and posterior fibers. The posterior fibers almost parallel the occlusal plane.[60] The most posterior and inferior fibers bend sharply at the root of the zygoma to insert on the mandible.[13] Posterior fibers immediately above these extend to the coronoid process and are important in positioning the mandibular condyle in its fossa.
Action: In general, the temporalis elevates the mandible to close the jaws. The posterior fibers retract the mandible. They are active on the side of lateral abduction as the mandible moves from side to side. When the mandible is brought back to the centerline, the posterior fibers on the opposite side are active.



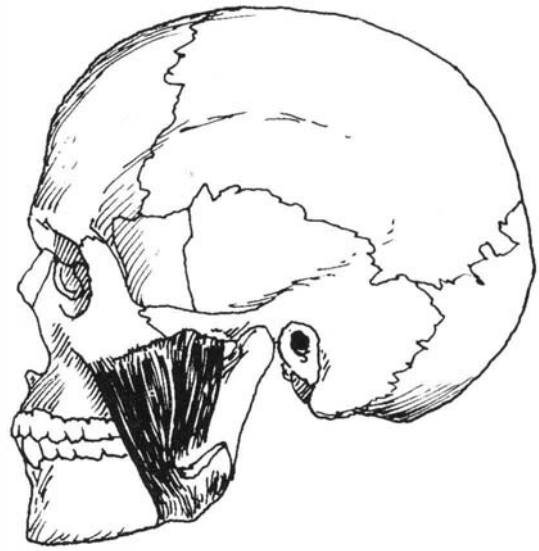
9—86. Temporalis muscle.

Masseter

Origin:
- **Superficial layer:** thick aponeurosis from the zygomatic process of the maxilla, anterior 2/3 of the lower border of the zygomatic arch.
- **Middle layer:** deep surface of the anterior 2/3 of the zygomatic arch and from the lower border of the posterior 1/3.
- **Deep layer:** deep surface of the zygomatic arch.

Insertion:
- **Superficial layer:** angle and lower half of the lateral surface of the ramus of the mandible.
- **Middle layer:** middle of the ramus of the mandible
- **Deep layer:** upper part of the ramus of the mandible and into the coronoid process.

Nerve supply: masseteric nerve from the anterior trunk of the mandibular division of the trigeminal nerve.



9—87. Masseter muscle with superficial layer sectioned to show middle and deep layers.

Action: The major action of the masseter muscle is to elevate the jaw and clench the teeth. There is minimal or no activity of the muscle in the resting position.[9] Other than its major activity in closing the jaws and giving force to occlusion and mastication, the masseter is not very active.

During applied kinesiology examination, the masseter muscle is frequently found to have neuromuscular spindle cell involvement, probably because of the great power this muscle can develop in normal actions and in bracing and bruxism. In addition, the masseter muscle

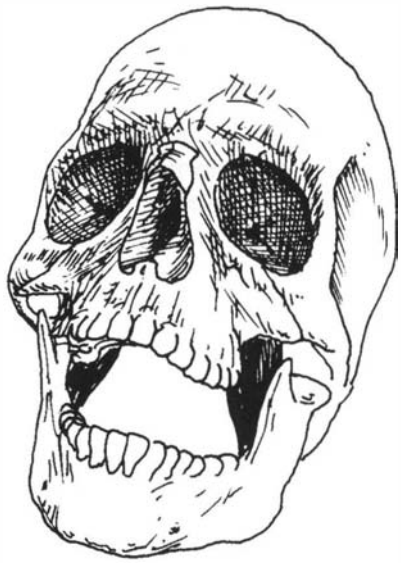
has the highest concentration of neuromuscular spindle cells of the muscles of mastication.[40] The apparently dysfunctioning neuromuscular spindle cell(s) can easily be located by palpation. This is one of the easiest areas in the body in which to conduct this type of examination. Along with the palpable mass of the neuromuscular spindle cell, there will usually be exquisite tenderness.

Masseter muscle action pulls on the zygomatic process, activating cranial bone motion. When function is normal, it appears that this action is beneficial to cranial motion. When there is malocclusion or muscle imbalance, it appears that the action is detrimental, perpetuating or creating cranial faults.

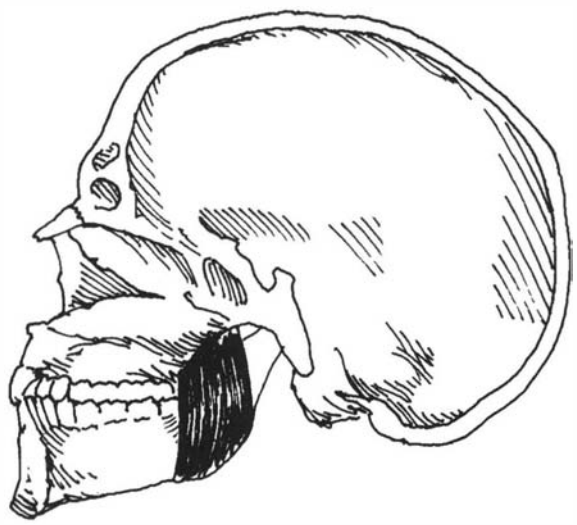
Internal (Medial) Pterygoid

Origin: medial surface of the lateral pterygoid plate and the pyramidal process of the palatine bone; a more superficial slip from the lateral surfaces of the pyramidal process of the palatine bone and tuberosity of the maxilla.
Insertion: inferior and posterior parts of the medial surface of the ramus and the angle of the mandible.
Nerve supply: medial pterygoid nerve of the mandibular division of the trigeminal nerve.
Action: protracts and elevates the mandible; active in rotary motion while chewing. In abduction of the mandible, there is activity of the contralateral muscle.[9,54]

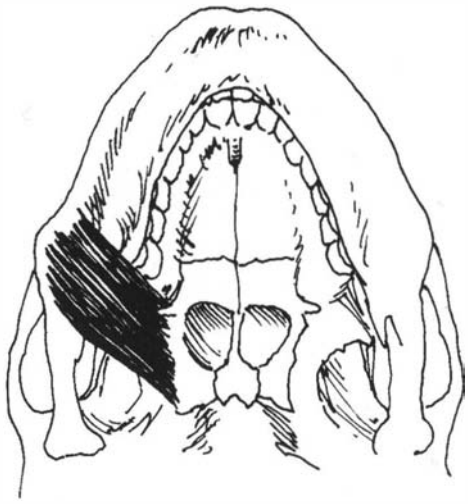
The internal pterygoid muscle's origin from the lateral pterygoid plate and pyramidal process of the palatine bone provides a lever for movement of the cranial bones. As with the masseter, when occlusion and muscle balance are normal, the action of the internal pterygoid balances cranial motion, in effect giving the cranial mechanism a treatment.



9—89. Internal pterygoid muscle.



9—88. Internal pterygoid muscle.



9—90. Internal pterygoid muscle.

Buccinator

Origin: There are three areas of origin — from the maxilla, the pterygomandibular raphe, and the mandible.

Maxilla: from the buccal surface of the alveolar process of the three molar teeth.

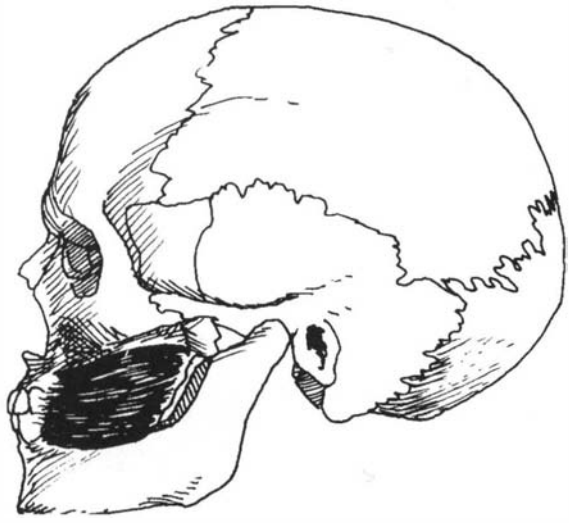
Pterygomandibular raphe: This is a tendinous inscription between the buccinator and constrictor pharyngis superior muscles that gives origin to the middle portion of the muscle. It is attached superiorly to the pterygoid hamulus and inferiorly to the posterior end of the mylohyoid line of the mandible.

Mandible: the lateral alveolar processes inferior to the three molar teeth.

Insertion: blends into the deeper layer of the muscle fibers of the lips, splits in the middle to join with the orbicularis oris of the upper and lower lips.

Nerve supply: buccal branches of the facial nerve.

Action: During mastication the buccinator acts to hold food between the teeth. It provides the foundation from which the orbicularis oris functions, and is a major part of the external envelope of the teeth. Balance between the tongue and the external muscular envelope is important in maintaining tooth position.[22,23,75]



9—91. Buccinator muscle.

External (Lateral) Pterygoid

Origin:

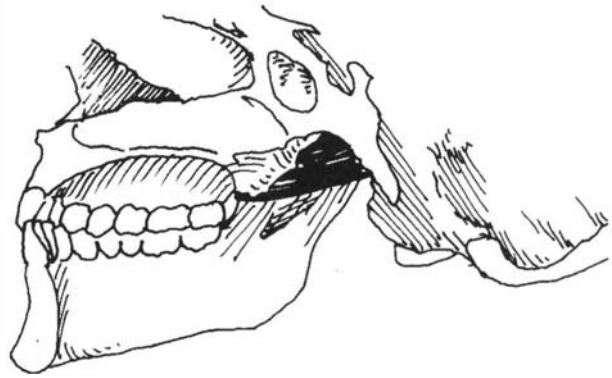
Superior head: infratemporal surface and infratemporal crest of the greater wing of the sphenoid.

Inferior head: lateral surface of the lateral pterygoid plate.

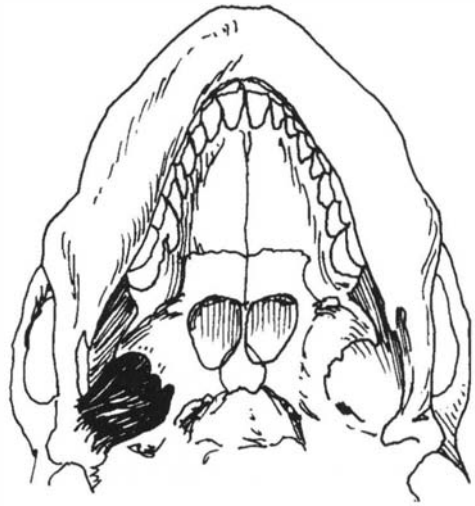
Insertion: anterior part of the neck of the condyle of the mandible, including the superior head,[48] and into the articular capsule and disc of the temporomandibular articulation.

Nerve supply: lateral pterygoid nerve of the anterior trunk of the mandibular division of the trigeminal nerve.

Action: The external pterygoid muscle is actually two muscles with antagonistic function,[32,49,51] separated by their own fascia in the anterior portion.[36] When the external pterygoid muscle is discussed in the literature as a single muscle, reference is usually to the inferior head, which is larger and active on jaw opening.[38] The superior head is smaller and is active in jaw closing to guide the disc,[16] keeping it in the most anterior rotatory position on the condyle permitted by the width of the articular disc space.[5] In normal function, the superior and inferior lateral pterygoid muscles demonstrate nearly reciprocal activity as indicated by EMG.[48] When there is symptomatic dysfunction of mandibular movement, there may be simultaneous contraction of these antagonistic muscles.[38]



9—92. External pterygoid muscle.



9—93. External pterygoid muscle.

Neuroanatomic Basis of Mandibular Movement

Mandibular movement within the complex of the closed kinematic chain of the stomatognathic system consists of complex activity when a person chews, swallows, and talks. The muscles of mastication provide this movement in an unerring manner. The precision of activity can be seen by widely opening one's jaw, including some lateral excursion, and then quickly snapping it shut. With normal function, the teeth come together in perfect intercuspation. Perry[55] proposes a hypothesis that is now generally accepted for the neuroanatomic basis of mandibular movement, and there is considerable electromyographic evidence that this hypothesis is correct. It suggests that mandibular motion is specifically guided by neuromuscular interaction to dictate the final closing position of the mandible toward intercuspation.

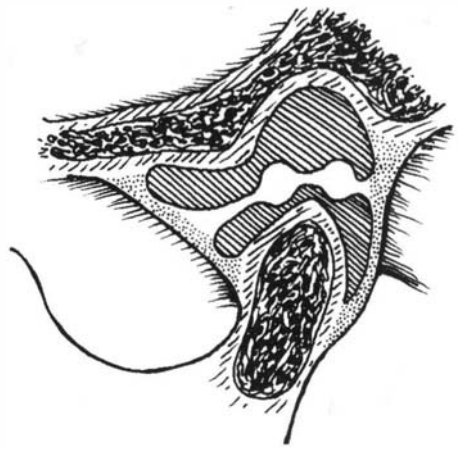
Teeth are anchored in the alveolar sockets by periodontal ligaments. Important in this discussion is the periodontal proprioceptor. When a tooth is tapped in alignment with its axis, there is an immediate silent period seen on electromyography of the jaw-closing muscles.[4] The signal for muscle inhibition comes from stimulation of the receptors in the periodontal ligament.[1] This provides protection for the teeth, similar to the way the Golgi tendon organ provides protection by inhibition of the homonomous muscle when excessive force is exerted.

When the teeth come together and one tooth contacts before the others, it is called a "prematurity." A prematurity can develop as a result of ill-fitting restorations or prostheses, or direct trauma to the tooth. The prematurity stimulates the periodontal ligament to send "...sensory signals into a reflex system which will guide the mandible by means of its musculature, away from the areas of noxious, premature contacts. This musculature is so positioned anatomically that movements in a guiding fashion can be instituted in both the lateral or sagittal plane."[55] The afferent supply from the periodontal ligament proprioceptors provides information for the development of the jaw-closing engram that allows the teeth to come into perfect intercuspation as the jaw snaps shut. DuBrul[13] makes the colorful statement, "This is perhaps an extreme example of the nicety of coordination in all normal bodily movements." As with any other activity in the nervous system, the engram is dynamic. If there is a slight change in the dentition over a period of time, the engram adapts to it. If, however, there is a rapid change in the dentition from a cranial fault, dental procedure, or trauma to a tooth, the engram cannot change rapidly enough to meet the immediate demand, and malocclusion develops.

Temporomandibular Joint

The temporomandibular joint is an encapsulated, compound synovial joint. The capsule is a highly vascular and innervated structure. It is lined with synovial membrane to provide lubrication for the articulation. The articular surfaces are lined with fibrous tissue rather than the usual hyaline cartilage of synovial joints. The fibrous tissue makes the temporomandibular joint less susceptible to degenerative joint disease. In addition, fibrous tissue has a much greater ability to repair and regenerate.[5] The joint ligaments limit the range of motion, but they are not responsible for maintaining the articular surfaces in contact; this function is the responsibility of muscle synergism.

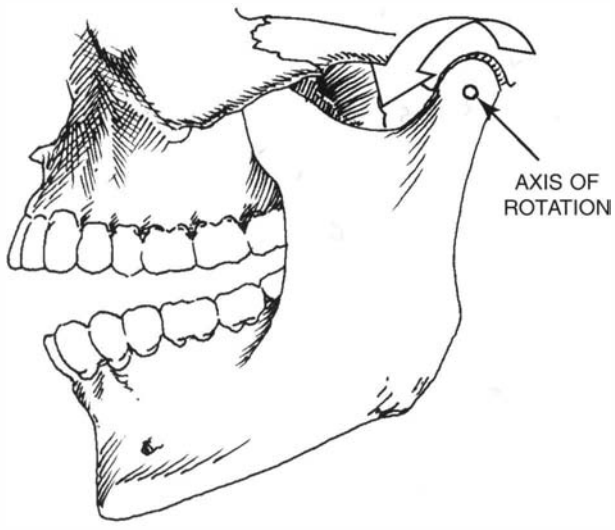
The temporomandibular joint is classified as a compound joint. By definition a compound joint requires the presence of at least three bones. An example is the knee, primarily comprised of the femur, tibia, and patella. In the temporomandibular joint there are only two bones. The articular disc functions as a non-ossified "bone." It has upper and lower facets articulating with the temporal bone and the mandible. The articular disc has been classified as an articular meniscus, which is misleading. The definition of a meniscus is "...a pad, commonly a wedge-shaped crescent of fibrocartilage or dense fibrous tissue, found in some synovial joints; one side forms a marginal attachment at the articular capsule and the other two sides extend into the joint ending in a free edge."[68] An example of an articular meniscus is the cartilage in the knee, with a firm attachment to the tibia and a free edge for articulation with the femur. The disc in the TMJ moves freely at its upper and lower surfaces. The function at each of these articulations is quite independent, with the upper articulation being a sliding joint and the lower a hinge joint. The joint is controlled by both muscular power and mechanical movement.



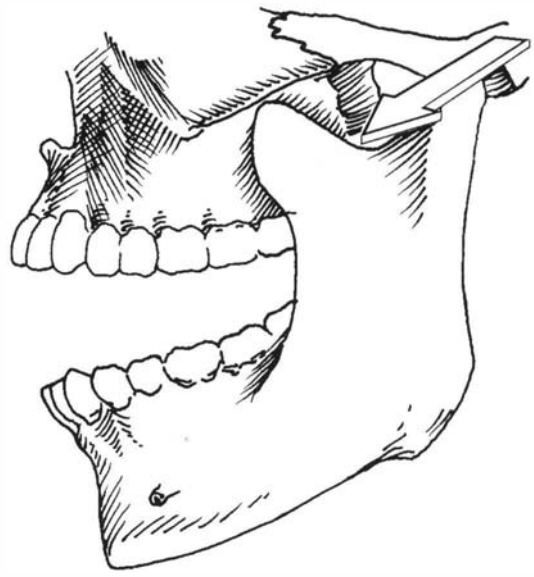
9—94. Sagittal section of the TMJ, with the potential cavities opened for demonstration.

Bell[5] describes three structures necessary for smooth TMJ transitory movements: the articular disc proper, the superior retrodiscal lamina, and the superior head of the external pterygoid muscle.

The motions of the temporomandibular joint are rotation and translation. The term "rotation" refers to a body rotating about its main axis. It is measured in degrees of an angle. "Translation" refers to displacing the total structure from one point in space to another, exhibiting a change in total position. Translatory movement is measured in linear units. Rotation in the temporomandibular joint refers to movement of the condyle around its central axis; translation refers to the sliding of the condyle on the articular eminence. Most mandibular movements are a combination of both. Rotation, a hinge-like action, can only be accomplished as an individual action during the first stage of mandibular opening. Normal TMJ activity depends on normally functioning muscles.



9—95. Rotation of condyle is around its main axis.



9—96. Translation is the condyle sliding on the articular eminence.

TMJ Examination and Treatment

The main applied kinesiology examination of the muscles of mastication is designed to evaluate the muscle proprioceptors. It is a combination of therapy localization to the temporomandibular joint with movement of the mandible in specific directions; a previously strong indicator muscle is tested for weakening.

Evaluation begins with therapy localization to the temporomandibular joint. There should be no weakening of the indicator muscle. Weakness indicates that there is probably some problem with the joint itself. There may be an internal derangement, arthritis, or pathology of adjacent tissues. If therapy localization is positive in the clear, one must differentiate its cause and eliminate it, if possible, before continuing with the muscle examination.

The jaw-opening and closing muscles are evaluated by having the patient open and close his jaws continuously while therapy localizing to the temporomandibular joints. One should take care that the patient does not click his teeth together or stretch the jaw wide open; either action causes other factors to be put into the test, such as stimulation of the periodontal ligament proprioceptors or stretching of the muscles pulling on the cranium to challenge cranial faults. If the indicator muscle weakens, there is probably poor organization of the muscles of mastication. Sometimes one wonders if the indicator muscle weakens simply because the patient cannot concentrate on contracting the muscle being tested while moving the jaw. If bilateral therapy localization to the TMJ is eliminated and the patient opens and closes the jaw repeatedly, the indicator muscle should test strong unless there is nutritional need for aerobic or anerobic muscle function.[29]

If an indicator muscle weakens with rapid movement of the mandible, there may be a need for pan-

tothenic acid, which is treatment for an anerobic muscle dysfunction (see page 190). If the indicator muscle weakens with slow mandibular movement, the aerobic fibers are involved and the indication is for low-potency chelated iron. If either aerobic or anerobic muscle dysfunction is the cause of the indicator muscle weakening during mandibular movement, the appropriate nutrition should eliminate the finding. Weakening of the indicator muscle will often be abolished by therapy localization to the neurolymphatic reflexes for the muscles of mastication, indicating a need for their stimulation.

9—97. Care should be taken that the patient is touching directly over the TMJ during therapy localization.

If the indicator muscle weakens with therapy localization while the mandible is moved, determine the muscle probably involved. Have the patient stop jaw movement in the open or closed position while continuing bilateral therapy localization to the TMJ. The muscles probably involved are those used just prior to stopping. For example, if the patient stops in the open position and an indicator muscle tests weak, the jaw-opening muscles are involved, probably the external pterygoid. Occasionally it is the anterior belly of the digastric muscle. If the indicator muscle weakens when the patient stops in the closed position without the teeth touching, a masseter, temporalis, or internal pterygoid muscle is probably involved. There is rarely a positive test on both opening and closing.

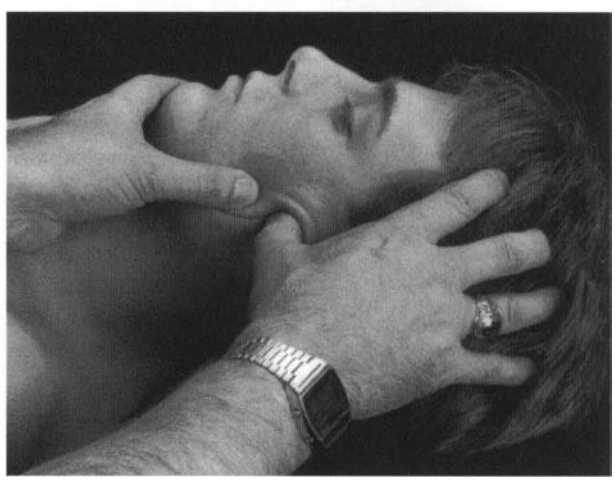
After determining the problem to be with opening or closing muscles, have the patient therapy localize to one side only; repeat the process of opening and closing the mandible and stopping in the position that previously tested positive. Either the right or left side will now test positive; rarely will both sides. The differentiation is now isolated to either opening or closing muscles on one side or the other.

Positive tests from movement of the mandible while therapy localizing the TMJ are usually caused by dysfunction of the neuromuscular spindle cell of the involved muscle. One can now palpate or therapy localize the neuromuscular spindle cell dysfunction. With jaw-closing problems it is usually in the masseter or posterior belly of the temporalis. With jaw-opening problems, it is usually in the inferior division of the external pterygoid muscle.

When neuromuscular spindle cell dysfunction is found in the masseter, a scissors-like manipulation is performed over the area by the physician's thumb, with his opposite thumb manipulating across the buccinator's fibers. Goodheart[24] established this as the method of correction, and it has been found clinically effective.

When the dysfunction is found in the temporalis muscle, the usual manipulation of spindle cells together to set down the muscle's activity is very often used. Because of the heavy fascia over the temporalis, discrete palpation may sometimes be necessary to locate the dysfunctioning neuromuscular spindle cell.

9—98. Arrows represent the direction of force applied to the masseter and buccinator muscles.



9—99. The treating thumbs move in opposite directions in a scissors-like motion to travel the length of the masseter and across the buccinator.

9—100. Neuromuscular spindle cell treatment for hypertonicity of the posterior fibers of the temporalis muscle.

The internal pterygoid muscle may be involved in a jaw-closing problem. In this case there will be positive therapy localization to the internal pterygoid muscle, usually located lateral and inferior to the pterygoid process. The physician makes a fast, sweeping motion over the area of positive therapy localization to treat the neuromuscular spindle cell.

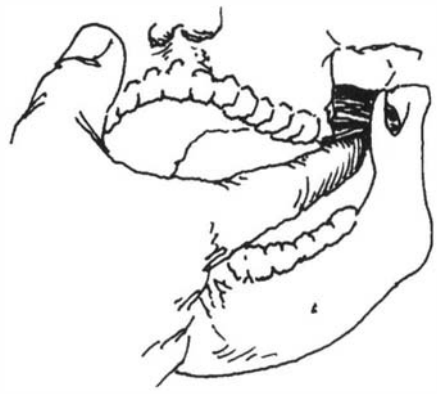
9—101. Palpation of internal pterygoid.

When the test is positive for jaw opening, the external pterygoid muscle is therapy localized or palpated. The muscle area is contacted by placing the forefinger along the buccal side of the alveolus of the last upper molar tooth. If the physician has large hands, it may be better to use the little finger. The disadvantage is that often the little finger is too short to effectively reach the muscle area. The finger is passed backward behind the tuberosity of the maxilla until the lateral surface of the external pterygoid plate is reached.[62] As the finger moves posteriorly, there will be exquisite pain on dysfunction. The amount of contact past the muscle's origin is limited, and there is coincident contact of the superficial head of the internal pterygoid muscle.[37] Bell[6] recommends evaluating the muscle for pain by having the patient clench the teeth firmly in maximum intercuspation to stretch the muscle, or to contract the muscle by biting hard against an object between the teeth.[78]

Treatment has been described in applied kinesiology as being to the neuromuscular spindle cells of the external pterygoid muscle. The amount the spindle cells can be contacted is speculative because of the restricted space through which the finger must go.[37]

Treatment to the external pterygoid muscle is applied by the physician moving his finger past the pterygoid process as described previously, and as far into the muscle as possible. Movement of the patient's mandible may make the muscle more accessible. When the muscle is contacted as best possible, the treating finger is quickly drawn out over the muscle. This procedure is usually very uncomfortable for the patient, so pressure is applied to the patient's tolerance level. If successful, there will no longer be positive therapy localization to the muscle area, or to the TMJ with jaw movement stopped on opening.

9—102. Medial approach to external pterygoid.



9—103. Lateral approach to external pterygoid.

Pterygoid Muscle Strain/Counterstrain Technique

Because of the interaction within the stomatognathic system's closed kinematic muscle chain and the system's interaction with the rest of the body, remote treatment is often effective in improving function of the muscles of mastication. Strain/counterstrain technique is often valuable in improving pterygoid muscle function and reducing pain. If the reader is not familiar with the technique, it is suggested that the section on page 201 be read.

Funakoshi and Amano[19] and Funakoshi et al.[20] established a neurologic relationship between the head-on-neck receptors with the muscles of mastication in decerebrate and labyrinthectomized animals. They found electromyographic response in the muscles of mastication with movement of the head and neck in various directions. Activity of the muscles of mastication was abolished when the first three cervical nerves were cut. Their conclusion was that the tonic neck reflexes influence the jaw muscles. Goodheart[30] postulates that this relationship is the basis for strain/counterstrain technique to improve function of the muscles of mastication, especially the pterygoids.

In addition, Goodheart[30] correlates the relationship between the head-on-neck receptors with the balancing effect and gait action of cranial nerve XI in its control of the sternocleidomastoid and upper trapezius muscles in their function of leveling the head, and facilitation and inhibition during walking.

Often when there is disorganization within the stomatognathic system's closed kinematic muscle chain, pterygoid muscle tenderness increases when the patient stands in a gait position. A consistent type of therapy localization is present under these conditions. The test is done with the patient supine. The temporomandibular joints are bilaterally therapy localized, and the patient pulls inferiorly on the tissue overlying the joint while simultaneously clenching the teeth in intercuspation. If a previously strong indicator muscle weakens, there is clinical evidence that strain/counterstrain technique to the painful pterygoid muscle(s) will be effective in reducing pain and improving muscle function.

The physician monitors pain in the pterygoid muscle area with his index or little finger, as previously described. The primary spinal motion for obtaining reduced tenderness in the pterygoid muscle is head and neck hyperflexion, with some lateral flexion and rotation. The position is changed until the maximum amount of pain is reduced in the pterygoid muscle. The patient remains passive while his head and neck are maneuvered to obtain the relief. When the optimal position is reached, the patient takes a deep inspiration and holds it as long as possible. The physician holds the position for 30 or more seconds and then slowly maneuvers the patient back to neutral while the patient remains passive. The physician controls all movement from beginning to end.

Re-evaluate for pain reduction on digital pressure to the pterygoid muscle area. There should no longer be positive therapy localization on inferior traction therapy localization to the TMJ, and there is usually a great increase in range of motion at the hip, spine, or other areas.

Another method of correcting the pterygoid muscles is to hold the tender area in the pterygoid pocket as previously described while tapping T2, 3, and 4, which are the Dvorak and Dvorak spondylogenic reflex levels for the pterygoid muscles as determined by Goodheart. Repeat the pterygoid muscle contact on the other side while tapping the three thoracic vertebra. Successful treatment significantly reduces or eliminates the pain in the pterygoid pocket.

Imbalance of the pterygoid muscles is a problem that may be difficult to correct in the stomatognathic system. Increased pain in the pterygoid muscle area when the patient is in a standing gait position indicates that remote factors are influencing the continuing problem. Evaluate for the need of gait, equilibrium synchronization, dural tension, and PRYT techniques, as previously discussed.

9—104. Passively find the position of cervical flexion that relieves the pterygoid muscle pain. Have the patient inhale and hold the position for 30 seconds. All of the movement is with the patient passive.

Challenge and Manipulation of Teeth

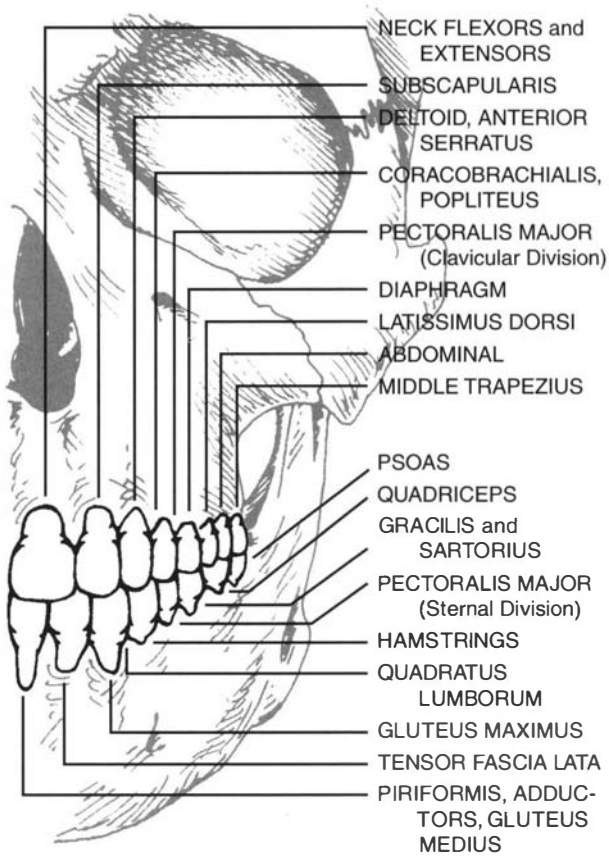
Proper afferent impulses from the periodontal ligament proprioceptors and nociceptors are important in establishing and maintaining the engram for action of the jaw-closing muscles. Positive therapy localization at the gingiva overlying a tooth's alveolar socket indicates there is either pathology in the area or disturbance of the periodontal ligament nerve receptors. If positive therapy localization reflects improper nerve function of the tooth, there will also be a positive challenge. When the therapy localization and challenge are both positive, the condition is called a "neurologic tooth" in applied kinesiology.

There are many reasons why a neurologic tooth develops. Specific trauma, such as biting on a seed in a piece of cherry pie, may injure the periodontal ligament from the violent tooth movement. Malocclusion due to cranial faults, muscle disorganization, or dental procedures can also traumatize the periodontal ligament nerve receptors.

Improper proprioceptive supply from a neurologic tooth can disturb the engram of closing and cause further malocclusion. Sometimes the problem with a neurologic tooth is pain unaccounted for by local dental pathology. This apparently occurs because improper stimulation of the nociceptors in the periodontal ligament continually signals pain. Treatment to the tooth often eliminates the pain or poor muscle interaction.

Ratner et al.[57] describe remote problems such as arm pain developing from infection of the maxillary bone, usually as a result of tooth extraction. They refer to Black and his colleagues,[2,7,76] who suggest a cause and effect relationship between alteration of the dentition and changes in the nervous system. Applied kinesiology clinical research has established homuncular representation of muscle-organ/gland association in the teeth. Sometimes when an associated muscle tests weak, it will return to normal function after a neurologic tooth is corrected. On the other hand, when an associated muscle dysfunctions in relation to its organ or gland, there may be disturbance of the associated tooth until the muscle-organ/gland relationship is corrected. For example, a sugar handling problem causing stress to the adrenal glands and consequent weakness of the sartorius and/or gracilis muscle may cause the lower 1st molar on the side of involvement to test positive as a neurologic tooth. The tooth involvement will not usually be permanently corrected until the muscle-organ/gland dysfunction is corrected.

A screening procedure designed by Blaich[8] has the patient bring his teeth gently into intercuspation, and a previously strong indicator muscle is tested for weakening. This procedure is done after cranial faults and TMJ dysfunction have been corrected. When positive, further evaluation is made to determine the exact tooth or teeth involved. The general area of tooth involvement can be found by therapy localizing several teeth together. Have the patient put his finger along the length of the teeth at the gingival line. When positive therapy localization is found, individual teeth are then therapy localized. When a tooth tests positive, it is then challenged to determine how correction should be applied. One specific vector of challenge will cause maximum weakening of an indicator muscle. It may be in the buccal, lingual, mesial, or distal direction, or toward the apex of the tooth. Occasionally it may even be traction on the tooth that shows a positive challenge. Immediately after challenging the tooth, while the indicator muscle is still weak, have the patient inspire and re-test the indicator muscle. If the tooth involvement is associated with inspiration, the indicator muscle will test strong. If not, repeat the procedure with exhalation. The tooth and its periodontal ligament are corrected by pressing on the tooth with 3-4 pounds of pressure in the direction of positive challenge on the phase of respiration that abolished the challenge. Repeat four or five times. The tooth is then therapy localized and challenged to determine if correction was obtained.



9—105. Muscle representation in the dental arches.

The homuncular association with muscles is present in edentulous patients, but not to as great an extent as in those with normal dentition. Examination and treatment are basically the same. The gingiva is challenged in various vectors to find the maximum weakening of an indicator muscle. Then find the phase of respiration that abolishes the positive challenge. Treat in the same manner as for a neurologic tooth.

If the correction for a neurologic tooth does not hold and there is no observable dysfunction in associated muscles, organs, or glands it is possible that zinc is needed. Zinc in small quantities along with other nutritional supplements, appears to improve this condition better than zinc supplement by itself. A neurologic tooth can also be perpetuated by malocclusion.

Hyoid Muscles and Function

Important to the stomatognathic system's closed kinematic muscle chain are the hyoid muscles. The hyoid bone is U-shaped and has no direct articulation with another bone. It is suspended by muscles and ligaments, and moves within this sling-type arrangement. It has a body with two greater and two lesser cornua.

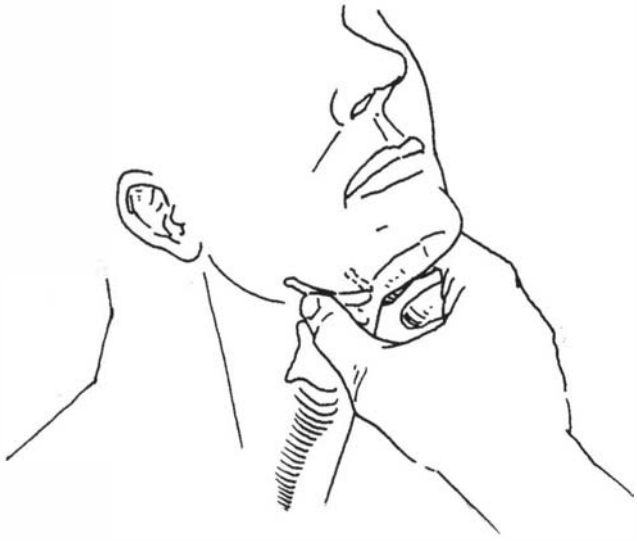
The hyoid is located in the receding angle between the chin and anterior part of the neck, on the level of the 4th cervical vertebra.

The hyoid can be palpated immediately above the thyroid cartilage. The anterior superior portion of the thyroid cartilage can be located by palpating the thyroid notch anteriorly. The hyoid bone is best palpated with the thumb on one side and the index finger on the other side.

Goodheart[25] has compared hyoid suspension with a gyroscope in a guidance system. A gyroscope is flexibly mounted in an object such as a missile, ship, or airplane so that it maintains its equilibrium. Sensors relay information about any change in position between the vehicle and the gyroscope, providing a type of feedback. This information is used by an automatic guidance system, such as an autopilot, or by a pilot to know the airplane's orientation in space. If the airplane and the gyroscope are not level with each other, the instrument will show the disparity.

The proprioceptors of the hyoid muscles appear to provide afferent information that is compared with total body position for orientation in space. The suprahyoid muscles have their origins on the skull and the mandible, while the infrahyoid muscles originate from the sternum, clavicle, scapula, and thyroid cartilage. Since the muscles originate from such varying locations, their afferent supply provides considerable information about the orientation of these structures with each other.

Applied kinesiology examination of the hyoid muscles requires moving the hyoid bone to stretch specific muscles, then therapy localizing the muscle. It is important that the examiner be familiar with muscle anatomy to properly perform the examination.



9—106. The hyoid is immediately above the thyroid cartilage. Palpate for the thyroid notch. Move up to the hyoid and locate its superior border.

Digastric Muscle

The digastric muscle is different from other hyoid muscles because it has two bellies, with an intermediate rounded tendon surrounded by a fibrous loop that connects the bellies to the hyoid.

Posterior belly:

Origin: mastoid notch (digastric fossa) on the posteromedial aspect of the mastoid process of the temporal bone.

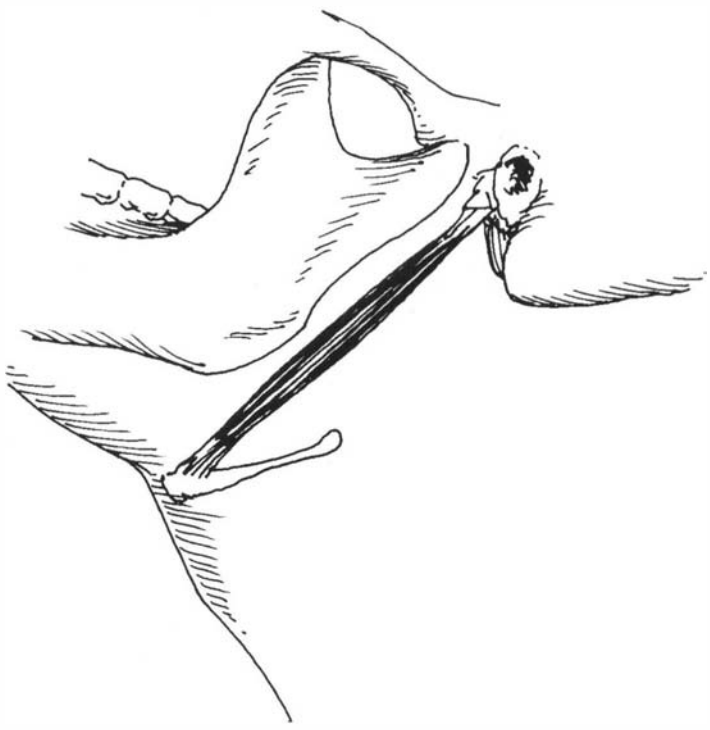
Insertion: into the intermediate tendon, which is attached to the lateral aspect of the hyoid body and greater cornu by a fibrous loop.

Anterior belly:

Origin: digastric fossa of the mandible, which is a depression on the inner side of the inferior border of the mandible, close to the symphysis menti.

Insertion: into the intermediate tendon, which is attached to the hyoid as noted above. The intermediate tendon perforates the stylohyoid muscle.

9—107. Inferior view of anterior and posterior bellies of the digastric muscle.



9—108. Stylohyoid muscle. The head is extended on the cervical spine to raise the angle of the mandible away from the muscle.

Nerve supply:

Anterior belly: mylohyoid branch of interior alveolar branch of the mandibular division of the trigeminal nerve.

Posterior belly: branch of the facial nerve.

Action: In general, the anterior belly of the digastric is a jaw-opening muscle. It is important in completing the opening movement. Acting together, the digastric bellies can elevate the hyoid bone.

Stylohyoid

Origin: posterolateral surface of the stylohyoid process near the base.

Insertion: body of the hyoid at the junction of the greater cornu above the omohyoid.

Nerve supply: facial nerve.

Action: moves the hyoid superiorly and posteriorly toward the styloid process.

Mylohyoid

Origin: from the mylohyoid line of the mandible, which extends from the symphysis menti to the last molar.

Insertion: into the body of the hyoid. The median edge of the muscle inserts into a fibrous raphe, which extends from the symphysis menti to the body of the hyoid; thus, the bilateral muscles join at an angle along their entire median length.

Nerve supply: mylohyoid branch from the inferior alveolar branch of the mandibular division of the trigeminal nerve.

Action: raises the hyoid, base of the tongue, and floor of the mouth. It is active in mastication, deglutition, sucking, and blowing. Because the bilateral muscles form the broad, flat muscular floor of the oral cavity, these muscles were previously known as the oral diaphragm.

Geniohyoid

Origin: inferior genial tubercle on the posterior surface of the symphysis menti.

Insertion: anterior surface of the body of the hyoid.

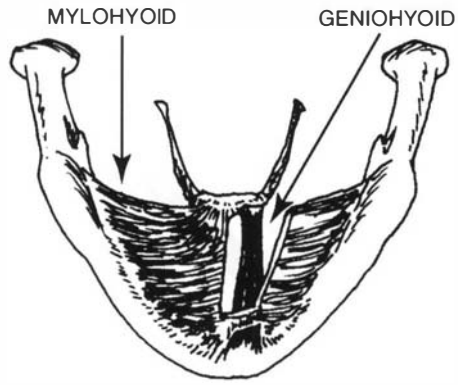
Nerve supply: branch of the 1st cervical nerve by way of the hypoglossal nerve.

Action: pulls the hyoid superiorly and anteriorly, or, if the hyoid is the fixed origin, pulls the mandible posteriorly and inferiorly. It is active in anterior movement of the tongue, determines the length of the floor of the mouth with the stylohyoid muscle.

Sternohyoid

Origin: posterior surface of the median end of the clavicle, posterior sternoclavicular ligament, and the upper and posterior parts of the manubrium.

Insertion: inferior border of the body of the hyoid.



9—109. Mylohyoid and geniohyoid muscles.

Nerve supply: branch of the ansa cervicalis, with branches from C1, 2, and 3.
Action: stabilizes and draws the hyoid inferiorly. It is active in mastication, speech, and deglutition.

Sternothyroid

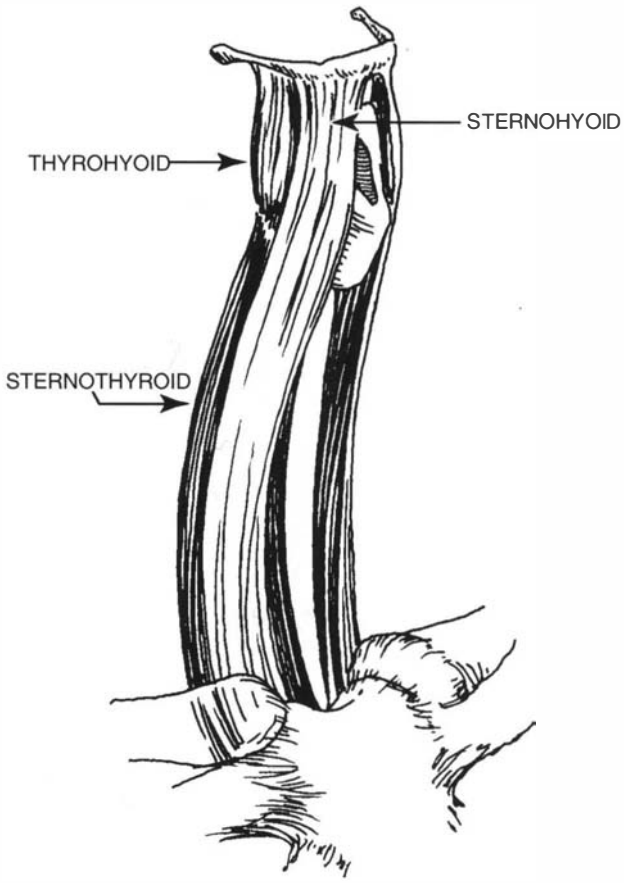
Origin: edge of the cartilage of the 1st rib, and from the posterior surface of the manubrium below the sternohyoid origin.
Insertion: oblique line on the lamina of the thyroid cartilage.
Nerve supply: branches from the ansa cervicalis, C1, 2, and 3.
Action: draws the larynx inferiorly after it has been elevated in swallowing or in vocal movements.

Thyrohyoid

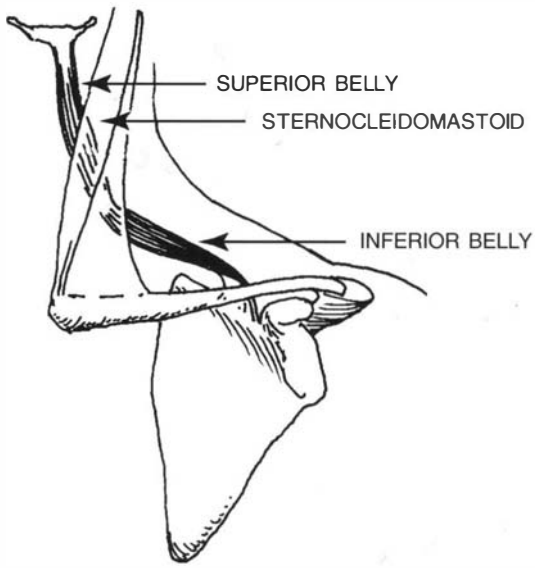
Origin: oblique line on the lamina of the thyroid cartilage.
Insertion: lower border of the greater cornu and adjacent part of the body of the hyoid.
Nerve supply: branch of the ansa cervicalis, with branches from C1 and possibly 2.
Action: draws the hyoid inferiorly or, if it is the fixed origin, draws the thyroid cartilage superiorly.

Omohyoid

Origin: from the upper border of the scapula near the scapular notch.
Insertion: inferior border of the body of the hyoid lateral to the insertion of the sternohyoid.
Nerve supply: ansa cervicalis, C1, 2, and 3.
Action: moves the hyoid and larynx inferiorly, steadies the hyoid from the inferior, and is suspected to be important in inspiration.



9—110. Anterolateral view of sternohyoid, sternothyroid, and thyrohyoid.



9—111. Omohyoid muscle.

Examination and Treatment

There are several types of body language that indicate hyoid neuromuscular balance should be evaluated. Whenever there is any dysfunction of the stomatognathic system's closed kinematic muscle chain, evaluate the hyoid muscles. They may be involved whenever phonation, deglutition, tongue movement, or change of head position causes a positive test in a remote area that was previously negative. Disorganization between the two sides of the body may be due to improper function of the hyoid neuromusculature, as evidenced by switching, reaction to bilateral brain activity, continued requirements for cross-crawl patterning, or problems in the gait mechanism.

The hyoid muscles are examined by moving the hyoid bone in various directions to stretch the muscles and testing a previously strong indicator muscle for weakening. It is assumed that a positive test is due to neuromuscular spindle cell dysfunction in the muscle being stretched. It is also possible that there may be fascial disharmony relating with Rolf's work,[61] or trigger points, which may be treated with Travell's technique[70] or by other methods.

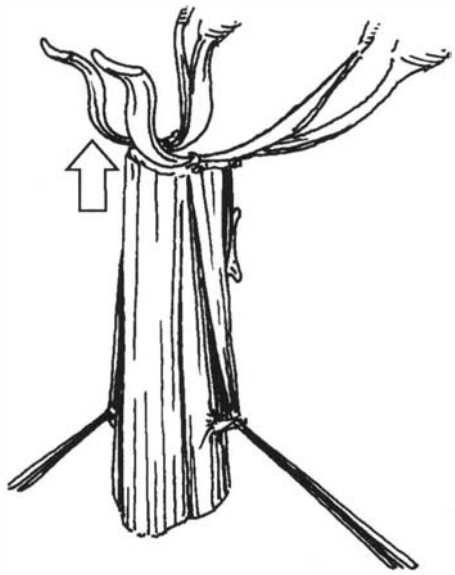
Movement of the hyoid can be done by the patient, the physician, or a support person. There should be no positive therapy localization when the hyoid is contacted without movement to stretch muscles. If there is, evaluate for local pathology or the possibility of Golgi tendon organ involvement.

The hyoid is moved into a position to stretch a muscle or group of muscles, and an indicator muscle is tested. Weakening of the indicator muscle indicates the hyoid muscle being stretched is hypertonic and needs to be returned to normal by neuromuscular spindle cell technique. When positive, evaluate the muscle(s) being stretched with accurate therapy localization. By having the patient therapy localize to the muscle with the tip of his index finger, the area of dysfunction can be located quite accurately. Treatment is directed to the neuromuscular spindle cell to push its ends together, if one can contact the area with two fingers; if not, a general massage over the area of positive therapy localization is used.

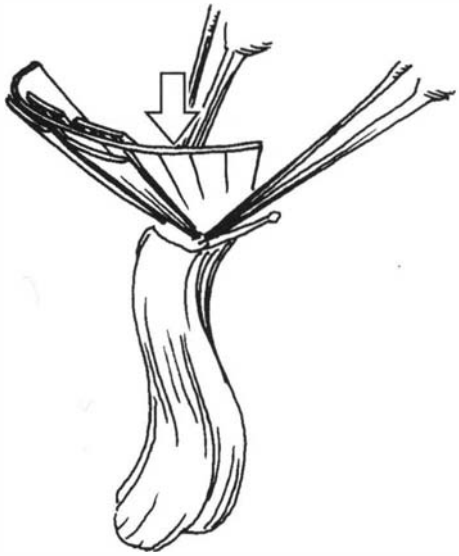
A combination of movements can quickly isolate the muscle probably involved. For example, if the hyoid is held forward and down and the indicator muscle weakens, hold the hyoid to one side or the other. If the indicator muscle weakens when the hyoid is held to the left, the right muscles are being stretched and the down and forward position indicates probable involvement of the posterior belly of the digastric or stylohyoid muscle. A few tests of this nature limit the number of muscles that must be therapy localized. Effective treatment is indicated by the elimination of positive therapy localization and by negative challenge.

9—112. Anterior challenge stretches the posterior belly of the digastric and the stylohyoid muscles.

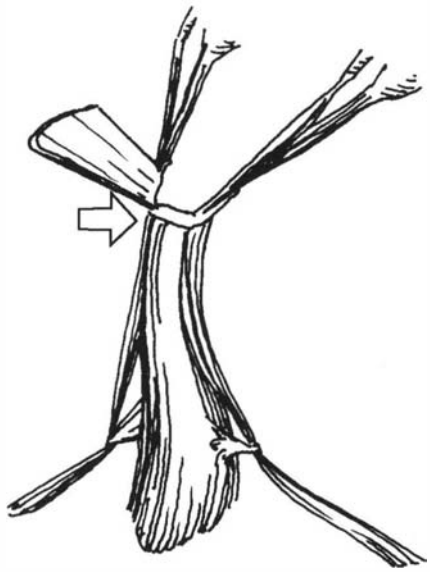
9—113. Posterior challenge stretches the anterior belly of the digastric, geniohyoid, and somewhat the mylohyoid muscles.



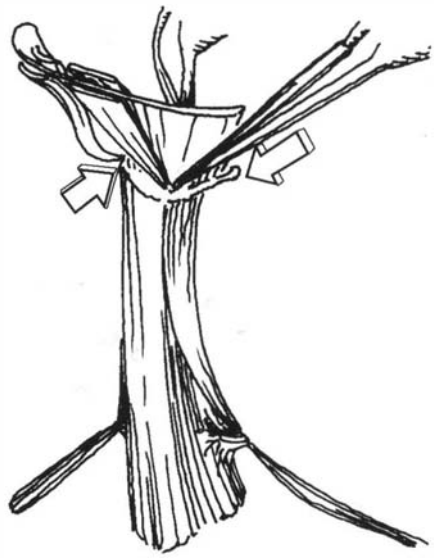
9—114. Superior challenge stretches the omohyoid, thyrohyoid, sternohyoid, and perhaps the sternothyroid muscles.



9—116. Inferior challenge stretches the mylohyoid, geniohyoid, both bellies of the digastric, and the stylohyoid muscles.



9—115. Lateral challenge stretches the posterior belly of the digastric, stylohyoid, mylohyoid, omohyoid, and perhaps the other hyoid muscles on that side.



9—117. Various combinations of hyoid challenge can be made to help isolate the muscle(s) involved.

Stomatognathic System Examination and Treatment Summary

The procedures presented in this text will improve a large percentage of cases having disturbance in the stomatognathic system. It has been emphasized that the stomatognathic system has many working parts within it. Cranial faults may cause poor function of the masticatory, hyoid, and/or cervical muscles due to nerve entrapment. Malocclusion may cause cranial faults, or vertebral subluxations or fixations may cause imbalance in the closed kinematic muscle chain. The interplay could go on and on, and we haven't even considered remote factors such as gait and posture influencing the stomatognathic system. In problem cases where dysfunction returns after effective correction, some aspect is not being corrected, causing the dysfunction to recur. It may be necessary to consult with a dentist for correction of the malocclusion, or for other dental procedures such as splint therapy. The reader is encouraged to pursue a more in-depth study of the stomatognathic system, such as that presented in *Applied Kinesiology, Volume II — Head, Neck, and Jaw Pain and Dysfunction — The Stomatognathic System* by this author.[75]

CHAPTER 9 — REFERENCES

1. Anderson, K.V., & P.E. Mahan, "Interaction of tooth pulp and periodontal ligament receptors in a jaw-depression reflex," *Exp Neurol*, Vol 32 (Aug 1971).
2. Anderson, L.S. et al., "Neuronal hyperactivity in experimental trigeminal deafferentation," *J Neurosurg*, Vol 35 (Oct 1971).
3. Baker, E.G., "Alteration in width of maxillary arch and its relation to sutural movement of cranial bones," *JAOA*, Vol 70, No 6 (Feb 1971).
4. Beaudreau, D.E., W.F. Daugherty, Jr., & W. Masland, "Two types of motor pause in masticatory muscles," *Am J Physiol*, Vol 216, No 1 (Jan 1969).
5. Bell, W.E., "Understanding temporomandibular biomechanics (an explanation)," *J Craniomandib Prac*, Vol 1, No 2 (Mar/May 1983).
6. Bell, W.E., *Orofacial Pains: Classification, Diagnosis, Management*, 3rd ed (Chicago: Year Book Med Publishers, Inc, 1985).
7. Black, R.G., "A laboratory model for trigeminal neuralgia," *Adv Neurol*, Vol 4 (1974).
8. Blaich, R.M., "Applications of neurological tooth involvement in resistant stomatognathic problems." Proceedings of Summer Meeting, ICAK, Detroit, 1981.
9. Carlsöö, S., "Nervous coordination and mechanical function of the mandibular elevators," *Acta Odontol Scand*, Vol 10, Supp 11 (1952).
10. Current advances in dentistry, Telephone Extension Program, University of Illinois College of Dentistry (1949).
11. De Jarnette, M.B., *Cranial Technique 1968* (Nebraska City, NE: privately published, 1968).
12. De Jarnette, M.B., *Cranial Technique 1979-80* (Nebraska City, NE: privately published, 1979).
13. DuBrul, E.L., *Sicher's Oral Anatomy*, 7th ed (St. Louis: The C.V. Mosby Co, 1980).
14. Duffy, C.A., "The nasosphenoid cranial fault — A conversation with Drs. Goodheart, Duffy, Schmitt, and Rogowskey," *AK Review*, Vol 7, No 1 (Spring 1997).
15. Duffy, D.H., "Glaucoma and the kinesiological approach," *Chiro Econ*, Vol 19, No 3 (Nov/Dec 1976).
16. Eversole, L.R., & L. Machado, "Temporomandibular joint internal derangements and associated neuromuscular disorders," *JADA*, Vol 110 (Jan 1985).
17. Frymann, V., "Relation of disturbances of craniosacral mechanisms to symptomatology of the newborn: Study of 1,250 infants," *JAOA*, Vol 65 (Jun 1966).
18. Frymann, V.M., "A study of the rhythmic motions of the living cranium," *JAOA*, Vol 70, No 9 (May 1971).
19. Funakoshi, M., & N. Amano, "Effects of the tonic neck reflex on the jaw muscles of the rat," *J Dent Res*, Vol 52, No 4 (Jul/Aug 1973).
20. Funakoshi, M., N. Fujita, & S. Takenana, "Relations between occlusal interference and jaw muscle activities in response to changes in head position," *J Dent Res*, Vol 35, No 4 (Jul/Aug 1976).
21. Gardner, W.J., "Trigeminal neuralgia," *Clin Neurosurg*, Vol 15 (1967).
22. Garliner, D., *Myofunctional Therapy in Dental Practice*, 2nd ed (Brooklyn: Bartel Dental Book Co Inc, 1974).
23. Garliner, D., *Myofunctional Therapy* (Philadelphia: W.B. Saunders Co, 1976).
24. Goodheart, G.J., Jr., *Applied Kinesiology 1976 Workshop Procedure Manual*, 12th ed (Detroit: privately published, 1976).
25. Goodheart, G.J., Jr., *Applied Kinesiology 1977 Workshop Procedure Manual*, 13th ed (Detroit: privately published, 1977).
26. Goodheart, G.J., Jr., *Applied Kinesiology 1979 Workshop Procedure Manual*, 15th ed (Detroit: privately published, 1979).
27. Goodheart, G.J., Jr., *Applied Kinesiology Workshop Procedure Manual*, 15th ed, Vol 2 (Detroit: privately published, 1979).
28. Goodheart, G.J., Jr., *Applied Kinesiology 1980 Workshop Procedure Manual*, 16th ed, Vol 1 (Detroit: privately published, 1980).
29. Goodheart, G.J., Jr., "Applied kinesiology in dysfunction of the temporomandibular joint," in *The Dental Clinics of North America*, Vol 27, No 3, ed H. Gelb (Philadelphia: W.B. Saunders Co, 1983).
30. Goodheart, G.J., Jr., *Applied Kinesiology 1984 Workshop Procedure Manual*, 20th ed (Detroit: privately published, 1984).
31. Granit, R., L. Leksell, & C.R. Skoglund, "Fibre interaction in injured or compressed region of nerve," *Brain*, Vol 67 (Jun 1944).
32. Grant, P.G., "Lateral pterygoid: Two muscles?" *Am J Anat*, Vol 138 (Sep 1973).
33. Greenman, P.E., "Roentgen findings in the craniosacral mechanism," *JAOA*, Vol 70, No 1 (Sep 1970).
34. Henningsen, M.G., "Living osteology of interest to the dentist: Part one," *Dent Digest*, Vol 63 (Oct 1957).
35. Henningsen, M.G., "Living osteology of interest to the dentist: Part two," *Dent Digest*, Vol 63 (Nov 1957).
36. Honee, G.L.J.M., "The anatomy of the lateral pterygoid muscle," *Acta Morphol Neerl-Scand*, Vol 10 (Dec 1972).
37. Johnstone, D.R., & M. Templeton, "The feasibility of palpating the lateral pterygoid muscle," *J Prosthet Dent*, Vol 44, No 3 (Sep 1980).
38. Juniper, R.P., "Temporomandibular joint dysfunction: A theory based upon electromyographic studies of the lateral pterygoid muscle," *Br J Oral Maxillofacial Surg*, Vol 22 (1984).
39. Kopell, H.P., & W.A.L. Thompson, *Peripheral Entrapment Neuropathies*, 2nd ed (Huntington, NY: Robert E. Krieger Publishing Co, 1976).
40. Lennartsson, B., "Number and distribution of muscle spindles in the masticatory muscles of the rat," *J Anat*, Vol 130, No 2 (1980).
41. Libin, B.M., "The cranial mechanism: Its relationship to cranial-man-

dibular function," *J Prosthet Dent*, Vol 58, No 5 (Nov 1987).
42. Magoun, H.I., Sr., "Osteopathic approach to dental enigmas," *JAOA*, Vol 62 (Oct 1962).
43. Magoun, H.I., "Whiplash injury: A greater lesion complex," *JAOA*, Vol 63, No 6 (Feb 1964).
44. Magoun, H.I., Sr., "A pertinent approach to pituitary pathology," *D.O.*, Vol 11, No 11 (Jul 1971).
45. Magoun, H.I., "Idiopathic adolescent spinal scoliosis: A reasonable etiology," *D.O.*, Vol 13, No 6 (Feb 1973).
46. Magoun, H.I., Sr., "Dental equilibrium and osteopathy," *JAOA*, Vol 74 (Jun 1975).
47. Magoun, H.I., ed, *Osteopathy in the Cranial Field*, 3rd ed (Belen, NM: privately published, 1976).
48. Mahan, P.E. et al., "Superior and inferior bellies of the lateral pterygoid muscle EMG activity at basic jaw positions," *J Prosthet Dent*, Vol 50, No 5 (Nov 1983).
49. McNamara, J.A., Jr., "The independent functions of the two heads of the lateral pterygoid muscle," *Am J Anat*, Vol 138, No 2 (Oct 1973).
50. Michael, D.K., & E.W. Retzlaff, "A preliminary study of cranial bone movement in the squirrel monkey," *JAOA*, Vol 74 (May 1975).
51. Miller, A.J., K. Vargervik, & G. Chierici, "Electromyographic analysis of the functional components of the lateral pterygoid muscle in the Rhesus monkey," *Arch Oral Biol*, Vol 27, No 6 (1982).
52. Mintz, V.W, "The orthopedic influence," in *Diseases of the Temporomandibular Apparatus — A Multidisciplinary Approach*, ed D.H. Morgan et al. (St. Louis: The C.V. Mosby Co, 1982).
53. Moses, A.J., & G.S. Skoog, "Cervical whiplash and TMJ — similarities in symptoms," *Trial* (Mar 1986).
54. Moyers, R.E., "An electromyographic analysis of certain muscles involved in temporomandibular movement," *Am J Orthod*, Vol 36, No 7 (Jul 1950).
55. Perry, H.T., "Muscular changes associated with temporomandibular joint dysfunction," *JADA*, Vol 54, No 5 (May 1957).
56. Pritchard, J.J., J.H. Scott, & F.G. Girgis, "The structure and development of cranial and facial sutures," *J Anat*, Vol 90 (Jan 1956).
57. Ratner, E.J., P. Person, & D.J. Kleinman, "Severe arm pain associated with pathological bone cavity of maxilla," *Lancet* (Jan 14, 1978).
58. Retzlaff, E.W. et al., "Nerve fibers and endings in cranial sutures," *JAOA*, Vol 77 (Feb 1978).
59. Retzlaff, E.W. et al., "Temporalis muscle action in parietotemporal suture compression," *JAOA*, Vol 78 (Oct 1978).
60. Robinson, M., "The temporomandibular joint: Theory of reflex controlled nonlever action of the mandible," *JADA*, Vol 33 (Oct 1, 1946).
61. Rolf, I.P., *Rolfing: The Integration of Human Structures* (Santa Monica, CA: Dennis-Landman Publishers, 1977).
62. Schwartz, L., & C.M. Chayes, "The history and clinical examination," in *Facial Pain and Mandibular Dysfunction*, ed L. Schwartz & C.M. Chayes (Philadelphia: W.B. Saunders Co, 1968).
63. Shore, N.A., *Temporomandibular Joint Dysfunction and Occlusal Equilibration*, 2nd ed (Philadelphia: J.B. Lippincott Co, 1976).
64. Stoner, F., *The Eclectic Approach to Chiropractic* (Las Vegas: F.L.S. Publishing Co, 1975).
65. Stowe, R.S., L.L. Lavoy, & N.A. Frigerio, "Measurement of bone torsion *in vivo* via biostereoroentgenography." XIII Int Congress Photogrammetry (Jul 11-13, 1976).
66. Strachan, W.F., & M.J. Robinson, "New osteopathic research ties leg disparity to malocclusion," *Osteo News*, Vol 6, No 2 (Apr 1965).
67. Sutherland, W.G., *The Cranial Bowl* (Mankato, MN: privately published, 1939).
68. Taylor, E.J., ed, *Dorland's Illustrated Medical Dictionary*, 27th ed (Philadelphia: W.B. Saunders Co, 1988).
69. Thompson, J.R., & A.G. Brodie, "Factors in the position of the mandible," *JADA*, Vol 29 (Jun 1942).
70. Travell, J.G., & D.G. Simons, *Myofascial Pain and Dysfunction — The Trigger Point Manual* (Baltimore: Williams & Wilkins, 1983).
71. Upledger, J.E., "The relationship of craniosacral findings in grade school children with developmental problems," *JAOA*, Vol 77 (Jun 1978).
72. Upledger, J.E., E.W. Retzlaff, & J.D. Vredevoogd, "Diagnosis and treatment of temporoparietal suture head pain," *Osteo Med* (Jul 1978).
73. Upledger, J., & J. Vredevoogd, *Craniosacral Therapy* (Chicago: Eastland Press, 1983).
74. Walther, D.S., *Applied Kinesiology — The Advanced Approach in Chiropractic* (Pueblo, CO: Systems DC, 1976).
75. Walther, D.S., *Applied Kinesiology, Volume II — Head, Neck, and Jaw Pain and Dysfunction — The Stomatognathic System* (Pueblo, CO: Systems DC, 1983).
76. Westrum, L.E., R.C. Canfield, & R.G. Black, "Transganglionic degeneration in the spinal trigeminal nucleus following removal of tooth pulps in adult cats," *Brain Res*, Vol 101 (Jan 9, 1976).
77. Wheeler, R.C., *Dental Anatomy, Physiology and Occlusion*, 5th ed (Philadelphia: W.B. Saunders Co, 1974).
78. White, L.W., "The lateral pterygoid muscle: Fact and fiction," *J Clin Orthodon*, Vol 19, No 8 (Aug 1985).

Chapter 10

Mental and Emotional Conditions

Chapter 10

Mental And Emotional Conditions

Within the framework of applied kinesiology there are many examination and therapeutic procedures that are intimately involved with the mental side of the triad of health. In my thirty-nine years of chiropractic practice, my attitude and therapeutic approach to mental and emotional health problems have changed considerably. In my early practice I referred patients with mental and emotional problems to psychiatrists, psychologists, or other counselors. I still do when necessary, but now it's less often. The overall results from referral were poor. In some instances, the patient's problem was compounded by his adverse reaction or addiction to prescription drugs. In other cases, the patient's problem was blamed on parental upbringing or some other family problem; that confrontation often created a schism in the family relationship.

With a broader knowledge of natural health, it became apparent that many mental and emotional problems are due to physiologic dysfunction. When the body is returned to normal function, the mental or emotional problem is often eliminated or the patient is better able to cope with it. There are those in the field of psychiatry who have come to the same conclusion. After several years of psychiatric practice, Philpott[37] began questioning the philosophical approach of probing deeply into a patient's hostility toward his father, mother, brother, sister, or uncle to arrive at a psychiatric diagnosis as the cause of the patient's neurosis, often causing psychosomatic conditions. "It is my conviction," Philpott states, "that diagnoses such as 'schizophrenic,' 'manic-depressive,' and other psychotic, neurotic, or psychosomatic labels are relatively meaningless and tend only to aggravate the illness. It is the underlying organic cause that is important."

Many readers may think they are not interested in this subject and do not care to treat patients with mental problems. This is probably not an option because in most general practices many of these patients are already being treated. Often they are the poorly responding patients who tend to feed negative aspects into the practice.

An experience in my clinic may help put in perspective treatment of a patient with mental and emotional problems. I have always encouraged my support personnel to discuss with me any problems they may feel are present in the treatment of our patients. If the support person feels I am overlooking a factor or not correctly examining or treating a patient, it is imperative that the subject be discussed so that appropriate measures may be taken. It may be, in fact, that I am failing in some aspect of the examination and/or treatment of a patient; on the other hand, the support person may not be aware of the therapeutic approach and its rationale.

One new employee in our office had previously worked in a clinic staffed by six psychiatrists. When she first began working for us, I informed her of my policy of discussing any problems she might feel existed in the treatment of patients. During her first weeks she frequently told me after a new patient's examination that that person really needed psychiatric care because of depression, hypochondria, or some other condition typically treated with psychotherapy. On numerous occasions during those first months of her employment we stayed after hours to discuss the cause of these conditions and my therapeutic rationale. Slowly but surely the criticism of m
became less frequent, and finally it stopped altogether. After she had worked for about four months, she said she wanted to talk to me about the mental problems of the patients we'd been caring for. I thought, "Oh, no — here we go again!" After patient treatment for the day was completed, we sat down in my office and I was mentally prepared to go into the same subject again. Instead she said, "Many of the patients who come in here are no different than the patients we treated in the psychiatric clinic. The difference is that here they get well." She became an excellent employee and a solid supporter of our therapeutic approach.

When working with patients with mental and emotional problems, one's limitations in dealing with the problem must be kept in mind. Patients may be suicidal but present no outward appearance of it. When the routine question about tendency toward suicide is asked in consultation or on a questionnaire, a surprising number of people answer positively. The same support person who questioned my therapeutic approach told me that there was rarely a month that one of the patients at the psychiatric clinic did not commit suicide. These patients may need a team approach[31] to effectively deal with the psychologic and physiologic factors of health.

The effect of the mental side of the triad of health on function can readily be observed with applied kinesiology testing. When a person concentrates on a subject that is emotionally detrimental, a previously strong indicator muscle will test weak. The muscles that appear to reflect the emotional thought processes most efficiently are the pectoralis major (clavicular division) muscles.

Change in muscle function with emotional disorders has been shown by Whatmore and Ellis.[50] They have demonstrated a low level of muscle activity in schizophrenics and depressives that is covert in most cases. It can only be observed by recording with low-level electromyographic equipment. The muscle action may be intermittent or continuous, static or phasic. This low level of muscle activity is called hyperponesis.

In a study of schizophrenics,[49] Whatmore and Ellis recorded a control group composed of hospital personnel who had never been afflicted with mental illness. The control group was divided into two sub-groups. One subgroup consisted of persons mostly free from functional symptoms of any kind; the other was composed of those more troubled with functional complaints. There was a statistically higher level of covert muscle action in those

with functional complaints over those without. The control group with higher hyperponesis had statistically lower activity than those with schizophrenia.

Whatmore and Kohli[52] have thoroughly discussed improper action-potential output from the pre-motor and motor cortex. This is called dysponesis, and it provides a model that partially explains the change in muscle function as observed by manual muscle testing.

Since early in applied kinesiology's development it has been observed that muscle function sometimes changes with different thought processes. Occasionally muscles change function as one discusses various subjects during an examination. This may be an indication of stress affecting the individual's health. During the examination the physician may change the conversation to various subjects that he thinks might be influencing the patient's health. For example, if the pectoralis major (clavicular division) muscle weakens when the discussion centers on a teenager, it indicates probable stress in that area.

The astute physician can often uncover considerable information about a patient's emotional relationship with regard to his finances, marital status, job stress, and general family problems by simply changing the conversation during examination and observing for disturbance in the neuromuscular system that develops as a result of specific thought processes. In addition to this subtle examination procedure, there are specific procedures used in evaluating fears and other emotional problems discussed later in this chapter. Other methods of evaluating the mental aspect of an individual's health are discussed in texts by Callahan[9,10] and Diamond,[15,16] both of whom use manual muscle testing in their psychology practices.

Psychological Reversal

Thought processes play a major role in an individual's health. Patients with terminal conditions have survived by changing their thought processes and lifestyles.[42] One of the fastest-growing fields in the healing arts is psychoneuroimmunology. Failure to regain health is sometimes due to subconscious self-sabotage.

A common psychological understanding is that when the consequences of getting well are greater than the consequences of staying sick, no therapy will be able to return health. Sometimes it is obvious that an "ill" patient receives attention from family and friends that he would not get if he were healthy. Some individuals may have a subconscious or conscious fear of interacting with the opposite sex. For example, they may be overweight to obscure their fear of sexuality. In either case, an individual may profess a desire to obtain good health or lose weight, but various therapies and diets fail miserably or only benefit temporarily.

The physician in charge may suspect a psychological cause for treatment failure; however, diagnostic methods to help confirm the suspicion have been limited. Compounding the problem is the difficulty in overcoming the psychological need to remain sick or overweight.

Callahan,[7] a psychologist using applied kinesiology methods, has developed a manual muscle testing technique to help confirm and treat this problem. Callahan calls the problem psychological reversal. This is when an individual's subconscious mind differs from what is consciously said. He first observed it in a woman who had been dieting for years with little success. In Callahan's words, "I asked her to picture herself thin the way she wished to be. I wasn't surprised when she went weak because I knew she had certain fears associated with being slender and sexually attractive. What surprised me was when I asked her to picture herself 30 pounds heavier than she was (she was already 35 pounds heavier than her desired weight), her muscle got extraordinarily strong. I had her say, 'I want to gain weight.' She tested strong."

The pattern of psychological reversal is seen when a strong indicator muscle becomes weak when an individual states or imagines a positive goal that he says he desires to achieve. When he states or imagines the failure or negative aspects of that goal, he tests strong. Both sides of the pattern must be present, i.e., testing weak on stating or imagining the positive goal, and testing strong when stating or imagining the negative aspects or failure of the goal. If the individual tests weak on both the positive and negative aspects of the goal, it may be due only to the stress associated with that subject.

Psychological reversal can be present with almost anything affecting a person's life. A person may repeatedly fail to succeed in his chosen occupation or profession. The Peter Principle states, "In a hierarchy every employee tends to rise to his level of incompetence."[35] One will quite often find that an individual is psychologically reversed at the point of failure. Blaich[5] has found that human performance in reading skills can be improved by clearing individuals of psychological reversal and neurologic disorganization.

Psychological reversal is either present or not, but it exists in varying degrees of severity. An overweight individual with psychological reversal may be 15 or 150 pounds overweight. In either case, the individual professes a desire to lose weight, but he is unsuccessful in obtaining or maintaining his goal. Callahan cites the example of psychologically reversed gamblers who consistently lose a significant but tolerable sum of money every time they gamble, while their more severely reversed brethren will lose everything they own, can borrow, or steal.

The better self-acceptance an individual has, the less tendency there is for psychological reversal. Some fortunate individuals may be in such good physical and

mental health that they are never psychologically reversed. Most people are occasionally psychologically reversed on some subject at some time. Some have massive psychological reversal, i.e., an individual weakens with anything good and tests strong with anything bad. Callahan recommends testing for this condition with the vague, general statements, "I want to have a good life," and "I want to be miserable." Most practicing physicians can recognize psychologically reversed individuals in their practices. These are often the individuals who respond poorly to treatment; when there is some improvement in a condition, they will dwell on the negative aspects. Even when the improvement is pointed out, they will immediately change the subject back to the negative aspects.

Psychological reversal can be very specific. For example, an excellent student who maintains a high grade average in all subjects except French will test negative for psychological reversal when stating, "I want to do well in my schoolwork" as the positive statement, and "I want to fail in my schoolwork" as the negative statement. However, when stating, "I want to do well in French" and "I want to fail in French," he will test positive for psychological reversal.

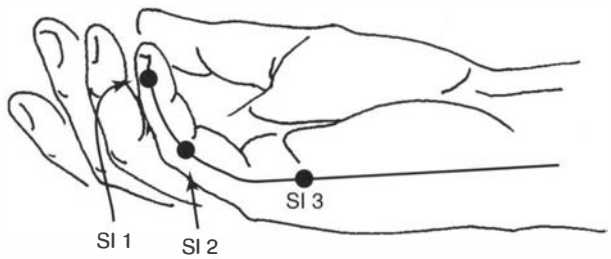
There are several aspects of neurologic disorganization that appear to relate with psychological reversal. Often when an individual's neurologic disorganization is corrected, as discussed in Chapter 5, the correction may be immediately lost when the reversal is induced by having him think positively about his reversal problem. This can happen even when the neurologic disorganization has been eliminated with structural correction of the cranium or feet. This is amazing to this structurally oriented author, and once more shows the great interplay in the triad of health.

When an applied kinesiology therapeutic approach is ineffective, one should consider psychological reversal. Callahan[7] relates his first observation of reversal resulting in a failed therapeutic effort. He was attempting to lower the glabella temperature of an anorexic patient with the applied kinesiology beginning and ending technique and having no success. Between the unsuccessful treatment and the patient's next visit, it occurred to him that she might be psychologically reversed. Upon testing for this and correcting it, the beginning and ending technique was effective in lowering the temperature 2°, after which the patient began eating three meals a day for the first time in fifteen years. Callahan finds the vigorous tapping on the face with the beginning and ending technique quite stressful, often causing psychological reversal or neurologic disorganization. If present, this must be corrected before successful treatment with B and E technique can be accomplished.

Treatment

Immediate elimination of the positive muscle tests in psychological reversal is obtained by simply having the patient state, "I profoundly and deeply accept myself with all my problems and shortcomings." Although this indicates that the individual is deficient in self-acceptance and excessively condemns and belittles himself, he is often unaware that he does so. Obviously one does not develop self-acceptance by merely verbalizing the statement, but it does give momentary relief. Establishing the cause of psychological reversal enables the physician to follow through and develop correction in the area causing psychological reversal.

In addition to attention to the psychological aspect of the reversal, Callahan has found that tapping acu-point SI 1 or SI 3 while the patient's mind is focused on the positive statement that caused the indicator muscle to weaken helps establish a much more permanent correction. The proper point, when therapy localized, will eliminate the weakening observed with the positive statement.



10—1. Small intestine meridian at the hand.

For example, if a patient weakens when saying, "I want to lose weight," there should no longer be weakening when SI 1 or SI 3 is therapy localized and the statement repeated.

Nutritional support with vitamin B and ribonucleic acid may be indicated. The Bach flower Rescue® Remedy is indicated, and should be taken every waking hour for two weeks. There is often a secondary involvement of the small intestine meridian after the psychological reversal has been corrected. Examine for it by therapy localizing to the alarm and/or pulse point. If there is a positive finding, correct the meridian imbalance with the usual methods of applied kinesiology meridian therapy discussed in Chapter 7. In addition, the same nutritional support indicated above may be needed. It is as if the small intestine involvement is being revealed at a different level. It requires treatment independent from that indicated above.

Correction of psychological reversal has many ramifications in a patient's total health picture. There are often poor results from treatment until psychological reversal is corrected. When uncorrected its adverse influence on body function can be widespread, from contributing to severe life-threatening pathological conditions to limiting human performance.

Blood Sugar

Blood sugar handling stress can be the cause of emotional disturbance. The balance within the endocrine system causes some people to be more susceptible than others to blood-sugar-handling stress as a result of dietary indiscretion. We inherit our glandular systems' strengths and weaknesses, just as we inherit our facial and body characteristics. Bieler[4] points out how endocrine balance influences the physical type a person will be. In a similar manner, the endocrine balance one inherits influences how efficiently stress will be handled. What is eustress to one person may be detrimental stress to another.[40]

There are specific mental characteristics that fit the diabetic, and characteristics that fit the hypoglycemic. True diabetes does not parallel asthma.[1] The diabetic is a happy-go-lucky person who usually has difficulty maintaining a diet because of an attitude that seems to indicate, "This really isn't important to me." On the other hand, the hypoadrenic-hypoglycemic often has asthma and/or other allergies.[6] Hypoglycemia usually relates with adrenal insufficiency, causing a failure of mobilization of glycogen and gluconeogenesis. In adrenal insufficiency there is a lack of the pro- and anti-inflammatory hormones,[39] leading to allergies.

The hypoglycemic tends to be depressed. After being established on a proper diet he usually follows it very closely, sometimes to the extreme of weighing foods and counting specific calories to be certain the diet is followed accurately.

If an individual diagnosed as diabetic does not follow the typical pattern but is consistently depressed, critical, and moody, one should think of dysinsulinism, a condition in which hyperglycemia is present during one portion of the day and hypoglycemia during another part of the same day.[1] We are not talking here about the insulin-dependent diabetic who develops hypoglycemia from excess insulin. Dysinsulinism refers to an individual who is not on insulin. Contributing to the problem may be a severely inadequate diet in which large quantities of refined carbohydrates are consumed throughout the day. The characteristics of dysinsulinism may be present in patients taking oral hypoglycemic medication, as illustrated with the following case.

A patient from my early practice (approximately thirty-three years ago) is an excellent example of this problem. She had been under my care for several years on a health maintenance basis. Prior to seeing me, she had been diagnosed as diabetic and treated with an oral hypoglycemic medication. The physician checked her blood sugar once a month. On every visit to my office she would report her blood sugar from the test, and I would dutifully enter it in my record. The blood sample was always taken in the morning, approximately two hours postprandial, and it consistently ran between 115 and 125 mg%. She seemed almost ecstatic when the blood sugar would drop 4 mg% from the previous reading, and was depressed if it was up. Her original diagnosis was made following a three-hour glucose tolerance test. As I learned more about blood sugar, I became aware that this patient did not fit the psychic pattern of a diabetic. She was constantly depressed, often experiencing long stages of deep depression. She had been hospitalized on numerous occasions for depression and "nervous breakdown." Treatment usually consisted of anti-depressants and counseling by her minister.

I was not proud of the poor results this patient was obtaining under chiropractic care. She constantly had some type of problem. Not only was she depressed, it depressed me to take care of her. In an effort to better understand her problem, I decided to start almost from the beginning. We sat down in my consultation room and I started questioning her about her health problems. Almost immediately she began crying and was completely unable to control her emotions. Her thought process was very poor. At that point I drew a blood sample, which was approximately four hours postprandial; it was 55 mg%. She was given some fruit to eat and almost immediately began to feel better. She was then scheduled to have a blood sample taken periodically throughout the day. The only time she had elevated blood sugar was about two hours postprandial; most of the rest of the day she was hypoglycemic. She discontinued the hypoglycemic medication. Eventually a six-hour glucose tolerance test was run and dysinsulinism was found. The dietary and applied kinesiology treatments for dysinsulinism are very similar to those for hypoglycemia, discussed in Chapter 12. Her emotions, especially the constant depression, improved within a very short time. Her general health also improved, and she no longer required the frequent chiropractic treatments she once had.

Emotional problems due to blood sugar handling stress are sometimes induced by dietary habits the patient places on himself, such as fad diets. An individual with a tendency toward hypoglycemia will suffer greatly from the low carbohydrate diet that periodically keeps coming back into vogue as a weight-reduction method. Although this discussion is centered on blood sugar handling stress and the emotions, one should consider the same principle in other dietary programs. Occasionally one will see a patient so fearful of gaining weight that s/he will eat nothing containing fat, if at all possible, thus creating a deficiency of the essential fatty acids discussed in Chapter 12.

Social or family pressure may be the factor that causes an individual to self-impose dietary restrictions that create emotional problems. A man I had taken care of for several years for work-related injuries and general spinal care stopped by the office unexpectedly and asked "...just to talk to the doctor for a couple of minutes." I saw him in my consultation room, and it was obvious that he was distraught and upset. He said he had a seri-

ous problem with his wife, and as he drove by my office the thought occurred that perhaps I could help him. He told me that his wife had become very depressed, irritable, irrational, and completely unreasonable with the children. Sometimes she would scream at them; other times she just didn't seem to care whether she disciplined them or not. This pattern, he said, had been present before but never to such an extent. He had grown desperate, and asked me if I could recommend a good psychiatrist.

A few years previously I had treated his wife, "Sally," for a shoulder injury. She appeared to be a very rational individual. She had a tendency to be plump, but was certainly the type of woman who would attract attention as she walked by. Having also treated several of her relatives — including brothers, sisters, and parents — I was aware of the family tendency toward obesity.

I suggested that possibly Sally's problem was physical, thus affecting her mental state, and that he should have her make an appointment with me for examination. Then we would determine if psychiatric care was indicated. My prior unsatisfactory experiences with psychiatric referrals did not make me anxious to refer Sally for psychiatric analysis and treatment.

Upon examination Sally was found to have many symptoms of hypoglycemia, not just the emotional depression with highs and lows. She had developed several additional health problems, including headaches, migrating pains, fatigue, and dizziness. A blood test revealed hypoglycemia. She also had a low level of total lipids, as well as hypoproteinemia. A dietary consultation revealed the real problem.

Sally had gained considerable weight since I saw her for the shoulder problem. She weighed 170 pounds and was very concerned about the gain. She had put herself on a diet that excluded fats and anything else she "thought" was fattening. To start her day she ate half a piece of toast and drank a cup of coffee; by 10:00 a.m. she was hungry and shaking. Unable to resist temptation any longer, she would eat a piece of pie or cake, which was always around the house because her husband enjoyed sweets. This started a snowballing effect of elevating and dropping blood sugar levels. She would feel all right temporarily, then become very depressed because she couldn't follow her unrealistic diet. After a short time her blood sugar would hit another low, and she would again bring the level up with something sweet.

Upon discussing nutrition with Sally, it became clear that she thought anything of a nutritious nature was also fattening. It was very difficult to get her to start eating a well-balanced diet, including proteins and fats. After much discussion — and almost with a guarantee from me that she would not gain any more weight on a proper diet — she began the process. Her emotions improved considerably within a very short time, and she didn't gain over her previous level of 170 pounds.

Additional insight into Sally's problem came a few months later when her husband injured his knee and came in for treatment. In the course of conversation, he said he was irritated with me and was seriously considering having Sally discontinue her treatment and find another doctor. Because of her excellent improvement, I was surprised. He related that because of my diet Sally had gotten very fat, and he hated fat people. He said it just made him sick to see her eating butter on her toast, snacking on nuts between meals, drinking whole milk, and eating all those fat things. At least before Sally came to me, he said, she tried to keep her weight down, but not any more. I showed him Sally's record from the time we started working with her blood sugar problem, emphasizing that she had not gained any weight since being on this diet but had actually lost some, and that she was now emotionally sound. The final outcome of this case several years later showed Sally functioning normally on an emotional basis, and maintaining her weight between 140 and 150 pounds.

As discussed in this book's hypoglycemia section, one of the most important aspects of your treatment is patient education. This is also necessary for members of the family.

The emotions and psyche of an individual are affected in many ways by hypoglycemia and relative adrenal insufficiency. The first and most obvious involvement is the inability of the nervous system — especially the brain — to function normally without adequate blood sugar. Thinking becomes irrational and sluggish; simple problems become complex and difficult to solve. The problems become more complex because of extrinsic factors. An individual may not be succeeding in his employment because of poor thinking ability. Constant stress causes increased relative adrenal insufficiency, as pointed out so well by Selye.[41] This constant pressure as a result of failure will create mental problems of many varieties. Frustrations may be taken out on family members in particular and society in general; the reactions of these people often go back to the individual, continuing to compound the problem.

When emotional problems develop as a result of physiologic disturbance, such as in blood sugar handling problems, treatment must be directed to the basic underlying cause. Treatment directed to the psyche alone often fails miserably.

Attention Deficit Hyperactivity Disorder and Learning Disabilities

Many children who have learning disabilities and/or attention deficit hyperactivity disorder (ADHD) are rapidly returned to normal with treatment directed to the subjects of this text. Treatment may be simply modifying the diet, or correcting extremely complex neurologic disorganization involving the cranial-sacral primary respiratory system and other causes of neurologic disorganization. Discussion in this section is limited to a brief association of some conditions that cause learning disabilities and attention deficit hyperactivity disorder. The reader can pursue each subject more thoroughly in the appropriate chapters of this text.

General Health Problems

Until an applied kinesiologist becomes established as one knowledgeable in the treatment of ADHD and learning disabilities, most children will be brought in with other health problems. This is common, because the same dysfunction causing ADHD and learning disabilities may also be responsible for headaches, neck pain, back pain, leg aches, asthma, allergies, and myriad other conditions.

Neurologic disorganization and hypoadrenia should routinely be examined for in all new patients. When found in children, questioning the parents will often reveal ADHD and/or learning disabilities about which the parents are well aware; however, they had no idea that chiropractic could be of help. It is important that parents understand how your overall approach to health, which is necessary to correct the chief complaint for which the patient is brought in, will also influence the learning disabilities and ADHD. It is impossible to treat the total body without influencing these factors.

It is not unusual for children with severe learning disabilities to change from failing students to ones with A's and B's within several months or a few grade periods. Many of these children are very intelligent. To them the classroom may be a jungle in which they have learned to cope by memorizing the recitations of other children. Reading one's own work may be difficult or impossible.

When the condition is found and treated in younger children, the response is more rapid and complete. If not treated until the student is in middle or high school, the child — even though physiologically functioning normally — must play a catch-up game because basics were not learned in the earlier grades.

The dramatic response in these conditions creates a large number of referrals. Almost every parent who has a child with ADHD or a learning disability knows several other parents facing the same problem. In addition, teachers, psychologists, social workers, and clergy are often intimately involved with the child's problem. In order to help more of these unfortunate children, do not hesitate to discuss the case with individuals who also have an interest in the child. Usually the parents will readily give permission to discuss the child's case with such a person. Correction of these problems is extremely gratifying; one can see a child's entire life turn around. What is it worth to be able to read and think clearly and to analyze problems of social interaction and employment?

Relative Hypoadrenia and/or Hypoglycemia

As discussed in Chapter 12, hypoglycemia and adrenal stress disorder often go hand-in-hand. A child may have inherited weak adrenal glands, causing lack of efficient mobilization of glycogen and gluconeogenesis. When hypoadrenia is the initiating factor, there will often be a history of allergies, frequent infections, fatigue, and sometimes even depression. Adrenal stress disorder can be secondary to a sugar handling stress. A child may eat large quantities of refined carbohydrates. When the blood sugar level rises rapidly, there is often an overreaction of insulin, pushing the blood sugar levels down and requiring glucocorticoids to bring it back up. The continual stress on the overworked adrenal glands eventually causes them to be depleted. The child often desires more high sugar items, worsening the condition.

The nervous system suffers from low blood sugar levels; the brain simply cannot function adequately. The child is inattentive, has a poor memory, and exhibits poor reasoning when confronted with analytical problems. He is often labeled as lazy when, in reality, he is unable to do any better because his mental processes cannot function without an adequate blood sugar level. Periods of depression and desire for isolation from other students may compound the problem. The child may be classified as a loner; other children may even ridicule him because of his inability in the classroom and his antisocial behavior. The resulting emotional stress continues to compound the condition by increasing the adrenal stress disorder, providing less ability to manage blood sugar levels properly. Distress in this child continues to be magnified if parents and school authorities, not understanding the reason for failure, pressure him to improve the quality of his schoolwork. Until the physiologic factor is corrected, the schoolwork will not improve; in fact, it will probably worsen because of increased stress magnifying the disability.

Neurologic Disorganization

Neurologic disorganization can affect a child's learning ability or coordination. Sometimes both problems are present but quite often one or the other is dominant.

The type of neurologic disorganization paramount in learning disabilities is that which comes from dysfunction of the cranial-sacral primary respiratory mechanism.

Cranial faults may result from birth trauma, with increased neurologic dysfunction as the cranium continues to develop and loses the great flexibility of infancy. Upledger[44] found a significant loss of cranial-sacral motion in grade school children with learning disabilities.

Ocular lock will often be present and can readily be identified using applied kinesiology techniques. This may lead to frank dyslexia, but one must remember that many reading disorders are grouped under this term.[24] Mirror writing or other commonly associated reversals, such as crossing a "t" at the bottom and reversing "d's" and "b's," are often exhibited in a person with a reading disorder. Diplopia may be so severe that two unrelated words may be superimposed on each other. Rapid correction is usually obtained using applied kinesiology methods for treating the cranial-sacral primary respiratory system.

Another type of reading problem associated with neurologic disorganization is what Goodheart has called the "B'nai B'rith" syndrome. The term developed because of an individual's inability to effectively read from left to right. When attempting to do so, a previously strong indicator muscle weakens. But if the person is asked to read backward, i.e., from right to left as the Jewish language is written, there is no weakening of the indicator muscle. This is an indication of neurologic disorganization; it is treated in the same way as any other evidence of neurologic disorganization. It will be temporarily eliminated by stimulation to acupuncture points KI 27 and the umbilicus. The optimal method of correction is to find the factor causing the neurologic disorganization, which is often in the cranial-sacral primary respiratory mechanism.

Children's health problems — including learning disabilities — are effectively treated using the AK approach to the cranial-sacral primary respiratory system presented in Chapter 9; there is a more thorough discussion of the stomatognathic system in *Applied Kinesiology, Volume II*.[46] Children are not usually as involved as adults with malocclusion complicating the stomatognathic system dysfunction, but it should be evaluated for and treated when necessary. A thorough examination of body organization including the feet, gait, and modular activity — such as treated with PRYT and the equilibrium synchronization techniques — must be done. As with all other conditions, the body functions in a totally integrated manner; all aspects must be considered.

Characteristics of Attention Deficit Hyperactivity Disorder

Attention deficit hyperactivity disorder (ADHD) often parallels learning disabilities. Children with blood sugar handling problems are especially susceptible to ADHD. A child may be moody. With low blood sugar, depression tends to occur; when the blood sugar goes up with refined sugar intake, there tends to be hyperkinesis. Ritalin® is a central nervous system stimulant often given to hyperkinetic children. Its mode of action in man is not completely understood.[27] Many of the side effects seem to indicate that part of its action is from stimulation of the adrenal cortex.

People outside the family often think a child with ADHD is the result of lack of parental authority and discipline. Several factors help distinguish the ADHD child from one who has poor parental guidance.

1. **Movement** is consistently excessive for the activity being accomplished. The child's hands and legs are constantly moving; he has a tendency to rock and dance with constant wiggles and jiggles. This overactivity is sometimes seen early in life when the child beats his crib, butts his head, and/or heavily rocks his crib.
2. **Aggression** toward his peers and parents is in the child's nature. He is compulsive in his disruption of others' activities; when obviously causing an interruption, he cannot be diverted from the action. He has a compulsion to touch everything and everyone. The child's acts of aggression are often dangerous to his safety, but he is incapable of recognizing the danger.
3. **Unpredictability.** It is difficult to determine how the child will react in specific situations. He is impulsive and may react differently at different times. He is highly excitable, especially when something does not go as he wishes.
4. **Impatience.** The ADHD child has many demands that he insists be quickly met. He sometimes cries for no apparent reason and becomes frustrated very easily.
5. **Short attention span**, with an inability to concentrate. The ADHD child rarely completes a project, and his ability to sit quietly through school, meals, or TV programs is very limited, even though he may be enjoying the activity. For example, the child might be enjoying a children's TV program but he cannot sit still and watch it. He gets up, moves to another chair, sits down on the floor, and goes back to his original seat — continually moving about throughout the program.
6. **Very poor coordination.** The child is often considered clumsy and inattentive to what he is doing; however, closer observation shows that he cannot coordinate the right side of his body with the left. Buttoning clothes and generally dressing is difficult; he has difficulty in writing and drawing because his eyes and hands fail to function together; he does poorly in sports that require catching and throwing a ball, and he frequently bumps into objects.

   Poor coordination extends also to the thought processes. A child may mean one thing and say exactly the opposite. He may be told to do something, yet he does the exact opposite. This is not failure to obey, but confusion within the mental processes.
7. **Poor sleep habits.** The child is usually a restless sleeper who does not want to go to bed. He will wake

up often throughout the night. Many ADHD children cannot get enough sleep for their bodies' needs.

Food and Chemical Sensitivity

Sensitivity or allergic reactions to food, especially food additives such as colorings and preservatives, can contribute to learning disabilities and/or hyperkinesis. Food additives as a cause of hyperkinesis have been well-documented by Feingold,[18] although there are many who dispute his findings. Applied kinesiology examination findings support his conclusions in some cases, but food and chemical sensitivity is not the cause of all cases of learning disabilities. The applied kinesiology technique of evaluating for food sensitivity is a definite asset in determining which cases may be involved and what products may be detrimental to a child.

Emotional Neurovascular Reflex

Interplay between the sides of the triad of health — physical, chemical, and mental — may perpetuate an obvious health problem while leaving the basic cause undiscovered. This is often true when the obvious problem is structural or chemical and the perpetuating factor is on the emotional side of the triad.

Structural problems may be perpetuated by muscular imbalance. Frequently a category II posterior ilium is secondary to poor function of the sartorius and/or gracilis muscles, associated with the adrenal glands. (Improving adrenal gland function is discussed in the section on adrenal stress disorder in Chapter 12.) Persistent hypoadrenia with associated sartorius and gracilis muscle dysfunction may be caused by stress, one type of which is emotional. If it is the basic cause of the hypoadrenia, there will be little or no adrenal gland response until the emotional stress is corrected. An example of emotional stress perpetuating a condition on the chemical side of the triad is some form of digestive disturbance, such as hyperchlorhydria.

The emotional side of the triad of health is the one frequently overlooked by applied kinesiologists. The technique described earlier of engaging patients in conversation about suspected emotionally associated subjects often helps reveal covert emotional stress that may cause loss of correction and perpetuate the health problem.

The emotional neurovascular reflex originally discussed by Bennett[32] and adapted into applied kinesiology by Goodheart is an effective adjunct to managing and overcoming emotional stress that interferes with an individual's health. There are two types of emotional stress treated by this reflex. The more common is that related with social interaction, employment, and self-esteem. In addition to treatment with the emotional neurovascular reflex, treatment with psychological reversal is of value. Of course, if an individual's stress relates with financial problems, a child on drugs, or some other specific problem, it must be approached directly.

The most dramatic effects from emotional neurovascular treatment occur when the problem relates with some specific incident an individual has experienced and cannot deal with realistically. It may be the vivid memory of a horrible accident, the death of a loved one, or personal embarrassment such as being humiliated while speaking in public. The list of subjects that can cause continued emotional disturbance is as long as what people deal with in their everyday lives. What may seem inconsequential to one may be horrifying and detrimental to another.

Many of the emotional problems dealt with by this technique are those found in general examination to determine the reason for a recurring posterior ilium subluxation or a digestive disturbance, as mentioned above. It is not necessary for a physician to know the precise emotional factor. Often it is best to simply say to the patient, "My examination indicates there is some type of emotional factor interfering with the maintenance of corrections that have been made. I am not interested in the specific emotional factor. I want you to think of what you feel may be emotionally detrimental to you." Sometimes the patient will volunteer information about a family problem or some other emotional conflict; others will simply reply that there is something bothering them. Ask the person to think about the problem with his eyes closed. Watch the eyelid for rapid eye movement (REM), which is often associated with the individual's emotional experience. Test a previously strong muscle for weakening. It can be the pectoralis major (clavicular division), which is strongly associated with emotions. If there is a specific problem, such as a recurrent posterior ilium, test the sartorius and/or gracilis for weakening. The REM and muscle weakening indicate that the emotional neurovascular technique will probably be of value. There will be positive therapy localization to the neurovascular reflex when it needs treatment.

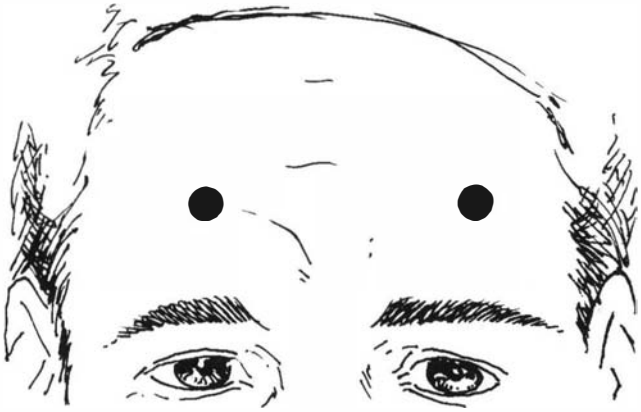
Treatment

The neurovascular reflex is located bilaterally on the frontal bone eminence and is treated with a light, tugging contact, as is usual for neurovascular reflexes. It is necessary to vary the vector of tug on the skin until maximum pulsation is felt. In some cases it may be necessary to hold the contact for several minutes. Indication that the reflex has been adequately treated is negative therapy localization to it.

Having the patient think about the emotional problem while the neurovascular reflex is being held appears to improve treatment effectiveness but is not necessary. If you ask the person to think about the problem, request

that he hear, see, smell, feel, and remember it.

The reflex appears more receptive to treatment when the conception vessel is first "run" to open it. The conception vessel is opened by the physician passing his hand



10—2. Emotional neurovascular reflex.

over the meridian from its beginning (CV 1) to its end (CV 24) at the lower lip; this weakens or opens the meridian. CV 1 is located at the perineum. It is not necessary to begin at CV 1; beginning over the symphysis pubis near CV 2 is adequate. This is opposite from running the twelve bilateral meridians, which are weakened by passing the hand over the meridian from its end to its beginning. After the neurovascular reflex is successfully treated, the conception vessel is closed by running it from its end (CV 24) at the lower lip to near its beginning (CV 2) at the symphysis pubis.

After the neurovascular point has been treated, have the patient again relive the emotional experience and retest the muscle(s) that weakened during the previous test to determine it remains strong.

Treatment of the emotional neurovascular reflex often produces very dramatic results. The physician may not be aware of how important the treatment is to the patient because the emotional problem was not discussed. The effectiveness of the treatment may initiate the referral of patients specifically for treatment of an emotional condition with which they cannot cope. An example is a retired gentleman who was referred to me because he could not overcome his wife's death. During the initial consultation he could not explain the reason he was in the office, although I was aware of it because the referring patient had discussed it with me. Every time the gentleman attempted to say that he could not get over his wife's death, he would break into tears and be unable to talk. He was able to tell me that he was so emotionally distraught that he could not raise a cup or glass to his lips without spilling the contents. Prior to treatment he was given a glass of water to attempt the action and, as stated, he spilled a large amount. After the neurovascular treatment he was able to bring a glass of water to his lips and drink it with minimum shaking. After three treatments he no longer had any tremor when thinking about his wife, and he could easily talk about her. He stated he was finally able, after nine months, to cope with her death.

Bach Flower Remedies

Edward Bach developed a system of treating patients based on their fears, anxieties, greeds, likes, and dislikes.[2] The remedies consist of thirty-eight homeopathic flower preparations developed in his later career.

Bach's life is told in an interesting story by Nora Weeks,[48] his assistant for many years. After his death she continued to make people more aware of his treatment methods. His work is now recognized and well-received by many in natural health care. Diamond,[14] who introduced the remedies to applied kinesiologists, states in Weeks' book, "The true medical advance will always be simple, natural, beautiful, loving, and poetic. This was very difficult in Bach's day and today it has become exceedingly rare as we are now faced with increasing regulation and the drive to conformity. The time for true far-reaching medical pioneering, such as Bach did, may well be over."

Bach's early career was as a pathologist and bacteriologist. He developed a series of vaccines from intestinal bacteria. The information was published and widely received. Later he changed the mode of vaccine administration from injection to an oral homeopathic preparation. He developed the treatment with seven groups of bacilli during this period in his career.

Bach recognized that patients could be grouped into personality types. Within a group of patients having the same condition, there are different responses to therapy depending on the personality group into which the patient falls. Bach thus recognized early in his education that an individual's personality is important in his response to treatment of disease. By observing people he rationalized that within a large group individuals would not suffer from the same kinds of diseases, but within the large group are smaller personality groups that would react the same or nearly the same to any type of illness. Thus began his search for treatment of emotional deviation as a method of combatting health problems.

Bach's direction was toward seeking purer remedies that were gentle, non-toxic, and effective in treating the patient instead of the disease. This was reflected in his own life's interest, a fondness for animals and nature in general. In searching for his natural healing method he left the scientific community, where he was well-accepted, to move around the fields and meadows in search of the

herbs that would effectively treat the emotional classifications in which he found people to fit. He was convinced that poisonous substances and plants could have no real function in healing the human body. In 1928, he discovered the first of what were to become known as the Bach remedies.

The Bach remedy used for treatment is determined by the emotional classification of the patient; thus, many think the remedies are specifically for emotional treatment. Bach saw it as much more. In his booklet, *The Twelve Healers and Other Remedies*,[2] which summarizes all the remedies, he states, "The mind being the most delicate and sensitive part of the body, shows the onset in the course of disease much more definitely than the body, so that the outlook of mind is chosen as the guide as to which remedy or remedies are necessary." In his case records,[48] there are many accounts of various physical conditions responding to the same remedy. The common factor among the patients with the various conditions was their emotional state.

The Bach remedies are used in applied kinesiology as adjunctive therapy. They help complete treatment to the triad of health. This writer has witnessed many occasions when a patient improves to a plateau and then fails to respond further until a Bach remedy is added to the regimen. Usually the patient is not told the purpose of the remedy; this helps eliminate any placebo effect that may be present.

Treatment for the emotional backache, discussed next, can be supplemented with impatiens. The key words for this remedy are impatience, irritability, and extreme mental tension.[13] This often describes the overachiever, typically the emotional backache sufferer. He is a striving individual, anxious to get as much accomplished as possible, and is quite often irritated that he has a backache; he wants to get over it as quickly as possible.

The Bach remedies are available in concentrate stock form. For use, two drops of the stock are placed in 1 oz. of 50% water and 50% brandy as a preservative.

In 1982 the Bach remedies were classified by the Food and Drug Administration as over-the-counter homeopathic drugs. This classification enables them to be imported by the American distributor from England, where they are produced. The classification also puts restrictions on formulation and re-labeling. Since states differ in their health laws, one should contact the State Board of Pharmacy to determine if formulation and re-labeling can be done by other than a licensed pharmacist. In most states, dispensing the unopened concentrate for the patient to mix is proper.

The Bach remedies may be ordered from Global Health Alternatives, Inc., 193 Middle Street, #201, Portland, ME 04101 (voice 207-772-7234, FAX 207-772-8493) or search the internet for other sources. There are various kits available; the remedy concentrates may be purchased in 5, 10, and 30 ml sizes.

Five or six drops of the treatment solution are put in the patient's mouth, ranging from several times an hour to a few times per day, depending on the severity of the condition and rapidity of results desired. There is no danger of overtreatment, or treatment with the wrong substance. Bach[2] states, "As all these remedies appear harmless, there is no fear of giving too much or too often, though only the smallest quantities are necessary to act as a dose. Nor can any remedy do harm should it prove not to be the one actually needed for the case." Kaslof[28] reviews the chiropractic use of Bach remedies by stating that "...Bach remedies may be classified as having a self-diminishing effect. As internal conflict is resolved, both the need for and the effectiveness of the remedy diminishes."

Although the remedies are non-toxic and in themselves have no side effects, there may be a minor reaction such as a rash or an accentuation of negative emotions. This appears to result from emotional unraveling, with an unconscious resistance to dealing with them. In addition, as some aspects clear, others that may be more deeply seated are uncovered. An emotional reaction can be secondary to one that is repressed. As the primary condition improves, the repressed one may surface, requiring a change in Bach remedy treatment.

One will often identify several emotions or moods in the patient, indicating several remedies. The applicable remedies can be mixed together in a single treatment bottle. Usually the number of remedies is not over six.

In addition to oral treatment, the Bach remedies may be applied topically. For example, in joint pain several drops of the remedy appropriate to the patient's emotions are put in water, which is used to wet a cloth that is put over the painful joint. This application is sometimes startling in its effectiveness.

For prolonged administration, one can put the appropriate remedies into a vaporizer for inhalation therapy. Usually one dropper full of the remedy is put in two quarts of water to vaporize in the bedroom while the patient sleeps.

Following is a brief description of the emotions and moods indicating the different Bach remedies. A more thorough description is available in Chancellor's work.[13] He presents numerous case histories of patients treated with the method. A cross-reference list indicating the remedy types for different emotional moods is presented by Wheeler.[53] Both of these reference works help identify the remedy appropriate for the patient.

Guide to the Bach Flower Remedies*

Agrimony. For those not wishing to burden others with their troubles and who cover up their suffering behind a cheerful facade. They are distressed by argument or quarrel, and may seek escape from pain and worry through the use of drugs and alcohol.

Aspen. For those who experience vague fears and anxieties of unknown origin. They are often apprehensive.

Beech. For those who while desiring perfection easily find fault with people and things. Critical and at times intolerant, they may overreact to small annoyances or the idiosyncrasies of others.

Centaury. For those who are overanxious to please, often weak willed and easily exploited or dominated by others. As a result they may neglect their own particular interests.

Cerato. For those who lack confidence in their own judgment and decisions. They constantly seek the advice of others and may often be misguided.

Cherry Plum. For those fearful of losing mental and physical control, of doing something desperate. May have impulses to do things thought or known to be wrong.

Chestnut Bud. For those who fail to learn from experience, repeating the same patterns or mistakes again and again.

Chicory. For those who are overfull of care for others and need to direct and control those close to them; always finding something to correct or put right.

Clematis. For those who tend to live in the future, lack concentration, are daydreamers, drowsy or spacey and have a halfhearted interest in their present circumstances.

Crab Apple. For those who may feel something is not quite clean about themselves, or have a fear of being contaminated. For feelings of shame or poor self-image. For example, thinking oneself not attractive for one reason or another. When necessary, may be taken to assist in detoxification, for example, during a cold or while fasting.

Elm. For those who at times may experience momentary feelings of inadequacy, being overwhelmed by their responsibilities.

Gentian. For those who become easily discouraged by small delays or hindrances. This may cause self-doubt.

Gorse. For feelings of hopelessness and futility; when there is little hope of relief.

Heather. For those who seek the companionship of anyone who will *listen* to their troubles. They are generally not good listeners and have difficulty being alone for any length of time.

Holly. To be used when troubled by negative feelings such as envy, jealousy, suspicion, revenge. Vexations of the heart, states indicating a need for more love.

Honeysuckle. For those dwelling in the past, nostalgia, homesickness, always talking about the good old days, when things were better.

Hornbeam. For the Monday morning feeling of not being able to face the day. For those feeling that some part of the body or mind needs strengthening. Constant fatigue and tiredness.

Impatiens. For those quick in thought and action, who require all things to be done without delay. They are impatient with people who are slow, and often prefer to work alone.

Larch. For those who, despite being capable, lack self-confidence or feel inferior. Anticipating failure they often refuse to make a real effort to succeed.

Mimulus. For fear of known things, such as heights, water, the dark, other people, of being alone, etc.

Mustard. For deep gloom that comes on for apparently no known reason, sudden melancholia or heavy sadness; will lift just as suddenly.

Oak. For those who struggle on despite despondency from hardships; even when ill and overworked, they never give up.

Olive. For mental and physical exhaustion, sapped vitality with no reserve. This may come on after an illness or personal ordeal.

Pine. For those who feel they should do or should have done better, who are self-reproachful or blame themselves for the mistakes of others. Hardworking people who suffer much from the faults they attach to themselves, they are never satisfied with their success.

Red Chestnut. For those who find it difficult not to be overly concerned or anxious for others, always fearing something wrong may happen to those for whom they care.

Rock Rose. For those who experience states of terror, panic and hysteria; also when troubled by nightmares.

Rock Water. For those who are very strict with themselves in their daily living. They are hard masters on themselves, struggling toward some ideal or to set an example for others. This would include strict adherence to a living style or to religious, personal, or social disciplines.

Scleranthus. For those unable to decide between two things, first one seeming right then the other. Often presenting extreme variations in energy or mood swings.

Star of Bethelem. For grief, trauma, loss. For the mental and emotional effect during and after a trauma.

Sweet Chestnut. For those who find they have reached the limits of their endurance. For those moments of deep despair when the anguish seems to be unbearable.

Vervain. For those who have strong opinions and who usually need to have the last word, always teaching or philosophizing. When taken to an extreme, they can be argumentative and overbearing.

Vine. For those who are strong-willed. Leaders in

**L.J. Kaslof, "Bach flower remedies: A unique adjunct to chiropractic care," Amer Chiro (Sept 1985). Reprinted with permission.*

their own right who are unquestionably in charge; however, when taken to an extreme they may become dictatorial.

Walnut. Assists in stabilizing emotional upsets during transition periods, such as puberty, adolescence, and menopause. Also helps one to break past links and emotionally adjust to new beginnings such as moving, changing or taking a new job, beginning or ending a relationship.

Water Violet. For those who are gentle, independent, aloof, and self-reliant, who do not interfere in the affairs of others, and when ill or in trouble prefer to bear their difficulties alone.

White Chestnut. For constant and persistent unwanted thoughts, such as mental arguments, worries or repetitious thoughts that prevent peace of mind and disrupt concentration.

Wild Oat. For the dissatisfaction with not having succeeded in one's career or life goal; when there is unfulfilled ambition, career uncertainty, or boredom with one's present position or station in life.

Wild Rose. For those who for no apparent reason, have resigned themselves to their circumstances. Having become indifferent, little effort is made to improve things or find joy.

Willow. For those who have suffered some circumstance or misfortune that they feel was unfair or unjust. As a result they become resentful and bitter toward life or toward those who they feel were at fault.

Emotional Backache

Low back and neck pain, characterized in applied kinesiology as "emotional backache," is considered as such due to the unique therapy localization and therapeutic response from adrenal treatment. There is no question that back pain can be a psychophysiological musculoskeletal disorder.[43] Patients with adrenal stress disorder, especially when there is a blood sugar handling problem, have highs and lows of feelings, from euphoric to depressed. It is well-established that more pain is reported by depressed patients,[20] and that men especially appear very neurotic, with high hysteria and a hypochondriasis when not working and functioning according to their social role.[47]

When considering emotional back pain, one must look at many factors. Some personality types are more prone to back problems and do not respond positively to treatment, either conservative or surgical.[36,54]

Care must be taken in determining that an individual has a psychosomatic back problem. When an individual presents a confused neurologic pattern that fails to relate with standard orthopedic tests, he is often classified as having a psychosomatic problem. Most applied kinesiologists have successfully treated individuals who have been referred to psychiatrists and psychologists for back pain treatment. The Minnesota Multiphasic Personality Inventory (MMPI) was used to evaluate patients with low back pain who were classified as organic, mixed, and functional.[19] The organic group consisted of persons in whom an orthopedic physician found an organic basis for their pain as determined by physical exam, x-ray, and surgery that correlated with the patients' descriptions of pain. The mixed group had an organic basis to explain the pain, but it was insufficient to account for the amount of pain and disability the patients reported. The functional group showed no organic basis for the pain. On the Fc scale of the MMPI, a significant difference between the functional and organic groups was expected by the researchers because, as they state, "...patients with completely functional problems tend to exhibit confused thought processes." They were surprised to find no difference between the mixed and functional groups. This does not seem surprising from an applied kinesiologist's point of view. Often those persons categorized as not having an organic basis for pain are neurologically disorganized on applied kinesiology examination. The individuals are frequently confused in their thought processes. Correction of the neurologic disorganization often eliminates the back pain with no psychological counseling, and the patients return to a functional status.

The back pain referred to in applied kinesiology as emotional back pain is functional; usually no disc or other pathological problems are found. There is a predictable recurrence with the eustress of the overachiever,[40] or any stress related with the four categories — emotional, physical, chemical, and thermal. In fact, the stress may not be major; it may be the accumulation of stress from each of the categories.

Goodheart[22] postulates that individuals with stressed or high output adrenal function produce constituents in adrenalin that inhibit normal ligaments. The ligaments of the lower spine and sacroiliac articulations appear particularly vulnerable to this type of adrenal stress. This is based on positive therapy localization to the sacroiliac articulations, which shows only when the gracilis or sartorius muscles associated with the adrenal gland are tested. Chewing adrenal concentrate eliminates the positive therapy localization and clinically improves an individual's back pain.

The upper cervical ligaments are sometimes involved with this low back condition. This involvement can be determined by having the supine patient therapy localize the sacroiliac articulations while lifting his head from the table. The physician applies caudal pressure on the vertex of the patient's head as he attempts to turn it right and left a few times. Positive association is the weakening of sternocleidomastoid muscles that were strong in

the clear. The same pressure application and head turning without sacroiliac therapy localization is negative.

The ligament stretch reaction (see page 200) is often present throughout the body in conjunction with the applied kinesiology emotional backache. Because of the ligament involvement, manipulation of the spine — as well as other articulations of the body — is contraindicated until adequate treatment to re-establish normal ligament function has been completed.

Effective treatment for this condition is nutritional support in the form of whole raw adrenal concentrate. Immediately upon chewing this substance, weakening of the adrenal-associated muscles with therapy localization to the sacroiliac is eliminated, as is the cervical test. Adrenal concentrate is generally needed on a more intensive basis than usual. The patient chews twelve to fifteen tablets per day for the first few days; the dosage is reduced as symptoms improve. The nutrition may be needed for two or more weeks, depending on the condition's severity. Prolonged activation of the neurolymphatic and neurovascular reflexes for the adrenal gland enhances the correction. Treatment for emotional backache should be correlated with adrenal stress disorder, discussed in Chapter 12.

Patients with emotional backache are usually aggressive individuals who work long, hard hours and are leaders in their communities. Typically, these individuals play as hard as they work.

Homolateral Gait and Crawl Pattern

Cross-pattern activity (flexion of contralateral arm and leg), discussed in Chapter 5, enhances general muscle function in the body under certain conditions, especially if an individual is neurologically disorganized. Cross-pattern training has been successfully used in applied kinesiology in treating individuals who have failed to develop properly through the neurologic stages that culminate in bilateral organization and, finally, unilateral dominance.

Some individuals have a neurologic organization in which cross-pattern activity generally causes strong muscles to weaken, while a homolateral pattern causes previously weak muscles to strengthen. This was first observed by Goodheart[21] and was correlated with schizophrenia. When an individual is enhanced by the homolateral pattern, he will also exhibit positive therapy localization to KI 27, but only when done cross-handed, i.e., with fingertips of the right hand touching the left KI 27, and fingertips of the left hand touching the right KI 27. Care must be taken that the two hands do not touch each other, causing a short circuit. One must also be careful that the patient actually therapy localizes to KI 27. There is a tendency to be too far from the midline, or to have the fingertips of one hand too low. KI 27 is a small point that must be therapy localized accurately. It is located at the junction of the sternum, clavicle, and 1st rib.

10—3. Cross-therapy localization to KI 27.

Cross-KI 27 therapy localization will be present in all schizophrenics, but it does not necessarily mean all who exhibit a positive cross-KI 27 have schizophrenia. There will be neurologic disorganization that exhibits the homolateral crawl pattern in every instance of positive cross-KI 27 therapy localization.

Disturbed muscle function in schizophrenics has been established by Whatmore and Ellis.[49] They found low levels of residual motor activity in schizophrenics endeavoring to rest and relax as completely as possible in a quiet, comfortable environment. The muscle activity is, in most cases, covert. It can only be observed by recording with low-level electromyographic equipment. This intermittent or continuous, static or phasic muscle activity is called hyperponesis. Whatmore and Ellis[51] propose the hypothesis that "Hyperponesis is a form of energy expenditure within the nervous system, a way of reacting to environment and self, that has detrimental effects on nervous system function. It can trigger a schizophrenic exacerbation in a nervous system prone to schizophrenic reactions through inherited or acquired characteristics. When the hyperponesis is diminished adequately, by whatever means, the nervous system is freed from this deleterious influence and can return to a state of better functioning." This mind-muscle integration seems to represent the neurologic disorganization clinically observed in applied kinesiology examination. It may be the "static" in the nervous system that causes the sensory aberrations and disturbed perception of the schizophrenic.

The original applied kinesiology treatment for schizophrenia was to pattern the patient homolaterally.[21] This treatment was supplemented with niacin or niacinamide and B_6, and other means of orthomolecular psychiatry. The homolateral patterning, along with the other appropriate treatments for schizophrenia, produced good

results; however, it was observed by this author[45] that after a period of time some patients did not progress as well and, in fact, complained of symptoms developing after the homolateral patterning. The symptoms related to sensory disturbances, feelings of disorientation, and sometimes muscular pain. When these complaints were aired, the patient was re-examined for the appropriateness of homolateral pattern activity. It was found that the homolateral activity no longer enhanced the patient; rather, he had shifted to a standard cross pattern. No longer was there positive cross-KI 27 therapy localization; KI 27 would either be negative or show a standard positive therapy localization for neurologic disorganization. It appeared that some therapeutic measure had shifted the patient from a cross-KI 27 homolateral pattern to a standard cross pattern. After shifting from the homolateral pattern, the patient may have positive or negative standard therapy localization to KI 27. If there is evidence of standard neurologic disorganization, the patient is treated for it as usual.

The shifting from homolateral crawl to cross pattern was present in schizophrenic patients who responded well, often completely recovering from the schizophrenia. Patients with poor response failed to shift from the homolateral pattern to the cross pattern; they continued to maintain positive cross-KI 27 therapy localization.

It appeared that the ultimate goal was to shift from the homolateral pattern to the standard cross pattern and eliminate positive cross-KI 27 therapy localization. Efforts were then begun to find the treatment that would eliminate the positive cross-KI 27. Conditions relating with standard neurologic disorganization are also responsible for the neurologic disorganization evidenced by positive therapy localization to KI 27. As indicated in the neurologic disorganization discussion (Chapter 5), the cranial-sacral primary respiratory mechanism is the most common cause of neurologic disorganization, followed by foot and gait dysfunction, poor modular interaction, and other less common causes. The most common therapeutic effort that will eliminate positive cross-KI 27 therapy localization is equilibrium synchronization, accomplished by cranial, upper cervical, and pelvic correction. In this author's experience, over 75% of the cases are corrected with equilibrium synchronization.

After correction of the suspected cause, the patient should be re-evaluated with cross-KI 27 therapy localization and homolateral patterning. If the correction fails to eliminate cross-KI 27 and the homolateral pattern, proceed to evaluate other factors, such as the feet, gait, PRYT pattern, and dural tension. Most patients shift from homolateral to cross-pattern activity with structural corrections, as indicated above. Some shift by chewing niacin or niacinamide[25,33] and vitamin B_6. Occasionally one finds that a severe sugar handling stress is the cause of the homolateral pattern.

When the patient shifts away from the cross-KI 27 pattern, there may or may not be positive therapy localization to KI 27 in the usual manner. If there is positive therapy localization to KI 27, proceed to neurologically organize the patient using the standard methods described in Chapter 5.

When procedures were initially developed to shift an individual from a homolateral to a cross pattern, the cross-pattern training was prescribed as a routine measure; it has since been found unnecessary in most cases. If cross patterning is prescribed, one must take care to warn the patient that if he feels bad during or after the patterning, he should discontinue it until re-examined. It is possible for the correction that eliminated the homolateral pattern to be lost. In this case, homolateral patterning will enhance the individual, and cross patterning will be detrimental. During the initial stages of treatment, one may have to correct the cause of the cross-KI 27 several times before proper and lasting organization is established.

As indicated previously, not all individuals with a cross-KI 27 and a homolateral pattern have schizophrenia, and it is not in the scope of practice of most physicians using applied kinesiology to diagnose the condition. Some cases that exhibit positive therapy localization to cross-KI 27 and a homolateral pattern have been previously diagnosed as schizophrenic by a psychiatrist or psychologist. In these cases, one can tell the patient and his family that this is a usual pattern seen in the condition. Emphasize that treatment is directed to total body organization and general health. Although this treatment is effective in schizophrenia, it is not a panacea for the condition. Never diagnose schizophrenia on the limited basis of positive therapy localization to cross-KI 27 and a homolateral pattern.

A relatively simple evaluation procedure for schizophrenia is the Hoffer-Osmond Diagnostic (HOD) Test,[26,29,30] which consists of 145 questions. Six scales or scores are used for evaluating the patient's status: the total score (TS), perceptual score (PerS), paranoid score (PS), depression score (DS), ratio score (RS), and a short form (SF) score.

The total score is the number of positive answers given to the questions. It provides an overall assessment of the patient's condition. There are cut-off scores for various age levels for differentiating the psychotic condition as schizophrenia or some other condition.

The perceptual score is calculated from fifty-three statements in the test. When there is a high perceptual score, it is observed in an applied kinesiology practice that the patient may not fully understand explanations of examination procedures or of his condition. Great care must be taken in providing the explanation so the patient will understand. It may be advisable to have a relative or friend present.

The paranoid score can give information to help manage the patient. A high paranoid score indicates that great care must be taken in communicating with the patient, as there is a tendency toward poor interpersonal relationships.

The depression score is particularly valuable in helping manage the patient and in determining whether psychiatric referral is necessary, possibly for anti-depressant drugs or custodial care. Although there are definite advantages in home care, the patient may be so deeply depressed that he may be suicidal and need the constant protection provided in an institution.[38]

The ratio score is mathematically calculated from the total and depression scores. It has been found to make a sharper discrimination between schizophrenic and non-schizophrenic psychiatric patients than any of the other scores. The short form is used for large population screening. It was found to have a better differentiation between schizophrenic and non-schizophrenic patients than the total test on a cross-validation study. It is not a replacement for the total test, as the latter provides more comprehensive information.

The HOD Test is available from suppliers[3] and is easily administered. Supplied with the test questions is a pamphlet giving a synopsis of the viability of the HOD Test, its administration, and grading. The questions are supplied on cards. The patient simply sorts the questions into two boxes labeled "yes" and "no." A score sheet is marked for the questions answered yes, and then overlay sheets are used to obtain the various scores.

The HOD Test has proven reliable in the evaluation of schizophrenia. It can be given repeatedly to an individual to evaluate improvement from the therapy, or possible worsening of the condition.

Many interesting experiences develop when patients who have positive cross-KI 27 therapy localization are routinely administered the HOD Test.

To justify the HOD Test to a patient, simply explain that his examination shows the possibility of an overactive sensory nervous system. Explain, "These questions will help me evaluate various aspects of your sensory nervous system and perception. This is not a test that you pass or fail, it just categorizes how your nervous system is working." If the patient has a high HOD total score, explain that the test confirms the overactivity of the sensory nervous system, and treatment will be applied to help improve balance. Refrain from correlating the score with schizophrenia unless trained to do so, or the patient has previously been diagnosed as schizophrenic by a psychiatrist or psychologist. It may be necessary to refer the patient to a mental health specialist for further evaluation and possible treatment.

When one discusses the symptoms identified by the HOD Test with the patient, it is not unusual for the patient to experience an emotional catharsis. He may state that he was afraid to tell the symptoms to anyone because he felt he was going crazy. The patient is often greatly relieved to find someone who understands what has been turmoil of increasing magnitude.

Body language indicating the need to test for cross-KI 27 and homolateral crawl is supersensitivity to stimuli of almost any type. The patient may react excessively to digital pressure almost anywhere on the body. One may think that the patient is simply afraid of manipulative efforts to the spine or some other articulation when, in reality, the sensory nervous system is supersensitive to the usually mild manipulative force.

When the neurologic disorganization of homolateral pattern and cross-KI 27 returns to normal, the symptomatic pattern improves dramatically. The HOD Test can be used repeatedly to determine improvement of the condition or the possibility of relapse.

Treatment for Phobias

Like other problems that relate with the mental side of the triad of health, many patients currently under an applied kinesiologist's care have a problem with one or more phobias but they have not told the doctor. Patients tell a doctor what they believe he needs to know. Most are unaware that there is an effective treatment for phobias in applied kinesiology. The examination and treatment procedures were developed by Callahan,[8] a psychologist using applied kinesiology techniques.

A phobia is an irrational fear. It can be directed to almost any factor in an individual's environment and day-to-day function. The key feature is that a phobia is an unrealistic or disproportionate fear of a situation. When dealing with a phobia, most psychiatrists and psychologists believe that "The fear that [the patient] feels in the presence of a certain object or experience is really the displaced fear of some anxiety-producing component within his own personality."[34] This, of course, indicates that the proper treatment would be directed to the anxiety-producing component. The phobia treatment developed by Callahan shows this need not be so. By testing patients with applied kinesiology methods, Callahan has found that phobias relate with improper energy flows within the body. He uses manual muscle testing to determine how a patient's neuromuscular system reacts to vocalized statements regarding the possible phobia. The disturbed energy patterns related with the phobia are found in the meridian system. Therapy localization to alarm points determines the meridian involved. Following treatment the patient is immediately relieved of the phobia, with no concern directed toward treating the personality. Callahan's finding — that disturbed function produces the phobia, not the individual's personality deviation — fits well with the discussion earlier in this chapter that mental health problems are often due to physiology gone awry.

Phobias can be directed toward almost anything.

There may be a severe fear of public speaking, objects, animals, interpersonal relations, diseases, activities — the list could go on and on.

If a phobia does not severely limit an individual's activity, it is called a "simple" phobia. This is a phobia that can be "worked around," such as fear of snakes, cats, spiders, airplanes, and elevators. These are things an individual can avoid. There may be a rationalization for the avoidance, such as "I much prefer to drive and see the beautiful scenery, that you can't see from an airplane." Simple phobias are the ones most easily treated by Callahan's method.

Other types of phobias severely limit an individual's life. An example is a fear of crowds and open spaces, called agoraphobia, which literally means fear of the market place. An agoraphobic may never leave his house because of these deep-seated fears. This type of severely limiting phobia is called a compound phobia; it is not as common as the simple ones.

Callahan's method of treating phobias is so simple and effective that it has been presented in books by Callahan[9] and Durlacher[17] for the lay public titled the *Five Minute Phobia Cure* and *Freedom From Fear Forever,* respectively. Callahan has been on many television and radio talk shows, effectively demonstrating the technique. Individuals who had previously screamed with fear of snakes were minutes later petting one. People with a fear of height, to the degree of not going up the second rung of a ladder, were minutes later climbing to the top rung with no fear. These demonstrations include the wide range of phobias that can be demonstrated before and after treatment.

The most significant evidence of the effectiveness of this treatment is the immediate elimination of the phobia, demonstrated by the individual confronting it with no fear. Fear of public speaking is a phobia that is easily tested on a radio talk show. After treatment directed by telephone, people with a fear of public speaking go on to communicate on the show in an effective and fearless manner.[11] Speaking with no fear on the radio when thousands are listening is a strong test of the treatment's effectiveness. Callahan's second edition of his lay book, *How Executives Overcome the Fear of Public Speaking and Other Phobias*,[10] has additional material for eliminating the fear of public speaking.

Examination and Treatment

The procedures discussed here vary somewhat from Callahan's description in his lay books.[9,10] Here the reader is expected to understand the applied kinesiology approach to the meridian system, eyes into distortion, right and left brain function, and accurate manual muscle testing, as discussed earlier in this text.

The first — and possibly most important — examination and treatment procedure is to clear the individual of any psychological reversal (see page 427). This is the most common cause of failure to effectively treat phobias. First examine the individual for massive reversal by having him state, "I want to have a good life," while testing a previously strong muscle; then test the individual with the statement, "I want to be miserable." The patient should remain strong on the former statement and weaken on the latter. Treatment for psychological reversal, if needed, must be administered at this stage before further testing is done.

The best muscle to test in evaluating for phobic reactions is the pectoralis major (clavicular division). Have the patient think of his fear and test the muscle. It should become weak during the thought process; if it does not, the patient is psychologically reversed specifically for the phobia. Further testing for psychological reversal relating to the phobia can be done with specific questions regarding the phobia. For example, if an individual has a fear of dogs, have him state, "I want to get rid of my fear of dogs." If psychologically reversed, he will weaken on the statement and be strong when saying, "I want to retain my fear of dogs." Treatment must be directed toward this specific psychological reversal. In this case, treat the same as indicated on page 428, except use the positive affirmation, "I profoundly and deeply accept myself with all my problems and shortcomings, including my fear of dogs." Be certain to follow through with all the other aspects of treating psychological reversal. When the psychological reversal has been effectively treated, the patient should test weak while thinking of his phobia.

At this point, the weakness observed while the patient thinks of his fear is no different than what has been observed for a long time in applied kinesiology. As discussed previously, when individuals concentrate on an emotionally distressing subject, previously strong indicator muscles test weak. This is most easily observed by testing the pectoralis major (clavicular division). Callahan's contribution is a diagnostic system that locates the area of disturbance in the meridian system. The most common imbalance is of the stomach meridian. While the patient concentrates on the fear and the indicator muscle tests weak, have him therapy localize to the stomach alarm point, which is CV 12 located halfway between the umbilicus and tip of the xiphoid. If the stomach meridian is involved, the indicator muscle will now test strong while the patient continues to concentrate on the fear. Approximately 95% of all phobias are associated with the stomach meridian. If the indicator muscle fails to strengthen with stomach alarm point therapy localization, find the alarm point that cancels the indicator muscle weakness while the patient thinks of his fear. The kidney meridian is the next most common involvement.

Before treatment it is important to have the patient rate his phobia on a scale from 1 to 10, with 10 being the maximum level of disturbance. Following treatment, the patient is again asked to rate his phobia. The following chart from Callahan[9] may help the patient rate his condition.

10 — The discomfort is the worst it can possibly be. I can't tolerate it. It puts me into a panic.
9 — Discomfort is very close to intolerable.
8 — My fear is very severe.
7 — My fear is severe.
6 — Fear is very uncomfortable.
5 — Fear is uncomfortable but I can stand it.
4 — Fear is noticeable and bothersome but I can stand it.
3 — I feel a slight degree of fear but I am totally in control.
2 — I am rather calm and quite relaxed with no fear.
1 — I am perfectly calm and totally relaxed.

The manner in which people react to their phobias varies. Some may develop sheer panic just by thinking about the phobia. Others may have no fear while thinking of the phobia, but on direct confrontation they panic. For example, an individual may think of cats with nothing more than distaste for the subject, but if a cat is brought into the room, he will do everything he can to get away from it. An important factor in phobia treatment is that an individual be able to confront the phobia immediately after treatment. This fixes the effectiveness of the treatment in the patient's mind. When the patient does not confront the object of fear for some time after treatment, effectiveness is lessened. When the patient develops a high level of fear from simply thinking of the subject or object, this creates no problem. Sometimes when a patient is treated in an office setting, the fear — such as flying in an airplane — cannot be created. In this case, treat the patient and then teach him how to perform the procedure so he can treat himself prior to confronting the phobia. Be certain to include how to treat for psychological reversal, since it may develop at any time. The patient may be clear of any psychological reversal while in the office and only thinking about the problem; it may develop when the actual situation is confronted.

When the involved meridian is established, the patient has graded his phobia, and there is no psychological reversal, treatment can begin. Treatment is so simple and effective that it is almost incredible. The primary treatment is simply to treat the involved meridian, usually the stomach, in any effective manner. The only difference in treating a phobia as opposed to balancing a meridian is that the patient concentrates on his phobia while the meridian is treated. Callahan recommends tapping the beginning and ending points of the meridian. In the case of the stomach, ST 1 is in the center of the infraorbital ridge and ST 45 is at the lateral nail point of the 2nd toe. Other methods of stimulation are also effective. Acu-aids can be used for prolonged stimulation. Remember that during the primary stimulation the patient must be thinking about his phobia.

After stimulating the meridian, the patient should test strong while thinking about his phobia. If he does not, there is probably an undiscovered psychological reversal or one that has returned just prior to treatment. In this case, treat again for psychological reversal and repeat the procedure.

When the patient tests strong while thinking about his phobia, have him rate the level of discomfort on the scale of 1 to 10. If the rating is above 2, proceed with further treatment using applied kinesiology techniques for uncovering subclinical disturbances.

Have the patient access the right brain by humming a familiar tune of his choice, while concentrating on the phobia. If the indicator muscle weakens, add therapy localization to the alarm point of the involved meridian, which should cause the indicator muscle to again test strong. Repeat the treatment to the meridian while the patient concentrates on the phobia and continues humming. Likewise, test while the patient accesses the left brain by doing simple multiplications aloud while concentrating on the phobia. Evaluate the meridian with therapy localization to the alarm point and treat it, if necessary. One may think that the patient tests weak because of the many actions used in accessing the brain, concentrating on the phobia, and attempting to resist the muscle test. It will readily be seen that this is not true, because after successful treatment the patient will be able to do all actions effectively.

Another method of accessing the brain to uncover subclinical problems is to concentrate on a problem with the eyes open or closed.[23] When an individual accesses the brain with the eyes open, the occipital and temporal cortices are accessed. When thinking about a problem with the eyes closed, the frontal lobes and temporal cortex are accessed. Have the patient concentrate on his phobia with the eyes open and test an indicator muscle for weakening. Next have the patient concentrate on the phobia with the eyes closed. The muscle may weaken with the eyes either open or closed while thinking of the phobia, but not both. Treatment consists of tapping the skull. The back of the head over the occiput is tapped if the indicator muscle weakened while thinking of the phobia with the eyes open. If the muscle weakened with the eyes closed while thinking of the phobia, tap the front of the head over the center of the frontal bone with the eyes closed. After tapping the skull twenty or thirty times at the appropriate location, the patient's muscle should not weaken while thinking of the phobia with the eyes open or closed.

The front or hindbrain examination and treatment are applicable to other conditions besides phobias, similar to the right and left bilateral brain function discussed on page 39.

Further evaluation for phobia treatment can be done with eyes into distortion and body into distortion, discussed on pages 43 and 45. If weakening of the indicator muscle is found while the patient concentrates on his phobia with either of these additional procedures, treat with the patient's eyes or body in the position that caused the indicator muscle to weaken.

When the patient's rating of his phobia is at the

lowest level possible on the scale of 1 to 10, confront him with the item toward which the phobia is directed, if possible. For example, if the patient has claustrophobia, have him go into a closet and close the door. The experience of being able to comfortably be in the confined area will reinforce the treatment's effectiveness and help to deeply establish a permanent cure.

When it is impossible to immediately confront the patient with the subject of the phobia, such as flying in an airplane, teach him how to treat for psychological reversal and tap the treatment points. Since there are no harmful effects from treatment, the patient can apply tapping to SI 1 while stating the applicable affirmation, and he can tap the points on the involved meridian while concentrating on the phobia. It may be of value to have the patient tap the points while humming or doing the multiplication tables, and to tap the points with the eyes down and to the left and down and to the right. Callahan recommends having the patient tap the acupuncture points while simultaneously rolling the eyes counterclockwise and then clockwise.

Many phobias develop from incidents that happen in childhood. These can easily be treated by parents knowledgeable in the technique, and may save a lifetime of suffering. Generally, this author is not in favor of lay people applying manual muscle testing and applied kinesiology principles. There is too much opportunity to abuse the system from lack of anatomical, physiological, and diagnostic knowledge; however, it seems reasonable to make people aware of the technique available to treat phobias and psychological reversal. Callahan's book[10] gives simple instructions regarding examination and treatment. The only major problem is that he describes the "arm pull-down test" that fails to isolate to the maximum an individual muscle, allowing recruitment and possible testing error. The major factor in the layman's application is that there are no adverse effects from improper treatment. The worst that can happen is failure to obtain results. Covering up symptoms, and perhaps a severe pathological condition, is not a problem. If the treatment fails, a doctor proficient in the procedure should be consulted.

Much of this discussion has been directed toward phobias that are interfering significantly with one's lifestyle. Indeed, phobias can cause the sufferer to refrain from activities that add pleasure to life. The claustrophobic cannot go to a restaurant or anywhere else where it is necessary to ride in an elevator. Just think of the restraints placed on his life! In addition to social and business restrictions, there may be activities or procedures that must be done. What if the person needs a CT or MRI scan? Being confined in "that large machine" would be terrifying.

With these techniques, one can rapidly relieve a patient of the fear of needles when a blood test must be done. Children can often be relieved of the fear of visiting a doctor when they have had a previous unpleasant experience. If the parents are knowledgeable about the technique, a child can be treated at home. The doctor or nurse may treat the child in the office where the child does not need to concentrate on the fear; he is already living it!

Often when an individual's phobia has been successfully treated with these techniques, it will not return. If it does, it can be immediately treated by tapping on ST 1, if that was the original treatment point. The patient does not need to concentrate on the phobia in this case because he is already experiencing it. Usually if there is a recurrence of the phobia, it will be slight and can easily be treated by simply tapping on the treatment point, even if the patient is at a social function. Rarely will anyone in the crowd notice the subtle tapping on the face. The fear will subside quite rapidly.

Anxiety neurosis, or free-floating anxiety, is a condition in which the individual cannot specifically identify the cause of his fear. An important part of the phobia treatment is for the individual to concentrate on his fear while the meridian is being treated. In this case there is no ability to identify the cause of the fear, but the individual nevertheless may rate the discomfort very high on the 1 to 10 scale. Callahan recommends that the individual concentrate on his discomfort while being treated. To test for psychological reversal have the patient state, "I want to live with no fear" and "I want to have fear." To treat for psychological reversal have the patient affirm, "I profoundly and deeply accept myself with all my faults, including any and all fears I may have." In treating free-floating anxiety, one may need to repeat the treatment more than once. Since there may be many fears in the subconscious mind, all may not be accessed with the original treatment. Have the patient think of all the fears he did not think of the first time. The success rate of phobia treatment is very high. If there is failure to obtain results, one should re-evaluate for psychological reversal, which is the most common cause for failure. Sometimes the patient will test negative for psychological reversal, but it develops immediately prior to treatment.

Sometimes treatment may be successful by one therapist but not by another. A strained psychological interaction between the therapist and patient may interfere with successful treatment application. It may be that the patient becomes psychologically reversed in the person's presence.

The reader is encouraged to pursue this subject further in books on the subject by Callahan[9,10,12] and Durlacher.[17]

CHAPTER 10 — REFERENCES

1. Abrahamson, E., & A. Pezet, *Body, Mind, and Sugar* (New York: Pyramid Books, 1951).
2. Bach, E., *The Twelve Healers and Other Remedies* (London: The C.W. Daniel Co Ltd, 1933).
3. Behavior Science Press, Institute for Social and Educational Research, 3710 Resource Dr., Tuscaloosa, AL 35401-7059.
4. Bieler, H.G., *Food Is Your Best Medicine* (New York: Random House, 1965).
5. Blaich, R.M., "Applied kinesiology and human performance." Proceedings of Winter Meeting, ICAK, Palm Desert, CA, 1987.
6. Blaine, T.R., *Goodbye Allergies* (New York: The Citadel Press, 1965).
7. Callahan, R.J., "Psychological reversal." Proceedings of Winter Meeting, ICAK, Acapulco, Mexico, 1981.
8. Callahan, R.J., "A rapid treatment for phobias." Proceedings of Winter Meeting, ICAK, Acapulco, Mexico, 1981.
9. Callahan, R.J., *Five Minute Phobia Cure* (Wilmington, DE: Enterprise Publications, Inc, 1985).
10. Callahan, R.J., *How Executives Overcome the Fear of Public Speaking and Other Phobias* (Wilmington, DE: Enterprise Publications, Inc, 1985).
11. Callahan, R.J., "Successful psychotherapy by telephone and radio." Proceedings of Winter Meeting, ICAK, Palm Desert, CA, 1987.
12. Callahan, R., with P. Perry, *Why Do I Eat When I'm Not Hungry?* (New York: Doubleday, 1991).
13. Chancellor, P.M., ed, *Handbook of the Bach Flower Remedies* (London: The C.W. Daniel Co Ltd, 1971).
14. Diamond, J., "Dr. Edward Bach: An appreciation," in *The Medical Discoveries of Edward Bach, Physician*, N. Weeks (New Canaan, CT: Keats Publishing, Inc, 1973).
15. Diamond, J., *BK — Behavioral Kinesiology* (New York: Harper & Row, 1979).
16. Diamond, J., *Life Energy* (New York: Dodd, Mead & Co, 1985).
17. Durlacher, J.V., *Freedom from Fear Forever* (Tempe, AZ: Van Ness Pub Co, 1994).
18. Feingold, B.F., *Why Your Child is Hyperactive* (New York: Random House, 1974).
19. Freedman, C., D. Calsyn, & J. Louks, "The use of the Minnesota Multiphasic Personality Inventory with low back pain patients," *J Clin Psychol*, Vol 32, No 2 (Apr 1976).
20. Gatchel, R.J. et al., "Quantification of lumbar function, Part 6: The use of psychological measures in guiding physical functional restoration," *Spine*, Vol 11, No 1 (Jan/Feb 1986).
21. Goodheart, G.J., Jr., "The schizophrenic pattern," *Chiro Econ*, Vol 13, No 1 (Jul/Aug 1970).
22. Goodheart, G.J., Jr., *Applied Kinesiology 1976 Workshop Procedure Manual*, 12th ed (Detroit: privately published, 1976).
23. Goodheart, G.J., Jr., *Applied Kinesiology 1982 Workshop Procedure Manual*, 18th ed (Detroit: privately published, 1982).
24. Grune, D., & A. Grune, "Dyslexia" (Letters to the Editor), *Sci Am*, Vol 257, No 3 (Sep 1987).
25. Hoffer, A., "Mechanism of action of nicotinic acid and nicotinamide in the treatment of schizophrenia," in *Orthomolecular Psychiatry — Treatment of Schizophrenia*, ed D. Hawkins & L. Pauling (San Francisco: W.H. Freeman & Co, 1973).
26. Hoffer, A., H. Kelm, & H. Osmond, eds, *Clinical and Other Uses of the Hoffer-Osmond Diagnostic Test* (Huntington, NY: Robert E. Krieger Publishing Co, 1975).
27. Huff, B.B., ed, *1987 Physicians' Desk Reference*, 41st ed (Oradell, NJ: Medical Economics Co, Inc, 1987).
28. Kaslof, L.J., "Bach flower remedies: A unique adjunct to chiropractic care," *Am Chiro* (Sep 1985).
29. Kelm, H., "The Hoffer-Osmond diagnostic test," in *Orthomolecular Psychiatry — Treatment of Schizophrenia*, ed D. Hawkins & L. Pauling (San Francisco: W.H. Freeman & Co, 1973).
30. Kelm, H., A. Hoffer, & H. Osmond, *Hoffer-Osmond Diagnostic Test Manual* (Canada: privately published, 1967).
31. Lemeshow, S., "Mental health — a total professional problem," in *Mental Health and Chiropractic*, ed H.S. Schwartz (New York: Sessions Publishers, 1973).
32. Martin, R.J., ed, *Dynamics of Correction of Abnormal Function — Terrence J. Bennett Lectures* (Sierra Madre, CA: privately published, 1977).
33. Meiers, R.L., "Relative hypoglycemia in schizophrenia," in *Orthomolecular Psychiatry — Treatment of Schizophrenia*, ed D. Hawkins & L. Pauling (San Francisco: W.H. Freeman & Co, 1973).
34. Noyes, A.P., *Modern Clinical Psychiatry*, 4th ed (Philadelphia: W.B. Saunders Co, 1948).
35. Peter, L.J., & R. Hull, *The Peter Principle* (New York: William Morrow & Co, Inc, 1969).
36. Pheasant, H.C. et al., "The MMPI as a predictor of outcome in low-back surgery," *Spine*, Vol 4, No 1 (Jan/Feb 1979).
37. Philpott, W.H., & D.K. Kalita, *Brain Allergies — The Psycho-Nutrient Connection* (New Canaan, CT: Keats Publishing, Inc, 1980).
38. Schwartz, H.S., "Physical, psychological and environmental aids," in *Mental Health and Chiropractic*, ed H.S. Schwartz (New York: Sessions Publishers, 1973).
39. Selye, H., *The Stress of Life* (New York: McGraw-Hill Book Co, 1956).
40. Selye, H., *Stress Without Distress* (New York: New American Library, 1974).
41. Selye, H., *Stress in Health and Disease* (Boston: Butterworths, 1976).
42. Siegel, B.S., *Love, Medicine & Miracles* (New York: Harper & Row, 1986).
43. Sternbach, R.A. et al., "Traits of pain patients: The low-back 'loser,'" *Psychosomatics*, Vol 14 (Jul/Aug 1973).
44. Upledger, J.E., "The relationship of craniosacral findings in grade school children with developmental problems," *JAOA*, Vol 77 (Jun 1978).
45. Walther, D.S., "An additional approach in the treatment of schizophrenia." Proceedings of Summer Meeting, ICAK, Detroit, 1980.
46. Walther, D.S., *Applied Kinesiology, Volume II — Head, Neck, and Jaw Pain and Dysfunction — The Stomatognathic System* (Pueblo, CO: Systems DC, 1983).
47. Watkins, R.G. et al., "Comparisons of preoperative and postoperative MMPI data in chronic back patients," *Spine*, Vol 11, No 4 (May 1986).
48. Weeks, N., *The Medical Discoveries of Edward Bach, Physician* (New Canaan, CT: Keats Publishing, Inc, 1979).
49. Whatmore, G.B., & R.M. Ellis, Jr., "Some motor aspects of schizophrenia: An EMG study," *Am J Psychiatry*, Vol 114, No 10 (Apr 1958).
50. Whatmore, G.B., & R.M. Ellis, Jr., "Some neurophysiologic aspects of depressed states," *Arch Gen Psychiatry*, Vol 1 (Jul 1959).
51. Whatmore, G.B., & R.M. Ellis, Jr., "Some neurophysiologic aspects of schizophrenia: An electromyographic study," *Am J Psychiatry*, Vol 120 (Jun 1964).
52. Whatmore, G.B., & D.R. Kohli, *The Physiopathology and Treatment of Functional Disorders* (New York: Grune & Stratton, Inc, 1974).
53. Wheeler, M.J., "The Bach remedies repertory," in *The Bach Flower Remedies*, compiled by Keats Publishing, Inc (New Canaan, CT: Keats Publishing, Inc, 1979).
54. Wiltse, L.L., & P.D. Rocchio, "Preoperative psychological tests as predictors of success in chemonucleolysis in the treatment of the low-back syndrome," *J Bone Joint Surg*, Vol 57A, No 4 (Jun 1975).

Chapter 11

Orthopedic Conditions

Introduction

Applied kinesiology adds a dynamic aspect to orthopedic examination. There is a wide range for its application, from examining individual joints and their support by the muscular system to the integration of different body modules. Some of the basic applied kinesiology techniques, such as PRYT, gait, equilibrium synchronization, ligament interlink, and dural tension, are presented in other chapters. These, as well as different types of muscle testing and treatment — muscle stretch reaction, aerobic/anerobic, and strain/counterstrain — should be integrated with the orthopedic tests and procedures indicated here.

It is important to correlate applied kinesiology tests with standard orthopedic testing. It is assumed that the reader is familiar with orthopedic and neurologic test procedures, which are outside the scope of this text. The optimal examination is a combination of standard orthopedic and neurologic tests, chiropractic tests, and applied kinesiology tests. Applied kinesiology adds an improved understanding of the functional nature of the condition. Sometimes the standard clinical work-up, including x-rays and orthopedic and neurologic testing, indicates a condition that is not correctable with the usual conservative methods, yet it responds quickly with AK techniques. Orthopedic tests indicating an intervertebral disc condition may rapidly become negative following the release of dural tension or hypertonic muscles. Visualization of a severely osteoarthritic knee on x-ray may indicate that the individual is destined to have pain when walking and moving. Often when muscle dysfunction, such as the vastus medialis, sartorius, and other knee-supporting muscles, is corrected, a severely arthritic knee becomes painless.

It is important to make a differential diagnosis because some orthopedic conditions are surgical emergencies, such as the compartment syndrome. Following exercise the pressure in a compartment of the leg or elsewhere can rise to a critical state that produces neurovascular ischemia; paralysis and necrosis may develop. It is conceivable that a physician not familiar with this condition may find weak muscles and be unable to correct them with applied kinesiology techniques. If the patient is sent home to return on another visit, permanent damage may result. If allowed to continue, significant irreversible damage may be done to the muscle within eighteen hours.[97]

Along with consideration of orthopedic, neurologic, and laboratory tests, applied kinesiology examination should include those tests that reveal dysfunction within the triad of health. Orthopedic structural conditions can be secondary to dysfunction on the chemical or mental side of the triad.

Weight-Bearing Tests

Patients are often tested at the doctor's convenience, i.e., in the doctor's office lying on an examination table. One should constantly keep in mind that a patient must be examined in correlation with the way he lives when working, playing, and relaxing. Often pertinent examination findings are missed when the patient is lying supine or prone on an examination table.

Initial clues about how to examine a patient may be uncovered during the examination. If the patient's chief complaint is headaches, ask when the headaches develop. Does he awaken with them in the morning, indicating a possible blood sugar handling problem, or do they develop with the day's activity and subside when he arrives home and sits down to relax? Do backaches develop during the day that are relieved by lying down? There are many factors to consider when a patient's condition develops throughout the day and eases when he arrives home from work. Emotional stress, chemical pollutants, and physical stresses must be considered. If the individual stands and walks all day and obtains relief when he sits or lies down, a weight-bearing problem is indicated. If the patient is examined in a prone or supine position, there may be few or no positive findings; the positions do not parallel the body's status when symptoms develop.

After obtaining general information about body function by testing in the usual prone and supine positions, have the patient sit and test some of the muscles again. If weakness develops, it may be due to spinal or pelvic subluxations that are not present in the reclining position. Further test the individual while he stands. If positive tests now develop, dysfunction in the feet, knees, or hips is indicated — usually in the feet.

Extra care must be taken when testing an individual in the weight-bearing position. It is usually much more difficult to stabilize the patient so that he cannot recruit synergistic muscles to change the test parameters. Good weight-bearing tests can often be obtained by having the patient lean against an upright hi-lo adjusting table. Tests that apply forces parallel to the sagittal plane, such as the hamstrings, rectus femoris, and bilateral neck flexors or extensors, are easily accomplished with little patient shifting. Tests that apply forces somewhat parallel to the transverse plane, such as the pectoralis major (clavicular division), psoas, and piriformis, require great attention to stabilization of the body against the hi-lo table.

Many health problems that relate to structural

11—1. Proper stabilization is of primary importance in weight-bearing tests.

imbalance are present only when the patient is in the weight-bearing position. Applied kinesiology provides one of the few methods for discovering these problems and reveals how integrated body structure is.

The orthopedic discussion begins with the foot, which well illustrates the body's integration. Many problems discussed later, such as knee pain and shoulder problems, may develop as a result of foot dysfunction. Almost any health problem can relate directly or indirectly to the feet. In an applied kinesiology practice it is not uncommon to eliminate an individual's headaches by correcting foot dysfunction.

Foot Reflexes and Reactions

In Chapter 5, "Neurologic Disorganization," there is discussion about how the foot proprioceptors send afferent information for muscle organization throughout the body. The example is given of having a patient stand on pencils placed under the 1st and 5th metatarsals to create artificial subluxations of the metatarsal arch. When this is done, neurologic disorganization develops in almost everyone, creating a lack of predictability of remote muscle function.

Knowledge of predictable muscle function resulting from stimulating the foot proprioceptors helps understand how subluxations and muscle dysfunction of the feet affect remote areas of the body. The two primary areas discussed in the literature are the positive support reaction[64,94,129] and the "magnet reaction."[64,69]

Positive Support Reaction

The positive support reaction was first observed in decerebrate animals. When pressure was applied to the plantar surface of the foot, the limb extended strongly enough to support the animal's body weight, leaving it standing in a rigid position.[94] The reaction was originally called the "magnet reaction" or "extensor thrust reaction." It is now often referred to as the positive support reaction, which is the term we will use here. The term "magnet reaction" will be applied more specifically to the foot reflexes later. Ruch[129] acknowledges that the reactions are present in normal animals and man, but they are more readily demonstrated in the decerebrate preparation.

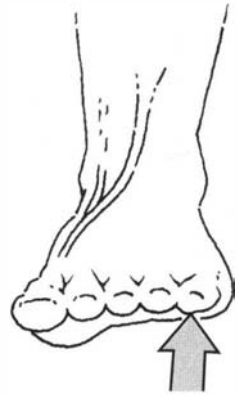
The positive support reaction comes from stimulation of muscle, joint, and cutaneous receptors in the foot. These combine to form a complex union of stimuli to provide facilitation and inhibition of muscles contributing to posture and gait. Weight bearing in the foot stretches the muscles and ligaments, stimulating the joint and muscle mechanoreceptors to provide facilitation of the postural extensor muscles. If the phalanges and metatarsals are squeezed together or flexed instead of being spread apart or extended, the extensor muscles are inhibited and all joints of the limb flex.[64] If there is failure of normal muscle and/or joint function during weight bearing, facilitation of the extensor muscles necessary for proper posture fails.

O'Connell[122] describes an experiment done in many physiology laboratories to demonstrate the positive support reaction. She used a swing arranged so that it could be raised and lowered various distances from the floor. The subject sitting in the swing was raised and lowered, and the swing's seat was randomly tilted to dump the subject toward the floor. The individual landing on his feet maintained an upright posture. After repetitions of the procedure, the individual was blindfolded and the procedure repeated with enough swinging and random elevation and lowering of the swing to disorient the subject. Again random spilling dumped the individual toward the floor. Correct posture was maintained when he landed on his feet, but with greater difficulty and slower recovery than when not blindfolded. Finally, the individual's feet were immersed in ice water for twenty minutes to produce local anesthesia, and the procedure was repeated. In this instance the individual was unable to maintain an erect posture and crumpled to the mat. The conclusion, of course, is that chilling of the feet interfered with proprioceptive communication to the body; consequently, there

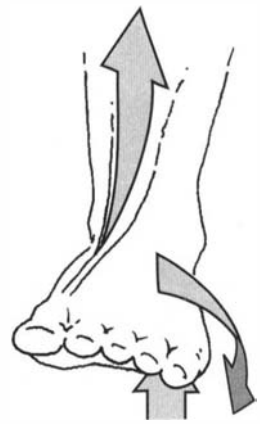
was no facilitation of the extensor muscles.

The positive support reaction should be tested in all patients with excessive AP spinal curves, which indicate poor function of the postural extensor muscles. The positive support reaction can easily be tested for normal function with manual muscle testing. With the patient prone, stretch the foot to spread the metatarsal and longitudinal arches. This simulates the stretching of the foot when weight bearing. Normal function is for the extensor muscles, such as the hamstrings, gluteus maximus, and neck extensors, to test equally strong or stronger. When there is a dysfunction of the positive support reaction, they will test weak. Failure of the positive support reaction is due to some type of foot dysfunction. It can be foot subluxations or dysfunction of the muscle proprioceptors, usually neuromuscular spindle cells. Foot correction as discussed here will usually correct the condition.

Sometimes muscle weakening will be observed when the foot is squeezed together. There is a normal action of facilitation of the limb flexors when the phalanges and metatarsals are squeezed together.[64] With the facilitation of the flexors, there is normally reciprocal inhibition of the extensors. When there are subluxations in the foot or dysfunction of the positive support reaction muscle proprioceptors, there is usually a neurologic disorganization that causes unpredictable muscle test results. The spreading challenge simulating weight bearing is the best method for testing the positive support reaction.



11—2. Stimulus to the lateral foot causes . . .

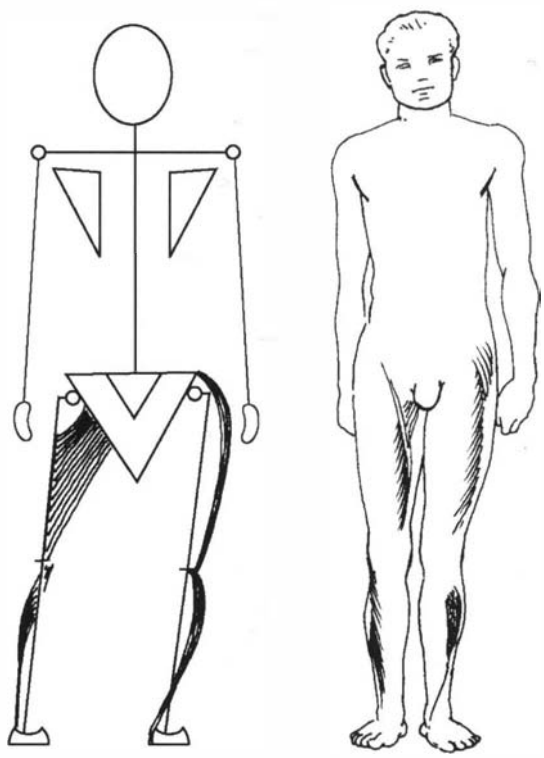


11—3. contraction of the foot inverting muscle.

Magnet Reaction

The magnet reaction (also called the "placing reaction") has no relation to electromagnetic activity. The reaction was so named because when the physiologist pressed on the decerebrate animal's foot, it followed the experimenter's withdrawing hand as if attached like a magnet. If pressure is applied to one side of the foot, the foot moves toward the source of stimulation in an effort to return balance to the foot position.[69] The ability to maintain balance is enhanced by these reactions, particularly in the biped. A simplified example is as follows. When one sways to the left, there is adduction of the left femur and abduction of the right one, with eversion of the left foot and inversion of the right one. With the sway there is stimulation to the muscle, joint, and cutaneous receptors. These impulses are interpreted to cause facilitation of the foot inverter muscles on the left, such as the tibialis anterior. At the right foot and ankle, there is facilitation of the peroneus group to cause eversion. At the pelvic and thigh levels there is facilitation of the left gluteus medius and tensor fascia lata, causing left hip abduction. There is also facilitation of the right adductors to cause hip adduction. This muscular action brings the body back to neutral from its lateral sway.[64]

In the early days of applied kinesiology, Edward Doss, Sr., D.C., of Stuttgart, Arkansas, reported to Goodheart his frequent observation that a lateral cuboid subluxation correlated with a weak tensor fascia lata muscle. Correction of the subluxation returned normal function to the muscle, as observed by manual muscle testing. Goodheart concurred with the observation, and also found that an adjustment of the medial longitudinal arch (medial cuboid) often corrected adductor muscles that tested weak. This logically fits very well with the available information about the magnet and the placing reactions. Lateral sway of the body that causes inversion



11—4. Muscle contraction to return body balance from lateral sway.

and eversion of the foot to regain balance would stimulate the cutaneous and joint receptors. A medial or lateral subluxation of the transverse tarsals would be equivalent to body sway as far as the joint receptors of the cuboid would be able to discern; consequently, impulses would be transferred to facilitate and inhibit muscles to return body balance.

The bones of the mid-tarsal arch can be challenged to determine if they are subluxated and causing the tensor fascia lata or adductor muscles to be weak. In the presence of weak muscles, challenge the bone(s) with various vectors and re-test the weak muscle(s) for strengthening. When a subluxation is challenged with the proper vector for correction, a weak associated muscle will test strong. Usually there will also be a weakening of a previously strong indicator muscle when the articulation is challenged in the opposite direction. The adjustive thrust is made in the direction that produced a strengthening of the weak muscle. Generally, it is a lateral-to-medial thrust on the cuboid for the tensor fascia lata, and a medial-to-lateral thrust on the navicular affecting the mid-tarsal bones. The latter is often called a medial cuboid.

After effectively correcting the subluxation to return normal function to the tensor fascia lata or adductor muscles, have the patient walk. Usually if the correction will not hold, it will be immediately lost with this action. It is best to have the patient walk in a figure-eight pattern. This includes both right and left turning, further stressing the foot. If the patient loses the correction, there is probably extended foot pronation or some other foot problem.

Foot Pronation

The general use of the term "foot pronation" connotes abnormal position or function of the foot. Actually, foot pronation during function is a necessary and important aspect of the gait cycle.

Slocum and James[138] describe the foot's joint motion and its importance in the gait cycle. At heel strike, the subtalar joint is supinated and the tibia is in external rotation. As the foot comes into full contact with the substrate rapid pronation begins, with the subtalar joint moving into a neutral position and the tibia coming out of external rotation. Pronation acts as a shock absorber mechanism for the forces being applied to the foot. During this stage, the tibialis anterior and posterior muscles are active in supporting the medial longitudinal arch. The joint configurations and flexibility allow the foot to adapt to the underlying surface. The ligaments, joint constraints, and muscles combine to dissipate stress to the foot and body, similar to a flexible band. The tibia follows the medial rotation of the talus as pronation continues. At 15-25% of the stance phase, the foot should begin to come out of pronation. At this time it starts to become a rigid lever for the toe-off propulsive stage of gait. "The foot that is not reconverted into a rigid lever by 75% weight bearing is defined as having increased pronation."[99] Normally 75% weight bearing is at approximately 25% of the stance phase.[126]

As mentioned, pronation is not bad; extended pronation or failure to resupinate is bad. The medial longitudinal arch is important as a shock absorber. During the stance phase of gait, it allows pronation from heel strike to flatfoot. If the foot does not pronate, difficulties may arise in the midfoot, hindfoot, tibia, and knee.[100]

Care must be taken not to overcontrol pronation with orthotics. Pronation in the amount of 4° provides the shock absorption necessary and accommodates the internal rotation of the leg. It also enables accommodation to changes in the angle of the substrate. Overcontrol of pronation is not unusual, especially when solid orthotics are prescribed. Artificially reducing the normal pronation decreases the foot's ability to act as a shock absorber and adapt to the changing substrate surfaces. Failure of normal pronation results in structural stress.[142] Overcorrection of pronation is often the cause of remote problems, as observed in applied kinesiology examination.

Etiology of Foot Dysfunction

Many factors come into play for normal foot function. Authors have individually stressed improper bony architecture, ligament integrity, and muscle and fascia function as causes of prolonged foot pronation. All these factors come into play, either singly or, usually, in combination. If the initial cause is only one factor, the other two will probably be secondarily involved if the condition is allowed to continue.

The importance of proper bony architecture in maintenance of the arch is emphasized by the fact that a completely paralyzed foot, if once normal, maintains its arch even when maximum weight is placed upon it.[72] It is obvious that the ligaments play an important role, for if they should fail to hold the bones together the arches would surely fall in a completely paralyzed foot.

Muscles play a more dynamic role in maintenance of the arch. They protect ligament ability to hold the bones together, as observed by Hamilton,[71] who notes that when the tibialis posterior tendon ruptures, a true fallen arch occurs with great rapidity and severity. It is important to note that the ligaments and muscles have separate roles in the maintenance of the arch. The paralyzed foot that is not required to constantly bear weight, as in walking and running, maintains its arches due to the integrity of the ligaments. If, however, there is a loss of muscle support when there is repeated weight bearing,

the arches are lost as the ligaments break down. When this happens, the muscles are unable to maintain the arches.

Neither the extrinsic or intrinsic muscles of the arch are necessary to maintain its normal position in a static stance, as indicated by electromyographic studies.[12,14,139] In contrast to a normal foot, the one with extended pronation has muscle activity, as observed by electromyography, in the static stance and excessive activity when walking.[66,68,103] The preponderance of data indicates that muscles are not important in static stance, supporting Basmajian's thought, "Muscles [are] spared when ligaments suffice." Even though muscles are not important in supporting the normal foot in static stance, they are important in maintaining the foot's integrity during walking and running. When muscles fail to support the dynamic arch the ligaments can be stretched, and the medial longitudinal arch of the foot is lost.[70] In addition, when excessive pronation is present from whatever cause, the muscles are abnormally active in an attempt to control the problem.

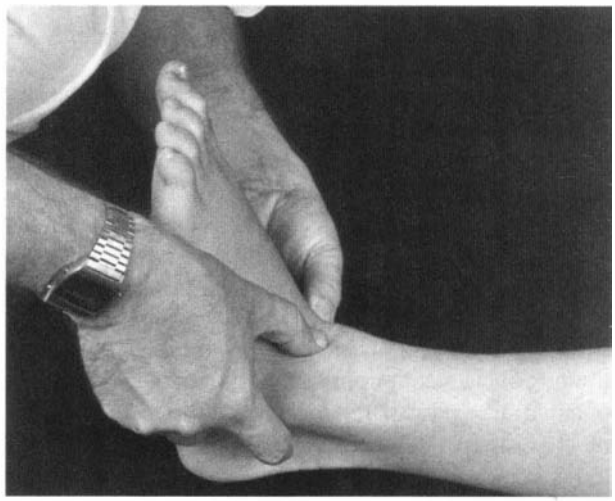
A major cause of extended pronation is a short triceps surae, often referred to as a short Achilles tendon. The triceps surae is composed of the gastrocnemius, the soleus, and their shared tendon. When the triceps surae is short, compensation must take place elsewhere during the middle of the mid-stance phase of gait.[141] As body weight is forced into the midfoot, compensation takes place by pronation of the subtalar and mid-tarsal articulations in order to compensate for the lack of ankle dorsiflexion. These joints must pronate more than normal, causing stretch to ligaments with the development of flexible flatfoot. The breakdown of the midfoot accommodates the short triceps surae. The triceps surae should allow 10° dorsiflexion at the ankle. When evaluating the amount of dorsiflexion, one must limit motion to ankle joint movement only. The examiner should grasp the calcaneus and midfoot to stabilize the calcaneus, navicular, and cuboid bones against the talus before moving the foot into dorsiflexion. It is with this action that there should be a minimum of 10° dorsiflexion. Remember that in extended pronation there is increased laxity of the subtalar and mid-tarsal articulations. If the entire foot is brought into dorsiflexion by applying pressure at the metatarsals, it will appear that there is much greater dorsiflexion than is being allowed at the ankle mortise. When limiting motion to the ankle, one is often amazed at how little there is. The foot may fail to reach a right angle with the tibia by as much as 25°. In normal feet and ankles, dorsiflexion may reach 20° beyond the right angle.

When the triceps surae is short, a surgical procedure to lengthen the Achilles tendon is sometimes done. Ryerson[130] points out, "The tendo-Achilles is not short. The muscle bellies are short." He recommends lengthening the muscle. In applied kinesiology there are many techniques to do this, including fascial release and intermittent cold with stretch techniques, and treatment to the muscle proprioceptors.

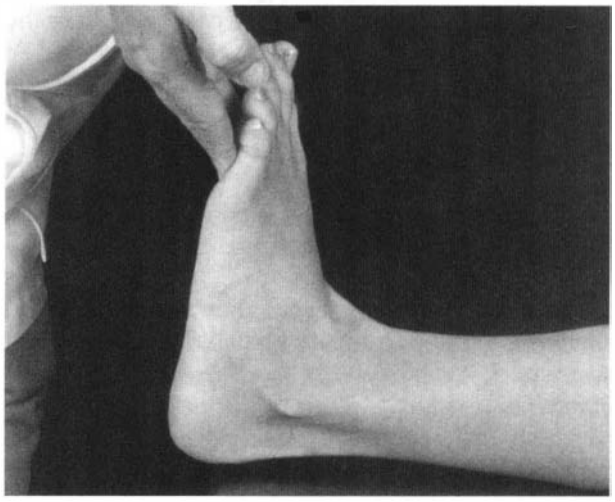
Examination

An excellent screening examination for extended foot pronation, subluxations, and other dysfunctions of the foot is the shock absorber test. When applying it to the foot, be certain to strike the foot with many vectors because of the numerous articulations that must be evaluated. Quite often when there is foot dysfunction, a previously strong indicator muscle will test weak. The psoas is a good muscle to evaluate because it appears to reflect foot dysfunction more often than other muscles.

The foot with extended pronation will usually cause remote muscles of the body to test weak when the patient is standing after walking, and it will also usually have



11—5. Limit ankle dorsiflexion by stabilizing the calcaneus, navicular, and cuboid on the talus.



11—6. Breakdown of the midfoot in extended pronation makes it appear that there is more movement at the ankle mortise than there is.

a positive challenge for the positive support reaction. When muscles weaken when the patient is standing, have him externally rotate his legs, putting weight into the lateral longitudinal arches. In most cases of static pronation, the muscles will become strong.

Generally one can observe static pronation as the patient stands. Helbing's sign[143] is a medial bowing of the Achilles tendon, i.e., the convexity of the curve faces medially. This is most easily observed with an imaginary line down the center of the leg to the center of the Achilles tendon, continuing into the center of the calcaneus. When Helbing's sign is positive, there will be a break in the line as it deviates laterally into the calcaneus. Confirming Helbing's sign is an appearance of the calcaneus in eversion.

11—7. Helbing's sign.

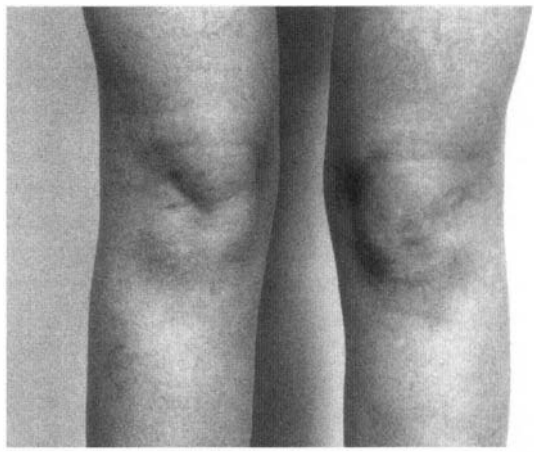
There is generally depression of the medial longitudinal arch with pronation; however, this can lead one astray because individuals with a very high arch can sometimes have extended pronation. If so, there will be tension on the plantar fascia in the weight-bearing position. In the case of the high arch, the examiner can put his fingers under the medial longitudinal arch and palpate for tension. Have the patient externally rotate the leg to put weight on the lateral longitudinal arch. If there is considerable relaxation of the plantar fascia in this position, pronation is probably extended.

Palpation of the ligaments of the joints making up the medial longitudinal arch can help determine chronicity and extent of the arch breakdown. Generally there will be exquisite tenderness with digital pressure on the ligaments. If not, the ligaments have been stretched to the point that they are no longer being strained during gait.

Shoe wear will be excessive on the posterolateral aspect of the heel. Often there will be a breakdown of the shoe's counter with it overlapping the heel.

11—8. Typical shoe wear with extended pronation.

With static pronation there will be internal leg rotation as the tibia and fibula follow the talus. The rotation is most easily observed at the knee, with the patella in internal rotation.

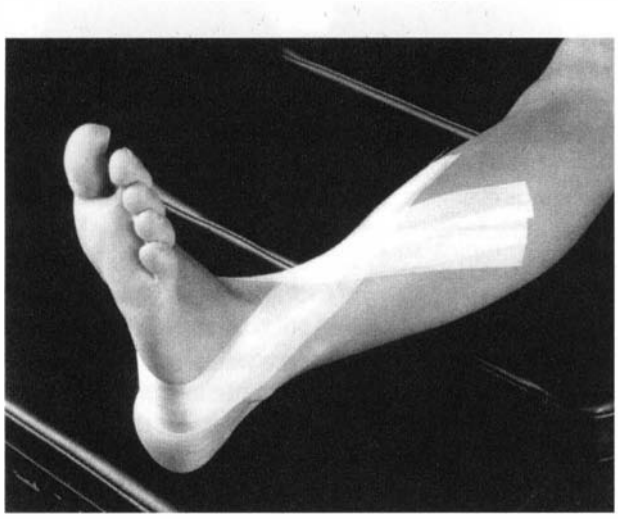


11—9. Medial knee rotation with extended pronation.

Sometimes individuals weaken with walking due to problems other than foot pronation. A figure-eight tape support to the longitudinal arches can help differentiate weakening from pronation and other causes. Taping is sometimes of value to help explain to a patient how foot pronation can cause remote problems. There may be a shoulder condition related with foot dysfunction. After walking numerous muscles in the shoulder may be weak; after applying tape support to the arches the patient can walk with no shoulder dysfunction.

To apply the figure-eight arch support, start with 1" tape over the distal third of the medial leg, cross in front of the ankle to the lateral midfoot and pass under the cuboid to the navicular, then cross in front of the ankle to anchor on the lateral distal third of the leg. Taping in this direction — from cuboid to navicular — provides better support to the medial longitudinal arch than in the other direction. Apply a second strapping somewhat over-

lapping the first. Sometimes the tape is valuable as a therapeutic trial to determine if foot pronation is contributing to the patient's symptoms, or if orthotics will help hold a failing correction.



11—10. Figure-eight tape support.

Treatment

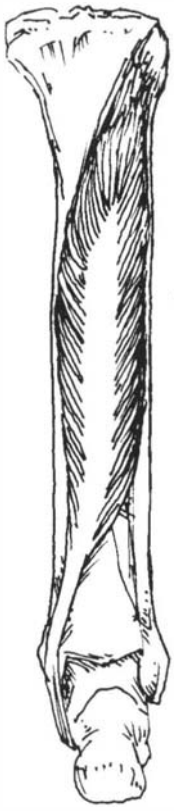
Treatment must be directed to three specific areas: muscle function, subluxations, and support. Muscles can be tested by direct muscle testing and by therapy localizing to determine treatment needed to the origin and insertion and muscle proprioceptors. Muscle dysfunction is treated with the appropriate applied kinesiology techniques. Any of the five factors of the IVF may be applicable.

Of particular interest is the tibialis posterior muscle. The design of the ankle mortise is such that during plantar flexion the narrower portion of the talus moves between the tibial and fibular articular surfaces. To accommodate this, the tibia and fibula are pulled closer together by the pinnate fibers of the tibialis posterior, which contracts to aid in plantar flexion. This maintains the integrity of the ankle mortise during gait plantar flexion.

If there is less than 10° dorsiflexion at the ankle mortise, the triceps surae must be lengthened. First obtain as much increase as possible in range of motion by applied kinesiology techniques directed to the muscles and remote neurologic factors. On a local basis application of intermittent cold with stretch, fascial release, and treatment to the muscle and cutaneous receptors will often increase the muscle's length. There are several techniques in applied kinesiology — such as PRYT, equilibrium synchronization, and relieving dural tension — that increase range of motion throughout the body.

It may be necessary to apply stretching procedures to the triceps surae; however, the major problem with this is that the foot must be used as a lever to apply the stretch. This tends to compound the problem of stretching the ligaments of the midfoot and breaking it down further.

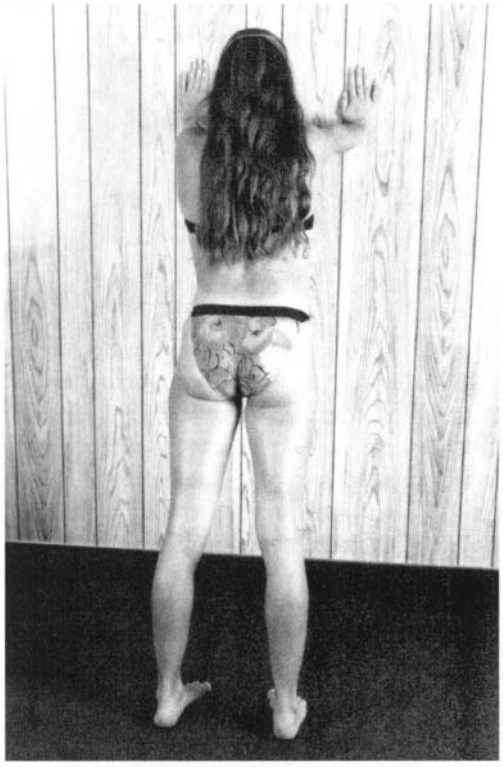
11—11. Superior view of transverse section of distal tibia, fibula, and talus.



11—12. Pinnate fibers of the tibialis posterior pull the tibia and fibula together to accommodate the articular surfaces of the talus during plantar flexion.

The muscles can best be stretched by a contraction, relaxation, and stretch technique. It has been shown to be more effective than a ballistic stretch.[149] In a ballistic stretch the muscle is rapidly stretched, activating the interfusal muscle spindles to reflexly cause a protective muscle contraction. The stretching procedure is done with the patient facing a wall with his feet approximately three feet from it, with the legs in internal rotation so the feet are in a "pigeon-toed" position. This helps lock the midfoot to reduce stretch on its ligaments. The hands are placed on the wall and the patient leans toward it, stretching the gastrocnemius and soleus almost to their limit. In this position an isometric contraction of the triceps surae is held for seven seconds. The muscles are then relaxed for two to five seconds, followed by moving the hips forward and flexing the arms to place maximum stretch on the gastrocnemius and soleus for seven to eight seconds. This is repeated five times on a daily basis. After thirty days of stretching, one can expect approximately 6° increase in dorsiflexion. When the desired range of motion is obtained, it can be maintained by a stretch series one time per week.[149]

Subluxations. The feet and ankles are one of the most common areas of the body for extraspinal subluxations. The challenge method of applied kinesiology determines exactly how correction should be made. With that information, plus an excellent knowledge of foot



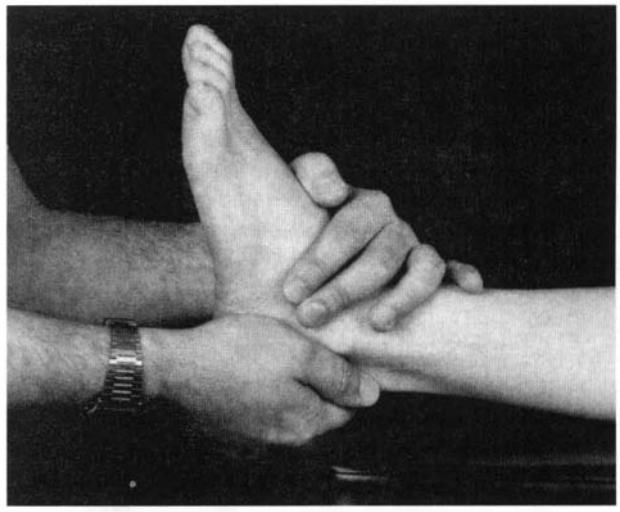
11—13. Triceps surae muscle stretch.

anatomy, manipulation should be relatively easy. Standard techniques for adjusting the feet can often be coupled with the challenge information to make the correction. It is sometimes necessary to develop a specific technique for the individual being adjusted to conform with the information challenge presents. This can only be accomplished when the physician has an excellent knowledge of foot anatomy.

The most common factor causing corrective effort to fail is not obtaining proper relaxation in the foot prior to the manipulative thrust. Only a small amount of force is required for effective correction when proper relaxation has been obtained. Most chiropractors can relate to obtaining proper relaxation when adjusting in the cervical spine. It becomes almost intuitive to know when to apply the corrective thrust. Sometimes the doctor can feel the patient signal the proper time by a sensation of "letting go" as relaxation is obtained. At other times it is necessary to distract the patient from the cervical area by having him put his hands on his abdomen or otherwise drawing attention away from the cervical spine. A similar technique should be used when adjusting the foot to obtain optimal relaxation for making the correction.

Bones of the foot should be thoroughly evaluated for subluxations with challenge and therapy localization. The most efficient method is challenge, since it gives evidence of the subluxation and also indicates the direction of optimal correction.

The talus is nearly always subluxated in the ankle mortise when there is extended pronation. The direction of correction is usually lateral to medial, with a slight cephalad vector. Challenge the talus to determine which vector of force causes a previously weak associated muscle to strengthen. The muscles most often involved are the peroneal group, tibialis anterior and posterior, psoas, or neck flexors. The description given here is when a talus challenge strengthens a weak associated muscle when done from lateral to medial. If there is a positive medial to lateral challenge, correction is the reverse of that described here. With the patient supine, the physician cradles the calcaneus in his right fingers and palm. The thenar eminence wraps tightly up under the lateral malleolus to contact the talus. It is emphasized that the contact must be as tight under the lateral malleolus as possible because of the limited lateral exposure of the talus. The left hand contacts the dorsum of the foot, with the little finger contacting the talus just distal to the tibia. Again, this contact should be as tight against the tibia as possible to contact the limited exposure of the talus and not be distal to Choppart's joint. With proper patient relaxation, the manipulation is performed in two quick maneuvers. First, both hands perform traction on the foot to open the ankle mortise. At the end of the traction thrust, the physician's right hand moves the talus medially and slightly superiorly in the direction of optimal vector challenge. This results in slight eversion of the foot. There will usually be an audible release, but it is not necessary to obtain correction.

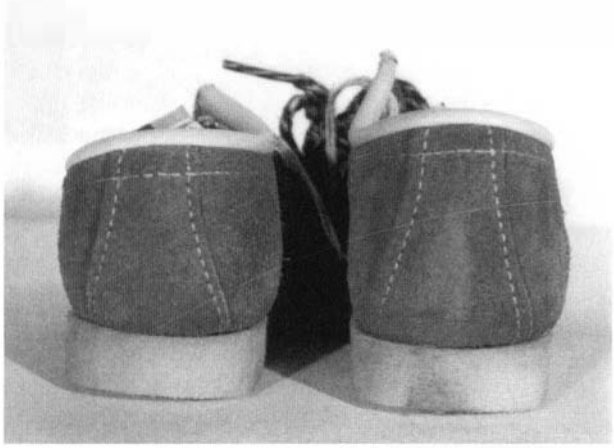


11—14. Starting position for talus adjustment.

Support. There are two types of support to consider regarding the foot: shoes and possibly orthotics. The first should always be considered; the second depends on how broken down the foot is and how successfully correction is obtained.

Many shoes — especially women's — seem to be designed to create foot problems. A shoe should be sturdy, low-heeled, and preferably laced. Two areas to routinely

evaluate in shoes are the strength of the counter and the shank. The counter must solidly support the medial and lateral aspects of the calcaneus. It should also be built squarely over the heel. Viewing even a new shoe from the back, one often sees the counter leaning medially or laterally, which may contribute to creating or perpetuating foot problems.



11—15. An imbalanced pair of new shoes which have not yet been worn.

The shank of the shoe can easily be tested by pressing down on the instep; it should not yield to the pressure. An alternative to a strong steel shank is a wedge-type shoe giving full-length support to the foot.

A common problem is shoes that are too narrow in the forefoot, which cramp the metatarsals. Often during foot correction the metatarsals regain normal motion and the foot widens, creating the need for a larger shoe. If the individual maintains the same shoe size when a larger one is needed, the therapeutic effort yields poor results.

If a foot is badly broken down from long-term extended pronation, it may be necessary to fit the patient with orthotics. When properly made, they support the foot and help maintain the correction.

All orthotics are not equal. From evaluating foot function and its effect on remote areas of the body, my experience has indicated that a flexible orthotic helps best

11—16. A weak shank such as this contributes to foot problems.

in maintaining the correction; more important, it usually does not create new problems. Solid orthotics tend to cause remote dysfunction in many areas of the body, as indicated by applied kinesiology testing and symptomatic patterns. Spinal Pelvic Stabilizers® made by Foot Levelers, Inc.,[50] have consistently provided good support for the foot and do not force it into positions that create remote problems. There are many models available for men and women to fit the person's life-style and activities.

Quite often when poor results are obtained with the Spinal Pelvic Stabilizers®, it is because of the poor quality or improper fit of the patient's shoes.

11—17. Support made by Foot Levelers, Inc.

Tarsal Tunnel

The most common peripheral nerve entrapment on branches of the sciatic nerve is on the tibial nerve at the tarsal tunnel. The tibial nerve is the larger terminal division of the sciatic nerve. Its source is the 4th and 5th lumbar and 1st, 2nd, and 3rd sacral nerves. As it descends the leg it is well-protected by muscle. It descends through the popliteal fossa and continues in the leg with the posterior tibial vessels to enter the tarsal tunnel. Usually within the tunnel the nerve splits into the medial plantar, lateral plantar, and calcaneal nerves. Prior to the tarsal tunnel there is little or no incidence of entrapment of this nerve because of the excellent protection the muscles provide it.

When there is entrapment of the tibial nerve in the leg, its source is usually a deep posterior compartment syndrome.[151] Compartment syndromes are associated with muscle activity in which the contents of the compartment enlarge past the closed compartment's ability to yield. An acute compartment syndrome can be a sur-

gical emergency; it must be differentially diagnosed from other conditions.

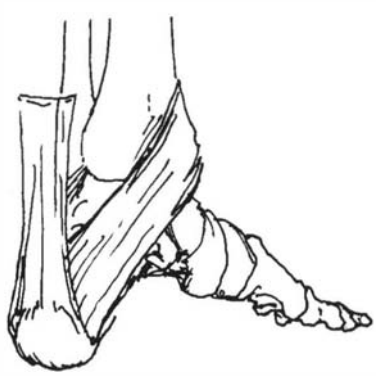
Lam[92] compares the early knowledge of the carpal and tarsal tunnel syndromes. He points out that the first description in the literature of the tarsal tunnel syndrome was in 1962, when two operative cases were reported. He goes on to state that in the early investigation of the carpal tunnel syndrome, advanced cases showed marked motor and sensory disturbance; at operation, pathological changes of the median nerve were evident. He states in his 1967 paper, "Nowadays the carpal tunnel syndrome is sufficiently well-recognized to ensure that most cases are treated before this stage is reached. The same pattern of events may evolve in the case of the tarsal tunnel syndrome." Unfortunately, there are still many people with unrecognized entrapment at the tarsal tunnel. Many physicians overlook the tarsal tunnel syndrome, and certainly in applied kinesiology experience many functional tarsal tunnel syndromes go untreated. This allows the plantar intrinsic muscles to become weak and causes increased development of extended pronation that affects total body function.

The term "functional tarsal tunnel syndrome" is used above to differentiate the condition from frank pathological entrapment of the tibial nerve, which may require surgical decompression. The functional tarsal tunnel syndrome highlighted in this discussion is neurapraxia nerve entrapment that is the minor type but still causes dysfunction. There is no wallerian degeneration, and recovery is rapid when the pressure is relieved. A classic experiment by Granit et al.[65] reveals how minimum pressure on a nerve changes its function. They demonstrated the creation of fiber interaction within a nerve from compression. The artificial synapse was produced by a pressure so gentle that it did not impair conduction of the original impulse. An important aspect of this investigation relates with therapeutic efforts; the nerve returned to normal after being decompressed and irrigated with a saline solution. Since Goodheart's[59] introduction of tarsal tunnel treatment into applied kinesiology, many have been successfully treating the syndrome. With an increased awareness of this type of tarsal tunnel syndrome we may yet, as Lam states, treat these conditions before development of marked motor and sensory disturbance with pathologic change.

Anatomy

The tarsal tunnel is located behind and inferior to the medial malleolus. The bones make up the floor, and it is covered by the laciniate ligament (flexor retinaculum). In this osteofibrous passageway, the tibial nerve passes with the tendons of the tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles. The other components of the neurovascular bundle are the posterior tibial artery and vein.[55]

The flexor retinaculum has several deep fibrous septa that blend with the periosteum, covering the medial side of the calcaneus. The neurovascular bundle in the tarsal tunnel is often attached to some of these septa, rendering itself more liable to minor degrees of traction with foot movement.[92]



11—18.

11—19.

11—20.

Vascular supply to the nerve may have a bearing on its susceptibility to compression. The median nerve at the wrist and the posterior tibial nerves have better arterial supply than the ulnar and lateral popliteal nerves. The ulnar and lateral popliteal nerves rarely have "spontaneous" compression symptoms, though they run through osteofibrous tunnels. The median and posterior tibial nerves have a much more common incidence of "spontaneous" compression. Their ample arterial blood supply may make them more susceptible to the effects of localized vascular insufficiency.[92] It is suggested by Fullerton[51] and Thomas[144] that sensory symptoms in nerve compression syndromes are due to arterial insufficiency. More slowly occurring motor paralysis is thought to be due to later structural changes produced within the nerve, and the paralysis is less likely to benefit from decompression. Lam states, "It is important therefore to make the diagnosis and treat the patient before the onset of demonstrable motor involvement."

In Lam's series of ten cases surgically treated,[92] one case had enlarged tortuous veins within the tarsal tunnel. There was no other demonstrable pathology in any of the cases. This points out the necessity in an applied kinesiology examination of determining whether actual paralysis develops in the muscle from nerve impingement,

or whether there is functional weakness as observed in the manual muscle test. This is readily determined by challenge and therapy localization. If one is unable to return muscle function to normal as observed by the manual muscle test, surgical intervention may be necessary. Denny-Brown and Brenner[40] have shown that when peripheral nerve entrapment is treated in its early stages, it is reversible.

Extended pronation is a factor in nearly all cases of tarsal tunnel syndrome treated by applied kinesiologists. Because of the posterior movement of the calcaneus on the talus during extended pronation, the flexor retinaculum is stretched. There is intermittent compression, then release, then compression, then release of the laciniate ligament on the posterior tibial nerve.[23]

Body Language

Often tarsal tunnel nerve entrapment is discovered by an astute examiner when the patient has no complaint of foot or ankle dysfunction. As discussed earlier, foot dysfunction plays a major role in total body organization. Recognizing asymptomatic tarsal tunnel entrapment is just as important as examining and finding the cause of the painful foot.

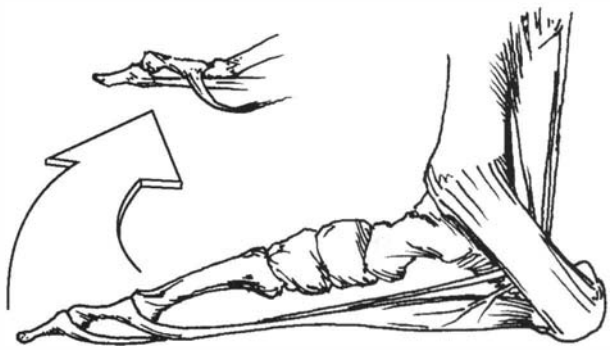
Patients' complaints of tarsal tunnel syndrome include burning pain and paresthesia in the toes and along the sole of the foot. Symptoms may be worse at night. Moving the limb, getting out of bed, or hanging the limb over the edge of the bed may provide relief.[92]

Pain may radiate up the leg from a tarsal tunnel syndrome. It may simulate a disc problem, peripheral vascular disease, or neuritis. Sometimes the pain is attributed to an existing condition, such as diabetes or peripheral vascular disease, when the problem is really a tarsal tunnel syndrome. This error frequently occurs with older patients.

Many aspects of an examination may appear normal when there is entrapment at the tunnel. There may be normal dorsalis pedis and posterior tibial pulses. Skin color, hair distribution, and capillary circulation may also appear normal.[86]

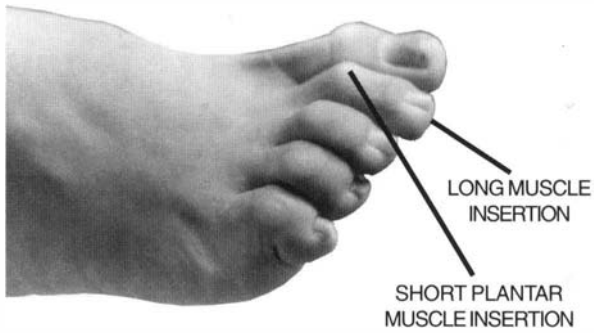
With chronicity the plantar muscles of the foot may atrophy, giving an appearance of a high arch. There is indication in the literature that sensory deficit develops prior to motor deficit; this does not agree with applied kinesiology findings. One often finds severe plantar muscle atrophy with no sensory deficit or hyperesthesia. The patient with tarsal tunnel entrapment will have weak plantar muscles, and the long toe flexors will probably be strong. This, of course, is because the long muscles receive their nerve supply prior to the tarsal tunnel, and the intrinsic muscles after the point of entrapment. With this muscle imbalance, the patient will have hyperextension at the metatarsophalangeal articulations and hyperflexion at the interphalangeal articulations. This is because the long muscles insert into the distal phalanx, and the intrinsic muscles into the intermediate phalanx.

11—21. Intrinsic plantar muscles.



11—22. Insertion of muscles to the toes.

There is a claw-like appearance to the toes, which is often called claw toes. When the individual stands or walks, one can see the distal phalanx gripping the substrate with elevation of the proximal phalanges. There will often be calluses on the distal ends of the toes.



11—23. "Claw toes" typically present in a tarsal tunnel syndrome.

Etiology

Often there is no known etiology for functional tarsal tunnel syndrome. Even in a surgical series of sixteen cases reviewed by Edwards et al.,[42] eight of the cases were "spontaneous" entrapment; that is, no space-occupying lesion was found. Five were post-traumatic fibrosis due to fracture, three were attributed to structural anomalies of an accessory or hypertrophied abductor hallucis muscle, and one was due to tenosynovitis.

Tenosynovitis within the tarsal tunnel can develop in runners[90] or from trauma from other causes.[89] This, in effect, causes a space-occupying lesion to produce pressure on the nerve. There will be crepitation at the tunnel and severe pain on digital pressure over the tendons.

Venous engorgement within the tunnel can develop as a result of proximal venous obstruction or valvular deficiency.[55] Some of the pain present with an obvious venous stasis or thrombophlebitis may be caused by encroachment on the posterior tibial nerve rather than pressure from the vein distension alone.[89]

When the tarsal tunnel syndrome is symptomatic, it must be differentially diagnosed from interdigital neuritis, dropped metatarsal heads, plantar calluses, arch strain, various types of arthritis, tenosynovitis, peripheral neuritis, peripheral vascular disease, and various other causes of sciatic pain.[2,55,89,92] Antidromic impulses can cause tenderness along the entire sciatic nerve. The pain may simulate root pain of spinal origin. Moloney[111] reports on a case where three spinal surgeries were done without permanent improvement of pain until the tarsal tunnel was diagnosed and operated, which eliminated the painful condition.

Examination

When there is frank entrapment of the tibial nerve in the tarsal tunnel, the Tinel sign is often present over the tunnel or the medial arch. Palpation over the retinaculum may reveal tenderness or a small fusiform swelling of the nerve. Hypo- or hyperesthesia may develop in the posterior tibial nerve branches (medial and lateral plantar nerves), or the calcaneal branches. Sensory disturbance should only be in this distribution, and it should not affect the dorsum of the foot except over the distal phalanges of the toes. There may also be loss of two-point discrimination. Pressure applied on the calf of the leg by inflating a sphygmomanometer cuff may reproduce symptoms on the affected side more quickly than in a normal foot.[24,92,98]

Maneuvering the heel into a valgus position narrows the tarsal tunnel and may increase pain; on the other hand, maneuvering the heel into a varus position may reduce pain.[89]

There is usually no complaint of muscle weakness in a tarsal tunnel syndrome[2]; consequently, muscle deficiency must be carefully examined for by inspection, palpation, and muscle testing. The abductor hallucis, located along the medial longitudinal arch, is inspected and palpated for atrophy along with the other intrinsic muscles of the foot. The ability to flex the toes at the metatarsophalangeal articulation is evaluated by muscle testing. There is no weakness on extension of the toes.

The flexor digitorum longus and brevis and the flexor hallucis longus and brevis should be evaluated. The long muscles will have improved function over the short ones. Challenge, usually directed to the calcaneus, may improve the function of the intrinsic muscles. There will be positive therapy localization over the tarsal tunnel at the area of entrapment, and probably at the subtalar articulation.

Electrodiagnosis should be done to determine nerve conduction in cases that are unresponsive to conservative care; however, there may be false positive findings from these studies. Gatens and Saeed[53] studied the adductor hallucis, extensor digitorum brevis, 1st dorsal interosseii, and abductor digiti minimi in seventy individuals with asymptomatic feet. They found that 38.6% had at least one of the four muscles examined showing abnormal potentials. They concluded that using needle EMG abnormalities in intrinsic foot muscles as a diagnostic criterion could be misleading. An interesting question regarding their study is what an applied kinesiology examination would have found on the individuals with positive tests. Functional problems are often found in asymptomatic feet. It is possible that their positive findings were in feet that were functionally inadequate but asymptomatic.

Treatment

Most cases of tarsal tunnel syndrome respond well to the conservative approach of applied kinesiology. If there are neoplasms or other space-occupying lesions that are irreversible with conservative care, surgical intervention will be necessary.

Most often included with, and perhaps the precipitating factor of, the tarsal tunnel syndrome is extended foot pronation. The first effort toward correction is to examine for and correct extended pronation, which includes subluxations of the foot along with the other causes of the dysfunction. Intrinsic muscles of the foot may not be corrected until specific adjustments are made for the tarsal tunnel because of interference with their nerve supply.

After correcting pronation, challenge the calcaneus in its relationship to the talus. The calcaneus will usually be subluxated posterolaterally, with its posterior surface somewhat superior. There are several methods for adjusting the calcaneus; it can be done with the patient supine or prone.

With the patient supine, the physician stands at the patient's feet facing the foot to be corrected. To adjust the right calcaneus, the physician solidly grasps the posterior superior surface of the bone with his left hand. His right hand makes a broad contact across the dorsal surface of the foot. A broad contact is important to avoid creating a subluxation with this stabilizing and controlling hand. The correction is an extension thrust directed

toward moving the posterior surface of the calcaneus in a generally inferior anterior direction. The vector of force is determined by challenge. It usually requires moving the inferior calcaneus laterally. It is often necessary to have the patient hold the side of the examination table to prevent slipping, especially if the table is covered with tissue paper. There is often an audible release of the calcaneus; however, it is not necessary for an adequate correction.

Another method is to flex the prone patient's knee to 45°. The physician stands on the side to be corrected. To correct the right calcaneus, the physician's right hand cradles the superior posterior aspect of the calcaneus between his thumb and forefinger. The other hand cradles the dorsum of the foot. The thrust is directed to the calcaneus, as previously determined by challenge. Usually the thrust is in an anterior inferior direction.

The intrinsic plantar muscles should be individually evaluated. They often require treatment to the muscle proprioceptors, origin/insertion technique, or fascial release. The muscles are often exquisitely tender in the area requiring treatment. It may be of value to use a vibrator for treatment, such as the G5 unit.

When conservative treatment fails, surgical release is often effective[114] if proper diagnosis has been made. Surgical release may disclose a small tag or tags of tissue,[91] often secondary to trauma.[42] At the point of entrapment there may be enlargement of the tibial nerve[86]; electrical stimulation may elicit poor nerve conduction past the point of constriction. In a group of sixteen patients who required surgical decompression, Edwards et al.[42] found that "Local injection of cortisone produced only transient, if any, improvement. Shoe modifications provided no improvement and arch supports always increased the severity of symptoms." Burning feet from tarsal tunnel entrapment do not improve with vitamin treatment.[9] When there is a sensation of burning feet without the nerve entrapment, it often responds to vitamin B complex.

Functional Hallux Limitus

Functional hallux limitus was first described by Dananberg.[31] It is the inability of the hallux to properly extend at the metatarsophalangeal articulation at the proper stage of the gait stance phase. The condition had gone unrecognized because there is complete range of motion at the metatarsophalangeal articulation when non-weight-bearing, as it is most often examined. The term "functional" differentiates this condition from one in which there is limited range of motion of the hallux at all times, such as in hallux rigidus.

It is obvious there is hallux extension in gait even with functional hallux limitus, noted by the upper shoe crease in this area. The failure of metatarsophalangeal joint extension is the timing. The restriction can be objectively observed with the Electrodynogram™ by the Langer Biomechanics Group,[93] which records foot forces with ground contact. Six sensors are applied to the following areas: the medial heel; lateral heel; 1st, 2nd, and 5th metatarsal heads; and the interphalangeal articulation of the hallux. A seventh sensor can be placed at the will of the investigator. The Electrodynogram™ makes a computer recording of the temporal and force patterns of foot contact. The first metatarsophalangeal restriction varies in length and may be less than 100 msec in duration and invisible to the naked eye.[31] Symptoms caused by functional hallux limitus can be almost anywhere in the body, but they are rarely in the foot. To understand this it is necessary to consider an abbreviated analysis of gait.

The gait mechanism is an excellent example of muscle action being conserved by the body when possible. It might seem that forward propulsion is primarily powered by muscle contraction, such as by the hip extensors of the stance leg; this is not the case. For example, the gluteus maximus is inactive except at the beginning and end of the stance phase.[13,37] Forward movement power is from the pendular kinetic energy of the swing leg. This pull phase is sufficient to move the center of the body up and over the ipsilateral leg.[104] Gluteus maximus contraction at the end of the stance phase may be to control the swing phase energy.

Movement into gait is started by a slight backward and then forward body movement as the swing limb flexes at the hip and knee. As the swing limb moves forward, movement in gait is powered by the kinetic energy of the swing leg. The forward motion of the limb moves the body's center of gravity forward, advancing it over the stance limb. The pendular kinetic energy of the stance leg is called the "pull force." The entire body weight pivots over the metatarsophalangeal articulation, which is fixed with the substrate. The kinetic energy of the swing limb continues to advance the body's center of gravity past the substrate contact. The stance leg produces rearward thrust on the substrate, attempting to push it backward. Since the substrate can't be moved, the pressure on the stance limb provides propulsion for forward motion without muscle action, except for stabilization. The greatest power input to the ground force for forward motion is at the point of weight bearing of the metatarsophalangeal pivot point. It is at this phase of stance limb motion that a momentary halt of hallux extension can disrupt the gait pattern.[34] There is maximum power to move the body forward at this point; limitation of hallux extension interrupts the proper sequential dissipation of energy, which must take alternate paths not in keeping with normal function.

The 1st metatarsophalangeal joint combines ginglymus and arthrodial type movement; that is, it has both

hinge and glide functions. The hinge motion is approximately 15-20° of the total motion and the glide makes up approximately 50°, providing the total of 65-70° extension range of motion. With this amount of action, the heel can adequately rise to advance the leg while maintaining digital ground contact. The mechanisms that stabilize the foot come into play, and the plantar aponeurosis is shortened to rise and resupinate the arch and externally rotate the lower leg. This keeps the lower leg synchronous with the external thigh and pelvic rotation brought about by the swing limb pull force, and the energy is sequentially dissipated within the foot and leg. When full extension of the 1st metatarsophalangeal joint is limited and/or the temporal pattern is disturbed, the forces are directed elsewhere; this ultimately causes remote dysfunction and symptoms that are often not recognized as being caused by foot dysfunction.

Foot Stabilization

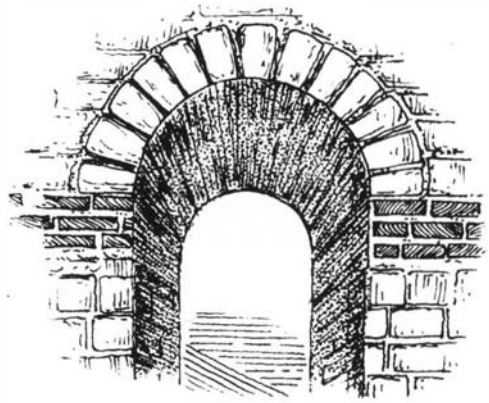
Dananberg[34] describes three distinct mechanisms that permit the foot to support the applied stress during gait: (1) calcaneocuboid locking secondary to aponeurosis tightening, (2) the locking wedge and truss effect, and (3) the windlass effect. They rely little, if at all, on muscle function and are referred to as "autosupport."

Calcaneocuboid Locking. Bojsen-Møller describes the transition from the anthropoid to human foot to provide a rigid lever for propulsion and protection of the tissues from the extreme repetitive forces of walking and running.[19]

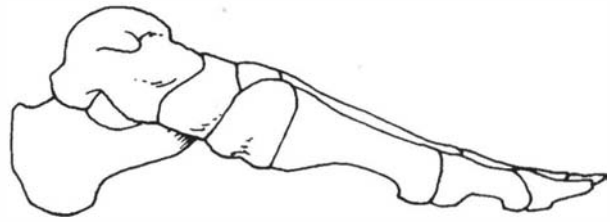
Relaxed, the ball of the foot is a soft and pliable pad. The plantar skin can be moved from side to side as well as proximally and distally. Rigidity of the tissue and bones comes with toe dorsiflexion. This is accomplished by the tissue arrangement divided into three transverse areas, each with a different mechanical function: (1) a series of transverse bands proximal to the metatarsal head in which the deep fibers of the plantar aponeurosis form 10 sagittal septa, eventually connecting to the proximal phalanges, (2) inferior to the head where vertical fibers form the joint capsules and the sides of the fibrous flexor sheaths to form a cushion below each metatarsal head with fat bodies, and (3) a distal area where the superficial fibers of the plantar aponeurosis insert into the skin. Metatarsophalangeal extension tenses the three areas, anchoring the skin firmly to the skeleton so that forces to the skin during push-off and breaking are transferred to the skeleton.[19,20]

The ball of the foot tissue is a complex network that provides plantar fascia tension to the calcaneocuboid articulation that becomes close-packed by pronation of the forefoot in relation to the hindfoot. It is congruency between the joint surfaces obtained in this position that provides strength. The calcaneus overhangs the cuboid dorsally, which stops the movement. The peroneus longus is a key to pronating the forefoot for high gear push-off and locking of the calcaneocuboid articulation. It assists in internal rotation of the crus, forcing the foot to use the transverse axes.[34]

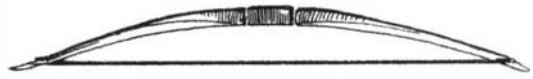
Truss and Locking Wedge Effect. In engineering a truss is a structure usually formed by a triangle or series of triangles. This is a stable arrangement because a triangle cannot be distorted by stress.[115] The triangles of the foot are made by the bones of the foot in combination with the plantar aponeurosis. The wedge effect is demonstrated by the stones in a Roman arch which, like some of the bones in the foot wedge, combine with compressive force to provide foot strength.



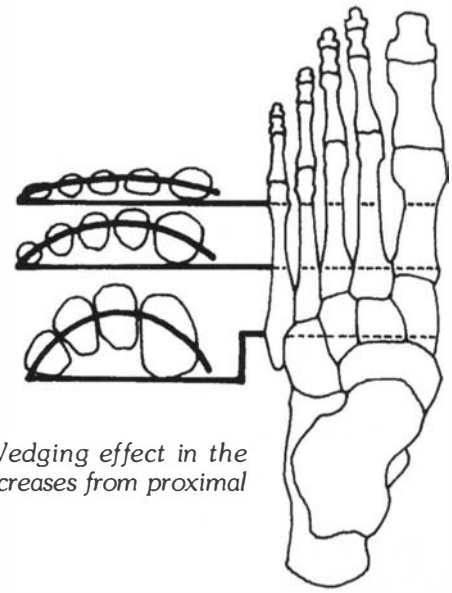
11—24. The wedging effect of a Roman arch.



11—25. Medial longitudinal arch.

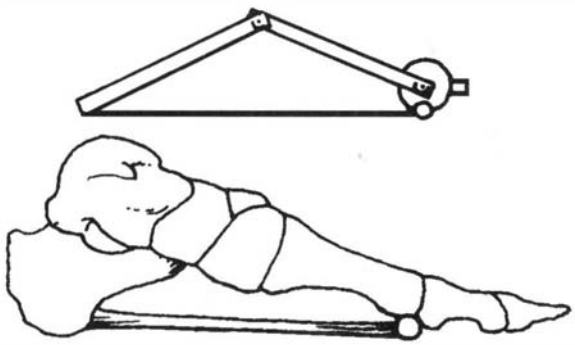


11—26. The bowstring is equivalent to the plantar fascia.

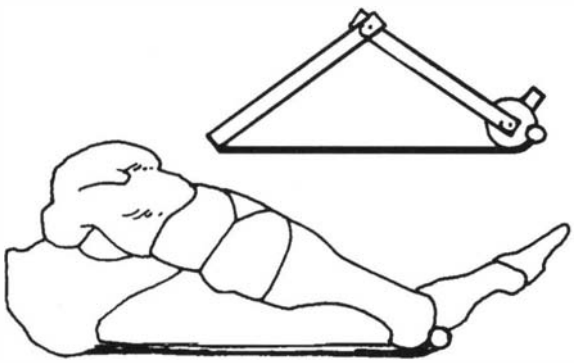


11—27. Wedging effect in the midfoot decreases from proximal to distal.

Windlass Effect. Hicks[76,77,78,79] studied the dynamic function of the foot longitudinal arches by dissection, x-rays of movement at sequential steps of the dissection, and x-rays of the living foot. He concluded that the normal foot arch is maintained by the plantar aponeurosis, with no contribution from the intrinsic or extrinsic muscles. The operative words here are "normal foot." There must be normal activity of the intrinsic and extrinsic muscles during running and walking. The mechanism that Hicks describes elevates the arch by tightening the aponeurosis with extension of the toes. The influence of toe extension on the plantar aponeurosis results from the attachment of the aponeurosis to the proximal phalanges. As the toe extends it pulls with it the plantar pad, which is an extension of the aponeurosis. The plantar pad, sliding around the metatarsal head, pulls on the plantar aponeurosis and tightens it as if it were a cable arrangement being pulled around the drum of a windlass. Maximum toe extension shortens the effective length of the aponeurosis by approximately 1 cm. This mechanism exists in all five toes, but it is most marked in the hallux. When the hallux is amputated, its role in the windlass is lost; the forefoot weight bearing shifts laterally throughout gait, and the height of the medial longitudinal arch diminishes.[105]



11—28. Hicks' windlass mechanism.

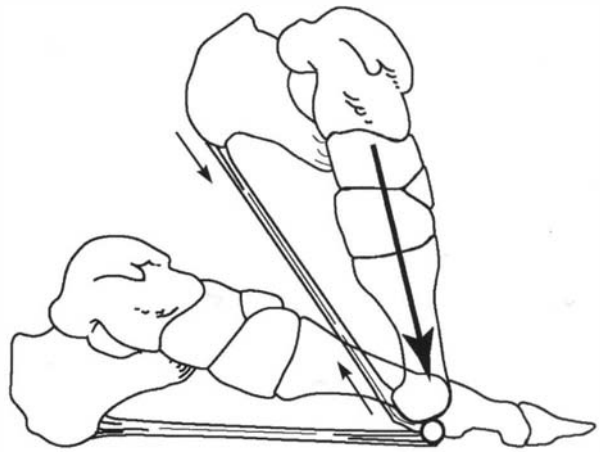


11—29. With hallux extension the plantar fascia shortens, increasing arch strength and height (after Hicks[78]).

The mechanism Hicks describes can be observed in the normally functioning subject. With the person standing in a neutral position, the arch will rise and the tibia will externally rotate. Failure of this action may be due to abnormal alignment of the first ray.[128] As described later, there is failure of the hallux to extend properly in this test when functional hallux limitus is present.

Sequence of Motion. Upon heel strike the subtalar articulation rotates into pronation for shock absorption and to accommodate internal leg rotation. As the heel unloads, weight is transferred through the foot with supination to accommodate external limb rotation. The plantar aponeurosis tightens, causing the calcaneus and cuboid to align in such a manner as to "lock" the foot and prevent arch collapse. The weight continues to move toward the toes and transfers to the second and first rays. Dananberg[36] calls the body weight flow an automatic natural arch support, noting the importance of its efficiency because forces can be two to three times body weight. As forward motion continues, the weight of the body moves over the metatarsophalangeal articulation. This extends and tenses the aponeurosis to bring the calcaneus closer to the toes, supporting the medial longitudinal arch by the windlass action. The windlass action also rotates the posterior part of the foot externally (supinates) to synchronize with the tibia external rotation.[78] Strands of the aponeurosis attach to the skin of the ball of the foot, which tightens the tissue as the metatarsal head rotates within it to prevent tissue trauma.

As the body moves over the stance limb, all of the forward movement is centered on the ball of the foot, with the most important weight bearing being carried on the firmly planted 1st metatarsophalangeal articulation. The metatarsals and midfoot become more vertical, and the body weight is carried more by compression of a column than by the arch.



11—30. As the body pivots over the metatarsophalangeal articulation the foot is stabilized and strengthened by the windlass effect, locking wedge effect, and compressive forces.

Compensation for Functional Hallux Limitus

Failure of hallux extension when the body is being pulled over the 1st metatarsophalangeal articulation during gait causes the kinetic energy to be dissipated in compensations that usually stress the weakest links. In the foot and ankle there are five alterations in movement that may be combined or individually present to adapt to functional hallux limitus: (1) altered heel lift, (2) vertical toe-off, (3) inverted step, (4) abducted toe-off, and (5) adducted toe-off.[35]

Heel lift occurs as the body weight moves forward over the metatarsophalangeal articulation. If there is failure of hallux extension the adjacent midfoot joints become obliged to move, giving the appearance of excessive pronation. Over a prolonged period the bones will remodel according to Wolff's law,[155] causing greater instability and extended pronation. When the heel cannot lift because of hallux extension failure, the next form of compensation — vertical toe-off — develops. Direct lifting of the toe from the substrate eliminates the thrusting forward of normal forward motion often seen as the slow, shuffling gait of the elderly.

The last three variations due to functional hallux limitus are avoidance compensations. Normally weight is transferred through the foot from the heel along the lateral longitudinal arch, across the metatarsals to the hallux. In the presence of hallux limitus there is prolonged weight bearing along the lateral longitudinal arch, with failure to move over to the hallux because of its inability to properly extend. The aponeurosis is never tightened by Hicks' mechanism. With heel lift there is a rapid stretching of the aponeurosis that has not been properly tightened. This results in trauma to the aponeurosis attachment at the calcaneus that may cause inflammation and ultimately a heel spur with the consequent pain. Typically there will be excessive lateral wear on the forefoot of the shoe.

The failure of the windlass mechanism arch strengthening and the weight bearing of compressive columnar features cause extended pronation to develop, producing another reason for plantar fascitis and heel spurs to develop.

It must be emphasized that pain may be located almost anywhere in the body, and there may be no foot complaint. Remote pain develops as a result of compensation that takes place to dissipate the kinetic force that is not properly used in the foot and leg for forward propulsion. When the forces of gait are dissipated abnormally in the body, there is recurring remote strain with each step, creating thousands of microtrauma at the area each day. The body responds to the trauma by inflammatory reaction, swelling, and stiffness. The resulting chronic inflammation is the hallmark of degenerative joint disease of old age, especially when it is combined with systemic problems that cause failure of tissue regeneration.[39]

The following table lists the normal motion that occurs during the second half of single support during gait and the compensations when functional hallux limitus is present.[36]

Joint	Normal Motion	Compensatory Motion
Mid-tarsal joint arch	Supination	Pronation
Ankle	Plantar flexion	Dorsiflexion
Knee	Extension	Flexion
Hip	Extension	Flexion
Lumbar spine	Lordosis	Lumbar flexion
Cervical	Lordosis	Cervical flexion

11—31. Compensatory motions that may take place during the second half of single support due to functional hallux limitus.

Symptoms

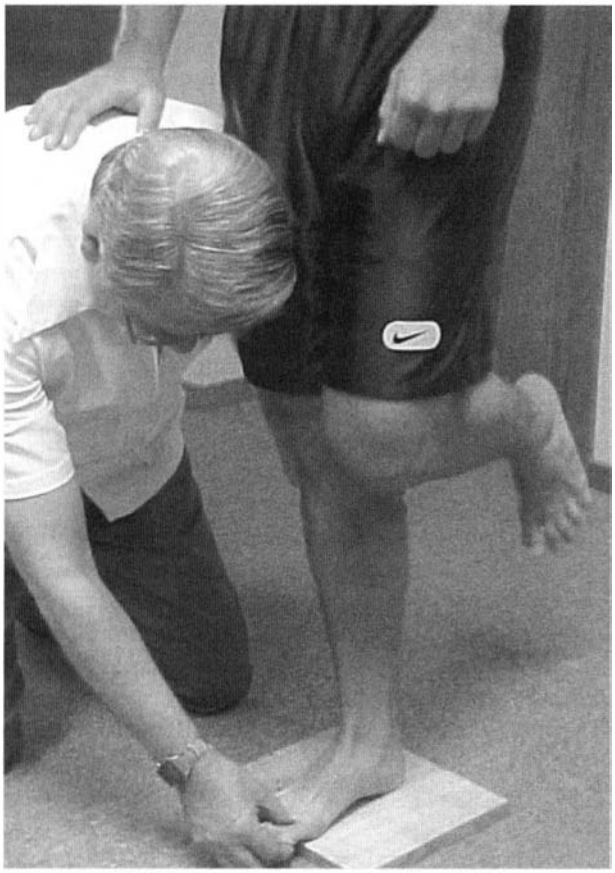
Functional hallux limitus has been overlooked for several reasons. Symptoms are rarely in the foot until well advanced; they are then attributed to the extended pronation, not to its original cause. In many cases gait may appear normal on visual observation, yet objective recording by the Electrodynogram™ clearly shows the disturbance. Finally, the hallux range of motion in the non-weight-bearing foot examination is completely normal.

Symptom possibilities are as follows: lower leg, knee, thigh, sciatic-type pain, lower back, neck, TMJ,[33,36] chronic headache, and other corrections not holding. Treatment to the areas of pain and dysfunction may help the symptoms; however, if the primary cause of gait dysfunction is not corrected, symptomatic relief is not long-lasting or may manifest in some other area of the body.

Examination

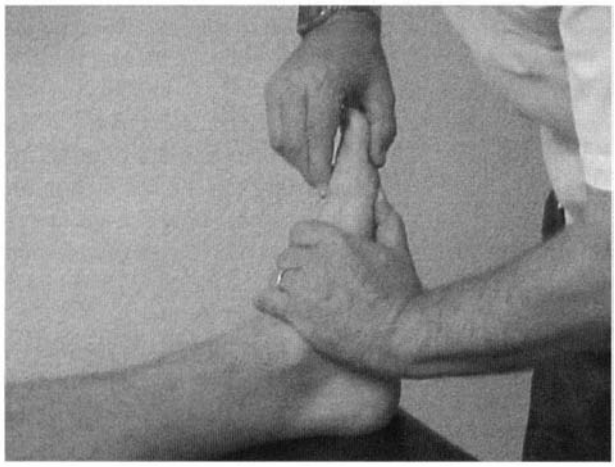
In the non-weight-bearing foot there should be full hallux range of motion. The condition is found with the patient weight bearing. With the patient standing in a neutral position, i.e., with the feet in the normal position of gait, the hallux is passively lifted into extension by the physician. Normally the medial arch will rise and the tibia will externally rotate.[128] An easy way to test for this is to have the patient stand on a platform, such as a 1"-thick board, with the ball of the foot close to the edge so the toes hang over. The patient stabilizes himself by holding on to the physician's shoulder or something else and stands only on the foot being examined. The physician attempts to lift the hallux into extension; failure of passive extension when weight bearing is a positive test.

Weight bearing can be simulated with the patient supine. With one thumb push up on the ball of the foot directly under the 1st metatarsal head until some resistance is felt. With the other thumb, attempt to extend the hallux while maintaining the constant upward pressure on the 1st metatarsal head. Resistance of the hallux to move into extension indicates the standing test will probably be positive.



11—32. Test ability of the hallux to be passively extended with full weight bearing.

The extensor hallucis longus and brevis muscles will test weak in the presence of flexor hallux limitus. This weakness causes secondary contraction of the flexor hallucis longus and brevis muscles. The contracted muscle is secondary to the weak antagonist muscle, a familiar finding in applied kinesiology. Applied kinesiology treatment for functional hallux limitus is directed to returning the weak extensor hallucis muscle(s) to normal.



11—33. Apply cephalad pressure on the 1st metatarsal head while simultaneously extending the hallux. There should be freedom of hallux extension in this simulated weight-bearing test.

Podiatry

There are several modes of treatment by podiatrists that specialize in gait analysis and the foot's relation to remote body function. Treatment may consist of orthotics, various pads and strapping, manipulation, and shoe modification.[38] Dananberg describes the Kinetic Wedge® orthotic[93] for correction of functional hallux limitus. The Kinetic Wedge® is also built into athletic shoes.[32]

A study of podiatric treatment of functional hallux limitus resulted in 46% of the patients reporting they were 75-100% better and 35% reporting 50-75% better, making a total of 77% to be at least 50% better.[39] The most interesting aspect of this study is that none of the patients complained of foot pain or discomfort. The primary symptoms were all remote from the feet.

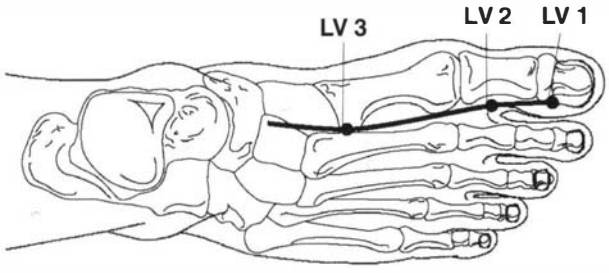
Primary Complaint	Percentage of Patients
Cephalgia/TMJ/Neck Pain	22%
Lower back pain	22%
Hip pain	22%
Knee/Leg pain	34%

11—34.

Applied Kinesiology

Examination and treatment of functional hallux limitus in applied kinesiology is directed to finding the cause and making lasting correction of extensor hallucis muscle weakness. A major factor in all corrections is that the weakness does not return after walking. Test the extensor hallucis with the ankle in dorsiflexion and the hallux in extension. Apply the testing force to flex the hallux only at the metatarsophalangeal articulation. The weakness may be uni- or bilateral. The extensor digitorum muscles are also tested and may or may not be weak. The weak muscles are treated with the five factors of the IVF involved with the weakness. All foot subluxations should be corrected before examining for functional hallux limitus. Correcting foot subluxations and the muscles will not usually correct functional hallux limitus; it is a separate entity. Any or most of the following may need attention for lasting correction.

Liver meridian. The liver meridian begins at the lateral nail point of the hallux. LV 3 is where the 1st and 2nd proximal metatarsal bones join, just lateral to the extensor hallucis tendon. Mann[102] places a high degree of importance on the LV 3 area. He discusses it as an area rather than an acupuncture point. Goodheart[63] noted that activity at LV 3 is often present in functional hallux limitus. Often a weak extensor hallucis temporarily strengthens with therapy localization to the liver alarm point (LV 14); when it does, stimulation of LV 3 strengthens the muscle. Stimulation can be done by any stan-



11—35.

dard method of acu-point stimulation, e.g., teishein, fingertip tapping, laser, needle, electrical, and others. When experience indicates that functional hallux limitus is present but the extensor hallucis is not weak, it will often show a subclinical weakness with therapy localization to the liver alarm point. When the extensor hallucis is strengthened by stimulation of LV 3, the improved function will often be lost after walking if other corrections are not made.

Origin and insertion technique. Therapy localize the origin and insertion of the extensor hallucis; if it strengthens, apply the hard digital pressure used in that technique. Most often the involvement will be at the origin along the middle one-half of the medial aspect of the fibula.

Repeated muscle activity – patient induced. If the extensor muscles weaken after the patient actively extends the toes ten times, origin and insertion technique is needed along with nutritional support with water for additional hydration and wheat germ oil t.i.d.

Muscle stretch reaction. If the extensor muscles weaken following stretching, apply fascial release, trigger point, or myofascial gelosis technique as indicated by examination. The most common need is for fascial release. Vitamin B_{12} may be necessary for lasting correction.

Maximum muscle contraction. If the extensor muscles weaken following maximum contraction, apply strain/counterstrain technique. The tender point will usually be at a more proximal area of the muscle. A collagen source of glycine may be needed for lasting correction.

Rib pump technique. Determine the need for rib pump technique by therapy localizing in the 4th and 5th anterior rib area. Strain/counterstrain technique often needs to be applied in this condition to ribs 4 and 5, both anteriorly and posteriorly, for adequate rib pump activity.

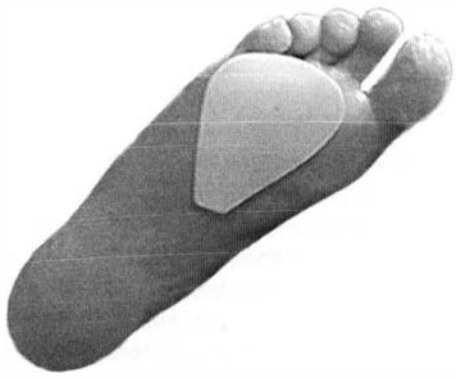
Deep peroneal nerve. The deep peroneal nerve may be entrapped by distal tibia and fibula spreading, much like nerve entrapment at the carpal tunnel by the spreading of the distal radius and ulna. Test for extensor hallucis strengthening by having the patient approximate the proximal one-third of the tibia and fibula by holding them together. The approximation could also be done at the distal ends, but most patients cannot reach that far down. The distal tibia is adjusted toward the fibula with the patient side-lying and the physician using a high velocity thrust.

Support. Most often the patient will lose the corrections immediately upon walking. If the corrections are lost with walking, support is added to give the corrections an opportunity to stabilize.

The correction at the tibia and fibula is supported by taping with porous adhesive elastic tape, such as Elastikon by Johnson and Johnson. Use two layers, taking care not to apply too much tension to the elastic and impede circulation. The patient should wear the support over the distal tibia and fibula for one week. He can bathe or shower without taking the tape off because it dries rapidly and will not loosen. Patients are not usually allergic to this type of tape. If there is a problem with allergy, place the first circular wrap with the sticky side out and then apply two more wraps over the first one in the usual manner.

A triangular adhesive felt or foam pad is placed under the 2nd-5th metatarsal heads. The felt pad provides a more solid support than the foam pad, but the foam is tolerated better than felt regarding comfort and usually provides adequate support. The pads are available in 1/8" or 1/4" thickness. They are 2" wide at the point under the metatarsal heads. The lateral border of the pad extends along the 5th metatarsal as the pad narrows to about 1/2" width at the end pointing toward the calcaneus. Most patients can easily accommodate the pad in their regular shoes. The metatarsal foam pad survives bathing or showering better than the felt one. Neither lasts as well as the adhesive tape wrap. It is advisable to give the patient several pads to take home with instructions for application. Caution the patient to not walk without the pad(s). If it is removed for bathing it should be replaced before walking. The pad(s) can be left in place when showering and then replaced before dressing.

The triangular pads are available from podiatric suppliers. An example is: Universal Footcare Products, Inc., 300 Wainwright Drive; Northbrook, IL 60062, (800) 323-5110



11—36. The wide end of the pad is placed under the 2nd through 5th metatarsal heads. No padding is placed under the 1st metatarsal.

Knee

The knee has always been a source of frequent orthopedic complaint. Being the joint between the longest and next-to-longest bones of the body, it is subjected to great stress and has great demands placed on it.

During what appears to be a hinge motion of flexion and extension, there is a gliding of the femoral condyles on the tibia, with some rotational motion. The axis of rotation continually changes. In final extension there is an increased rotation called the "screw-home" motion, which is primarily a function of the ligaments.[95] This motion locks the knee in extension, providing stability. Stability throughout the range of motion and in full extension is primarily a function of the ligaments. Muscles provide support during motion, but when varus or valgus forces are applied to the static knee, stretching the ligaments, there is no activation of muscle function to help protect the knee.[6,124]

The menisci have numerous important functions in the knee. They improve joint stability by adapting to incongruity between the surfaces of the tibia and fibula. This also improves weight distribution by effectively enlarging the contact area between the bones. They act as shock absorbers and provide a ball-bearing action, facilitating the rolling-sliding forward of the lateral femoral condyle as it rotates medially during the screw-home motion. An important function is that of load-bearing. The lateral meniscus distributes 70% and the medial meniscus 50% of the load, acting on the respective sides of the joint.[134]

With the advent of increased physical activity due to fitness awareness, knee trauma and strain are becoming more prevalent. Knee injuries can be classified into three types. (1) There is acute, direct trauma, often resulting in ligament or meniscus injury. (2) There may be acute, non-contact trauma, such as an individual running, quickly cutting, and having the knee give out. The run and cut may appear the same as those done hundreds of times before, but for some reason this time the knee fails. Here, too, there may be ligament and meniscus damage. (3) Finally, there is insidious knee pain that develops from constant stress to the tissues from bony misalignment, poor muscle support, or stress put into the knee from remote dysfunction.

Treatment for knee trauma and dysfunction has dramatically changed in the past several years. The advent of the arthroscope and MRI has provided a much better understanding of the acutely injured knee. The amount of internal derangement can be more specifically determined, enabling the physician to do a partial meniscectomy or to choose to do no further procedure. Noble and Turner[121] cite studies that indicate knee investigation by arthrography and arthroscopy combined achieves a 98% accuracy in diagnosing meniscal tears. Goodfellow, in an editorial entitled "He Who Hesitates Is Saved,"[54] makes a plea for using arthrography and arthroscopy to limit meniscectomy to major conditions. He urges "...that these techniques...be employed to reassure the patient and the doctor that the knee is not so badly damaged that it could not be made worse by an operation."

There is significant evidence that the large number of meniscectomies done in the past was not only unnecessary, but also undesirable. When the meniscus is removed, pressure at the articular cartilage increases greatly.[134,137] Osteoarthritis is more often than not a sequel to a meniscectomy.[83] Extensive postmortem studies indicate that a significant meniscal lesion does not necessarily mean that symptoms had arisen or would arise from it.[121] Noble and Erat[120] point out that "...the risks of removing a normal meniscus far exceed those of leaving a tear in the posterior third."

The standard orthopedic examination for ligament integrity and meniscus tears should be done on all patients. Even when there is no history of acute trauma, there may have been something long ago that the patient has forgotten. The physician should be familiar with these tests, which are outside the scope of this text. There are numerous excellent texts that describe the tests and give the rationale for their use.[74,81,116,117,135] The pivot-shift test is most sensitive in testing for anterior cruciate ligament rupture in acute knee trauma. Next is the Lachman test, and the least sensitive is the anterior drawer sign. This shows the change that has taken place in knee examination. The classic anterior drawer sign has been replaced by the newer tests. Treatment, too, has dramatically changed, with less extensive surgery being done.

Applied kinesiology's contribution to knee examination and treatment is primarily in the second and third classifications of knee disorders: the acute, non-contact type, and insidious knee pain.

Muscle Support

The first step in an applied kinesiology knee examination is to determine the knee-supporting muscles' function in the clear. Postural analysis may provide clues that the muscles are functioning improperly. With genu valgus, the sartorius and gracilis are often weak. Genu varus may be associated with weak adductors and/or the tensor fascia lata. Genu recurvatum is often associated with poor anterior support by the quadriceps muscles or poor posterior support by the popliteus and/or gastrocnemius.

Correcting knee pain is often as simple as correcting the muscles that stabilize the knee. This may require attention directly to the muscle, its reflexes, and/or the organ/gland association. The initial palpation examination may indicate poor muscle support. An example is the hypoadrenic who has medial knee pain. Digital pressure at the insertion of the sartorius and gracilis will produce pain. There will typically be a category II posterior ilium pelvic subluxation. It is not unusual for the pain to

be completely relieved by treating the muscle with the usual five factors of the IVF, correcting the pelvic subluxation, and having the patient chew the proper nutritional support. The condition may tend to recur until normal adrenal gland function is obtained.

Poor muscle support to the knee may not be observable in the clear. A common cause of knee problems is muscle reactivity. Examine the muscles that support the knee for reactivity in the manner discussed on page 65. Common reactive muscles are the sartorius, gastrocnemius, popliteus, adductors, tensor fascia lata, quadriceps, and hamstrings. The activity may be of muscles within the group, such as quadriceps weakening after hamstring contraction, or muscles of the group reacting to contraction of remote muscles, such as the quadriceps weakening after abdominal contraction. Body language indicating that a reactive muscle is present is seen when an individual develops knee pain with certain types of activities. Often the muscles involved can be determined by analyzing the activity. If knee pain develops from a sport that requires considerable side-thrusting, such as tennis, test the medial and lateral knee-stabilizing muscles for reactivity.

Positive ligament stretch reaction in the knee can cause failure of the supporting muscles. Blaich[17] found that the ligament stretch reaction can be limited to the knee; that is, when the ligaments of an involved knee are stretched, only those muscles crossing the knee joint weaken on manual muscle testing. Therapy localization to an adrenal reflex — usually the neurolymphatic — abolishes the stretch reaction, as does chewing whole adrenal concentrate. In a study of ten patients with the condition, he found that seven had an immediate decrease in pain at the involved knee, and nine had an immediate increase in range of knee motion.

Reactive muscles, ligament stretch reaction, or remote subluxations can cause a knee to suddenly lose muscle support, sometimes resulting in severe injury. This is often called a "trick" knee; it can develop when an individual walks or turns a corner, or from the severe strain of a running cut in football. This is the type of injury indicated earlier in which an individual repeats a maneuver done uneventfully many times before; this time, the knee gives out and severe injury results. In a sport like football, it may occur as a result of an injury a few plays or days before in which a muscle proprioceptor was damaged, causing reactivity of the sartorius or some other knee-supporting muscle.

Durlacher[41] recommends screening athletes, industrial workers, and others subject to joint strain from twists, falls, and various types of strain for ligament stretch reaction as a preventive measure against injury. This is especially applicable to an athlete because of the high degree of stress often put on the adrenal gland from the excitement and demand of the game.

When poor function develops well into the physical activity and results in joint pain or poor performance, the muscles may be failing due to their repeated activity. This is evaluated in applied kinesiology with the aerobic/anerobic muscle testing technique and repeated muscle activation patient-induced. Evaluate the activity the individual is performing and test those muscles continually active with the repeated muscle-testing procedures. Treat as indicated on pages 187-191.

Lack of muscle support to the knee can develop from the muscle stretch reaction, but it is not as common as that indicated above. In this case, a muscle is usually short and hypertonic; it weakens after it is stretched. Most physical activities that cause knee strain from lack of balanced muscle support develop as a result of strain to the joint, as in the ligament stretch reaction, or from contraction of the muscles. In the muscle stretch reaction, it is necessary for the muscle to be stretched to its full length before it will weaken.

When there is positive stretch reaction of muscles that support the knee, pain in the knee, thigh, and/or leg is most likely due to trigger points in the stretched muscle(s). The most common site for trigger points that cause knee pain is in the oblique fibers of the vastus medialis, according to Travell and Simons.[147] Failure to evaluate the muscles for ligament and muscle stretch reaction will miss a lot of the dysfunction causing knee, thigh, and leg pain.

Finally, there can be failure of muscle support to the knee because of remote subluxations, either spinal or extraspinal. The activity engaged in may cause what is, in essence, a challenge to the foot or some other joint. This may create poor muscle function in the body, just as if an examiner were challenging a joint and testing general indicator muscles for poor function.

Foot Dysfunction

Foot dysfunction of almost any type can cause secondary knee dysfunction. Subluxations or muscle dysfunction in the foot can cause neurologic disorganization with subsequent muscle weakness, improper temporal pattern, or hypertonicity.

Extended pronation causes internal rotation of the tibia while the femur is externally rotating, causing strain to the knee and subsequent inflammation.[90] This may cause patellofemoral stress syndrome, and perhaps eventually chondromalacia patella.

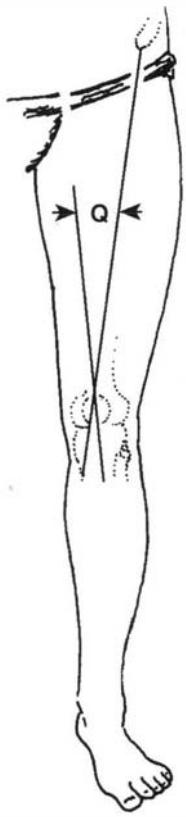
Patellofemoral Stress Syndrome

Improper tracking of the patella in relation to the condyles of the femur can cause a functional and eventually pathologic condition. Patellofemoral stress syndrome and chondromalacia patella are caused by the same mechanical factors. Chondromalacia patella indicates that there are observable pathologic changes in the articular cartilage that are not present in patellofemoral stress syndrome. In the latter, arthroscopy reveals no visible articular damage. The pain associated with patellofemoral stress syndrome is from increased pressure

caused by abnormal patellar tracking in the groove between the femoral condyles, not by cartilage damage. This is the most common cause of "runner's knee." It often develops in a novice runner who is increasing his mileage.[22]

When the knee is fully extended, the patella is above the femoral condyle. As the knee flexes, the patella glides over the condyle. When the quadriceps lifts the body weight and any other weight that might be involved, there is considerable compression force of the patella against the condyles. Balance of the patella within the femoral groove depends on the height of the condyles, muscular forces, and ligamentous support.

Q Angle. Poor patellar tracking is often secondary to an increased Q angle. The Q angle is measured from two lines, one originating from the anterior superior iliac spine and the other from the tibial tubercle, with both lines intersecting in the middle of the superior border of the patella. The Q angle is typically greater in females because of their wider pelvises. Vahl[148] considers the angle normal at 13° for males and 18° for females, while other authors consider the Q angle not to be abnormal until above 20°.[22,133] In any event, the greater the Q angle, the more laterally the patella tracks.



11—37. Q angle.

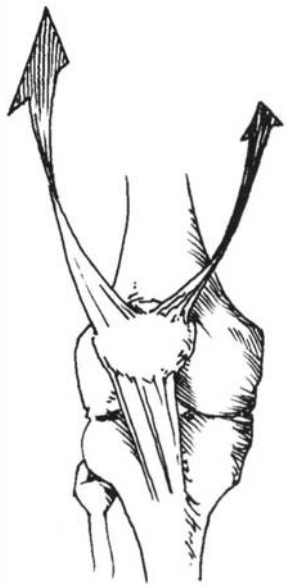
Symptoms are increased with any activity that increases the compressive forces of the patella on the condyles, including excessive stair climbing, hill running, squatting and kneeling, and quadriceps exercises. Symptomatically there is retropatellar pain after jogging a specific distance, e.g., two miles. The pain may persist for hours after an activity and even disturb sleep. There may be slight swelling, crepitus, transient locking, and instability in more severe cases.

There will be pain and possibly crepitus with the patellar compression test and pulling the patella laterally. The pain is aggravated by positions and activity requiring quadriceps contraction, such as going upstairs, and often with quadriceps muscle tests.

Running should be reduced and limited to flat, smooth terrain; icing the knee before and after activity helps. In severe cases, running should be eliminated until the condition is brought under control. Substitute activities, such as swimming, can maintain the aerobic base.

All muscles should be examined with applied kinesiology techniques for weakness, shortness, and hypertonicity. Particular attention should be given to the distal fibers of the vastus medialis, which pull the patella medially.

Vastus Medialis. The distal fibers of the vastus medialis are referred to as the vastus medialis oblique. They change direction, angling toward the patella giving the muscle considerable ability to pull the patella medially. The transverse and oblique fibers of the vastus lateralis and medialis oppose each other to suspend the patella between them. If there is imbalance between the muscles, the patella deviates toward the tight or stronger side.



11—38. Weakness of the vastus medialis allows the patella to track laterally.

There have been several studies comparing the antagonist strength and timing between the vastus lateralis and vastus medialis. One study with fine wire EMG electrodes did not find timing or intensity differences between the two muscles in symptomatic subjects,[125] but it did find an overall reduction of quadriceps activity in the symptomatic subjects. This is supported in another study[145] that also documented a reduction of jumping height in comparison to controls.

A study by Morrish and Woledge[113] found that in the normal group the reflex time of the vastus medialis oblique is significantly shorter than the vastus lateralis. In the symptomatic group the vastus medialis oblique lags behind the vastus lateralis. The temporal pattern of muscle contraction is noted in another study.[153] that revealed there is a delay in the vastus medialis reflex time in comparison to the vastus lateralis in the symptomatic group. From an applied kinesiology point of view, this

indicates there may be a reactive pair between the vastus lateralis and vastus medialis as they have antagonistic action on the patella. Reactivity between the muscles is occasionally found when the vastus medialis is strong in the clear.

The medialis is often the location of trigger points and neuromuscular spindle cell dysfunction, both of which can cause knee pain. Travell and Simons[147] indicate that a trigger point develops first, then muscle weakness follows weeks or months later. They refer to the vastus medialis muscle as the "quitter" because of its weakness that causes a propensity for the knee to "give out." The patient often says, "I can just be walking along and all of a sudden it feels like my leg will crumple under me."

In an applied kinesiology practice, the vastus medialis weakness is often found on the first examination of knee pain. The weakness is usually due to neuromuscular spindle cell dysfunction. In addition, there can be shortness of the iliotibial track and lateral retinaculum causing additional lateral deviation of the patella.

The dysfunctioning neuromuscular spindle cells in the oblique fibers can usually be palpated as a soft nodular area that is painful. Digital pressure does not usually cause radiation of pain as a trigger point does. If there is radiation of pain to the knee, it is usually eliminated with the proper neuromuscular spindle cell treatment. Therapy localization to the neuromuscular spindle cell will often not only strengthen the muscle, but also allow the muscle test to be performed without pain. For example, if the lower fibers of the vastus medialis are at fault due to neuromuscular spindle cell dysfunction, therapy localization over the spindle cells may allow the muscle to be tested with no pain and good strength, although it is severely painful and weak without TL.

Treat the neuromuscular spindle cell by digital pressure to pull apart from the middle to the ends of the area identified by palpation and therapy localization. If needed, nutritional support is provided by phosphatase found in sources made from raw bone.

Make appropriate corrections for any pelvic and/or foot faults, and check and correct all the leg muscles with the usual applied kinesiology examination and treatment techniques.

Nicholas et al.[118] recommend correction of hamstring weakness and quadriceps tightness in patellar pain syndromes. The vastus lateralis especially may need to be lengthened with the various applied kinesiology techniques.

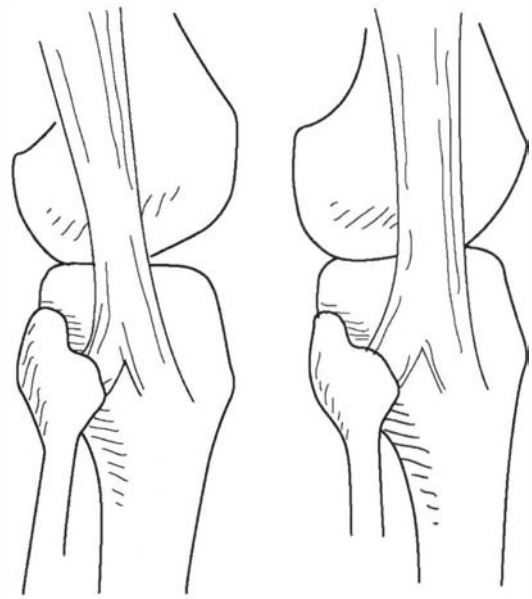
Exercise. In an applied kinesiology practice, it is observed that quadriceps boot lifts and other exercises are ineffective if there is poor muscle function that can be corrected with AK techniques. I have treated football players with enormous quadriceps group muscles who have been doing quadriceps boot lifts for months, yet they test extremely weak with manual muscle testing. A few minutes of treatment to the muscle proprioceptors and other AK factors result in dramatic change to the muscle so that it produces enormous strength on manual muscle testing.

Patellofemoral Pain Syndrome. Goodheart[62] describes an involvement of patellofemoral pain that requires support in addition to the corrections indicated above. The symptoms are diffuse ache in the subpatellar area with exacerbation on stair climbing. On extended knee flexion there may be pain upon straightening the knee. There is usually crepitus with knee motion that can be felt by the examiner placing his hands on the sides of the knee. There may be a mild swelling that is rather diffuse and can be lateral or medial or both. The same sensation of the knee giving out as described above is present and sometimes it actually does, causing the patient to fall.

Examination and treatment consist of the same procedures indicated for the vastus medialis weakness noted previously. In addition, rib pump treatment at the 7th costocartilage junction and costovertebral location posteriorly may be needed.

Iliotibial Band Friction Syndrome

The iliotibial band friction syndrome is an overuse injury generally found in long-distance runners. At 30° of knee flexion, the posterior fibers of the iliotibial band abut on the lateral epicondyle. The condition is characterized by tenderness over the lateral epicondyle of the injured knee; occasionally swelling is present. The pain may affect the individual's gait. It can usually be relieved by walking with a stiff knee.[119]



11—39. With knee flexion and extension, the iliotibial band glides back and forth over the lateral femoral condyle.

The condition usually develops in novice runners with bowlegs, tibia vera, and hyperpronated feet. Brody[22] recognized that worn outer soles of running shoes are associated with iliotibial band friction syndrome, and considered that they may be causative. He recommends foot correction, possibly including orthotics. Worn outer soles are an indication of functional hallux limitus, which should be considered as previously discussed.

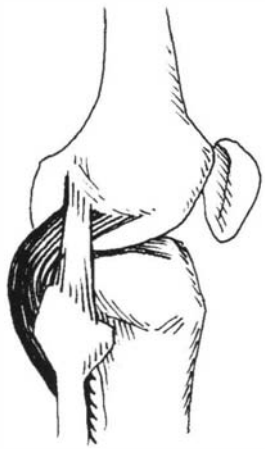
Krissoff and Ferris[90] indicate the etiology to be a "...tight foot, calcaneal varus, cavus foot, or valgus heel-forefoot alignment. The foot is unable to absorb the stress of the support phase of running and transmits stress to the lateral side of the knee." "Orthotics are often contraindicated in this rigid foot, because it needs more, not less motion."

The length of the iliotibial band can be evaluated with Ober's test.[123] With the patient side-lying with the affected leg up, the examiner stabilizes the pelvis. The thigh is abducted and extended, with the knee flexed to 90°. While maintaining stabilization of the pelvis, the leg is allowed to adduct. Failure to adduct beyond the neutral position is a positive test.

The tensor fascia lata and gluteus maximus muscles help tighten the iliotibial band. Examine for muscle stretch reaction and involvement of the muscle proprioceptors. Treat as indicated to lengthen the muscle. The iliotibial band may need to be lengthened with stretching exercises.

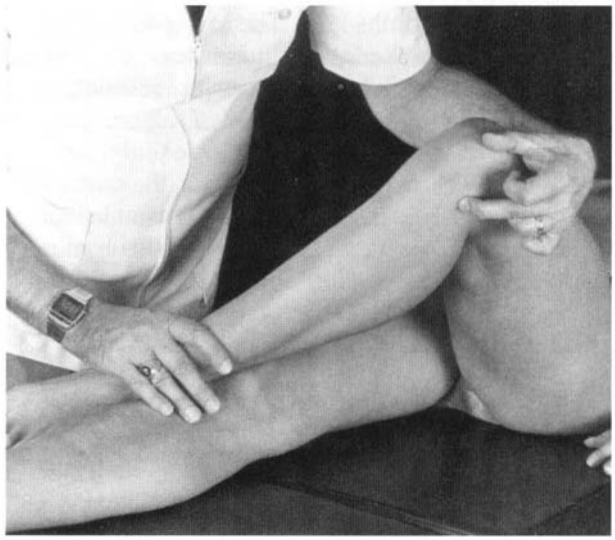
Popliteus Tendinitis

The popliteus muscle helps prevent forward dislocation of the femur on the tibia, which is especially important when running downhill. Longitudinal forces in the femur tend to displace the condyles anteriorly on the tibial plateau during mid-stance. In checking this motion, the popliteus can become overstrained.[22] There is exquisite tenderness at the popliteus insertion. Put the leg into the FABERE Patrick position so the tenderness can be easily palpated. Locate the proximal fibular head and palpate up the fibular collateral ligament to a point just proximal to the lateral joint line. The insertion of the popliteus is slightly anterior to the fibular collateral ligament at this level.



11—40. Popliteus action of limiting forward motion of the femur on the tibia.

Popliteus tendinitis must be differentiated from iliotibial band friction syndrome, torn ligaments, cysts, and lateral meniscus involvement. The tenderness from the iliotibial band friction syndrome is usually over a larger area. Tenderness in popliteus tendinitis is localized to its small area of insertion. When weight bearing, internal rotation of the femur on the tibia often causes pain to be accentuated or reproduced.[90]



11—41. Locating the point tenderness of the popliteus insertion.

Extended pronation increases internal rotation of the tibia. Its correction, along with all aspects of popliteus muscle correction, is necessary to improve the condition. Runners should limit themselves to flat, even terrain during rehabilitation.

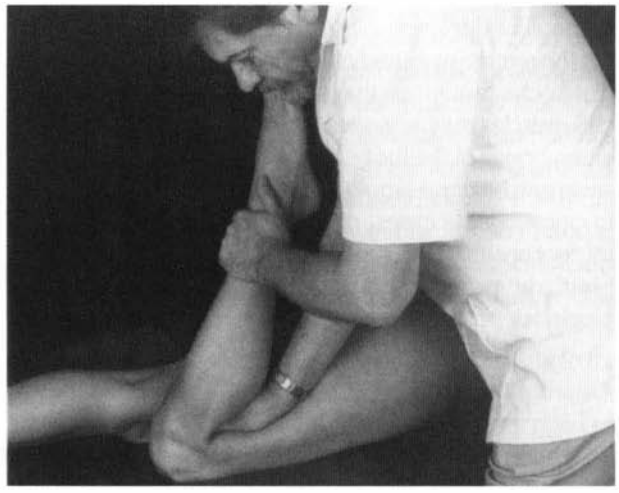
Proximal Fibula Subluxation

The proximal head of the fibula is often found subluxated with the tibia, especially when there is extended pronation. This is probably due to the torsion put on the leg from the extended pronation. There may or may not be pain from digital pressure on the articulation. There will be positive therapy localization and challenge. The best challenge is to strengthen associated knee muscle weakness by challenging the fibular head. This provides the best vector for correction of the subluxation. If no knee muscles are weak in the clear, one can challenge the fibular head to find the direction that causes a knee muscle to weaken. In this case, manipulation will be in the direction opposite the challenge that caused the weakness.

Generally the proximal fibular head will be subluxated posteriorly. It is easily manipulated in either the prone or supine position.

For supine manipulation of the left fibular head, the physician stands beside the treatment table facing the patient's head. The patient's knee and hip are flexed past 90°, and the physician puts the palm of his left hand just inferior to the patient's knee crease, with his thumb on

11—42. Adjusting proximal fibula head anteriorly with patient supine.



11—43. Adusting proximal fibula head anteriorly with patient prone.

the posterior fibular head. His left hand grasps the patient's ankle, and a gentle thrust is applied with the right thumb in the posterior-to-anterior vector that gave the maximum strengthening of an associated muscle. Simultaneously, the physician's right hand slightly increases the flexion of the knee and hip.

Prone manipulation is done in a similar manner. To adjust the right fibula, the physician stands beside the patient. The palm of his right hand is on the distal posterior thigh, just above the knee, while his thumb contacts the posterior aspect of the fibular head. His left hand grasps the ankle, and a thrust is applied with the right thumb in the vector of optimal challenge while the left hand slightly increases knee flexion.

In both types of manipulation there is usually an audible release, but it is not necessary for effective correction. There should be negative therapy localization, challenge, and strengthening of the associated muscles, indicating an effective correction. Re-evaluate after the patient walks, as the correction is often lost in the presence of extended pronation or other foot problems.

Shoulder and Shoulder Girdle

A major difference between man and lower animals is the effectiveness with which man can use his hand. The unique features of the hand are lost to a great extent if the shoulder and arm cannot put it in the proper position to be used. A comparison of the shoulder and hip puts in perspective the greater motion the shoulder has over the hip, but at a sacrifice of stability. The femur head is solidly located in the acetabulum, and all hip motion comes from that articulation. The shoulder obtains its motion from the integrated motion of five articulations and numerous muscles that must work together harmoniously.

Due to the integration of action at the shoulder, it is often subject to disturbed function that may limit range of motion, with accompanying pain. Dysfunction can be at the shoulder joint itself, the associated articulations, local muscles of the shoulder, or of some other associated structures, such as the bicipital tendon.

The shoulder is vulnerable to trauma because of its flexibility. The arm, being a long lever, can put damaging forces into the shoulder that may be especially traumatic when it is in a vulnerable position. Finally, the shoulder is the recipient of much referred pain from systemic conditions that must be differentially diagnosed.

Shoulder dysfunction is often secondary to remote problems. Often the remote problem may not be symptomatic, but it causes a neurologic disorganization that interferes with shoulder muscle harmony. When a patient's chief complaint is shoulder pain, the examination obviously begins around the shoulder. During this initial observation, one must be constantly aware of remote conditions that may be causing secondary shoulder dysfunction. It is not unusual to find the cause of shoulder dysfunction in the pelvis. A category I pelvic fault may cause adaptive faults in the shoulder girdle, breaking up the rhythm of muscles moving the five joints necessary for full range of motion at the shoulder. Subluxations, extended pronation, and other foot dysfunction can improperly stimulate the proprioceptors. The resulting neurologic disorganization interferes with proper shoulder muscle function, again breaking up the harmony of shoulder motion.

The effect of remote problems can often be observed

in shoulder dysfunction by challenging or therapy localizing the remote area and observing the improved function of muscles that move the shoulder. For example, rotator cuff muscles may test weak and immediately strengthen when bones of the foot are challenged. Usually the challenge can be done simply by generally forming the arches. In questionable cases, or to determine which bones of the foot are involved, specific challenging may be required. Similarly, properly placing the patient on DeJarnette blocks for pelvic correction may strengthen weak shoulder muscles. Nearly all the applied kinesiology techniques discussed in this text may be applicable to correcting shoulder problems. Many of the techniques that deal with modular interactions, such as PRYT and equilibrium synchronization, are prime factors to consider in shoulder dysfunction.

Other conditions that must be considered, which are outside the scope of this text, are the various types of thoracic outlet syndromes, cervical spine dysfunction, and the many kinds of referred pain.

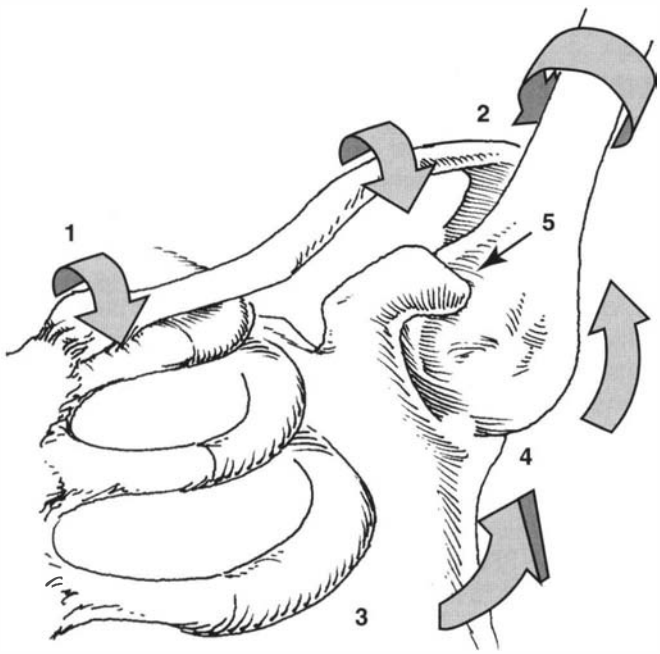
Five Joints of the Shoulder

The entire shoulder girdle moves during most shoulder movements. There are five articulations considered here in shoulder motion. Three of the articulations — the glenohumeral, acromioclavicular, and sternoclavicular — are bony in the general sense of the term. The subacromial and scapulothoracic articulations are not bone-to-bone articulations; rather, they are soft tissues.

A routine that includes all possible facets of dysfunction should be developed to examine the shoulder. Because of the interaction of structures in shoulder movement, there may be several areas in the complex that contribute to the total problem. Sometimes only a portion of the problem is found; the patient is brought to a plateau of improvement, but the condition is not completely corrected.

Particular attention should be paid to the muscles as one examines the shoulder girdle. The control of the muscles over the shoulder girdle is demonstrated by the fact that nearly normal shoulder action is maintained after resection or removal of the clavicle if muscle function is carefully preserved.[87] This, of course, depends on the muscles having the proper temporal pattern working in the harmonious symphony for which they are designed.

A basic principle throughout shoulder examination is comparison of the normal and abnormal sides. Generally one can stand behind the patient, with palpation beginning at the sternoclavicular articulation, moving out the clavicle, around the shoulder, and to the scapula, all the time comparing the two sides.



11—44. Overview of five functional articulations of the shoulder: 1) sternoclavicular; 2) acromioclavicular; 3) scapulothoracic; 4) glenohumeral; 5) subacromial. Dysfunction of any one disturbs normal function.

Sternoclavicular Articulation

The sternoclavicular articulation is the only true articulation the shoulder has with the trunk. It is a saddle-shaped joint, giving a double-gliding action.

Palpation is done directly over the joint capsule. Pain relating to the joint is markedly localized, with the patient easily able to circumscribe the pain location. When there is a subluxation of the sternoclavicular articulation, there will be positive therapy localization and challenge. Care must be taken to differentiate the positive therapy localization from that of KI 27 and the neurolymphatic reflex to the subclavius, and intrinsic spinal muscles that are in the same location.

When there is considerable tenderness of the sternoclavicular articulation, evaluate for a category I or II pelvic fault. Often there will be positive therapy localization or even challenge that is eliminated when the pelvic fault is corrected. The tenderness on digital pressure will be dramatically relieved. It is not unusual for a patient with a pelvic and shoulder problem to have complete range of motion after a pelvic correction, with no treatment administered to the shoulder.

If there is a primary subluxation at the sternoclavicular articulation, challenge to find the vector that causes optimal strength of weak associated muscles. When a weak associated muscle cannot be located, adjust in the direction opposite the challenge that causes a previously strong indicator muscle to weaken.

Acromioclavicular Articulation

Following evaluation of the sternoclavicular articulation, palpate the origin of the sternocleidomastoid at the sternum and progress out the clavicle to palpate the muscle's clavicular origin. Tenderness or nodules indicate a possible involvement. Sternocleidomastoid muscle dysfunction is often secondary to disturbance in the stomatognathic system, which should always be screened in shoulder dysfunction.

Continue to palpate distally on the clavicle until reaching the maximum posterior curve. From that point drop down approximately 1" to the coracoid process, located under the pectoralis major muscle. Tenderness of the coracoid process indicates involvement of the pectoralis minor muscle. If the muscle is weak or tight, it can cause neurovascular bundle compression or poor lymphatic drainage.

Tightness of the pectoralis minor muscle is often associated with weakness of its antagonist, the lower trapezius muscle. Bilateral lower trapezius weakness is associated with a dorsolumbar fixation.

Shoulder-arm symptoms can be caused by poor thoracic duct or right lymphatic duct drainage. The patient should be evaluated with retrograde lymphatic technique.

Proceeding with palpation from the posterior curve of the clavicle, go distally along its superior border to a point where the fingers drop slightly down over a ledge. This ledge represents the acromioclavicular articulation, with the acromion being slightly lower than the clavicle. While palpating the joint, have the patient move the shoulder girdle to help identify the joint. Observe for crepitation with the movement. Pain relating to the acromioclavicular joint is usually localized to and about the joint on digital pressure. The patient can easily localize the pain by pointing directly to the joint.

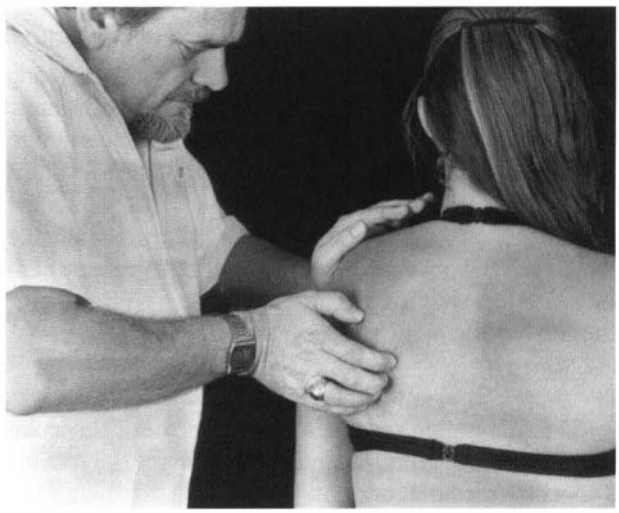
The clavicle rotates with shoulder abduction and flexion, requiring considerable motion at the acromioclavicular articulation. At full elevation of the arm, the joint is at its greatest strain; consequently, pain increases during the final range of elevation. The last 30° of elevation is called the "acromioclavicular painful arc."[87] This painful arc contrasts with the "subacromial painful arc" of between 60-100° of elevation.

Examination of the acromioclavicular joint must differentiate between acute and chronic infection, rheumatoid arthritis, post-traumatic osteoarthritis, osteolysis, and occupational osteoarthritis from structural functional conditions.[108]

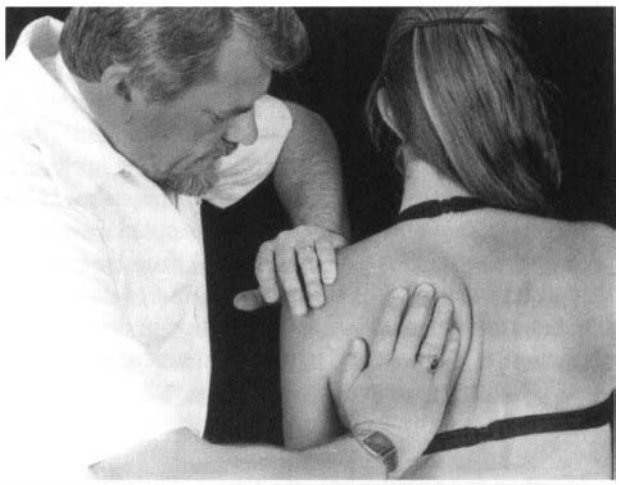
Kessel[87] points out five important factors in diagnosing acromioclavicular joint disturbance: (1) history of trauma, often caused by occupation or sporting activities, (2) localized and specifically circumscribed pain over the joint without radiation, (3) increased pain in the last 30° of arm elevation, (4) increased pain by passive adduction, particularly adduction and extension, and (5) positive x-rays taken specifically of the acromioclavicular joint.[157]

Sprain. The acromioclavicular sprain quite often results from force being transmitted into the shoulder from a fall or a blow. Allman[4] classifies sprains by grade, referring to the amount of ligament damage, x-ray findings, and therapeutic approach. When sprain of the articulation is suspected, stress x-rays should be taken with the patient holding 10-15 pound weights in each hand to distract the joint. Treatment of an acromioclavicular sprain ranges from simply protecting the articulation while it heals to surgical repair.

Goodheart[56] describes a method of treatment to the acromioclavicular joint that is applicable when there is not too much tissue damage. The joint is challenged by contacting the inferior angle of the scapula with one hand



11—45. Challenge to approximate acromioclavicular articulation.



11—46. Challenge to separate acromioclavicular articulation.

and the acromion process with the other. The scapula is then moved in various directions to challenge the articulation. The challenge that strengthens weak associated muscles in a separation or sprain is generally toward approximation of the articulation.

In some dysfunctioning acromioclavicular articulations, there is an approximation of the joint that must be separated by challenge to cause weak associated muscles to strengthen. An appropriate treatment is to simply hold the scapula to approximate or separate the articulation, as indicated by challenge. It is often necessary to hold pressure for a prolonged time (four to five minutes) to obtain effective correction. In case of trauma, this type of non-irritating manipulation corrects the joint without producing further tissue damage.

An effective method of supporting the articulation in the case of separation is tape applied to the contralateral shoulder and across the chest to the involved shoulder. Before applying the tape, put another piece of tape — sticky side to sticky side — over the tape crossing the chest. Using this technique, the two shoulders are connected by the tape, with no contact across the chest. Instruct the patient to pull the contralateral shoulder back when pain develops in the involved acromioclavicular joint. This causes the tape to pull on the involved shoulder to approximate the joint. Thus the patient can give himself a treatment whenever needed, which is effective in reducing pain and promoting healing in the proper position.

Scapulothoracic Articulation

Examination proceeds from the acromioclavicular joint to the scapula and the muscles responsible for moving it in its six cardinal motions.

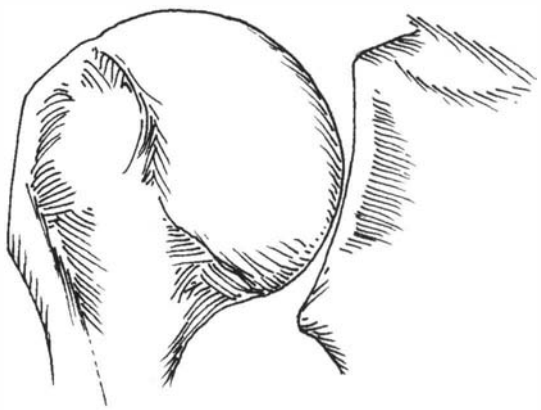
First, observe the scapula's position and compare it with its contralateral partner. A flared vertebral border of the scapula indicates probable weakness of the serratus anticus or rhomboid muscles. When the superior lateral portion of the scapula is inferior and the head and neck are tilted away from the side of involvement, the upper trapezius muscle is probably weak. A depression of the coracoid process indicates probable weakness of the lower trapezius. Continue to analyze the scapula's position for muscles that may be weak or hypertonic.

An additional method of evaluating the muscles that move the scapula is to have the patient bilaterally elevate the scapula by hunching his shoulders. There should be symmetry of movement between the scapulae. Failure of proper movement may be due to weak muscles or tight, shortened muscles that do not allow the motion. In a similar manner evaluate scapular motion in depression, abduction, adduction, and inferior and superior rotation.

Correcting any dysfunction of muscles that move the scapula is paramount in treating shoulder dysfunction. The techniques of applied kinesiology to strengthen or lengthen the muscles are effective in obtaining correction. Exercise or stretching procedures are rarely needed in correcting shoulder dysfunction.

Glenohumeral Articulation

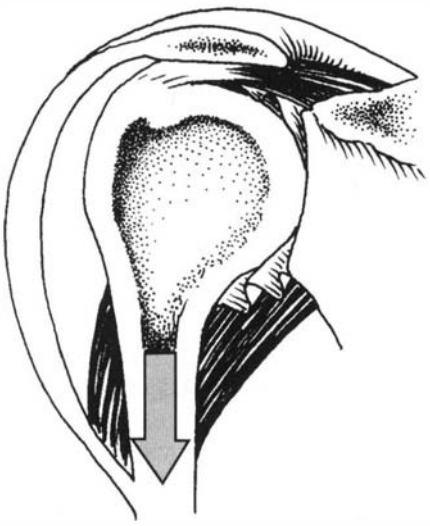
The glenoid fossa faces laterally, superiorly, and anteriorly. It is a shallow fossa that is deepened somewhat by the glenoid labrum, which is an extension of the anterior capsule. The motion of the head of the humerus in the glenoid cavity is not a true ball-and-socket arrangement, but more a gliding motion because of the shallowness of the glenoid cavity. Only a small portion of the humeral head contacts the glenoid cavity. Because of the small surface contact, instability, and great freedom of motion, this joint requires significant muscular support with precise harmony of muscle action during joint movement. The upper portion of the joint capsule is tight when the arm hangs loosely at the side of the body. The lower portion of the capsule is tight when the arm is fully abducted on the scapula. In between these two extremes the articulation is the weakest and most likely to dislocate, especially if the muscles are not working harmoniously and providing balanced support. The joint capsule is lax enough that with some abduction the humeral head can be drawn out from the glenoid cavity 2 or 3 cm.[152] The anterior portion of the joint capsule is weak and accounts for 90% of shoulder dislocation. In a study of the elbow and shoulder muscles by fine wire EMG, Basmajian[11] observed minimal activity of the muscles when the arm was hanging freely at the side. When the subject held a 16-pound load to the point of unbearable fatigue, there was activity in the supraspinatus and posterior deltoid muscles. When a sudden and sustained heavy downward pull was applied to the arm by the investigator, the supraspinatus and posterior fibers of the deltoid contracted appreciably. There was no activity in the vertical muscles supporting the shoulder, such as the middle deltoid. The reason the horizontal fibers of the supraspinatus and deltoid are responsible for prevention of downward dislocation of the humeral head is the angle of the glenoid cavity. From superior to inferior, the glenoid cavity slopes laterally. For the head of the humerus



11—47. Because of the small humeral head contact in the glenoid fossa, muscle balance is important.

to dislocate inferiorly, it must move laterally. This is prevented by contraction of the horizontal fibers.

In the mid-positions of shoulder motion, the capsule becomes extremely loose and the shoulder joint depends on the integrity of the rotator cuff muscles.



11—48. Caudal traction on the humerus activates the supraspinatus and posterior deltoid, but none of the vertical fibers of the deltoid.

Subacromial Articulation

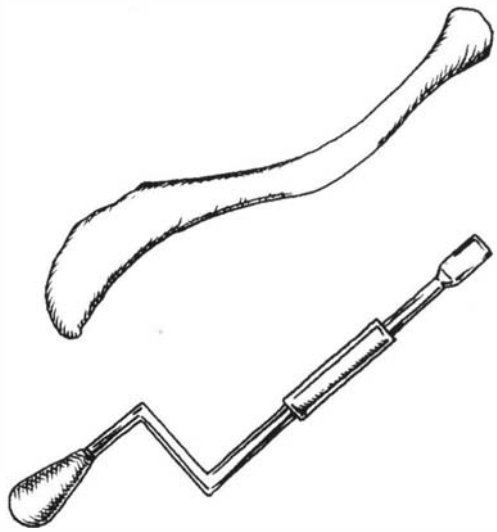
The subacromial joint is not formally recognized by anatomists as a true joint because there is no complete capsular enclosure between the acromion and upper trapezius.[67,152] In functional analysis it is necessary to consider it as a joint because it has all the characteristics of a true joint except for the capsule. Above, the joint is formed by the acromion process, acromioclavicular joint, and the coracoacromial ligament, which form an arc and combine with the deltoid. "The distal or inferior aspect is formed by the two tuberosities traversed by the transverse humeral ligament over the tendon of the long head of the biceps. The short rotator muscles and their conjoint tendons attached to the surgical neck of the humerus intervene and the large subacromial bursa acts as the joint cavity."[87]

When considering the subacromial articulation, one must pair it with the glenohumeral joint. Movement in one dictates movement in the other. The subacromial articulation becomes very important in arm abduction. A symphony of muscular action is necessary to pull the head of the humerus down and rotate it while abduction is taking place. Also necessary is appropriate laxity of soft tissue.

Shoulder Motion Evaluation and Treatment

Motion of the shoulder is dependent on normal clavicular, scapular, and glenohumeral motion. The first 20° of humeral abduction is primarily glenohumeral motion. During this phase the deltoid and supraspinatus, as well as other synergists, must act as a couple for efficient operation. During abduction it is necessary for the subscapularis, infraspinatus, and teres minor to keep the humeral head from rising up into the suprahumeral space and encroaching upon the coracoacromial arch. Failure of these muscles to resist the superior pull of the deltoid may be a cause of subacromial bursa irritation that might ultimately end in bursitis.

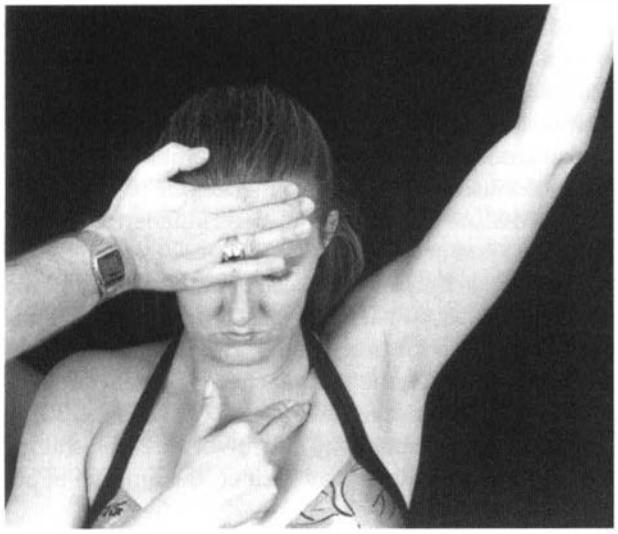
During complete abduction, the distal end of the clavicle elevates 60°. Thirty degrees of the elevation is at the sternoclavicular articulation. This elevation is in its final position when the arm reaches 90° abduction. It is limited by the costoclavicular ligament. The final 30° of distal clavicle elevation is by rotation of the crank-shaped clavicle. Failure of this rotation limits the amount of abduction that can be obtained. It appears that the subclavius muscle is important in rotating the clavicle. In many cases of "frozen shoulder," great improvement can be made by treating the subclavius muscle.[58]



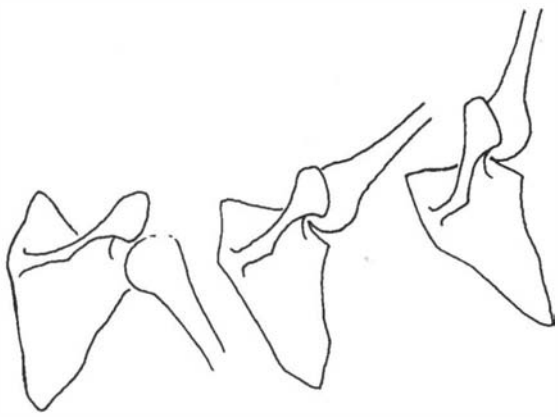
11—49. The distal end of the clavicle elevates with 30° of hinge-type movement at the sternoclavicular articulation, and rotation of the crank-shaped clavicle gives an additional 30° for a total of 60° elevation.

Since the subclavius muscle cannot be directly tested, it must be evaluated by observation of clavicle movement and therapy localization over the muscle. Treatment of the subclavius muscle is usually directed to the neuromuscular spindle cells, Golgi tendon organ, or origin/insertion. The digital pressure applied must be rather heavy to contact as much of the muscle as possible.

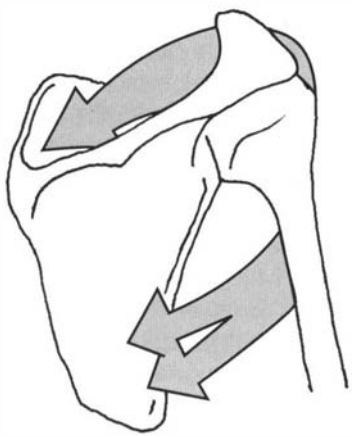
When the humerus is abducted to 20°, scapulohumeral rhythm develops. The scapula rotates to point the glenoid cavity superiorly. The upward rotation of the scapula takes place around an axis approximately 1" below the spine of the scapula and 2" from the medial border. The upper trapezius lifts up on the lateral portion of the scapula's spine. The middle and lower portions of



11—50. The subclavius muscle can only be evaluated by therapy localization and observation of clavicle movement.



11—51. Scapula motion.



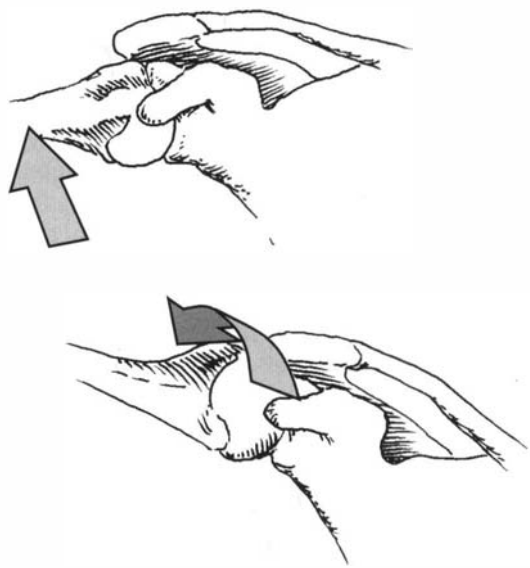
11—52. Muscle couple to keep the humeral head from too forceful movement into the subacromial articulation.

the trapezius show great electrical activity during abduction of the arm. The middle portion of the trapezius stabilizes the scapula near its axis of rotation, while the lower trapezius pulls the medial border down. The serratus anticus rotates the inferior border of the scapula anterolaterally. Upward movement of the scapula is checked by the stretching of the latissimus dorsi, pectoralis major, pectoralis minor, and somewhat by the rhomboids and levator scapula.

At approximately 90° of abduction, external rotation of the humerus must take place. This rotation is necessary to delay the locking of glenohumeral motion as a result of the greater tuberosity hitting the superior margin of the glenoid. The rotation is affected by teres minor and infraspinatus contraction.

Observation of the scapulohumeral ratio, clavicular movement, and muscle testing should reveal the cause of poor shoulder motion. In addition to testing for weak muscles, test for muscle stretch reaction. Fascial release or intermittent cold with stretch technique may be needed to lengthen shortened muscles. Trigger points are frequently found in the rotator cuff muscles.

As a final thought, remember to test for remote dysfunction influencing the shoulder girdle. For example, sometimes there is a weak latissimus dorsi on the side opposite shoulder dysfunction. This weakness appears to cause hypertonicity in the latissimus dorsi on the side of dysfunction, limiting abduction of the humerus.



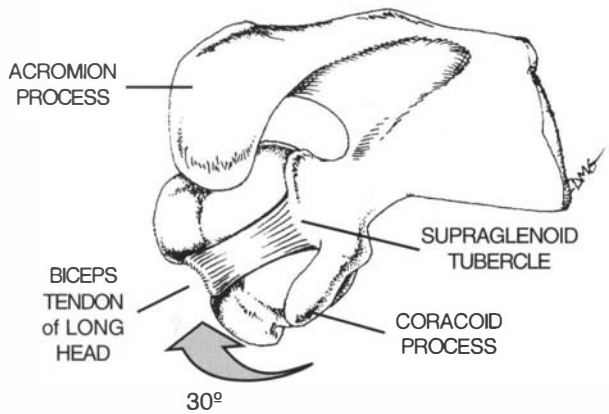
11—53. The greater tuberosity must externally rotate to avoid hitting the superior margin of the glenoid fossa.

Slipped Bicipital Tendon

The tendon for the long head of the biceps brachii muscle originates from the supraglenoid tubercle of the scapula and passes over the head of the humerus, descending into the intertubercular groove. It is retained in the groove by the transverse humeral ligament and by a fibrous expansion from the tendon of the pectoralis major. The tendon can sometimes slip from the groove with minimum or no trauma, depending on its morphology and soft tissue containing factors.

A slipped bicipital tendon is very often effectively treated with applied kinesiology techniques.[56] It may slip as a result of a jerk when the biceps contracts, especially when the humerus is in internal rotation. This may happen when a worker is carrying an object and his partner drops the other end. It may also happen from less violent activity, such as reaching into the back seat of a car from the driving position. The position of the arm in elevation and rotation predisposes the tendon to slip.

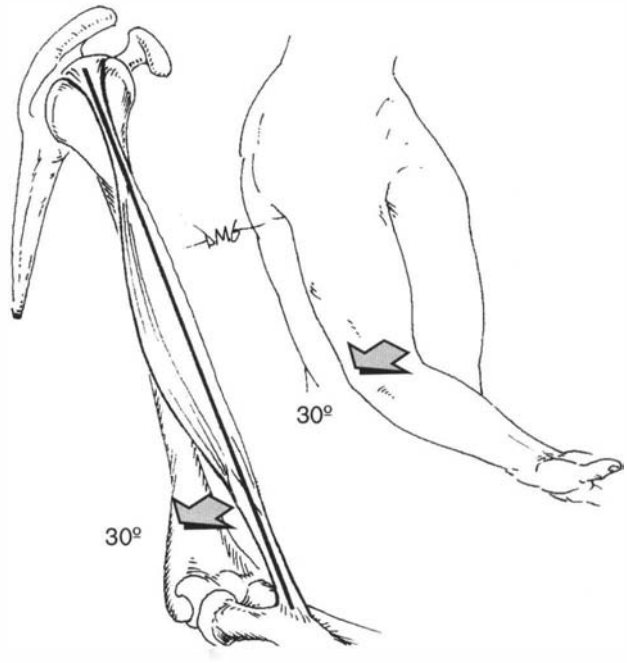
The groove for the bicipital tendon varies in width, depth, and configuration of the walls. When the groove's morphology is shallow or its walls do not slope deeply, there is greater chance for a slipped bicipital tendon to occur. When this is combined with occupations requiring considerable biceps activity, the chance of slippage becomes even greater.



11—54. Superior view of bicipital tendon with 30° external humerus rotation.

The biceps tendon does not move in the groove when the biceps contracts to flex the elbow. When there is biceps muscle contraction with the humerus in internal rotation, the biceps tendon tightens against the medial wall of the bicipital groove. This creates a mechanical strain on the tendon. With increased external rotation of the humerus, the tendon tightens against the central floor of the groove, giving greater efficiency and less strain to the tendon.[87]

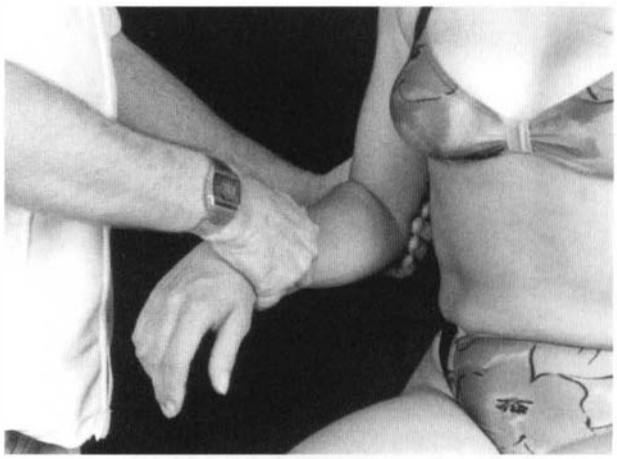
To locate the biceps groove for palpation, flex the elbow to 90° and use it as an indicator to externally rotate the humerus to 30°. In this position the biceps groove is directed forward. There will be change of contour and tenderness with tendon slippage.



11—55. Bicipital tendon with 30° of external humerus rotation.

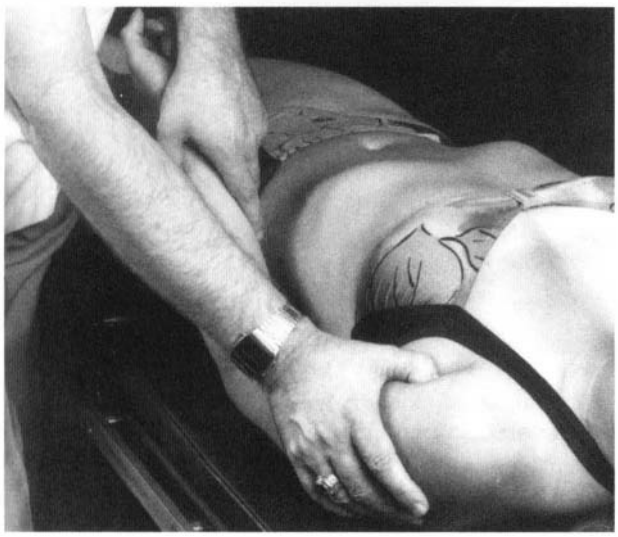
Rupture of the tendon is indicated by the characteristic biceps bulge. Yergason's sign[156] indicates tendinitis or slippage. It is positive when there is pain and tenderness over the bicipital groove with elbow flexion and supination against resistance.

When there is biceps tendon slippage, the biceps will frequently test weak with manual muscle testing.



11—56. Yergason's sign — patient supinates against resistance.

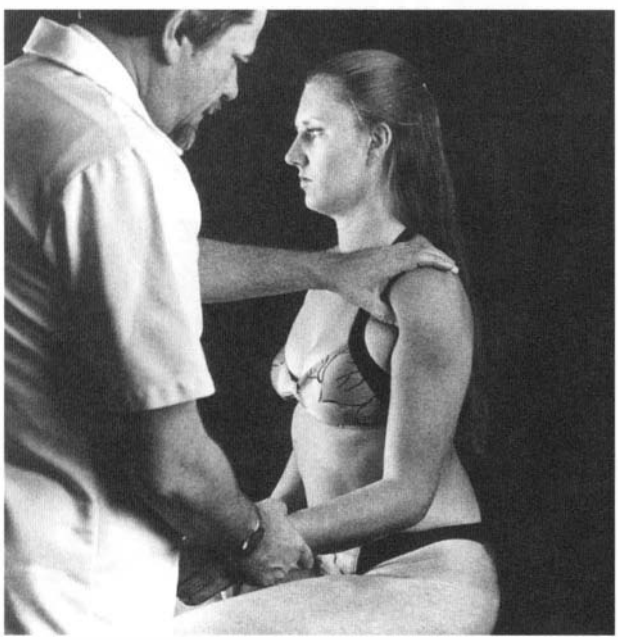
Positive indication of slippage is when the previously weak muscle tests strong with static challenge on the tendon toward the groove. There will also be positive therapy localization over the tendon.



11—57. The biceps brachii will usually test weak with a slipped bicipital tendon. When the tendon is held toward correction, the muscle will usually test strong.

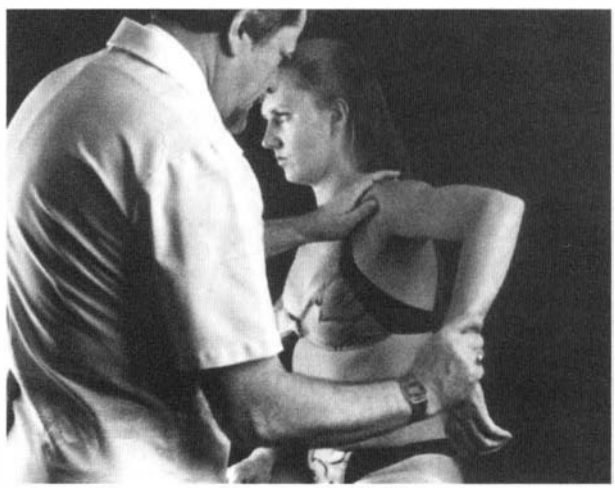
Conservative treatment consists of manipulating the tendon into the groove and balancing the rotator muscles of the shoulder. The tendon quite often slips medially, but occasionally there is a lateral slip.

The slipped bicipital tendon can be manipulated back into the intertubercular groove with the patient supine or seated. If supine, the patient should be positioned near the edge of the table so that his shoulder can extend over it, as arm extension will be necessary in the manipulation. The same is applicable if the patient is seated in a chair with a backrest. If there is no backrest on the chair, it is often necessary to have an assistant support the patient's upper thorax and shoulders; during the extension phase, there is a tendency for the patient to move backward, making the manipulation difficult.



11—58. Step 1 correction of slipped bicipital tendon. Obtain solid purchase on the tendon in direction toward correction.

11—59. Step 2 is to stretch the tendon by extending the arm at the shoulder while maintaining pressure on the tendon toward correction.



11—60. Step 3. Abduct and medially rotate the humerus while maintaining pressure on the tendon toward correction. The tendon slipping into the groove will usually be felt when the maneuver is successful.

To manipulate the tendon back into the groove, the patient's arm should be loosely at his side, with the elbow flexed at 90° and the physician supporting the forearm. Pressure is applied to the tendon toward reduction. With continuous pressure on the tendon in the direction

of positive challenge toward the groove, the arm is slowly extended by the physician moving the elbow posteriorly with his contact on the forearm. When maximum arm extension is reached, the arm is slowly abducted and internally rotated with continued pressure on the tendon. A slipping back of the tendon into the bicipital groove of the humerus is usually observed. The arm is slowly returned to its starting position. Successful manipulation of the tendon into the groove is indicated by the biceps muscle testing strong and negative therapy localization over the tendon. When therapy localizing, one must do so over the groove and in the area where the tendon would slip. In general, the entire shoulder area should be free of positive therapy localization. It may be necessary to remanipulate the tendon on subsequent visits. The tendon should be traction-taped to help prevent recurrence of the slip.

Test the biceps brachii for muscle stretch reaction. If present, the muscle can be lengthened with fascial release or sometimes intermittent cold with stretch technique. The muscle may also be lengthened with various forms of physical therapy.

Elbow

Generally the elbow is considered a single joint, but it is made up of a complex of the humeroulnar, humeroradial, and proximal radioulnar articulations. For a complete elbow examination, all three articulations must be evaluated. If the reader is not familiar with the orthopedic and chiropractic examinations of the elbow, the subject should be pursued elsewhere.[28,29,73,81,112] The discussion here will be primarily limited to the humeroulnar articulation and a few common conditions of the elbow, giving a general overview of applied kinesiology examination of this joint complex.

Examination

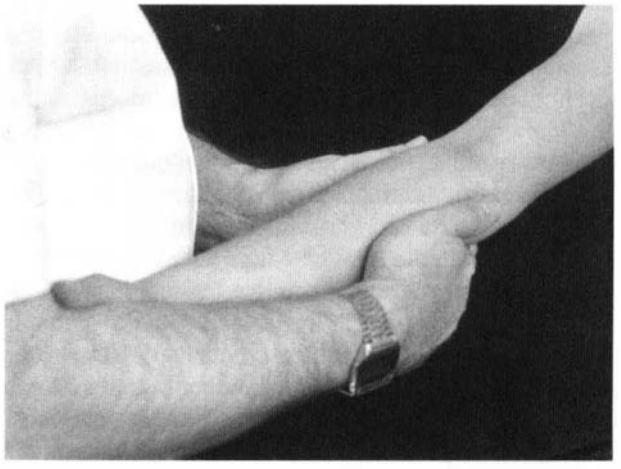
A comprehensive elbow examination includes structure and function from the cervical spine to the fingers. Poorly defined pain at the elbow region is usually referred from above, but it can be antidromal from a carpal tunnel or pisiform hamate syndrome.

Following general inspection and palpation of the elbow articulation, the elbow is moved through its complete range of motion, both passively and against the patient's muscular resistance. Passive movement evaluates for joint pathology; movement against resistance evaluates for muscular involvement.[28]

The elbow is first examined with the passive motions of flexion, extension, pronation, and supination. One evaluates for full range of motion, observing for pain, crepitation, and a hard or soft limitation to the range of motion. Pain during these passive movements indicates joint involvement.

Minor or passive movements are allowed at the humeroulnar articulation by the trochlea's looseness in the trochlear notch, due partly to the presence of the articular cartilage. It is exaggeration of these minor movements that especially promotes elbow dysfunction. The minor or passive movements include abduction and adduction of the ulna on the humerus, and internal and external rotation of the ulnar longitudinal axis in relation to the humeral longitudinal axis.

The abduction-adduction motion can be observed with the patient's elbow close to full extension. The examiner stabilizes the patient's distal humerus with one hand, while the other grasps just above the wrist and applies abduction and adduction forces. Another method is to put the patient's wrist between the physician's arm and chest to stabilize it while he uses both hands — one on either side of the elbow — to apply the abduction and adduction motions to the elbow. An adduction lesion is indicated if there is limited motion in quality and quantity of abduction, and vice versa. Typically, there will be a positive challenge in the direction of limited motion.



11—61. Apply adduction and abduction forces to the elbow to evaluate motion. Challenge is done in the same manner.

Rotation of the ulna about its axis with the humerus can be somewhat evaluated in the passive actions of supination and pronation. A more exact evaluation is done with applied kinesiology challenge, discussed later. The elbow is next tested against resisted movement. Most of the resisted movement tests described can be done simultaneously with manual muscle testing. When muscle dysfunction is present, the standard methods of applied kinesiology treatment are usually effective in eliminating the pain.

With the patient's elbow at approximately 20°, the

physician stabilizes the forearm to provide resistance against the patient's efforts of flexion, extension, supination, and pronation. Stabilization is held against the distal forearm to exclude action of the muscles controlling the wrist. The reason for this will become clear with the examination of the wrist extensors in tennis elbow, discussed later.

Pain on resisted flexion indicates involvement of the biceps or brachialis muscle. Differentiation is made by testing resisted supination, which will produce pain with biceps involvement but not with the brachialis.

The biceps muscle may be involved at its long head, in its belly, at the lower area of the musculotendinous junction, or at its insertion. When at the insertion, there is a distinctive and localized pain that begins in the cubital fossa and radiates down the front of the forearm as far as the wrist. This pain is elicited on resisted flexion and supination. Passive pronation also elicits the pain when the elbow is held in flexion, indicating that tenderness is evoked by pressing the tuberosity of the radius against the ulna; thus, the tendon insertion at the tuberosity is irritated.

If there is no pain on resisted supination but there is on flexion, the brachialis muscle is involved. This happens less frequently than involvement of the biceps brachii.

Pain on resisted extension of the elbow is not as common as on resisted flexion. If pain arises in the shoulder from resisted extension, it is because the triceps is lifting the humeral head into the subacromial bursa; this has the same significance as a subacromial painful arc.

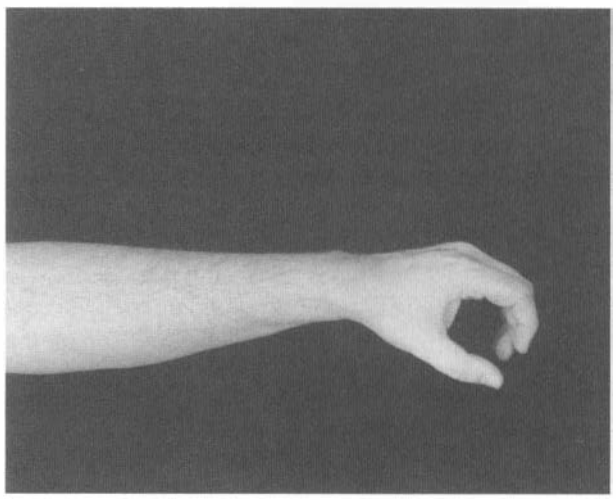
Pain on resisted supination is generally present when there is also pain on resisted flexion, indicating the biceps brachii muscle is involved, as noted earlier. When flexion against resistance is painless, the supinator muscle is probably involved. It is best tested with the elbow in full extension to remove biceps activity. Weakness on supination may be due to entrapment of the posterior interosseous branch of the radial nerve as it passes through the supinator muscle.[2] Cause of the entrapment may be repeated forceful supination, such as screwdriving.

Pain on resisted pronation indicates golfer's elbow. It is located at the origin of the pronator teres muscle, which merges with the common flexor tendon. Confusion may develop because there may be pain from resisted pronation as well as resisted flexion of the wrist. Pain on resisted pronation may be due to the pronator teres syndrome.[150] Dysfunction may be manifested by motor dysfunction of the hand.[88]

Tennis Elbow

Tennis elbow is a lesion located near the elbow in the extensor muscles controlling the wrist. Snijders et al.[140] prefer the term "epicondylalgia lateralis humeri" to "tennis elbow," since they state that only 5% of the people suffering from the condition actually play tennis. On the other hand, Cyriax[28] prefers the term "tennis elbow" because the disturbance is more often in the muscle adjacent to the elbow, rather than at the epicondyle. Reference here will be to tennis elbow, recognizing that the condition can be caused by other activities. The condition can develop from many types of athletic endeavors, as well as screwdriving, wringing laundry, writing, or any other activity that requires strenuous grasping with the hand or fingers. It is basically an overuse condition.

It is easy to recognize trauma in and about the extensor muscles controlling the wrist when forceful wrist extension is a major activity. But what causes the problem in the absence of wrist extension, such as in screwdriving and handwriting? During finger pinching and grasping with the hand by the flexor muscles, the extensors are also active in providing equilibrium of movements; they are synergists in the action of grasping.[5] Without the wrist extensor activity, there would be wrist flexion with gripping by the hand or with finger pinching. The same biomechanical model that explains the origin of tennis elbow in activities other than tennis can be used to explain the creation or aggravation of wrist complaints, because the force into the wrist is a summation of the flexor and extensor muscles during gripping or finger pinching.[140]



11—62. Extensor muscle action is necessary during hand flexion to stabilize the wrist.

According to Cyriax, the reason for tennis elbow lies in the common extensor tendon in nine out of ten patients. When this is the case, movement that hurts the elbow is resisted extension of the wrist.

There is some problem in diagnosing tennis elbow, since there are several conditions that cause pain in the same general area. Remote problems may also be erroneously blamed for this condition. The common age for tennis elbow is 40-60 years. Because of the higher age range, there is a greater frequency of cervical spondylosis that may erroneously be judged as causing the pain. The wrist movements of Cozen's and Mill's tests help differentiate the conditions.

Cozen's test. The patient flexes his elbow to approximately 45°, then extends his wrist and flexes his fingers to the maximum. The physician stabilizes the patient's elbow with one hand, and attempts to force the wrist into flexion with his other hand while the patient resists. Pain in the general area of the lateral epicondyle indicates a positive test.

Mill's test. The starting position is with the patient's elbow, fingers, and wrist completely flexed, with the forearm in pronation. The patient then extends his forearm against resistance. The test is positive when there is pain in the general area of the lateral epicondyle from stretching the extensor and supinator muscles.

Kaplan's test. Kaplan's test compares the strength generated when gripping a hand dynamometer, with and without support added by the physician distal to the origin of the involved muscles. Pain and grip are noted without support, and then the physician firmly encircles the patient's forearm with both hands, about 1-2" below the antecubital fossa. If there is reduced pain and an increased grip with the support, tennis elbow is indicated.

Restricted movement tests. In tennis elbow the pain is located at the elbow; however, there is a full range of motion and otherwise normal joint movement on passive motion. The positive sign is pain at the elbow reproduced on resisted extension, but not resisted flexion, of the wrist. Remember that in the earlier discussion of resisted movement testing, the patient's distal forearm was grasped to avoid movement at the wrist. Now, in testing for tennis elbow, movement of the wrist is included with the resisted movement tests. When testing wrist-resisted movement, the elbow must be kept in full extension. There is also pain on resisted radial deviation, but not on ulnar. The resisted movements described earlier, excluding the wrist extensors, are painless. As noted, the pain develops from forced wrist extension, indicating the lesion is in one of the radial extensor muscles of the wrist. Quite often the tenderness is at — or close to — the epicondyle, indicating the lesion is not in the long radial extensor originating at the supracondylar ridge. The most frequent involvement is at the origin of the extensor carpi radialis brevis muscle. The trauma may be to the musculotendinous junction or at the origin of the tendon at the periosteum. It may be relatively mild microtrauma or significant tears of the tissue. A common problem is that as the tear begins to heal, the patient reactivates the muscles and re-injures the healing area, resulting in scar formation at the muscle's origin. The onset of tennis elbow is usually not with the initial injury. It is with repeated tears that chronic inflammation sets in at the scar and causes symptoms.

Cyriax[28] indicates there will be a spontaneous cure within one year. It results from gradual widening of the gap between the two edges of the site of trauma, causing the gap to be filled with fibrous tissue; finally, it is healed with permanent lengthening. This reduces the strain on that part of the tendon connected to the extensor carpi radialis brevis muscle.

Lymphatic drainage. A common finding in tennis elbow is involvement of the common neurolymphatic reflexes for arm drainage, located over the pectoralis minor muscle and posterior to the areola. The reflex posterior to the areola is also for the adductor muscles. Goodheart[61] postulates that a contributing factor to the development of tennis elbow with tennis is the extensive side-thrusting of the game and activation of the adductor muscles. This stress overloads the lymphatic system, causing an active neurolymphatic reflex. Because of its common association with the arm neurolymphatic reflex, poor lymphatic drainage develops in the arm, making the person more vulnerable to trauma.

The patient should also be evaluated with retrograde lymphatic technique. Drainage from the arm can be specifically tested by elevating the arm and head and testing an associated muscle for weakening (page 541).

Trigger points. A trigger point in the extensor carpi radialis longus often radiates pain to the lateral epicondylar area and may be misdiagnosed as tennis elbow. Trigger points in the extensor carpi ulnaris and extensor carpi radialis brevis contribute to a painful, weak grip. Trigger points are located in the proximal forearm and are distal to, but near, the lateral condyle. Intermittent cold with stretch technique is done with the elbow extended and the wrist flexed to stretch the muscles. The intermittent cold is applied proximal to distal.[146]

Meniscus Injury

A confusing condition to differentially diagnose from tennis elbow is an intra-articular meniscus lock. Mennell[107] states that 10% of the population has an intra-articular meniscus in the elbow joint. It may cause symptoms in a manner similar to those of the menisci of the knee. Mennell describes the maneuver as similar to that of Mill's test. Elbow motion is evaluated from 90° flexion to extension when the forearm is held in full pronation, with full flexion of the wrist. As the motion goes toward full extension and the extensor muscles tighten, the patient feels pain; movement is resisted as if the joint were locking. Tenderness to digital pressure is closely related in both a meniscus injury and a tennis elbow. In the meniscus injury, the pain is directly over the head of the radius. The maximum pain on palpation of tennis elbow is proximal to, and more lateral than, the radial head.

Manipulation

Two types of manipulation are applied to the elbow. One is to adjust the articulation(s) to improve function. The other is to forcefully tear the scar at the extensor carpi radialis common tendon.

Generally the applied kinesiology challenge of the joint is done at the same time the passive movement tests are done in the early phase of examination. The abduction and adduction motion evaluation tests can be followed by testing a previously strong indicator muscle. It

is usually best to test a muscle associated with the joint, such as the biceps or triceps. If muscles associated with the elbow are weak, it is best to challenge to find the direction that causes the muscle to strengthen. The adjustive thrust is done in the direction that causes an associated muscle to strengthen, or opposite the direction that causes a previously strong indicator muscle to weaken.

Manipulation for abduction and adduction subluxations of the elbow can be simply done by stabilizing the wrist and applying the adjustive force to the elbow. Another less traumatic method is to apply constant force of abduction or adduction while the elbow is slowly moved from an extended position to about 90° of flexion, and back to extension again. This gives the patient's articulation the opportunity to find its optimal release point. Properly controlled manipulative thrusts rarely strain the joint. The second method reduces the possibility of traumatizing the joint.

Rotational subluxations between the radius and ulna in their axial plane are most easily challenged by the physician directing medial or lateral force to the tip of the olecranon process with the patient's elbow close

11—63. The important contact in adjusting an axial rotation subluxation is on the olecranon process. Apply the proper rotation at the end of elbow extension.

to full extension, but not fully extended. Immediately after the challenge, test a patient's weak or strong muscle associated with the joint for strengthening or weakening, respectively. Adjust in the direction that caused a weak muscle to strengthen, or opposite the direction that caused a strong muscle to weaken. The patient can be supine, seated, or standing. The physician assumes the same position beside the patient, facing in the same direction. To adjust the patient's right elbow, the physician cradles the elbow in his left palm and contacts the medial olecranon tip with his thumb to correct external rotation. His other hand grasps the distal forearm, putting it into maximum pronation. The manipulative maneuver is to rapidly extend the patient's elbow. At the end of extension, increased pronation is put into the forearm and a lateral thrust is applied to the olecranon tip with the physician's thumb. There will usually be an audible release; however, it is not necessary. To correct internal rotation of the ulna, reverse the procedure; the physician supinates the forearm and contacts the lateral olecranon tip with his middle finger.

Secondary subluxations at the wrist or proximal radioulnar subluxations are often automatically corrected when the humeroulnar subluxation is corrected.

The purpose of Mill's manipulation is to pull apart the two surfaces of the painful scar of the extensor carpi radialis brevis muscle's tendon. Treatment by manipulation is directed toward pulling apart the edges of the tear, thus relieving the painful scar from tension and imitating the mechanism of spontaneous recovery, discussed earlier.

The patient is seated, with the physician standing behind him. The patient's arm is positioned to put maximum stretch on the common extensor tendon, accomplished by adducting the patient's arm and maximally flexing his wrist. As tension is placed on the tendon, the patient slightly flexes his elbow to relieve the tension. It is mandatory that the wrist flexion be maintained throughout the manipulation. With a quick force, the elbow is manipulated to full extension. Since the purpose of the manipulation is to stretch the already painful tendon, the manipulation is painful but brief. Mennell[107] adds full pronation of the forearm to the manipulative position. Four to twelve manipulations may be required. Re-evaluate for full extension and the other criteria prior to each manipulation.

Before manipulating, it is important to determine that the patient has full range of extension and that this type of tennis elbow is present. Manipulation when full extension is not available may cause traumatic arthritis. If there is no scarring of the tendon, this type of forceful manipulation is of no value and may traumatize the joint.

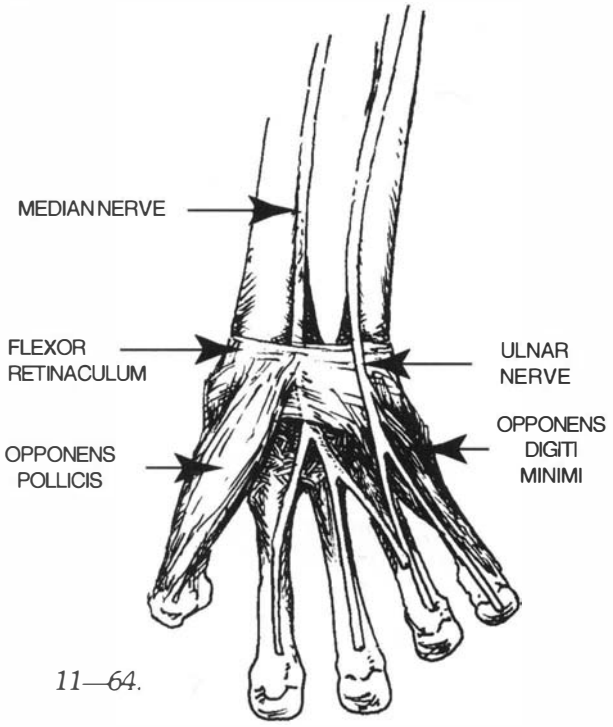
Carpal Tunnel Syndrome

The carpal tunnel syndrome results from median nerve entrapment as it passes through a tunnel in the wrist. The tunnel is formed by a strong flexor retinaculum bridging the volar surface of the carpals and the distal ends of the radius and ulna. This is the most common site for nerve entrapment in the upper extremity.

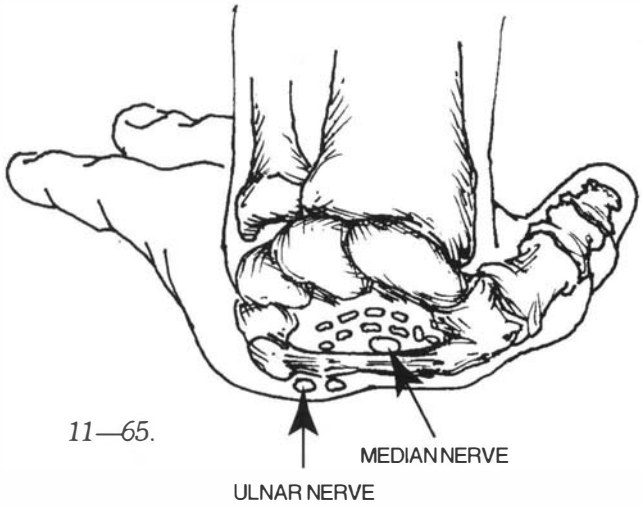
The carpal tunnel is considered by most to be formed by the volar surface of the carpal bones bridged by the transverse carpal ligament. This ligament attaches medially to the pisiform and hamulus of the hamate, and

laterally with a superficial layer to the tubercles of the scaphoid and trapezium; there is also a deep layer to the medial lip of the trapezium.

The superficial part of the flexor retinaculum is the palmar carpal ligament. Distally, it blends with the transverse carpal ligament; proximally, it attaches medially and laterally to the styloid processes of the ulna and radius. The palmar carpal ligament is not considered part of the carpal tunnel by many authorities. Applied kinesiology clinical evidence indicates that it plays a significant role in carpal tunnel entrapment. This is supported by the improvement of median nerve function when a subluxation that separates the distal radioulnar articulation is corrected.



11—64.



11—65.

Passing through the carpal tunnel are the tendons of the flexor digitorum profundus and superficialis, flexor pollicis longus, and flexor carpi radialis, as well as the median nerve. Superficial to the transverse carpal ligament are the ulnar nerve and artery. Within the carpal tunnel there is little latitude for alteration due to structural change, whether from trauma, neoplasms, or other factors.

Symptoms and Etiology

A carpal tunnel syndrome may have a history of violent trauma, such as stopping a fall with an extended hand. The trauma to the hand may be insidious, such as wrist extension when waiters/waitresses hold heavy trays of dishes over their shoulders, or repeatedly strike swinging doors with the flat of the hand. Repeated motion such as working on a computer keyboard has recently become a common reason for a workers' compensation claim.

Other types of trauma occur from activities such as a mechanic straining his wrist when pulling on a wrench, a carpenter using a heavy hammer, a driver hanging his wrist over the top edge of the steering wheel for prolonged periods, and from a person leaning on his hand when scrubbing a floor. Determining and eliminating such activity is often very important in obtaining permanent results from carpal tunnel syndrome correction. Improper use of the wrist may cause the condition to return.

Vitamin B_6 deficiency has been found to cause the symptoms of carpal tunnel syndrome.[45,136] In a double-blind study, Wolaniuk et al.[154] were able to identify the improvement in patients receiving pyridoxine treatment for carpal tunnel syndrome as opposed to those receiving a placebo. "In all patients taking pyridoxine, decreases in motor latency and increases in the conduction of the velocity of the median nerve were observed. It is well known that the most sensitive electrodiagnostic measurements for confirming the presence of the carpal tunnel syndrome are the distal sensory and motor latency periods."

When vitamin B_6 deficiency is part of the carpal tunnel complex, the nutrient should be added regardless of how the carpal tunnel syndrome is treated. Applied kinesiology methods or surgery may relieve the symptoms of the syndrome; it does not relieve the B_6 deficiency.[46]

Other symptoms may be present in B_6 deficiency, such as edema in the feet and ankles sufficient to warrant a larger shoe size. There may be pain in the elbows and knees. Other conditions may be improved when the carpal tunnel syndrome is treated with pyridoxine. One study showed that after the use of pyridoxine, paraarticular tissues of the knees appeared more pliable and elbow pain was reduced. The patients stated they could "...squat better and get up and down better."[44]

Adequate dosage of vitamin B_6 must be given, but overdosage must be avoided. In addition, it may be necessary to continue a maintenance dosage to avoid recurrence of the condition. Folkers et al.[49] demonstrated some improvement in symptoms with a dosage of 2 mg per day

of pyridoxine for eleven weeks. When the dosage was increased to 100 mg per day for twelve weeks, the condition was corrected. In this single patient of their larger study, a placebo was given after the twelve weeks. The condition returned within seven weeks. It was again corrected with the administration of 100 mg pyridoxine per day.

The use of vitamin B_6 as a treatment for carpal tunnel syndrome is put in excellent perspective by an extensive study of patients (n1075) treated conservatively. In this study, Kasdan and Janes[85] found that conservative care provided only 14.3% satisfactory alleviation of symptoms prior to the use of B_6. With its addition, the percentage improved to 68%. They found that a dosage of 100 mg twice daily was enough to obtain results. After noticeable alleviation of symptoms, with the condition apparently stable, the dosage was reduced to 50-100 mg per day for maintenance. Conservative care prior to the addition of vitamin B_6 consisted of wrist splints, job changes, anti-inflammatory agents, and steroid injections. Treatment with vitamin B_6 was either that alone, or with wrist splints and/or a job change. Kasdan and Janes state that they seldom use steroid injections in treating carpal tunnel syndrome because of lack of long-term success with the procedure, and increasing reluctance on the part of their patients to receive the injections.

Vitamin B_6 must be given long enough to obtain results, but it must be closely monitored during this period and also while patients are on a maintenance dose following successful treatment. Using the specific activity of the enzyme erythrocyte glutamic oxaloacetic transaminase (EGOT), as determined in the laboratory, Ellis[43] has found that it takes four weeks for vitamin B_6 deficiency to be corrected with a dosage of 100-200 mg of pyridoxine daily; it takes twelve weeks of therapy at this dosage level to improve or completely relieve the signs and symptoms of carpal tunnel syndrome.

In a large study of pyridoxine overdose neuropathy syndrome, Dalton and Dalton[30] found a raised serum B_6 level present in 172 women, of whom 60% had neurological symptoms that disappeared when B_6 was withdrawn. The mean dosage taken of B_6 by the women with neurological symptoms was 117 +/- 92 mg compared with 116.2 +/- 66 mg in the control group. There was a significant difference ($p<0.01$) in the average duration of ingestion of B_6 in the neurotoxic group of 2.9 +/- 1.9 years compared with 1.6 +/- 2.1 years in the controls.

Paradoxically, many of the symptoms that developed in the neurotoxic group parallel those of persons successfully treated with B_6 when it is deficient. Symptoms included paresthesia, which was more noticeable at night and limited to the extremities, with the upper limbs three times more commonly affected than the lower ones. Hyperesthesia, described as various types of sensation, predominated in the extremities with stocking glove distribution. There was also hypersensitivity in the vulva, vagina, or nipple. Muscle weakness caused difficulty in typing, playing the piano, eating pastry, and maintaining a grip on a steering wheel — many of the same symptoms present in carpal tunnel syndrome. There was fasciculation described as twitching, jangling, restlessness, fidgeting, and movements within.

There were no withdrawal symptoms when B_6 was discontinued. All patients recovered completely; however, the dosage taken by subjects in this study was less than that in other studies where complete recovery did not occur.

Vitamin B_6 occurs in nature as pyridoxine, pyridoxal, and pyridoxamine. These must be converted to the coenzyme form of pyridoxal-5-phosphate (P-5-P), which is accomplished by phosphorilation and oxidation. In order for this to take place, magnesium, zinc, riboflavin, and phosphorus must be present. It may be that individuals who become toxic from vitamin B_6 are unable to convert it to the usable form of P-5-P. Schmitt[132] has developed an effective method for evaluating vitamin B_6 and its conversion to P-5-P using applied kinesiology testing techniques.

There may be a history of Colles' or other types of fracture, dislocation of a carpal bone, or a neoplasm. All these space-occupying factors must be differentially diagnosed so that proper procedures can be instituted as soon as possible. Decompression surgery may be required. In the absence of this type of involvement, applied kinesiology conservative management is highly successful in correcting the carpal tunnel syndrome.

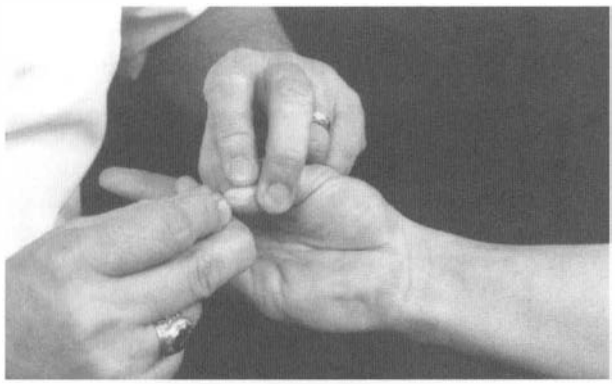
One of the most common symptoms of a carpal tunnel syndrome is nocturnal pain that interferes with sleep. A common description given by the patient is that his hand must be held over the edge of the bed, or it requires recurrent rubbing during the night in order for him to sleep. There is often a complaint of hand weakness. If the condition is chronic, atrophy may be observed at the opponens pollicis and flexor pollicis brevis muscles.

Often there is pain involved, usually of radial distribution in the hand. It covers the palmar surface of the thumb, index, middle, and half the ring finger. When conducting a neurologic evaluation, one should remember that there may be communication between the radial and ulnar nerves (Martin-Gruber connection).

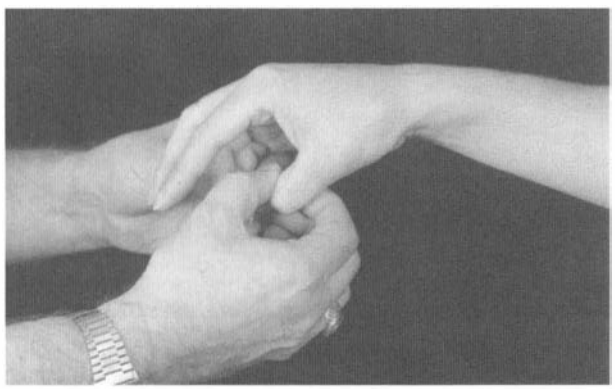
Differential Diagnosis

A carpal tunnel syndrome must be differentially diagnosed from all other peripheral nerve entrapments. These occur from the radix of the nerve at the intervertebral foramen down to the intermetacarpal tunnel. Manual testing of the muscles usually innervated by the median nerve below the carpal tunnel, compared with those innervated by the median nerve above the tunnel and by other nerves, gives differential evidence; however, it must be remembered that there are congenital variances that can change these patterns and cause confusion. The combination of manual muscle testing with the applied kinesiology procedures of challenge and therapy localization helps locate the entrapment.

In a hand with the usual medial and ulnar innervation of muscles, one would expect to find weakness of the opponens pollicis and abductor pollicis brevis on manual muscle testing in a carpal tunnel syndrome. The long muscles innervated by the median nerve proximal to the carpal tunnel, such as the flexor pollicis longus, flexor digitorum superficialis, and radial side of the flexor digitorum profundus, should test strong. The muscles innervated by the ulnar nerve, such as the opponens digiti minimi, flexor digiti minimi, and the adductor pollicis, will test strong because the ulnar nerve is superficial to the carpal tunnel and not involved in its entrapment syndrome.



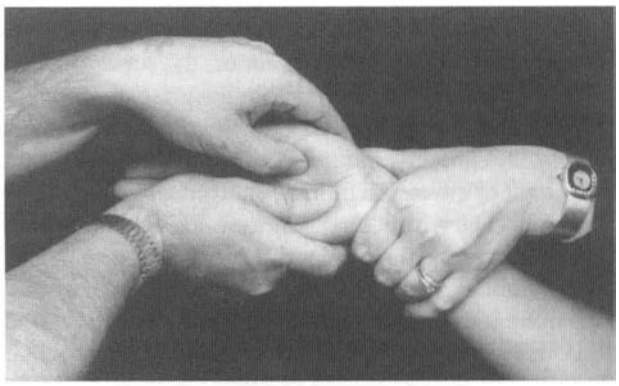
11—66. For gross screening, test the patient's ability to hold the thumb and little finger together.



11—67. Test in different position. Some may test weak, while others test strong.

When the pattern of muscle weakness indicates a carpal tunnel syndrome, confirmation can usually be obtained by challenge and therapy localization to the carpal tunnel. Challenge is accomplished by having the patient or support person give manual support to the carpal tunnel at the distal ends of the radius and ulna. Force is applied to approximate the bones and form an increased carpal arch. With this supportive challenge, the muscles weak as a result of the carpal tunnel syndrome should test strong.

Therapy localization to some aspect of the carpals,



11—68. A structural fault is indicated when support to the distal radius and ulna and/or the carpal arch strengthens a muscle.

ligaments, or the distal area of the radius and ulna will also cause the weak associated muscle to regain strength on testing, in most cases.

Median nerve peripheral nerve entrapment can develop at many locations from the cervical spine down to the intermetacarpal tunnel. Included are the various forms of thoracic outlet syndrome, pronator teres syndrome, lacertus fibrosis syndrome, flexor digitorum superficialis syndrome, and anterior interosseous nerve syndrome.[150] When there are multiple areas of entrapment it is called "double crush," which makes the distal nerve more susceptible to entrapment.

The pronator quadratus muscle often appears to be involved with a carpal tunnel syndrome. Entrapment of the median nerve at a higher level may be the reason for the pronator quadratus involvement allowing a secondary carpal tunnel entrapment to develop due to double crush. The pronator quadratus may be involved from trauma or repetitive motion. The working hypothesis is that the pronator quadratus weakens from improper nerve supply due to proximal peripheral nerve entrapment or trauma to its muscle proprioceptors, thus interfering with its action. The muscle proprioceptors — Golgi tendon organs and neuromuscular spindle cells — are usually traumatized by overcontraction of the muscle. An example of this is a mechanic twisting hard or repeatedly on a

11—69. The pronator quadratus gives support to the distal radius and ulna.

screwdriver. Weakness of this muscle allows the radius and ulna to separate, causing tension on the superficial aspects of the flexor retinaculum. In this case, therapy localization to specific aspects of the pronator quadratus will usually cause the intrinsic muscles of the hand, weak as a result of the carpal tunnel syndrome, to strengthen.

Testing the muscles described and evaluating them with challenge and therapy localization are effective means for differentiating a functional carpal tunnel syndrome. When the tests are positive, the carpal tunnel syndrome responds rapidly to the applied kinesiology approach. If there is a space-occupying problem, such as a tumor, tenosynovitis, rheumatoid arthritis, or edema, there is poor response to AK testing. Additional methods of diagnosis are necessary.

Conditions that cause edema can cause nerve entrapment at the carpal tunnel, especially when the nerve is close to compromise and the edema provides the last bit of pressure necessary to produce symptoms. An example is the fluid buildup from the toxicity of an ileocecal valve syndrome. The body's natural reaction to toxicity is to retain water to dilute the toxicity. In this case, correction of the ileocecal valve syndrome provides relief within a short time.

Treatment

Goodheart[57] introduced carpal tunnel syndrome treatment into applied kinesiology in 1967. The therapeutic approach has been very effective when a correct diagnosis is made. Most cases that don't respond fail to do so because the median nerve is entrapped at a higher level. In some instances there is entrapment at both the carpal tunnel and a higher level. Additionally, failure may result from lack of recognition of a neoplasm or other space-occupying lesion.

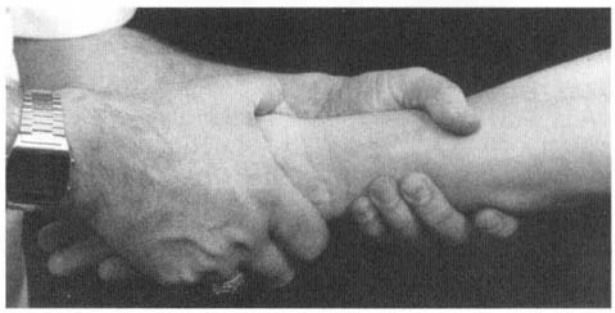
Most cases of carpal tunnel syndrome are a result of subluxations of the carpals and the distal radioulnar articulation. This is usually confirmed by challenge and therapy localization, which provide justification for treatment. If there is no supportive evidence of the condition's functional nature, effort should be directed toward differentiating the condition from neoplasms, tenosynovitis, hypothyroidism, and secondary edema as a result of ileocecal valve syndrome, among other conditions.

Subluxations include separation of the radioulnar articulation, or of carpal bone malposition or fixation. Challenge is done by holding the bones toward a suspected direction of correction and re-testing muscles weak as a result of the condition. Usually a weak opponens pollicis muscle will strengthen when the bones are held in the correct direction.

If the subluxation is separation of the radius and ulna, the pronator quadratus muscle will usually be weak. A simple way to test for this is to therapy localize at its origin and insertion or in the belly for Golgi tendon organ or neuromuscular spindle cell dysfunction. If present, treat the Golgi tendon organs with pressure toward the belly and the neuromuscular spindle cell away from the center of the belly before attempting to manipulate the radius and ulna.

If the carpal bones are subluxated, manipulate them in the direction of challenge that caused the involved muscle(s) to strengthen.

To adjust the radius and ulna, approximate by gripping around their distal ends with one hand while the patient's hand is tractioned away from the radius and ulna with the other hand. After separating the wrist from the radius and ulna in this manner, the physician uses his second hand to reinforce contact at the radius and ulna, making quick, approximating force around the bones. There will often be an audible release, but it is not necessary. Evidence of a successful adjustment is strengthening of the opponens pollicis and abductor pollicis brevis observed by manual muscle testing.

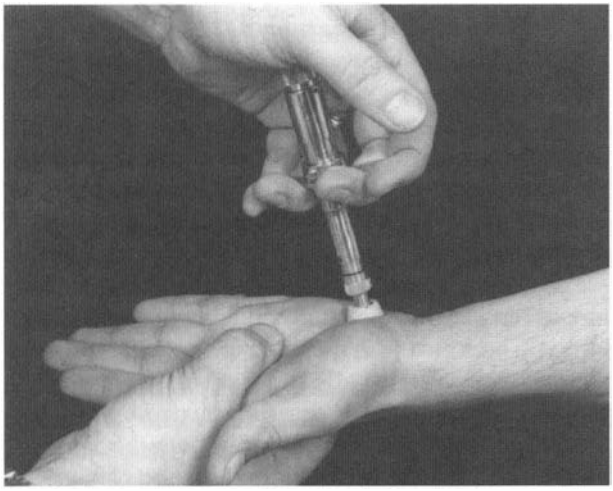


11—70. Step 1: separate by traction.

11—71. Step 2: correct by approximation.

Another method of radius and ulna manipulation is to use a toggle recoil adjustment with a dropping mechanism in the chiropractic table. The patient's wrist is placed on the table with the medial edge of the arm in contact. With a pisiform contact, the physician adjusts the radius and ulna for approximation.

If there are carpal bone subluxations, they are adjusted in the direction that strengthens the weak associated muscles. This can be accomplished with various types of thumb or pisiform contacts that are effective for the physician. Another method of adjusting the individual carpal bones is to use the activator instrument.[1] The contact tip is held on the carpal bone and the instrument is aligned in the vector of challenge that created the great-



11—72. Use of the activator instrument to correct subluxations in the carpal arch.

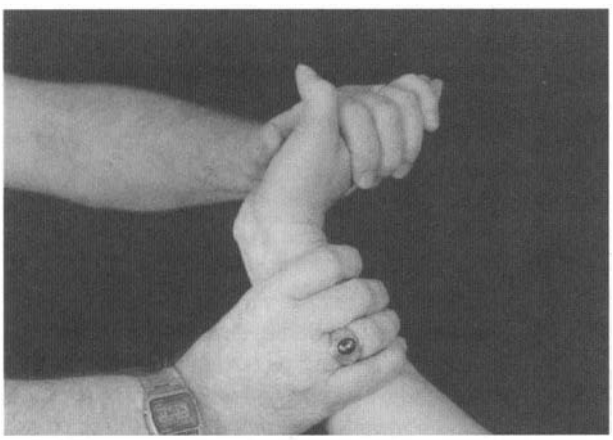
est strength. The instrument is then used to direct percussive force into the carpal bone.

After correction has been obtained and the muscles test strong, determine if the correction may be easily lost. This is done by putting the patient's wrist through extreme ranges of motion. Hyperextension is the motion that is most likely to cause the correction to be lost. If the syndrome returns, it is necessary to use a wrist support for approximately two weeks so that the structure can heal.

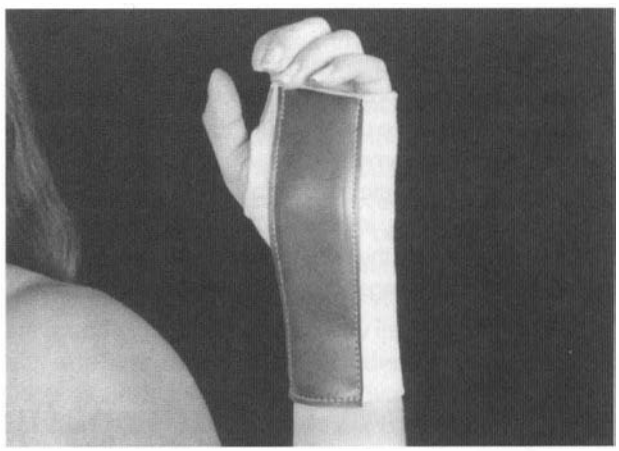
Early in applied kinesiology treatment of the carpal tunnel syndrome, it was nearly always necessary to support the wrist. Since evaluation and treatment of the pronator quadratus muscle in the carpal tunnel syndrome has been added,[60] it is often found that no additional support is needed.

The best support is one that encircles the wrist and hand and has a metal or plastic strip to limit wrist motion. Another type of support wraps around the wrist to maintain approximation of the radius and ulna. It may be elastic, leather, or heavy canvas. A particularly good one is manufactured for treatment of tennis elbow (epicondylitis); it can be applied to the wrist. Care must be taken that the wrist support is not so tight that it impedes circulation.

If repeated correction is necessary, nutritional support may be indicated, especially if the pronator quadratus muscle weakens again and requires further treatment to its proprioceptors. Typically, raw bone concentrate is indicated. It is applicable for both ligamentous support and support to the muscle proprioceptors. Usually three tablets per day, chewed, provide adequate support.



11—73. Move the wrist through its full range of motion to determine if correction is easily lost.



11—74. Wrist support to aid in maintenance of carpal tunnel correction.

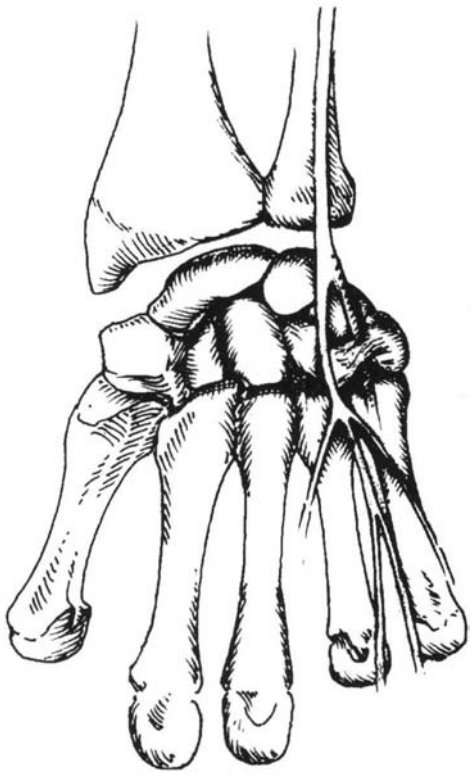
Pisiform Hamate Syndrome

The most common peripheral nerve entrapment of the ulnar nerve affecting the hand is at the pisiform hamate tunnel. The symptoms of ulnar entrapment at the wrist-hand will generally include pain, and perhaps tingling and numbness of ulnar distribution. The muscles innervated by the ulnar nerve distal to the wrist may be weak, and the patient may be unable to perform tasks requiring dexterity. There may be inability to press the tips of the thumb and little finger together forcefully from weakness of the adductor pollicis and 1st dorsal interosseous muscles.

The most common cause of ulnar neuropathy below the wrist is trauma to the hand. This may result from occupational activities, such as operating a pneumatic hammer, using a staple gun, pressing hard on various types of levers, using pliers or hammers, and other factors.

Diagnosis

Entrapment of the ulnar nerve at the wrist and hand must first be differentially diagnosed from the more proximal entrapment neuropathies. When the involvement is



11—75. Ulnar nerve at the pisiform hamate tunnel.

limited to the hand, there will be a normal muscle test of the flexor digitorum profundus muscle in the 4th and 5th fingers, which receive ulnar innervation but proximal to the wrist. If the entrapment is due to a subluxation, usually of the pisiform or hamate but possibly of other carpal bones, challenge and therapy localization will cause weak associated muscles to regain their strength, as observed on manual muscle testing. Findings at the elbow and shoulder outlet will be negative.

Treatment

Most involvements of the ulnar nerve below the wrist respond to applied kinesiology techniques of examination and correction. It is usually necessary to adjust the pisiform or hamate or other carpal bones to re-establish normal function of the structure. This is accomplished in a manner similar to that described for carpal adjustment in the carpal tunnel syndrome. The carpal bones are challenged until the vector of force is found that improves muscle function, as observed on manual muscle testing. The structure is then manipulated in that direction, either by various adjusting techniques or with an activator instrument.[1] It is occasionally necessary to add tape support to, and possibly padding over, the damaged area to enhance healing. Nutritional support of raw bone nucleoprotein extract or concentrate may be necessary.

11—76. Challenge the carpal bones to find direction that restores strength to weak muscles and adjust accordingly.

Idiopathic Scoliosis

The literature abounds with papers on the etiology, treatment, and natural course of scoliosis. There are many types of scoliosis appearing within different age ranges. The two peaks of onset are in infancy and adolescence. Least susceptible are the ages of 7-8 years. The spine grows slowly from about 7-10 years of age. During this period the increase of scoliotic angle ranges from 3-5° a year. When the spine grows more rapidly between the ages of 10-15, the scoliotic deformity worsens about 1° per month. When vertebral growth is completed, as indicated by the cessation of an increase in sitting height, the scoliotic deformity becomes essentially static[127]; however, it is possible for scoliosis to develop in the adult or elderly individual despite having had a straight, normal spine after cessation of growth. Infantile idiopathic scoliosis is common in Europe but rare in North America, where there is an increased incidence of adolescent idiopathic scoliosis. Scoliosis developing after 3 years of age rarely spontaneously disappears. "...In infants 90% of idiopathic structural curves appearing in the first year disappear spontaneously."[96] There is an increased frequency of idiopathic scoliosis in children born to women over 30 years of age.[84] A hereditary factor has been established in scoliosis, especially from father to daughter.[27]

This discussion is directed toward idiopathic scoliosis, the most common of all forms of lateral deviation of the spine.[110] When the mechanism of the condition is clear, such as congenital anomalies or scoliosis developing in the presence of another condition, e.g., cerebral palsy and the muscular dystrophies, applied kinesiology treatment is usually not applicable. Much research has been directed toward understanding idiopathic scoliosis, but finding the answer to its etiology has been an enigma, as exemplified by James'[84] statement, "Few of us even

have a hypothesis of causation. I certainly have not, though I have been observing this disease for twenty years and must have seen some 2,000 cases." Research has been directed toward heredity, postural equilibrium, the central nervous system, the intervertebral disc, growth rate, muscle function, asymmetrical vertebral growth rate, and bone histology.[110]

Idiopathic scoliosis has been studied extensively in chiropractic. Barge[10] reviews some of the chiropractic concepts, heel lifts, and manipulation for its treatment. Aspegren and Cox[8] report on the use of manipulation and transcutaneous neuromuscular stimulation in the treatment of idiopathic scoliosis. Although there has been much research directed toward finding the etiology of idiopathic scoliosis, a single cause remains unknown.

Some basic principles in applied kinesiology examination and treatment appear to supply answers to some of the questions raised by idiopathic scoliosis. It appears there is no single cause of the condition; clinical evidence indicates there are several contributing factors found by AK examination that must be corrected to stop the progress of and improve the scoliosis.

Applied kinesiology treatment of idiopathic scoliosis must be put in proper perspective. A developing scoliosis must be recognized early and proper treatment initiated. Late efforts in correcting scoliosis, when there is wedging of the vertebrae and distortion of the ribs, result in minimal — if any — success.[18] The conservative AK approach may be the primary treatment in early cases of idiopathic scoliosis. In more advanced cases AK methods may be combined with other recognized treatment, such as electrical stimulation or bracing for optimal effectiveness. Until objective studies are done to compare methods of treatment, it is best to consider all therapeutic options for each individual case.

Although the definitive cause of idiopathic scoliosis has not been strongly established, many have concluded that muscle imbalance is a primary etiology. This appears obvious in conditions such as cerebral palsy and the muscular dystrophies, where James[84] concluded that scoliosis may be caused by unequal pull of unequal muscles. He goes on to state, "As in so many conditions, the answer to scoliosis will almost certainly be simple and obvious — with hindsight." On the other hand, Farkas[48] states, "Idiopathic scoliosis is probably the most complex disease medicine knows." As more knowledge is developed in the applied kinesiology approach to idiopathic scoliosis, it appears that James' conclusion of simple and obvious — with hindsight — will be correct.

During twenty-five years of observing the condition, Risser[127] noted muscle imbalance in his scoliosis patients. He developed tests for the abdominal, pectoral, and serratus anticus muscles, among others. He noted the weak pectoral and abdominal muscles on the side of convexity and stated, "In my opinion weakness of these muscles does not cause the curve. They are merely what I call 'clue' muscles which indicate weakness of the spinal muscles posteriorly since both muscle groups are supplied by the same segmental nerves." Although he routinely examined for weak muscles, he had no way to treat them. He believed that most scoliosis was actually due to undiagnosed poliomyelitis, which was a relatively common view in the 1950s and 60s. Unfortunately, idiopathic scoliosis continues with a similar incidence after poliomyelitis has been controlled.

The applied kinesiology approach is to normalize the nervous system's control of muscle function and balance. There are numerous examination and treatment methods for accomplishing this. Local treatment of the muscles may include neuromuscular spindle cell and Golgi tendon manipulation, fascial release, trigger points, and origin and insertion technique, among other methods. Techniques to organize the dura mater with the bony vertebral column are often needed, as is the correction of neurologic disorganization.

One of the early methods used in applied kinesiology for treating idiopathic scoliosis was manipulation of the muscle proprioceptors, usually neuromuscular spindle cells. Using vibratory electromyography, Hoogmartens and Basmajian[80] studied the stretch sensitivity of neuromuscular spindle cells in the paraspinal muscles of subjects with minor or major degrees of scoliosis. They state, "Our general but basic conclusion is that idiopathic scoliosis may be caused by an asymmetrical stretch sensitivity of the spindles in the left and right deep spinal muscles. This study provides several arguments for stretch hypersensitivity at the concave side of the thoracic curve."

Manual muscle testing reveals weakness on the convex side of the curve; however, electromyography consistently shows more activity on that side.[3,82] Zuk[159] measured the paraspinal and external oblique abdominal muscles in idiopathic scoliosis with electromyography. He found higher electrical activity in muscles on the convex side of the curve. He concluded that these muscles were more active because they were weak; they were compensating for their weakness by high activity in an attempt to correct the curve. His basis for this theory is that a large, strong muscle will have less frequent and smaller potential than a smaller muscle lifting the same weight.

Applied kinesiology examination reveals neurologic disorganization in nearly all cases of idiopathic scoliosis. A common cause of neurologic disorganization is disturbance in the stomatognathic system. The entire stomatognathic system is often involved in idiopathic scoliosis. Magoun[101] makes a strong case for disturbance in the cranial-sacral primary respiratory mechanism being the common factor present in all cases of idiopathic scoliosis. James,[84] in his detailed description of the condition, shows pictures of plagiocephaly in infants, which is remarkable facial and cranial distortion. He states that it "...is present in all babies with infantile idiopathic scoliosis, even though resolving later." The plagiocephaly is not present in the newborn infant, but develops within the

next few days or weeks after birth; thus, it cannot result from intrauterine position.

Malocclusion is a common condition accompanying idiopathic scoliosis. It is so common that Blount[18] recommends that the patient be examined by an orthodontist to document the malocclusion before starting Milwaukee brace treatment, so that the brace will not be blamed for any jaw or bite deformity. More than half the persons who have scoliosis also have malocclusion of the teeth.[127]

Cranial faults or upper cervical subluxations or fixations appear to cause conflicting afferent supply from the labyrinthine receptors, visual righting, and head-on-neck reflexes. Sahlstrand and Petruson[131] compared labyrinthine dysfunction in children with idiopathic scoliosis with children who have normal spines. In the scoliosis group there was a high percentage ($p < 0.001$) of spontaneous or positional nystagmus, indicating labyrinthine dysfunction. This study implies the possibility of an asymmetric postural tone in the trunk during conditions when the labyrinths are exposed to stimulation. The postural nystagmus indicates that the imbalance may be present even at rest. They formed no conclusion as to whether the vestibular imbalance might be a contributing factor to adolescent idiopathic scoliosis, or whether the vestibular findings are secondary to the spine's deformity.

The head-on-neck reflexes are located in the ligaments of the upper three cervical vertebrae.[106] These reflexes are intimately integrated with the muscles of mastication,[52] which must be functioning in a balanced state to maintain harmony within the stomatognathic system. The tonic neck reflex is the head-on-neck reflex prior to its maturing.[64] The asymmetrical tonic neck reflex is seen in infants up to approximately six months of age, and in children with neuromotor dysfunctions. Connolly and Michael[25] have found a significant ($p < .05$) correlation between the asymmetrical tonic neck reflex and evidence of scoliosis, indicated by standard orthopedic screening methods in children between the ages of 10-14 years. The asymmetrical tonic neck reflex has also been demonstrated in healthy adults under stress[75] and in children with learning disabilities, or those classified as having minimum brain dysfunction. These are conditions that applied kinesiology routinely finds in neurologic disorganization, discussed in Chapter 5.

Organization within the nervous system of scoliotic subjects was studied by Cook et al.[26] They found that the upper limbs of scoliotic subjects perform inferiorly on a proprioceptive basis to a normal control group. This failure of body organization appears to be throughout the body of scoliotic subjects, and correlates with the disorganization found with applied kinesiology testing. They state, "Awareness of human body position in space is a highly developed sense resulting from vestibular, visual, and proprioceptive neural pathway input." The question arises whether the neurologic deficit in the upper extremity of scoliotic subjects is responsible for the scoliosis or results from it. Because this study was done on the upper extremity, it suggests that the deficit occurs above the curve level. Their conclusion is that the deficit precedes the deformity rather than results from it.

Farkas[47] observed on moving pictures of children with idiopathic scoliosis that they showed a marked increase in abnormal gait. His analysis is that scoliosis might be caused by the compression and rotation forces of the abnormal gait. He states "...that these forces are enormously increased in gait. It might be stated with some certainty that the mechanical factor bringing about all forms of acquired scoliosis is gait." The presence of disturbed gait in neurologic disorganization, as observed in applied kinesiology, is easily recognized. In addition to the improper temporal pattern of the extremity muscles of gait, it should be obvious that there is an improper temporal pattern of the individual muscles of the sacrospinalis.

Examination

The examination follows the usual approach of evaluating for congenital anomalies and neuromuscular conditions as a cause of scoliosis. The applied kinesiology portion of the examination provides additional information about the functional nature of the scoliosis. It is imperative that repeated measurements of height and curvature angle be made to evaluate improvement, lack of improvement, or worsening of the condition. Low-dose x-ray techniques can be used to minimize exposure to the breasts and gonads.[7] These projections provide adequate visualization to determine degrees of angulation, rotation, and exclusion of developmental defects. The low-dose techniques provide significant reduction of radiation, even if small areas of the spine must be re-examined for more detail by other techniques.

The first phase of examination documents the extent of the scoliosis. This is done by the standard methods of x-ray, generally using Cobb's method of measurement. The height of the patient should be meticulously measured. It is an evaluation that can be immediately re-measured to determine treatment effectiveness. With proper applied kinesiology treatment, there is usually an immediate increase in height as strain and rotation are removed from the patient's scoliosis. In addition to determining treatment effectiveness, one can determine whether the correction is holding. It is not unusual to observe an increased height of 1/4" to 1/2" from treatment, but it may be rapidly lost as the patient walks and otherwise moves about. In this case the corrective efforts are appropriate but some other factor is present, causing the loss of improvement.

Another effective method of immediately evaluating scoliosis treatment effectiveness is bilateral weight measurement. Normally a right-handed individual will be 5-10 pounds heavier on the right, due to heavier muscular development and the liver being on that side. A left-handed individual will normally be 0-5 pounds heavier

on the right. Seventy-five percent of the total body weight should be on the heels. Zatkin[158] found a higher percentage of categories I and II and upper cervical fixations in individuals who varied from the normal weight distribution. As the scoliosis improves, weight balance improves.[10] The improvement in weight balance may result from a double curve developing from a C curve, providing better balance but not necessarily improvement in the scoliosis. Almost anything treated in applied kinesiology can contribute to idiopathic scoliosis. It is outside the scope of this text to go over all therapeutic approaches that may be required in treating the condition.

First — and probably foremost — is to evaluate for and correct neurologic disorganization, including all aspects of the stomatognathic system and its relationship with the spine and pelvis. Evaluate the individual using the standard methods of therapy localization to KI 27 and ocular lock with the patient supine, sitting, and standing. Examine for need of equilibrium synchronization technique that often reveals disturbance in the cranial-sacral-pelvic mechanism that might otherwise go undetected. Normal function of the hyoid muscle proprioceptors is important in organization between the two sides of the body. Routinely evaluate them when examining the stomatognathic system.

Careful attention should be given to mandibular movement. The muscles of mastication must be organized within the stomatognathic system, and the patient should be evaluated for malocclusion. It may be necessary to consult a dentist regarding the malocclusion.

In difficult cases the answer will often be found in hidden neurologic disorganization. Evaluate the patient as he performs different activities, as indicated in Chapter 5. Along with neurologic disorganization, evaluate for proper right and left brain function; correct as needed.

All aspects of the gait mechanism may be involved in idiopathic scoliosis. First evaluate the six patterns of gait muscles with the patient supine; correct as indicated. Next, evaluate the shoulder girdle, neck and head gait pattern while the patient stands. Test for PiLUS muscle function and corrrect as indicated. Finally, have the patient walk and run; re-evaluate for neurologic disorganization or other dysfunction that may develop from the activity.

Foot dysfunction is often the cause of neurologic disorganization, especially when there is gait dysfunction. Imbalance of the psoas major is often present with foot problems. The psoas should be evaluated for length, as it is often hypertonic and shortened on the side opposite psoas muscle weakness. Michele[109] has given an extensive presentation on the iliopsoas and its relation to developmental anomalies in man. Its imbalance may be a perpetuating factor in scoliosis, if not the initial cause. In addition to strengthening the weak psoas muscle, it may be necessary to use fascial release, strain/counterstrain, trigger point therapy, and muscle stretching techniques on the contralateral muscle to obtain balance.

A common finding in scoliosis is positive aspects of the PRYT technique. Most common is one or more of the three yaw patterns.

The individual muscles of the sacrospinalis should be evaluated with special muscle tests developed by Beardall.[15,16] In addition, the quadratus lumborum and oblique abdominal muscles are often involved. The dural tension techniques of applied kinesiology, such as dural torque and filum terminale cephalad lift, are often required in idiopathic scoliosis. The rapid progression of the scoliosis is during periods of growth spurt. Throughout growth the spinal column progresses faster than the spinal cord; thus the conus medullaris is located at the 2nd lumbar vertebra. It may well be that the scoliosis develops to release tension on the dura mater. Breig,[21] known for his spinal surgical procedures designed to relieve tension on the dura mater, states, "Scoliosis alone rarely generates neurological manifestations." He goes on to state, "In most cases of *true* scoliosis, the reversal of the curve above and below the main deformity prevents pathological tension developing in the pons-cord track, as there is no increase in the total length of the canal. However, on applying therapeutic traction to the spine in order to straighten the scoliotic curvatures, pathological tension may be set up in the soft tissues in the spinal canal that can lead to neurological deficit." When applied kinesiology dural tension techniques are applied, one often sees a considerable increase in the patient's height, indicating that release of dural tension has allowed the scoliosis to straighten.

Throughout the applied kinesiology examination, continue to correlate with the usual chiropractic examination for subluxations, fixations, and pelvic distortions, especially leveling of the head and sacral base. It may be necessary to use orthotics or heel lifts for foot correction and adaptation to an anatomically short leg.

CHAPTER 11 — REFERENCES

1. Activator Methods Seminar Sales, Inc., 3714 E. Indian School Rd., Phoenix, AZ 85018. Telephone: 1-800-598-0224.
2. Aguayo, A.J., "Neuropathy due to compression and entrapment," in *Peripheral Neuropathy*, Vol I, ed P.J. Dyck, P.K. Thomas, & E.H. Lambert (Philadelphia: W.B. Saunders Co, 1975).
3. Alexander, M.A., & E.H. Season, "Idiopathic scoliosis: An electromyographic study," *Arch Phys Med Rehabil*, Vol 59 (Jul 1978).
4. Allman, F.L., Jr., "Fractures and ligamentous injuries of the clavicle and its articulation," *J Bone Joint Surg*, Vol 49A, No 4 (Jun 1967).
5. Anderson, J., *Grant's Atlas of Anatomy*, 7th ed (Baltimore: Williams & Wilkins, 1978).
6. Andersson, S., & B. Stener, "Experimental evaluation of the hypothesis of ligamento-muscular protective reflexes — II," *Acta Physiol Scand*, Vol 48, Supp 166 (1959).
7. Ardran, G.M. et al., "Assessment of scoliosis in children: Low dose radiograhic technique," *J Manip Physiol Ther*, Vol 3, No 2 (Jun 1980).
8. Aspegren, D.D., & J.M. Cox, "Correction of progressive idiopathic scoliosis utilizing neuromuscular stimulation and manipulation: A case report," *J Manip Physiol Ther*, Vol 10, No 4 (Aug 1987).
9. Babbage, N.F., "Burning foot syndrome: Bilateral occurrence with radical cure," *Med J Aust*, Vol 1 (May 22, 1965).
10. Barge, F.H., *Scoliosis*, Vol III (Davenport, IA: Bawden Printing Inc, 1981).
11. Basmajian, J.V., "Weight-bearing by ligaments and muscles," *Can J Surg*, Vol 4 (Jan 1961).
12. Basmajian, J.V., & J.W. Bentzon, "An electromyographic study of certain muscles of the leg and foot in the standing position," *Surg Gynecol Obstet*, Vol 98 (Jun 1954).
13. Basmajian, J.V., & C.J. De Luca, *Muscles Alive — Their Functions Revealed by Electromyography*, 5th ed (Baltimore: Williams & Wilkins, 1985).
14. Basmajian, J.V., & G. Stecko, "The role of muscles in arch support of the foot — an electromyographic study," *J Bone Joint Surg*, Vol 45A, No 6 (Sep 1963).
15. Beardall, A.G., "Differentiating the muscles of the low back and abdomen," in *Selected Papers of the International College of Applied Kinesiology*, ed B.C. Markham (Detroit, MI: ICAK, 1981).
16. Beardall, A.G., *Clinical Kinesiology, Vol V: Muscles of the Lower Extremities — Calf and Foot* (Lake Oswego, OR: privately published, 1985).
17. Blaich, R.M., "Ligament stretch-adrenal stress syndrome related to knee disorders." Proceedings of Summer Meeting, ICAK, Detroit, 1980.
18. Blount, W.P., "Early recognition and prompt evaluation of spinal deformity," *WI Med J*, Vol 68, No 8 (Aug 1969).
19. Bojsen-Møller, F., "Calcaneocuboid joint and stability of the longitudinal arch of the foot at high and low gear push off," *J Anat*, Vol 129, No 1 (1979).
20. Bojsen-Møller, F., & K.E. Flagstad, "Plantar aponeurosis and internal architecture of the ball of the foot," *J Anat*, Vol 121, No 3 (1976).
21. Breig, A., *Adverse Mechanical Tension in the Central Nervous System* (New York: John Wiley & Sons, 1978).
22. Brody, D.M., "Running injuries — prevention and management," *Clin Symp*, Vol 39, No 3 (1987).
23. Carrel, J.M., & D.M. Davidson, "Nerve compression syndromes of the foot and ankle: A comprehensive review of symptoms, etiology and diagnosis utilizing nerve conduction testing," *J Am Podiatry Assoc*, Vol 65, No 4 (Apr 1975).
24. Cipriano, J.J., *Photographic Manual of Regional Orthopaedic Tests* (Baltimore: Williams & Wilkins, 1985).
25. Connolly, B.H., & B.T. Michael, "Early detection of scoliosis — a neurological approach using the asymmetrical tonic neck reflex," *Phys Ther*, Vol 64, No. 3 (Mar 1984).
26. Cook, S.D. et al., "Upper extremity proprioception in idiopathic scoliosis," *Clin Orthop*, No 213 (Dec 1986).
27. Cowell, H.R., J.N. Hall, & G.D. MacEwen, "Familial patterns in idiopathic scoliosis," *J Bone Joint Surg*, Vol 51A (Sep 1969).
28. Cyriax, J., *Textbook of Orthopaedic Medicine, Volume One — Diagnosis of Soft Tissue Lesions*, 8th ed (Philadelphia: Bailliere Tindall, 1982).
29. Cyriax, J., *Textbook of Orthopaedic Medicine, Volume Two. Treatment by Manipulation, Massage and Injection* (Philadelphia: Bailliere Tindall, 1984).
30. Dalton, K., & M.J.T. Dalton, "Characteristics of pyridoxine overdose neuropathy syndrome," *Acta Neurol Scand*, Vol 76 (Jul 1987).
31. Dananberg, H.J., "Functional hallux limitus and its relationship to gait efficiency," *J Amer Podiatric Med Assoc*, Vol 76, No 11 (Nov 1986).
32. Dananberg, H.J., "The Kinetic Wedge" (Letter to the Editor), *J Am Podiatr Med Assoc*, Vol 78, No 2 (Feb 1988).
33. Dananberg, H.J., "Temporomandibular joint (TMJ) dysfunction syndrome as a result of gait malfunction: A case study," *Current Podiatric Med* (Jul 1988).
34. Dananberg, H.J., "Gait style as an etiology to chronic postural pain. Part I. Functional hallux limitus," *J Am Podiatr Med Assoc*, Vol 83, No 8 (Aug 1993).
35. Dananberg, H.J., "Gait style as an etiology to chronic postural pain. Part II. Postural compensatory process," *J Am Podiatr Med Assoc*, Vol 83, No 11 (Nov 1993).
36. Dananberg, H.J., "Foot malfunction and its relationship to craniomandibular disorders," in *New Concepts in Craniomandibular and Chronic Pain Management*, ed H. Gelb (London: Mosby-Wolfe, 1994).
37. Dananberg, H.J., "Lower extremity mechanics and their effect on lumbosacral function," *Spine: State of the Art Reviews*, Vol 9, No 2 (May 1995).
38. Dananberg, H.J., A.J. Phillips, & H.E. Blaakman, "A rational approach to the nonsurgical treatment of hallux limitus," in *Advances in Podiatric Medicine and Surgery*, Vol 2 (Mosby-Year Book, Inc, 1996).
39. Dananberg, H.J., M. Lawton, & D.R. DiNapoli, "Hallux limitus and non-specific gait related bodily trauma," in *Reconstructive Surgery of the Foot and Leg*, update, ed D.R. DiNapoli (Tucker, GA: Podiatry Institute Publishing Co, 1990).
40. Denny-Brown, D., & C. Brenner, "Paralysis of nerve induced by direct pressure and by tourniquet," *Arch Neurol Psychol*, Vol 51, No 1 (Jan 1944).
41. Durlacher, J.V., "Injury susceptibility in hypoadrenia." Proceedings of Summer Meeting, ICAK, Detroit, 1977.
42. Edwards, W.G. et al., "The tarsal tunnel syndrome — diagnosis and treatment," *JAMA*, Vol 207, No 4 (Jan 27, 1969).
43. Ellis, J.M., "Treatment of carpal tunnel syndrome with vitamin B_6," *Southern Med J*, Vol 80, No 7 (Jul 1987).
44. Ellis, J.M. et al., "Vitamin B_6 deficiency in patients with a clinical syndrome including the carpal tunnel defect. Biochemical and clinical response to therapy with pyridoxine," *Res Comm Chem Pathol Pharmacol*, Vol 13, No 4 (Apr 1976).
45. Ellis, J.M. et al., "Survey and new data on treatment with pyridoxine of patients having a clinical syndrome including the carpal tunnel and other defects," *Res Comm Chem Pathol Pharmacol*, Vol 17, No 1 (May 1977).
46. Ellis, J. et al., "Therapy with vitamin B_6 with and without surgery for treatment of patients having the idiopathic carpal tunnel syndrome," *Res Comm Chem Pathol Pharmacol*, Vol 33, No 2 (Aug 1981).
47. Farkas, A., "The pathogenesis of idiopathic scoliosis," *J Bone Joint Surg*, Vol 36A, No 3 (Jun 1954).
48. Farkas, A., "Scoliosis starting years after cessation of growth," *Bull Hosp Joint Dis*, Vol 30 (Apr 1969).
49. Folkers, K. et al., "Biochemical evidence for a deficiency of vitamin B_6 in the carpal tunnel syndrome based on a crossover

clinical study," *Proc Natl Acad Sci*, Vol 75, No 7 (Jul 1978).
50. Foot Levelers, Inc., P.O. Box 12611, Roanoke, VA 24027-2611. Telephone 1-800-553-4860.
51. Fullerton, P.M., "The effect of ischaemia on nerve conduction in the carpal tunnel syndrome," *J Neurol Neurosurg Psychiatry*, Vol 26 (1963).
52. Funakoshi, M., & N. Amano, "Effects of the tonic neck reflex on the jaw muscles of the rat," *J Dent Res*, Vol 52, No 4 (Jul/Aug 1973).
53. Gatens, P.F., & M.A. Saeed, "Electromyographic findings in the intrinsic muscles of normal feet," *Arch Phys Med Rehabil*, Vol 63, No 7 (Jul 1982).
54. Goodfellow, J., "He who hesitates is saved," *J Bone Joint Surg*, Vol 62B (1980).
55. Goodgold, J., H.P. Kopell, & N.I. Spielholz, "The tarsal-tunnel syndrome — Objective diagnostic criteria," *N Engl J Med*, Vol 273, No 14 (Sep 30, 1965).
56. Goodheart, G.J., Jr., "Arm and shoulder pain," *Chiro Econ*, Vol 7, No 2 (Sep/Oct 1964).
57. Goodheart, G.J., Jr., "The carpal tunnel syndrome," *Chiro Econ*, Vol 10, No 1 (Jul/Aug 1967).
58. Goodheart, G.J., Jr., "The 'frozen shoulder' syndrome, *Chiro Econ*, Vol 12, No 1 (Jul/Aug 1969).
59. Goodheart, G.J., Jr., "Tarsal tunnel syndrome," *Chiro Econ*, Vol 13, No 5 (Mar/Apr 1971).
60. Goodheart, G.J., Jr., *Applied Kinesiology 1973 Research Manual*, 9th ed (Detroit: privately published, 1973).
61. Goodheart, G.J., Jr., *Applied Kinesiology 1977 Workshop Procedure Manual*, 13th ed (Detroit: privately published, 1977).
62. Goodheart, G.J., Jr., *Applied Kinesiology 1992/93 Workshop Procedure Manual* (Grosse Pointe Woods: privately published, 1993).
63. Goodheart, G.J., Jr., *Applied Kinesiology 1997-1998 Workshop Procedural Manual* (Grosse Pointe Woods: privately published, 1998).
64. Gowitzke, B.A., & M. Milner, *Understanding the Scientific Bases of Human Movement*, 2nd ed (Baltimore: Williams & Wilkins, 1980).
65. Granit, R., L. Leksell, & C.R. Skoglund, "Fibre interaction in injured or compressed region of nerve," *Brain*, Vol 67 (Jun 1944).
66. Gray, E.R., "The role of leg muscles in variations of the arches in normal and flat feet," *Phys Ther*, Vol 49, No 10 (Oct 1969).
67. Gray, H., *Anatomy of the Human Body*, 29th Am ed, ed C.M. Goss (Philadelphia: Lea & Febiger, 1973).
68. Gresczyk, E., "Electromyographic study of the effect of leg muscles on the arches of the normal and flat foot." Thesis, U VT (Apr 1965).
69. Guyton, A.C., & J.E. Hall, *Textbook of Medical Physiology*, 9th ed (Philadelphia: W.B. Saunders Co, 1996).
70. Hamilton, J.J., & L.K. Ziemer, "Functional anatomy of the human ankle and foot," in *Symposium on The Foot and Ankle*, ed R.H. Kiene & K.A. Johnson (St. Louis: The C.V. Mosby Co, 1983).
71. Hamilton, W.G., "Surgical anatomy of the foot and ankle," *Clin Symp*, Vol 37, No 3 (1985).
72. Harris, R.I., & T. Beath, "Hypermobile flat-foot with short tendo Achillis," *J Bone Joint Surg*, Vol 30A, No 1 (Jan 1948).
73. Hearon, K.G., *What You Should Know About Extremity Adjusting* (Sequim, WA: K.G. Hearon, 1981).
74. Helfet, A.J., ed, *Disorders of the Knee*, 2nd ed (Philadelphia: J.B. Lippincott Co, 1982).
75. Hellebrandt, F.A. et al., "Tonic neck reflexes in exercises of stress in man," *Am J Phys Med*, Vol 35 (Jun 1956).
76. Hicks, J.H., "The function of the plantar aponeurosis," *J Anat*, Vol 85 (1951).
77. Hicks, J.H., "The mechanics of the foot, I. The joints," *J Anat*, Vol 87 (1953).
78. Hicks, J.H., "The mechanics of the foot II. The plantar aponeurosis and the arch," *J Anat*, Vol 88 (Jan 1954).
79. Hicks, J.H., "The foot as a support," *Acta Anat*, Vol 25 (1955).
80. Hoogmartens, M.J., & J. Basmajian, "Postural tone in the deep spinal muscles of idiopathic scoliosis patients and their siblings," *Electromyogr Clin Neurophysiol*, Vol 16 (1976).
81. Hoppenfeld, S., *Physical Examination of the Spine and Extremities* (New York: Appleton-Century-Crofts, 1976).
82. Horn, C.V., "Electromyographic investigation of muscle imbalance in patients with paralytic scoliosis," *Electromyography*, Vol 4 (1969).
83. Jackson, J.P., "Degenerative changes in the knee after meniscectomy," *Br Med J*, Vol 2 (Jun 1968).
84. James, J.I.P., "The etiology of scoliosis," *J Bone Joint Surg*, Vol 52B, No 3 (Aug 1970).
85. Kasdan, M.L., & C. Janes, "Carpal tunnel syndrome and vitamin B_6," *Plastic Reconstruct Surg*, Vol 79 (Mar 1987).
86. Keck, C., "The tarsal-tunnel syndrome," *J Bone Joint Surg*, Vol 44A (Jan 1962).
87. Kessel, L., *Clinical Disorders of the Shoulder* (New York: Churchill Livingstone, 1982).
88. Kopell, H.P., & W.A.L. Thompson, "Pronator syndrome — a confirmed case and its diagnosis," *N Engl J Med*, Vol 259, No 15 (Oct 9, 1958).
89. Kopell, H.P., & W.A.L. Thompson, *Peripheral Entrapment Neuropathies*, 2nd ed (Huntington, NY: Robert E. Krieger Publishing Co, 1976).
90. Krissoff, W.B., & W.D. Ferris, "Runners' injuries," *Physician Sportsmed*, Vol 7, No 12 (Dec 1979).
91. Lam, S.J.S., "A tarsal-tunnel syndrome," *Lancet*, Vol 2 (Dec 29, 1962).
92. Lam, S.J.S., "Tarsal tunnel syndrome," *J Bone Joint Surg*, Vol 49B, No 1 (Feb 1967).
93. Langer Biomechanics Group, The Electrodynogram System™, Deer Park, NY 11729.
94. Langworthy, O.R., *The Sensory Control of Posture and Movement — A Review of the Studies of Derek Denny-Brown* (Baltimore: Williams & Wilkins, 1970).
95. Lieb, F.J., & J. Perry, "Quadriceps function — An anatomical and mechanical study using amputated limbs," *J Bone Joint Surg*, Vol 50A, No 8 (Dec 1968).
96. Lloyd-Roberts, G.C., & M.F. Pilcher, "Structural idiopathic scoliosis in infancy," *J Bone Joint Surg*, Vol 47B, No 3 (Aug 1965).
97. Lueck, R.A., & R.D. Ray, "Volkmann's ischemia of the lower extremity," *Surg Clin North Am*, Vol 52, No 1 (Feb 1972).
98. Lusskin, R., "Peripheral neuropathies affecting the foot: Traumatic, ischemic, and compressive disorders," in *Disorders of the Foot*, Vol 2, ed M.H. Jahss (Philadelphia: W.B. Saunders Co, 1982).
99. Lutter, L.D., "Pronation biomechanics in runners," *Contemp Orthop*, Vol 2, No 8 (Nov 1980).
100. Lutter, L.D., "Forefoot injuries," in *Symposium on The Foot and Ankle*, ed R.H. Kiene & K.A. Johnson (St. Louis: The C.V. Mosby Co, 1983).
101. Magoun, H.I., "Idiopathic adolescent spinal scoliosis: A reasonable etiology," *D.O.*, Vol 13, No 6 (Feb 1973).
102. Mann, F., *Reinventing Acupuncture — A New Concept of Ancient Medicine* (Oxford, England: Butterworth-Heinemann Ltd, 1992).
103. Mann, R., & V.T. Inman, "Phasic activity of intrinsic muscles of the foot," *J Bone Joint Surg*, Vol 46-A, No 3 (Apr 1964).
104. Mann, R.A., G.T. Moran, & S.E. Dougherty, "Comparative electromyography of the lower extremity in jogging, running, and sprinting," *Am J Sports Med*, Vol 14, No 6 (Nov/Dec 1986).
105. Mann, R.A., N.K. Poppen, & M. O'Konski, "Amputation of the great toe — a clinical and biomechanical study," *Clin Orthop*, No 226 (Jan 1988).
106. McCouch, G.P., I.D. Deering, & T.H. Ling, "Location of receptors for tonic neck reflexes," *J Neurophysiol*, Vol 14 (May 1951).
107. Mennell, J.McM., *Joint Pain* (Boston: Little, Brown & Co, 1964).

108. Meschan, I., *An Atlas of Normal Radiographic Anatomy*, 2nd ed (Philadelphia: W.B. Saunders Co, 1959).
109. Michele, A.A., *Iliopsoas — Development of Anomalies in Man* (Springfield, IL: Charles C. Thomas Publisher, 1962).
110. Moe, J.H., & J.A. Byrd, III, "Idiopathic scoliosis," in *Moe's Textbook of Scoliosis and Other Spinal Deformities*, 2nd ed, ed D.S. Bradford et al. (Philadelphia: W.B. Saunders Co, 1987).
111. Moloney, S., "Tarsal tunnel syndrome pain abated by operation to relieve pressure," *CA Med*, Vol 101 (Nov 1964).
112. Morrey, B.F., ed, *The Elbow and Its Disorders* (Philadelphia: W.B. Saunders Co, 1985).
113. Morrish, G.M., & R.C. Woledge, "A comparison of the activation of muscles moving the patella in normal subjects and in patients with chronic patellofemoral problems," *Scand J Rehab Med*, Vol 29 (1997).
114. Murphy, P.C., & D.E. Baxter, "Nerve entrapment of the foot and ankle in runners," *Foot & Ankle*, Vol 6, No 6 (Jun 1986).
115. *New Encyclopaedia Britannica, The*, Vol X (Chicago: Encyclopaedia Britannica, 1979).
116. Nicholas, J.A., & E.B. Hershman, *The Lower Extremity & Spine in Sports Medicine, Vol 1. Basic Science* (St. Louis: The C.V. Mosby Co, 1986).
117. Nicholas, J.A., & E.B. Hershman, *The Lower Extremity & Spine in Sports Medicine, Vol 2. Regional Considerations* (St. Louis: The C.V. Mosby Co, 1986).
118. Nicholas, J.A., A.M. Strizak, & G. Veras, "A study of thigh muscle weakness in different pathological states of the lower extremity," *Sports Med*, Vol 4, No 6 (Nov/Dec 1976).
119. Noble, C.A., "Iliotibial band friction syndrome in runners," *Am J Sports Med*, Vol 8, No 4 (1980).
120. Noble, J., & K. Erat, "In defence of the meniscus," *J Bone Joint Surg*, Vol 62B, No 1 (Feb 1980).
121. Noble, J., & P.G. Turner, "The function, pathology, and surgery of the meniscus," *Clin Orthop*, No 210 (Sep 1986).
122. O'Connell, A.L., "Effect of sensory deprivation on postural reflexes," *Electromyography*, Vol 11, No 5 (Dec 1971).
123. Ober, F.R., "Back strain and sciatica," *Clin Orthop*, No 219 (Jun 1987).
124. Petersen, I., & B. Stener, "Experimental evaluation of the hypothesis of ligamento-muscular protective reflexes. III," *Acta Physiol Scand*, Vol 48, Supp 166 (1959).
125. Powers, C.M., R. Landel, & J. Perry, "Timing and intensity of vastus muscle activity during functional activities in subjects with and without patellofemoral pain," *Phys Ther*, Vol 76, No 9 (Sep 1996).
126. Ramig, D. et al., "The foot and sports medicine — biomechanical foot faults as related to chondromalacia patellae," *Bull Sports Med Section, APTA*, Vol 3, No 2 (Summer 1977).
127. Risser, J.C., "Scoliosis: Past and present," *J Bone Joint Surg*, Vol 46A, No 3 (Jan 1964).
128. Rose, G.K., "Pes Planus," in *Disorders of the Foot*, Vol 1, ed M.H. Jahss (Philadelphia: W.B. Saunders Co, 1982).
129. Ruch, T.C., "Brain stem control of posture and orientation in space," in *Physiology and Biophysics, I: The Brain and Neural Function*, 20th ed, ed T. Ruch & H.D. Patton (Philadelphia: W.B. Saunders Co, 1979).
130. Ryerson, E.W., in discussion of article by R.I. Harris & T. Beath, "Hypermobile flatfoot with short tendo Achillis," *J Bone Joint Surg*, Vol 30A, No 1 (Jan 1948).
131. Sahlstrand, T., & B. Petruson, "A study of labyrinthine function in patients with adolescent idiopathic scoliosis. I. An electronystagmographic study," *Acta Orthop Scand*, Vol 50 (1979).
132. Schmitt, W.H., Jr., "Making B-6 work (Activating pyridoxine to pyridoxal-5-phosphate)." Proceedings of Winter Meeting, ICAK, Palm Desert, CA, 1987.
133. Scott, W. et al., eds, *Principles of Sports Medicine* (Baltimore: Williams &Wilkins, 1984).
134. Seedhom, B.B., D. Dowson, & V. Wright, "Functions of the menisci. A preliminary study," *Ann Rheum Dis*, Vol 33 (Jan 1974).
135. Segal, P., & M. Jacob, *The Knee* (Chicago: Year Book Med Publishers, Inc, 1983).
136. Shizukuishi, S. et al., "The carpal tunnel syndrome as a probable primary deficiency of vitamin B_6 rather than a deficiency of a dependency state," *Biochem Biophys Res Communications*, Vol 95, No 3 (Aug 14, 1980).
137. Shrive, N., "The weight-bearing role of the menisci of the knee," *J Bone Joint Surg*, Vol 56B, No 2 (May 1974).
138. Slocum, D.B., & S.L. James, "Biomechanics of running," *JAMA*, Vol 205, No 11 (Sep 9, 1968).
139. Smith, J.W., "The activity of leg and foot muscles in standing," *J Anat*, Vol 85 (1951).
140. Snijders, C.J. et al., "Provocation of epicondylalgia lateralis (tennis elbow) by power grip or pinching," *Med Sci Sports Exer*, Vol 19, No 5 (Oct 1987).
141. Subotnick, S.I., "Equinus deformity as it affects the forefoot," *J Am Podiatry Assoc*, Vol 61, No 11 (Nov 1971).
142. Subotnick, S.I., *Podiatric Sports Medicine* (Mt. Kisco, NY: Futura Publishing Co, 1975).
143. Taylor, E.J., ed, *Dorland's Illustrated Medical Dictionary*, 27th ed (Philadelphia: W.B. Saunders Co, 1988).
144. Thomas, P.K., & P.M. Fullerton, "Nerve fibre size in the carpal tunnel syndrome," *J Neurol Neurosurg Psychiatry*, Vol 26 (Dec 1963).
145. Thomee, R. et al., "Patellofemoral pain syndrome in young women. II. Muscle function in patients and healthy controls," *Scand J Med Sci Sports*, Vol 5 (1995).
146. Travell, J.G., & D.G. Simons, *Myofascial Pain and Dysfunction — The Trigger Point Manual* (Baltimore: Williams & Wilkins, 1983).
147. Travel, J.G., & D.G. Simons, *Myofascial Pain and Dysfunction — The Trigger Point Manual. The Lower Extremities*, Vol 2 (Baltimore: Williams & Wilkins, 1992).
148. Vahl, R.J., "Some causative aspects of knee stress injuries in females," *Chiro Econ*, Vol 30, No 4 (Jan/Feb 1988).
149. Wallin, D. et al., "Improvement of muscle flexibility — a comparison between two techniques," *Am J Sports Med*, Vol 13, No 4 (Jul/Aug 1985).
150. Walther, D.S., "Upper extremity peripheral nerve entrapment," in *1982 Selected Papers of the International College of Applied Kinesiology*, ed W.H. Schmitt, Jr. (Detroit, MI: ICAK, 1982).
151. Whitesides, T.E., "Compartment syndromes," in *Disorders of the Foot*, Vol 2, ed M.H. Jahss (Philadelphia: W.B. Saunders Co, 1982).
152. Williams, P.L., & R. Warwick, eds, *Gray's Anatomy*, 36th Br ed (Philadelphia: W.B. Saunders Co, 1980).
153. Witvrouw, E. et al., "Reflex response times of vastus medialis oblique and vastus lateralis in normal subjects and in subjects with patellofemoral pain syndrome," *JOSPT*, Vol 24, No 3 (Sep 1996).
154. Wolaniuk, A., S. Vadhanavikit, & K. Folkers, "Electromyographic data differentiate patients with the carpal tunnel syndrome when double blindly treated with pyridoxine and placebo," *Res Comm Chem Pathol Pharmacol*, Vol 41, No 3 (Sep 1983).
155. Wolff, J., *The Law of Bone Remodelling* (New York: Springer-Verlag, 1986).
156. Yergason, R.M., "Supination sign," *J Bone Joint Surg*, Vol 13 (1931).
157. Yochum, T.R., & L.J. Rowe, *Essentials of Skeletal Radiology*, Vol 1 (Baltimore: Williams & Wilkins, 1987).
158. Zatkin, A., "Weight balance and structural involvement," in *1987 Selected Papers of the International College of Applied Kinesiology*, ed D.H. Schusterman (Shawnee Mission, KS: ICAK, 1987).
159. Zuk, T., "The role of spinal and abdominal muscles in the pathogenesis of scoliosis," *J Bone Joint Surg*, Vol 44B, No 1 (Feb 1962).

Chapter 12

Systemic Conditions

Chapter 12

Ileocecal Valve Syndrome

Examination and treatment of the ileocecal valve syndrome (ICV) was introduced into applied kinesiology by Goodheart in 1967.[162] Correction of ICV dysfunction has been responsible for improving myriad symptoms.

The ileocecal valve is also called the ileocolonic valve and the ileocecal sphincter. Its primary function is to control movement between the small and large intestines. The ileocecal valve is composed of two aspects. One is a sphincter-like thickening of the circular muscles at the distal end of the ileum, just prior to the cecum. The second aspect is at the point of joining where there is a valve-like structure formed by two transverse folds or lips, one above the other, on the cecal aspect of the ileocolic orifice. The sphincter-like portion is under neurologic control; the valvular portion is shaped to provide a mechanical one-way valve.[30,187]

12—1. Ileocecal valve.

There has been some controversy regarding the importance of ileocecal valve function. Occasionally one who treats this syndrome still encounters those holding the view that ileocecal valve function is not necessary. This is usually supported by statements such as, "You can see that everyone has movement both ways through the ileocecal valve section because barium in a colon study consistently passes into the ileum." *Gray's Anatomy*[420] supports this by stating, "...Radiological evidence contradicts the concept of an effective valve at this junction." Others conclude from roentgen studies that the ICV has an effective valve function.[140] Furthermore, when referring to a possible sphincteric action, *Gray's Anatomy* comments, "It should be noted that all such sphincteric mechanisms must be balanced by an opposite, dilatory activity." As we will see, there are many studies supporting the neurologic activity of the ileocecal valve and its competency.

Among those who recognize the ileocecal valve's function, there has also been controversy about whether the function is due to the muscular action of the sphincter-like portion or to the mechanical action of the valvular flaps. Most agree that both portions contribute to the control of food material leaving the ileum to enter the cecum, and preventing its return to the ileum. Best and Taylor[30] state, "The functions of the ileocolic valve appear to be: (a) to prevent the contents of the ileum from passing into the cecum before the digestive processes have been completed, and (b) to serve as a barrier which prevents the bacteria-laden contents of the large bowel from contaminating the small intestine." A review of ileocecal valve studies helps put in perspective the necessity of its proper function, and why some people may hold the view that its function is not necessary.

Studies of the ileocecal valve *in vivo* as opposed to by dissection give a completely different view of its function. *In vivo* human studies of the ileocecal valve are usually done by direct observation during surgery, or through a stoma left from a previous surgical procedure. In a study of five patients where there was ready access to the ileocecal junction, the conclusion was "...that a true sphincter exists at the human ileocecal junction."[90] These investigators presented support for the hypothesis that the valve's action is to control movement of material from the ileum into the colon and to prevent reflux of material from the colon back into the ileum.

Visualization of the human ileocecal valve *in vivo*[406,407] revealed a tightly closed, puckered opening, marked by a stellate configuration. Prior to death, the ICV had more substance and more direction; it looked like a cervix protruding into the vagina. Strong air pressure introduced into the colon did not flatten the valve, but it did produce about 1 mm dilation of the orifice. Thirty minutes after death the cecal wall was completely flaccid and collapsed. The papilla was flaccid and responded immediately to slight changes in air pressure. Fifty-five minutes after death the orifice was open 12 mm.

In an *in vivo* study of two humans, the ileocecal valve contracted with any mechanical stimulation.[17] In another human study[52] in which the ileocecal valve was exteriorized, any mechanical stimulation caused strong contraction. A finger inserted into the valve was held steady. When a strong stimulus was applied, the patient had a vague epigastric distress, at times amounting to pain. Prolonged tonic contraction characteristic of sphincter action was observed.

Numerous animal studies have been done. There are anatomical differences between animals and man. For example, in dogs the small intestine passes continuously into the colon, as opposed to the right angle junc-

tion of the ileum with the cecum in humans. The value of animal studies is that the neurologic and humeral control appears to be the same as that in humans. According to Gazet and Jarrett,[152] "There is ... no reason to suppose that the innervation of the sphincter in man is different from that in the experimental animals." The sphincters in man, monkey, cat, and dog react similarly to sympathomimetic and parasympathomimetic drugs.

Animal studies are usually done using various methods of bringing the ileum and the colon to the abdominal wall to give access to the ileocolonic junction from both directions. By pulling water-filled balloons attached to transducers through the area, a zone of elevated pressure is observed. The pressure varies at different times, suggesting that the area is closed at times and open at others.[227]

Control of Valve Function

In applied kinesiology, both "open" and "closed" ileocecal valve syndromes are recognized. In the open ileocecal valve syndrome, there is regurgitation of colon contents into the small intestine; in the closed or spastic ileocecal valve syndrome, material fails to pass from the ileum into the colon.

Several factors control the ileocecal valve's opening and closing: (1) a difference in pressure gradation between the ileum and cecum,[228] (2) neurologic imbalance from subluxations, or (3) neurohumeral causes. The structural, chemical, and mental sides of the triad of health have been found to be causes of ileocecal valve dysfunction in applied kinesiology.

Weisbrodt[415] states, "Tone of the ileocecal sphincter is basically myogenic in origin. It is modified, however, by nervous and humeral factors. Distention of the colon causes an increase in sphincteric tension,[228] a reflex probably mediated via intrinsic myenteric nerves. Distention of the ileum causes relaxation, also probably mediated via the intrinsic nerves." This is what one would expect in normal function. From the frequency of clinically observed ileocecal valve syndromes, there must be rather common causes of its dysfunction.

Neurologic control of the sphincter between the ileum and cecum in the cat was illustrated as early as 1904 by Elliott.[131] The sphincter contracts with the stimulation of the sympathetic nerves,[347] while the circular muscle in the adjacent walls of the ileum and colon relaxes. In the cat there is no flap-like mechanical action of the valve. The separation of contents between the ileum and colon is the responsibility of the sphincter. Removal of the spinal cord abolishes the separation between the two bowels.

Spinal anesthesia blocks the reflex closing of the valve when there is distention of the large intestine, indicating a spinal reflex arc.[304] Control of the valve is partly under the vagus nerve.[303] Depending upon the frequency of stimulation, the vagus nerve causes contraction or relaxation of the sphincter.[219,407]

Spinal reflex control of the ileocecal valve has been established, but there is definite sphincter action in isolated ileocecal sphincter preparations of cats, even after these have been denervated. Section of the nerve supplying the valve causes only temporary paralysis, thus indicating that extrinsic nerves have only a regulating function. The intrinsic, rhythmic function is under the control of the myenteric plexus (plexus of Auerbach).[407]

"Parasympathetic stimulation, in general, increases the overall degree of activity of the gastrointestinal tract by promoting peristalsis and relaxing the sphincters, thus allowing rapid propulsion of contents along the tract."[187] The sphincter muscle has two properties: (1) it has a resting property greater than the adjacent segments of the alimentary canal, and (2) it relaxes (opens) to the appropriate stimulus so that flow can occur from one compartment to the next.[194] It is generally considered that the ileocecal valve is closed by sympathetic and opened by parasympathetic action.[187] It is observed in applied kinesiology that patients with relative hypoadrenia and hyperadrenia tend to have open and closed valves, respectively.[45,215]

Many factors appear to affect the ICV. Distention of the cecum intensifies contraction of the ileocecal sphincter and ileal peristalsis is inhibited greatly, delaying emptying of chyme from the ileum. Any irritant to the cecum, such as an inflamed appendix, can cause intense spasm of the ileocecal sphincter and paralysis of the ileum to the point that it blocks emptying of the ileum. "These reflexes from the cecum to the ileocecal sphincter and ileum are mediated both by way of the myenteric plexus in the gut wall itself and through extrinsic nerves, especially reflexes by way of the prevertebral sympathetic ganglia."[187] As will be discussed later, the kidneys and psoas muscles, as well as the entire digestive system, should routinely be evaluated when there is an ileocecal valve syndrome.

Humeral control of the ileocecal valve was observed in human subjects who had previously had a colon bypass that allowed ready access to the ileocecal junction. Castell et al.[66] report, "Instillation of HCl into the stomach resulted in a decrease in lower esophageal sphincter pressure and a simultaneous increase in ileocecal sphincter pressure, whereas gastric alkalinization and pentagastrin injection resulted in marked decreases in ileocecal sphincter pressure and increased lower esophageal sphincter pressure."

It is surprising that there are still some who discount the importance of ileocecal valve function. In 1934, Wangensteen[414] concluded that the check valve arrangement of the ileocecal valve precludes regurgitation into the small intestine. His conclusion developed as a result of observing patients with acute obstruction of the pelvic colon who did not have the slightest trace of ileum distention at operation. In some cases, he observed the cecum perforate or become gangrenous

without the small intestine participating in the distention. Wangensteen recognized that roentgenologists would disagree with this because of their observation of the "incompetent ileocecal sphincter." He goes on to comment about the high pressure employed by roentgenologists when examining the colon.

Although many roentgenologists will comment on the incompetence of the ileocecal valve, there are many studies indicating that competence is the normal and desired function. X-ray studies show that when the valve is competent it prevents reflux into the ileum,[267] even when there are large bowel obstructions and colon pressure becomes extremely high. Ulin et al.[407] comment that barium enemas forcing the ileocecal valve open far exceed the pressure observed clinically, even in large bowel obstruction. In various disease processes, the ICV attempts to continue its major purpose of separating colon contents from the ileum. In 367 cases of colon cancer, a competent ileocecal valve was found in 61% of the cases.[112,267] This is of great importance because it causes a threat of a "closed loop" colic obstruction, creating a potential surgical emergency to prevent cecal perforation. Barium studies reveal that there is a wide variation in the reflux resistance of the human ileocecal valve.[334] Proper function of the valve is for it to prevent reflux, and to open at the proper time to allow ileal contents to enter the cecum. One tends to think in terms of improper neurologic control of the valve when it dysfunctions; however, there is the possibility that one or the other of the ileocecal valve lips is congenitally short, causing incompetence.[112] This is believed to be overemphasized in some analyses because the type of fixatives and preservatives used to preserve cadavers distorts the cecum and valvular structure, giving a false conclusion of the true anatomy of the structure.[407]

Valve Function Importance

Studies revealing the importance of the ileocecal valve have primarily been done in human disease states, such as cancer, where a colostomy has removed the ileocecal valve. Loss of the valve causes various types of problems. There have been animal experiments to design methods of limiting the damage done by removing the ICV.

Gazet and Kopp[153] comment on a human study showing that frequent loose movements of the bowel were a persistent complication in over 50% of right hemicolectomies. The corresponding figure for left-sided hemicolectomies was 5%. This diarrhea is not a transient feature; should it persist over a year post-operatively, it is likely to be a permanent disability. Gazet and Kopp[153] studied the subject in dogs, and it was clearly shown that preservation of the sphincter prevents an increase in the bacterial contamination of the ileum at some distance from the sphincter. Cats and monkeys lost the most weight when a classical hemicolectomy was performed. It is suggested that whenever possible in colonic surgery, the ileocolonic sphincter should be preserved as a means of preventing morbidity following a classical right hemicolectomy.

In an effort to eliminate the problem when it is necessary to remove the ileocecal valve, Glassman[155] constructed artificial ileocecal valves during ileocolostomies on dogs. This was done by surgically elevating flaps of the serous muscular coats of the intestine. The surgery was compared with standard ileocolostomies, and a comparison of the bacteria in the ileum was made. The dogs with the regular ileocolostomies had a greater number of organisms and at intestinal levels higher than occurred normally. The dogs with the valvular reconstruction had bacteria essentially unchanged and, in some instances, even showed a smaller number of organisms than before surgery. In addition, general observation of fourteen dogs with the ordinary ileocolostomy revealed that nearly all had an increased number of bowel movements, liquid stools, and — as a consequence — the appearance and symptoms of dehydration. The dogs with the artificial valves showed an appearance and condition of defecation that deviated little from the normal state.

In the human, the ileocecal valve helps prevent malabsorption and diarrhea. Kalser et al.[225] state, "If a segment of terminal ileum and the ileocecal valve can be left intact, nutritional deficiencies will be minimal." The slowing effect of the ileocecal valve holds the chyme so there can be water absorption in the ileum. "An intact ileocecal valve and a functioning ileal mucosa are important in fat absorption."

Body Language and Examination

It is believed in applied kinesiology that many symptoms of the ileocecal valve syndrome occur as a result of absorption of toxic products by the ileum that have been regurgitated from the colon. As the skin and lungs take over detoxification, there is usually a fetid breath and possibly body odor. In an open ileocecal valve condition, there may be the combination of dehydration and fluid retention. Fluid retention is the body's natural method of attempting to reduce the effects of the toxicity by diluting it with water. There will often be evidence of liver problems with standard diagnostic methods in severe conditions. Positive therapy localization over the ileocecal valve is a standard finding.

The symptoms of an open and a closed ileocecal valve syndrome are very similar. Some of the symptoms that may be caused by an ICV problem are palpitation and a feeling of the heart fluttering, chest pain with activity, pseudo-Meniere's syndrome, migraine headache, dependent edema, right shoulder pain simulating bursitis, neck stiffness, mid-afternoon dizziness, tinnitus, nausea, faintness, pseudo-sinus infection, sudden thirst, dark circles under eyes, and general achiness. The closed ICV poses an exception to the similarity of symptoms; it causes increased symptoms upon arising in the morn-

ing that ease with movement. When the patient becomes active after arising, the headache and other pains improve; this is probably associated with increased bowel activity. These individuals become much worse if they sleep late when the opportunity arises, e.g., on a weekend. One can often see a difference between two individuals with open and closed ileocecal valves. The person with the closed ileocecal valve will have the closed, pent-up body language described by Fast[134]; the person with the open valve will have open body language.

Because of all the symptoms that accompany an ileocecal valve syndrome one should examine for it routinely, regardless of the patient's complaint. Sometimes the major focus of pain at the area of complaint is very severe and remote from the digestive system. The physician's primary examination is obviously directed toward the pain. This may cause him to overlook the ICV syndrome. An example is the individual who has severe sciatic neuralgia, with signs of intervertebral disc protrusion or herniation. As we will see, the main treatment may need to be directed to the ICV to reduce fluid retention.

Generally there are no major changes in a patient's bowel habits or other complaints of the digestive system. If there has been chronic constipation or diarrhea, it will tend to continue. If there is a bowel change with an open ICV, it will usually consist of the stool changing to small balls of feces, or it may be full length but small in diameter in comparison with the individual's normal stool. Usually a patient will not complain about the bowel.

The major complaint of the patient may be of peripheral nerve entrapment, such as at the carpal tunnel, or an apparent intervertebral disc syndrome. Either of these can be from fluid retention by the body to help reduce the effects of toxin. The median nerve at the carpal tunnel may become entrapped and produce an effect similar to the swelling of rheumatoid arthritis, putting pressure on the nerve. Swelling of an intervertebral disc as a result of fluid retention may be the cause of low back pain with sciatic radiation. Vigorous vertebral adjusting is contraindicated when the disc is under such pressure. Along with this there is often a feeling of the right leg giving way, and pain in the groin at the location of the neurolymphatic reflex.

Occasionally a patient's symptoms can be improved dramatically by simply pushing in on the lower right quadrant of the abdomen and pulling up toward the left shoulder. A headache or other pain may be quickly reduced. Sometimes when there is low back pain, with or without sciatic neuralgia, the patient may be able to stand and walk with the cecum held in this manner.

The simplest method for determining the probability of an ileocecal valve syndrome is to have the patient therapy localize over the lower right abdominal quadrant. A previously strong indicator muscle will weaken. The tensor fascia lata, associated with the large intestine, is usually a good indicator muscle. A positive challenge cancels the indicator muscle's weakness as a result of the therapy localization. Pressure applied to the lower portion of the cecum and directed toward the left shoulder tends to close an open ileocecal valve. If this challenge strengthens the indicator muscle weakened by positive therapy localization, it is evidence of an open ileocecal valve. When the valvular area is pulled down in an open ICV, a previously strong indicator muscle will usually weaken. The latter is not as easy and effective a challenge to obtain as the former. Challenge for a closed ileocecal valve is limited to strengthening a muscle that tests weak as a result of therapy localization over the ICV, or as a result of the syndrome. Pulling down on the valvular area causes the muscle to strengthen.

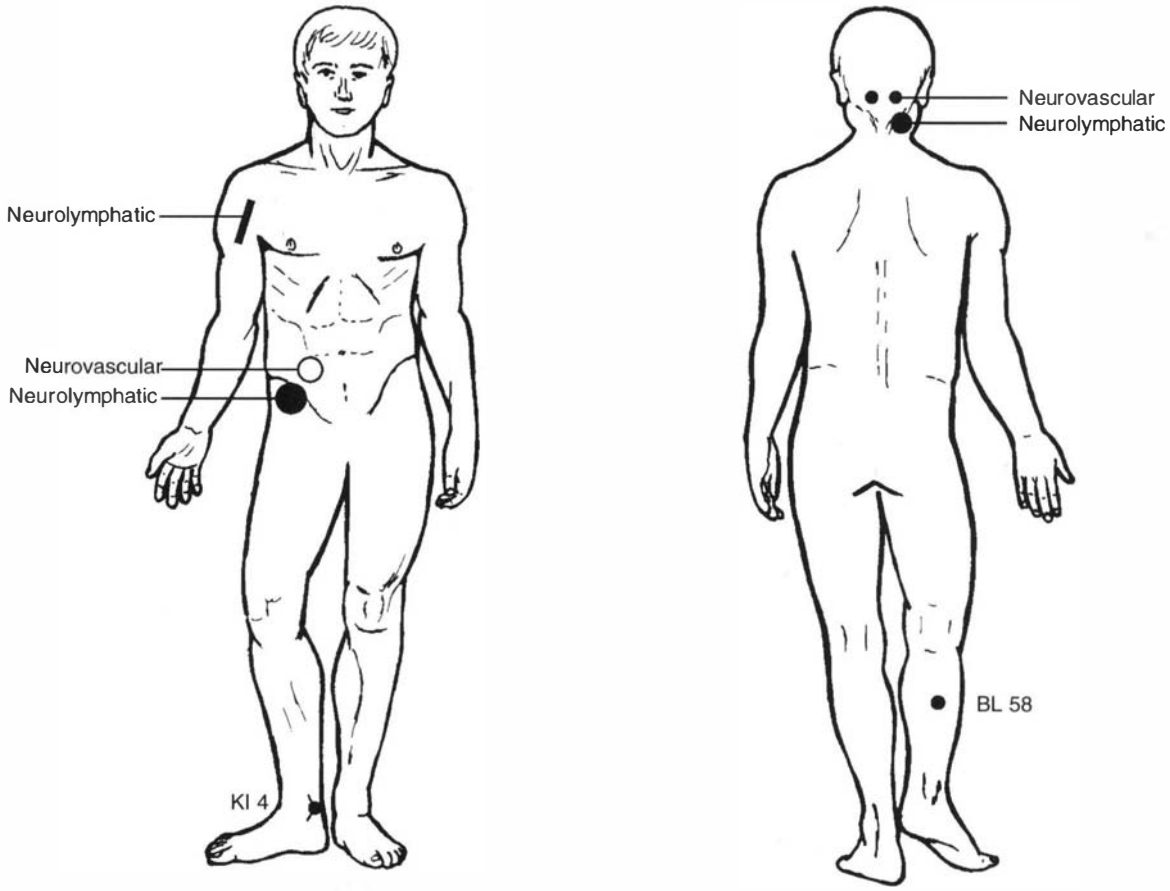
The directions for challenging the ileocecal valve just described are generally applicable. One may have to apply various vectors to find a positive challenge, especially if there is scar tissue from an appendectomy or some other surgery. One must remember that the superficial scar is only external evidence of adhesions that may traverse deep into the body.

Treatment

There are three neurolymphatic reflexes for the open ICV: below the right anterior superior iliac spine, at the right bicipital groove (approximately 3" long), and adjacent to the right 3rd cervical lamina. The neurolymphatic reflexes for the closed ICV are the same as for the rectus abdominis and quadriceps femoris. The neurolymphatic reflexes are usually very tender with an ileocecal valve syndrome. The tenderness may be greatly reduced when the valve is held open or closed. First evaluate the tenderness and have the patient grade it on a scale of 1 to 10, with 10 being the worst. Then have the patient pull the ICV closed and re-evaluate and grade the tenderness. If it lessens considerably, an open ICV is indicated; if not, re-evaluate by having the patient pull the valve open.

The neurovascular reflex for the ileocecal valve is located where a transverse line from the crest of the ilium meets the rectus abdominis.

When a patient has headache or neck symptoms associated with an ICV syndrome, the head stress receptor for the ICV is nearly always involved. It is located 1/2" lateral to the posterior occipital protuberance for both an open and a closed condition. The hand stress receptor is on a line at LI 4. LI 4 is "hoku," a point often used in acupuncture treatment. It is the sedation point for the large intestine meridian. The point is treated either as an acupuncture point, if so indicated, or as a hand reflex point if it has directional challenge and respiratory correlation. Further evaluation of the head or hand stress receptors and LI 4 can be made by first observing positive therapy localization over the ileocecal valve and then applying two-handed therapy localization, i.e., one hand



12—2. KI 4 and BL 58 are meridian luo points. They are usually used in the ileocecal valve syndrome to transfer energy between the coupled kidney and bladder meridians. The luo point is stimulated on the deficient meridian.

on the ICV and the other on the head or hand stress receptor (LI 4) to determine if it cancels the ileocecal valve positive therapy localization. All reflexes and stress receptors should be treated until they no longer show positive therapy localization.

The ileocecal valve syndrome is frequently involved with psoas muscle dysfunction and kidney meridian imbalance. The usual situation is for the kidney meridian to be overactive in a closed syndrome and underactive in an open one. Inability to cause the psoas muscle to test weak by stimulating the sedation point (KI 1) is evidence of a closed ileocecal valve.

Correction of psoas dysfunction and kidney meridian imbalance are important factors in this condition. KI 4 and BL 58 are the luo points for the kidney and bladder meridians, respectively. These points are often used in applied kinesiology to transfer energy between coupled meridians or bilateral counterparts (see Chapter 7). Treating these points, when necessary, is often the difference between success and failure in obtaining long-term results in the ileocecal valve syndrome. The psoas muscle may need fascial release or strain/counterstrain technique, especially in a closed ICV.

The quadratus lumborum is often involved with the ileocecal valve syndrome.[45] The structural imbalance may exaggerate pain at the level of the 12th thoracic vertebra, known as Rogoff's sign in hypoadrenia. It may also cause lower, and sometimes upper, thoracic spinal pain.

The spinal column and the cranium should be thoroughly evaluated, and corrected if dysfunctioning. The usual spinal subluxation is L1 or L2 in an open condition, and an anterior L3 in a closed condition. There is consistent tenderness of the inferior tip of the vertebra's spinous process in an anterior subluxation.

It is important to evaluate the autonomic nervous system for imbalance. The most common finding is relative adrenal insufficiency, in which positive ICV therapy localization is neutralized by gustatory stimulation with adrenal concentrate or nucleoprotein extract. If the patient is parasympathetic-dominant, sucking on choline will cause generalized muscle weakness throughout the body.

Normally, emotional excitement increases the frequency of food being ejected into the cecum.[30] Emotional stress may be associated with either hypo- or hyperadrenia, causing a recurrent open or closed ICV syndrome, respectively. When there is recidivism, evalu-

Ileocecal Valve Syndrome
Differential Diagnosis and Treatment Summary

	Open	Closed
Challenge:	Pulling up on valve strengthens muscle. Pulling down on valve weakens strong muscle.	Pulling up on valve causes no change in weak muscle. Pulling down on valve causes weak muscle to strengthen.
Therapy localization:	Over cecum in lower right quadrant of the abdomen.	Same
Muscular involvement:	Test general indicator muscle with therapy localization.	Weak right rectus abdominis and quadriceps femoris. Hypertonic psoas.
NLR:	Below right ASIS, right bicipital groove (3"), and adjacent to right C3 lamina.	NL for rectus abdominis and quadriceps femoris.
NVR:	Halfway between lateral border of rectus abdominis and ASIS.	Same
Stress receptor:	1/2" lateral to posterior occipital protuberance.	Same
Vertebral involvement:	Usually L1 or possibly T12 or L2.	Anterior L3 and Lovett reactor (C3); L5 (associated point for small intestine).
Meridian:	BL 58 and/or KI 4, luo points for kidney and bladder meridians to balance meridians.	BL 58 or KI 4, luo point and sedation point for kidney meridian to drain kidney meridian. (Test to see if method is appropriate.)
Cranial:	Evaluate total cranium. Zygomatic and lambdoidal faults are often present.	Evaluate total cranium. Universal fault is often present, and sometimes zygomatic and lambdoidal faults are present.
Nutrition:	Chlorophyll, and sometimes digestive aids.	Calcium, vitamin D; sometimes hydrochloric acid when calcium cannot be absorbed.
Diet:	Off roughage, raw fruits and vegetables and spicy foods, caffeine, cocoa, and alcohol.	Same

12—3.

ate the patient with the applied kinesiology methods described in Chapter 10.

The type of food an individual eats may relate with an ileocecal valve syndrome. A high percentage of vegetarians have problems with an ICV. The condition is associated with certain times of the year when dietary patterns change. In the late summer and fall when abundant supplies of fresh fruits and vegetables are available at low cost, people may increase their consumption to the point of creating a problem with the valve. At Christmas and other holidays, sugar increase may be the causative factor. McBride[279] recommends challenging patients with suspected foods immediately after correcting an ileocecal valve syndrome, to determine if positive therapy localization and other evidence of its return are produced by chewing and ingesting the food. If so, temporarily eliminate that food from the diet and begin correcting the autoimmune system.

Treatment nearly always includes dietary management, and often nutritional supplementation. First, the patient should be instructed to eat a low-roughage, nonirritating diet for two weeks following ICV treatment. Roughage includes food such as coarse cereals, popcorn, peanuts, potato chips, and raw vegetables and fruits (vegetables and fruits may be eaten if cooked). Spicy foods such as chiles, tacos, pepper, and most condiments

should be eliminated. The patient should avoid alcoholic beverages during recovery. When one discusses the dietary changes with the patient, a clue often appears as to why the condition developed when the patient says, "Boy, I'm glad I ate that big bowl of popcorn last night!"

Emphasize to the patient that the diet you are putting him on is not a healthy one; it is only temporary, to avoid aggravating the already irritated area while it heals. Rarey[327] points out the necessity of transitioning the patient properly to a high-roughage diet. Many times a poor quality, low-roughage diet is the reason — or partial cause — an ileocecal valve syndrome develops.

A nutritional supplement for an open ICV that has been found effective in applied kinesiology is chlorophyll. It is given primarily to help soothe and heal the bowel. For the closed ileocecal valve condition, the nutritional requirement is calcium. Consider the patient's absorption and use of calcium. Sometimes in calcium deficiency there is a need for hydrochloric acid. This must be correlated with the acid/alkaline balance of the gut, discussed next. Routinely evaluate the levator scapula muscle and its association with the parathyroid glands to determine possible correlation with calcium involvement.

The acid balance of the gut is very important in controlling an ICV syndrome. If an acid/alkaline imbalance of the gut is contributing to the ICV syndrome, it will be too alkaline in an open condition and too acid in a closed one. Normally, the bowel contents should be close to neutral at the rectum. Pruritus ani usually indicates that the bowel is too acid; however, it can also be caused by too alkaline a bowel.[354] Poor anal control (feces bubble out) probably results from alkalinity. When the bowel is too alkaline because of a lack of hydrochloric acid, first attempt to improve its production by correcting any cranial faults that may be influencing the vagus nerve. The common type of fault is torsion of the cranium, such as the temporal bulge cranial fault. When the bowel is too acid, causing a closed ICV, pancreatic enzymes may help by improving the alkaline production from the pancreas.

For home treatment, the patient can put a cold pack (not ice pack) over the cecum. This is easily accomplished by simply filling a plastic bag, such as obtained from a grocery store, with cold tap water and putting a few ice cubes in it. The bag can be tied off with string or any other method to seal it. Allow the cold pack to slowly come to room temperature. Colonics and the usual enemas are detrimental to an ileocecal valve syndrome. An enema that is effective is a normal saline solution given slowly (over approximately twenty minutes) and with quite cool water. This helps "tone" the valve. These are given twice a week for at least a month, even though the symptoms are gone.[162]

Colon

All too often the colon is thought of as a "garbage dump." Treatment directed to the colon, outside of severe pathology, is usually only when the garbage is dumped too rapidly (diarrhea) or too slowly (constipation). A close look at colon function and dysfunction reveals the etiology for many health problems on the rise today. The physician should evaluate every new patient entering natural health care for colon function, at least on the consultation level.

The diseases associated with colon dysfunction are primarily those that have developed within this century, paralleling the dietary change that is the major cause of colon dysfunction. There are two primary causes for the rapid rise in health problems relating with the colon: (1) an increased use of refined carbohydrates, especially white sugar and white flour, and (2) the reduction of dietary roughage.

These diseases are primarily in the industrialized nations. They began to appear when the roller mills replaced stone grinding of grains in 1870.[55] Appendicitis did not become common until after 1880; diverticular disease was not a major problem until the 1920s; obesity was rare among those not of royalty until approximately 200 years ago; and hemorrhoids and tumors of the colon and rectum are common only in nations consuming refined carbohydrate diets. The common denominator is the refining of foods.[55]

Epidemiological studies of tribal communities in Africa and of the Japanese who have retained their original diets show a low incidence of diseases associated with colon dysfunction.[55] Both of these groups consume diets high in fiber and low in refined carbohydrates. When these ethnic groups migrate to Great Britain or to the United States and change their dietary habits, they become just as susceptible to diseases related to colon dysfunction as the British and Americans.

Burkitt et al.,[54] in a review of the literature on bowel transit times in epidemiology, found a major correlation to decreased fiber intake in several disease processes. The western diet slows bowel transit considerably. They state, "The fiber-deficient food residues of western man may remain in the bowel for several days and yet be associated with a daily bowel movement. Thus there is evidence to support the truth of the old adage that the patient may be regular but five days late." The daily stool in North America is approximately one-fourth the amount of that of rural Ugandans.

One method of studying bowel transit time is to have the subject swallow radio-opaque plastic pellets about the size of rice grains. The next five or six stools are saved in plastic bags and then x-rayed to count the pellets. Studies show that five out of eight geriatric pa-

tients retained the plastic pellets for two weeks.[54]

In another study reviewed by Burkitt et al.,[54] transit times were slowed by twenty-four hours in patients eating white bread as opposed to brown bread. The study by Burkitt et al. indicates that appendicitis, colon diverticulosis, and both benign and malignant tumors of the colon — as well as coronary artery disease, gallbladder disorders, and constipation — develop from low-fiber diets. They point out that coronary artery disease, colon diverticulosis, appendicitis, and gallbladder disease appeared or increased in England when roughage was refined out of the food in the early 20th century. Astronauts eat an almost fiber-free diet to develop constipation so they will have no bowel movements for five or six days.

The following conditions have a common etiology. Treatment is similar and relatively simple, considering the major implications of the conditions involved.

Colon Cancer

Activity in the colon varies greatly between those on high-roughage and low-roughage diets. An individual on a high-roughage diet has voluminous bowel movements and the material moves rapidly through the colon, with 80% removed from the bowel in less than a day and a half. Those on a refined low-roughage American-type diet have a transit time through the body of about three to four days, with the material in the colon most of that time.

Hill et al.[196] compared the biochemical contents of the stools in subjects in countries with a high rate of colon cancer (England, Scotland, and the United States) with those of three countries with a low rate (India, Uganda, and Japan). The westernized countries, with their refined, processed diets, had an increase in anerobic bacteria and a greater concentration of steroids in the feces. There was also a greater concentration of acid steroids derived from bile salts. The basic constituents of the feces were the same for all diets, but the concentrations were different. They conclude, "The geographical variations in the incidence of carcinoma of the colon seem to be correlated with variation in the fat content of the diet. It was postulated that intestinal bacteria might be able to produce carcinogens from dietary fats or from acid steroids, and that the variations in the incidence of colon cancer might depend partly on differences in the composition of the intestinal bacterial flora brought about by the difference in diet."

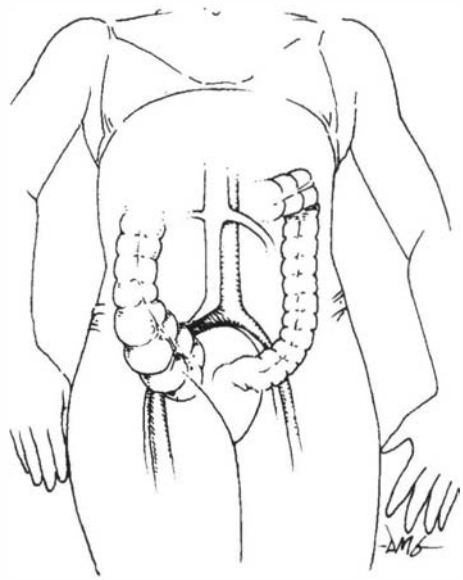
It is bad enough for carcinogens to develop in the colon, but when there is stasis from low-fiber content the chemicals are in contact with the colon walls for long periods. This increases the possibility of colon cancer.

Support for colon stasis increasing the chance of cancer is given by the incidence of cancer increasing along the progression of the colon, with the highest incidence at the rectum. The only exception to this is the cecum, which has a high occurrence of cancer. It is possible that a chronic or recurring ileocecal valve syndrome may be the cause of increased cecum cancer.

The answer to the problem is, again, prevention. It appears that regaining normal bowel chemistry and bacteria, along with rapid removal of waste products, is one answer to maintaining colon health. Often it is as simple as increasing the dietary fiber intake to a proper level.

Circulation

Hemorrhoids, varicosities, and phlebitis may be caused by colon dysfunction in several ways. It has long been known that constipation aggravates, and will indeed create, hemorrhoid problems. Colon stasis from a low-fiber diet causes accumulation of dry, hardened feces in the rectal area, causing pressure on the veins that, in turn, causes the veins to distend and eventually balloon the hemorrhoidal plexus. There is considerable pressure and strain when a compacted stool is passed, increasing the pressure in the vena cava and causing an additional distention and ballooning of the hemorrhoidal plexus.



12—4. Colon stasis can cause a large accumulation of feces to put pressure on the iliac vein, impeding blood flow.

Varicosities and phlebitis may be caused by portal stasis. In addition, there may be a blood flow obstruction as a result of the distended iliac portion of the colon and the cecum exerting pressure on the external iliac vein. The iliac vein lies posterior to the iliac artery. When pressure is applied to the vessel complex, the higher pressure of the artery keeps it from collapsing while the pressure collapses the vein, causing distention in the veins of the legs. This interferes with the muscular pump of the legs for effective venous return. Some investigators have indicted the pressure of straining at the stool,

causing high vena cava pressure, as a cause of the breakdown of the valves in the veins of the legs. The venous stasis could predispose an individual to phlebitis and consequent thrombosis.[55]

Cholesterol Level

Bacteria in the dysfunctioning colon act upon bile acids to form lithocholate, which acts back on the liver and cuts down the conversion of cholesterol to bile acids. As less cholesterol is eliminated by the liver as bile, it builds up in the bloodstream.[400,401]

Improving colon function by increasing fiber and reducing refined carbohydrates changes the type of bacteria that dominate the colon. The improved flora do not attack the bile and break it down to lithocholate the same way as the bacteria that dominate the abnormally functioning colon. Furthermore, the bile salts are removed through the bowel much more rapidly in the healthy colon and are eliminated from the body with less time for the bacteria to work on them for conversion to lithocholate.

Heaton[191] presents compelling evidence that gallstones form as a result of the liver secreting an excess of cholesterol into the bile and/or a deficiency of the accompanying bile salts that normally hold the cholesterol in solution. He has advanced the theory that these conditions of the bile arise from the consumption of refined carbohydrates. Also, lack of fiber causes an intestinal stasis, allowing extra bacterial conversion of a major bile salt, chenodeoxycholate, into lithocholate, a substance believed capable of depressing bile salt synthesis by the liver — hence the solubility of cholesterol. This also relates with the hypercholesterolemic effects of low roughage noted previously.

Diverticulosis and Diverticulitis[83]

When the diet is highly refined, there is slower passage of the colon contents and increased contractions of the colon to move the hard, dry material. Peristalsis along the narrow colon stretches and eventually causes the pouches of diverticulosis.

It is a short step to diverticulitis as a result of a piece of hardened fecal matter or a fecalith blocking the opening to the diverticula. Inflammation, infections, and abscesses can occur. The same etiology is associated with appendicitis.

The highly refined carbohydrate diet most often present with the loss of fiber frequently causes adrenal stress disorder, as discussed later in this chapter. As a result, there may be is a lack of mineralocorticoids and glucocorticoids that are the pro- and anti-inflammatory hormones of the adrenal; thus there is less natural ability to resist the inflammatory processes that may develop in the colon. In addition to making dietary changes and treating colon dysfunction, one should evaluate the adrenal, and treat and support it if necessary.

Obesity

Although obesity is commonplace today, it has not always been so. In fact, even today in some tribal areas of Africa and other areas where an unrefined diet is consumed, obesity is almost unknown. Although not the only ones, there are two important causes of obesity today as a result of the popular refined diet and decreased roughage. First are the so-called "empty calories" that add to the body's calorie count but provide no material for building healthy tissue. These items are primarily white sugar, white flour, and alcohol. The second cause is a lack of fiber in the diet. A fibrous diet that includes raw vegetables and fruit requires more chewing effort and slower intake; this consequently satisfies an individual with lower caloric consumption. The additional roughage in the diet causes more fat, a high-calorie item, to be excreted in the stool.[83] The amount of chewing necessary for a high-roughage diet increases the amount of saliva and digestive juices mixed with the food. The improved digestive function and bulk give an individual a feeling of fullness that satisfies the hungry feeling.

Infection from Bowel Dysfunction

There is extensive documentation that appendicitis was virtually unknown in Africa until the introduction of the western diet. In a South African study of 18-to-20-year-old senior students, the rural blacks versus the urban whites had 0.5% versus 16.5% incidence of appendicitis, respectively.[55]

Cleave[83] has found epidemiologic and historic evidence suggesting that E. coli infections, such as appendicitis, primary infections of the urinary tract, and diverticulitis, are the result of ingestion of large quantities of refined carbohydrates and a lack of fiber in the diet. Stasis, because of the lack of fiber and the culture medium provided by the refined carbohydrate, accounts for the change in the colon flora. During three years of treating these conditions in the Royal Naval Hospital in Hong Kong, Cleave changed the diet of patients with these conditions to one high in fiber and low in refined carbohydrates. There was a subsequent elimination of the infections. This treatment was provided before the days of antibiotics.

Dietary Change

The major key to the previously discussed health problems resulting from colon dysfunction lies in increasing the fiber content of the diet and reducing refined carbohydrates. It is less difficult today to get patients to change their diets. A story previously told in the chapter on nutrition bears repeating. Thirty-five years ago I was using the approach recommended here. On several occasions when I recommended to patients with colitis and diverticulosis that they change from their prescribed bland diet to one high in roughage, their allopaths strongly voiced their disapproval, stating that I was less

than an intelligent individual. One physician even said that if the patient changed to my recommended diet, it would surely kill her. Fortunately, the patients followed my recommendations, primarily because they were getting worse on the bland diet; all had improved function with the change. Today anyone who has watched television is aware that the American Cancer Institute recommends the high-fiber diet I was recommending long ago. In addition, two of those doctors who are still practicing now recommend the approach with which I corrected their patients over thirty years ago. As time moves on we see some positive changes regarding nutrition, but they are much too slow.

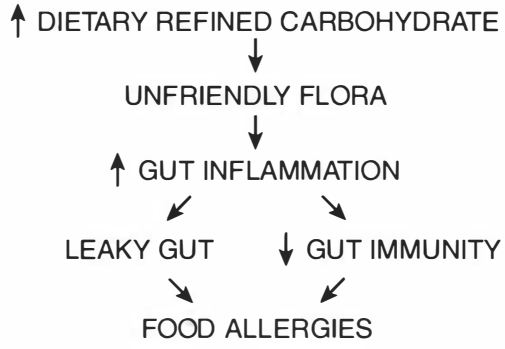
Although many people are more aware of nutrition today, it is still often difficult to get patients to change to an optimal diet. Many have been raised on refined and ultra-processed foods. Many of these foods are high-profit items and are thus widely advertised and promoted in the supermarket.

Dietary Roughage. The easy answer is to include what have previously been considered "waste products" from grains in our diet. Bran can be purchased very economically from any health food store, and some types of bran are included in the supermarkets' cereal section. Bran can be added to water or juice and consumed as a drink, or it may be mixed with such foods as meat loaf and cereals. It is relatively easy to get into the habit once a week of making bran muffins or some other bread items, supplying enough for the entire week.

The amount of bran necessary for normal colon function varies with individuals and their diets. Start with one heaping teaspoon three times per day and increase until the desired results are obtained. Some individuals will obtain very good results with only three teaspoons per day; others will require many tablespoons per day. The basic criterion is that the individual excretes a well-formed soft stool on a daily basis. No odor to the stool is a classic criterion for adequate bowel action. This gives indication of the bacterial content, as well as whether putrefaction is taking place.

When a person first starts using bran, he may become gaseous for a week or so. If the gaseousness is extremely objectionable, he should cut down on the amount of bran but not exclude it from his diet. As the bowel becomes more effective in its function, the gaseousness will diminish and the bran can be increased.

Refined Carbohydrate. White sugar, bread, and other highly refined carbohydrates set up a medium in the gut for proliferation of unfriendly bacteria, fungi, and other imbalances called dysbiosis. Eventually the gut health deteriorates and leaky gut with body toxicity develops. Decreased gut immunity may lead to allergies. Usually adrenal stress disorder is associated with the high refined carbohydrate diet. Examining for and correcting elevated cortisol:DHEA ratio as discussed later in this chapter is almost always necessary with colitis and the other inflammatory conditions of the colon.



12—5. After Schmitt, with permission.[366]

Post-Antibiotic Therapy

Antibiotics, unfortunately, are incapable of determining good bacteria from bad. When antibiotics are prescribed for an infection, they also affect the flora of the intestines. It is sometimes necessary after antibiotic therapy has been used to re-implant bacteria or provide a better culture medium for their growth. Lactic acid yeast is an excellent nutritional support for proper bacterial growth. Some lactic acid yeast products contain bacteria culture. A good quality product is dated, indicating the maximum shelf life, to ensure good bacterial count, and requires refrigeration for storage. Unsweetened yogurt provides a good culture medium for improving the flora.

Adrenal Stress Disorder†

One of the most common conditions encountered in a natural health care practice is adrenal stress disorder. Nearly everyone who has been ill or under other types of stress for any length of time will have this problem to some degree. Although it is often encountered by those aware of the condition, it is frequently overlooked in general health care practices. Many physicians do not recognize the condition because they are unaware of it. In general, endocrinologists do not consider an adrenal problem to be present unless an individual has Addison's disease or Cushing's syndrome. In fact, in a "Statement on Hypoglycemia," the American Diabetes Association, the Endocrine Society, and the American Medical Association state, "The diagnosis of [adrenal insufficiency] requires sophisticated measurements of adrenal hormones in blood and urine." They further state that it is "...an uncommon condition, [and] is a

† For additional subject information see Schmitt, *Adrenal Stress Diagnosis and Treatment Option.* Video program.[367]

rare cause of hypoglycemia. The treatment of adrenal insufficiency has been clarified and made routine by the discovery of cortisone and similar hormones over twenty-five years ago."[8] In this statement they are obviously talking about Addison's disease or other frank pathological conditions. This fails to take into account the condition discussed here as adrenal stress disorder that causes an imbalance of the hormones necessary for the body to function in an optimal manner. There is a continuum between frank disease and optimal function. An individual somewhere between the two extremes may not have disease, but the body still fails to function properly.

Unfortunately, much health care in the United States is directed toward crisis situations. The adrenal gland's function is a good example of this. With Addison's disease one is concerned with a life or death situation, whereas with adrenal stress disorder one is concerned with an adrenal gland that is not functioning to meet the demands of the body and promoting a high level of health.

Adrenal stress disorder is often caused or aggravated by factors commonly encountered in most people's current life-styles. In some respects, the widespread incidence of adrenal stress can be considered a disease of modern civilization.

The adrenal glands — located on each of the two kidneys — can be considered part of the body's military system. They instigate and direct reaction to stresses of all kinds, which may be intrinsic or extrinsic.

Triad of Stress

Selye[375] observed as early as 1925, while he was still a student in medical school, that there are common symptoms present in nearly all diseases. Regardless of the type of infection, most individuals have fever, an enlarged spleen or liver, inflamed tonsils, skin rash, and other symptoms. Looking at these common involvements — regardless of the patient's condition — Selye classified the situation to himself as the syndrome of "just being sick."

Selye later discovered a triad that is always present in the "just being sick" syndrome. His research was directed toward subjecting rats to various types of stress. After sacrificing them, dissection revealed a triad that consisted of (1) adrenal cortex enlargement, (2) atrophy of the thymus, spleen, lymph nodes, and all other lymphatic structures, and (3) deep, bleeding ulcers in the stomach and duodenum. Selye was able to determine what constituted stress by subjecting the rats to a wide variety of noxious stimuli. Rats were put in water and forced to continually swim to stay alive. They were frustrated by having their legs tied down, or by being put into a cage next to an angry cat. The rats were subjected to various types of chemical stimuli, placed in rarefied air, or confined in quarters where they had to live with their own excrement. All these factors and many more were evaluated and found to create the triad of stress.

Selye's studies help explain the many causes of adrenal reaction, helping to put in perspective the frequently found adrenal stress disorder. I have classified the forms of stress to facilitate explanation to patients. The four categories of stress include physical, chemical, thermal, and emotional, but they are not inclusive of all types of stress.

Types of Stress

1. **Physical**. Long hours of work, heavy physical exertion, loss of sleep, and postural and general structural strain are all primary in this category. Postural and localized structural strain are of primary importance, correction of which relates closely with applied kinesiology and chiropractic. Almost any type of trauma is a physical stress. Proper treatment in the acute phase and rehabilitation are important in removing stress to the adrenal glands.
2. **Chemical**. Chemical stress can be nutritional in nature, occurring when an individual eats an imbalanced diet or excessive amounts of refined foods. Stimulants such as coffee, tea, colas, chocolate, nicotine, alcohol, drugs (including over-the-counter, prescription, and recreational) drive the adrenal gland. Food additives, such as food colorings, preservatives, and artificial sweeteners, contribute to making a nutritional problem worse. The poisons that are in our environment — including air pollution from hydrocarbons, insecticides, and general industrial waste — are almost impossible to avoid. This includes urban areas and farmland where insecticides, weed control, and artificial fertilizers are used in abundance.

   Imbalance of the gases we breathe is a chemical stress. Until studying Selye's work, it was difficult for me to understand why my adrenal-involved patients who were doing quite well developed acute manifestations when going on vacation to the mountains. I accused these patients of not following their diets and not taking their nutritional supplements, only to have them deny my accusations. Selye found that rats put into an environment with a lowered oxygen level developed the triad of stress. My patients were simply being stressed as they went to higher altitudes, thus having less oxygen to breathe. Since recognizing that factor of stress, I have found that many individuals can determine when they reach a particular altitude simply by the way they feel. The patient's symptoms act almost as accurately as an altimeter.
3. **Thermal.** Thermal stress can be either overheating or overchilling. Adrenal function is partly responsible for adaptation to thermal changes. Sometimes the change in the body may be extreme, such as when an individual gets into a car that has been closed in the summer sun. Immediately the body

is subjected to an extremely high temperature. The individual usually rolls down the windows or turns on the air-conditioning to have cool breezes brushing over the perspiring skin, causing a rapid shift in the temperature to which the body is subjected. Going in and out of air-conditioned places or food coolers that may be a condition of employment for many food or grocery store workers may be a constant unavoidable daily stress.

Hot baths or showers often taken when a person has a lot of body pain activate the adrenal glands. Swimming for exercise in a cold pool or going from a hot tub to a cold plunge creates thermal stress.

Some individuals who are close to symptoms as a result of hypoadrenia may aggravate the condition by simply going outside for a short time, e.g., taking out the trash on a cool spring morning when one would not think a sweater necessary.

4. **Emotional.** This has been one of the most difficult categories to mitigate in patients. Improved applied kinesiology techniques to deal with emotional stress have helped us understand and correct these causes of adrenal stress. Often nothing can immediately be done about emotional stress, such as the death or prolonged illness of a loved one. More insidious, but often lasting over a longer period, are situations in which an individual is deeply in debt and in a job that provides a fixed salary with no opportunity for advancement. This person would be in a much better position if he could control his income and do something about the emotional stress of his indebtedness.

There are many situations in modern life in which the body cannot function as it is designed to handle stress. A natural function of stress is to prepare an individual to meet the demands placed upon him. For example, early man walking through a jungle sees a saber-toothed tiger. He runs for his life and finally ends up sitting in a tree with his heart pounding and the blood circulating through the adrenals, giving an opportunity for them to rebuild. The long-term, never-ending emotional and chemical stress often found in modern society creates adrenal stress disorder, primarily because it does not give the adrenal glands a chance to rebuild.

Stress is cumulative. If an individual has mild stress in all four categories, the accumulation is significant enough to create the triad of stress and the subsequent symptoms that develop. The chiropractic profession[122,422] and others have taken steps to combat the development of stress in the workplace, in personal interaction, and from the environment. Understanding the sources of stress and their cumulative nature allows one to recognize the problem in patients and recommend appropriate life-style changes and therapeutic procedures.

General Adaptation Syndrome

Earlier it was mentioned that there is a continuum between ideal adrenal function and frank disease processes, such as Addison's disease. Selye[376] classified the progression of stress on the body and its influence on the adrenal gland and other bodily functions. These classifications, called the "General Adaptation Syndrome (GAS)," help put the progression of a patient's condition in perspective. The three stages of the general adaptation syndrome are the alarm reaction, resistance, and exhaustion.

Alarm Reaction is the first stage of response; it is a generalized call to arms of the defense mechanism. The adrenal cortex creates its hormones to the point of depletion.

The alarm reaction can be extremely rapid and responsive. This reaction is the mechanism that enables a 98-pound mother to lift an automobile off her baby. It gives the after dinner speaker the ability to think faster on his feet and deliver an exciting lecture. A student taking a critical examination will perform better because of the stress (if his adrenal glands are functioning adequately, which often they are not).

Resistance Stage. Under conditions of continual stress, an individual moves from the alarm reaction into a stage in which the body prepares to continue the fight of adaptation on a prolonged basis. This is the stage that begins the adrenal hypertrophy and other factors of the triad of stress. As the body attempts to meet the demands placed upon it, imbalance of adrenal hormone production begins. With chronic stress the imbalance worsens, and dysfunction is manifested in loss of health. This is the category most often revealed in our patients by the adrenal stress index test discussed later.

Exhaustion. With prolonged stress, the adrenal glands eventually can no longer meet all the demands placed upon them. They reach a point of depletion, similar to the end of the alarm reaction before resistance develops. The general adaptation syndrome is often in the state of exhaustion in an individual who has had a chronic health problem, long-term nutritional deficiency, and/or long-term emotional problems.

Our modern environment and social cultures force the adrenals to react to stress on an almost continual basis. If enough time for recuperation is allowed between bouts of stress, the glands are again ready to handle each task. If, however, the stresses accumulate, the glands ultimately become depleted; in other words, the third stage of the general adaptation syndrome develops. As the problem continues, it takes longer and longer for the adrenals to respond to pressure put upon them. There are many degrees of adrenal stress disorder. The condition may only mildly affect an individual, or it may create severe health problems.

Hereditary Factors

All people have biochemical individuality.[421] A major factor in how individuals react to stress is their glandular heredity. The physical characteristics of an individual, such as large nose, hair color, and other body features, are easily recognized as inherited. The heredity of glandular balance is not usually recognized by the general population; however, the characteristics created by the glandular balance are obvious when one is familiar with the associations of a strong or weak adrenal, thyroid, or pituitary gland.

Bieler[31] lists the physical characteristics of the typically strong adrenal-type person:

Hair:	Of head, coarse and often curly; of body, coarse and thick, often characterized by a "hairy ape" appearance.
Features:	Coarse, large, and heavy.
Eyes:	The iris shows ample pigmentation, either dark blue, brown, or black; the pupil is small and reacts instantly.
Forehead:	Low, usually with a low hairline.
Nose:	Well-developed, with large nostrils.
Lips:	Full, with strong color and warmth due to ample circulation.
Teeth:	Large, especially the canines, yellowish in color, extremely hard and resistant to caries; the dental arches are full and round; the third molars usually erupt normally.
Tongue:	Thick, wide, and clean; papillae coarse and thick.
Palate:	Low-arched and wide.
Skull:	Wide across the temples. Lower jaw heavy, solid, and often protruding.
Ears:	The lobe thick, large, and long.
Skin:	Thick, dry, and warm.
Neck:	Thick and short; the characteristic "bull" type.
Chest:	Broad and thick, containing large heart and lungs.
Abdomen:	Wide, thick, and often protuberant.
Genitals:	Large.
Extremities:	Thick and short; fingers and toes stubby; nails short, thick, and with moons small or lacking.

When one is familiar with the features and physical characteristics of various glandular patterns, it is easy to recognize those who are subject to hypoadrenia, hypothyroidism, and other glandular problems. Considering the subject in another perspective may help this recognition. Animals are selectively bred to develop specific characteristics that follow glandular patterns. The draft workhorse has relatively short legs, large muscles, and heavy, coarse hair, characteristic of the strong adrenal type. On the other hand, the racehorse has long legs, sleek muscles, and finer hair, characteristic of a strong thyroid. The personality of the animal also reflects the glandular balance. The workhorse is docile and easy-going compared to the fidgety, excitable racehorse.

Adrenal Physiology

The adrenal gland is first recognizable in the embryo at the sixth week.[178] At birth the gland is about one-third the size of the kidney, whereas in the adult it is only about one-thirteenth. The adult gland is little larger than it is at birth.[420] The almost full adult development of the adrenal glands at birth seems to indicate how essential they are for normal body function.

There is some evidence that the adrenal glands of the fetus can supplement the mother's system through the placenta when she is in the resistance stage or adrenal exhaustion. This is probably the reason that postpartum depression develops. If the mother is in a chronic state of adrenal exhaustion, the child can be born with a similar adrenal exhaustion, subjecting the infant to allergies and other problems related to adrenal stress. The answer, of course, is to keep the mother functioning in a balanced state throughout her pregnancy for both the mother's and child's health.

The adrenal glands are comprised of two parts. The internal portion, or medulla, produces epinephrine and norepinephrine, which are collectively called catecholamines. Surrounding the medulla is the adrenal cortex. It is responsible for producing the corticosteroids, of which there are three basic categories: the mineralocorticoids, glucocorticoids, and small amounts of sex hormones.

Medulla

Epinephrine (adrenalin) and norepinephrine (noradrenalin) are secreted by the adrenal medulla. It receives stimulation by way of the sympathetic splanchnic nerves.[404] The small splanchnic nerves supplying the adrenal medulla make no ganglionic connection prior to their termination; thus, the nerve supply is entirely cholinergic. In the adult human, epinephrine is found in five times greater supply than norepinephrine.[30] In prenatal life, the adrenal contains only norepinephrine; after birth epinephrine appears. The two hormones are present in approximately equal proportions by about one year of age. Epinephrine gradually increases until it becomes the major amine of the adult adrenal.[404]

The secretions of the adrenal medulla have a wide

effect on body function. Epinephrine is responsible for increased uptake of amino acids into tissue cells from the bloodstream, and for glycogenolysis. Adrenal cortical secretions are increased by sympathetic stimulation.

The adrenal medulla is best known for its influence on the autonomic nervous system. The sympathetic and parasympathetic nerve endings secrete one of two neurotransmitters, acetylcholine or norepinephrine. Fibers that secrete acetylcholine are said to be cholinergic, and those secreting norepinephrine are adrenergic. Both pre- and post-ganglionic neurons of the parasympathetic system are cholinergic, while the pre-ganglionic neurons of the sympathetic system are cholinergic and post-ganglionic neurons are adrenergic. The innervation to most of the sweat glands and to a few blood vessels is an exception to post-ganglionic neurons being adrenergic; in this case, they are cholinergic.

The adrenal medulla receives sympathetic innervation through a branch of the greater splanchnic nerve. The nerve fibers go directly to the medulla without synapsing, so they are entirely pre-ganglionic. At the medulla, the neurons end directly on special cells that secrete epinephrine and norepinephrine directly into the circulatory blood. The secretory cells are derived from the neural crest and are analogous to post-ganglionic neurons.[187]

When there is sympathetic stimulation of an organ, norepinephrine is released at the terminal nerve ending to cause action. Simultaneously, norepinephrine and epinephrine are released from the adrenal medulla, to be distributed by the blood vascular system. The target of the sympathetic stimulation is thus activated by two mechanisms, providing a safety factor should one mechanism fail. Total loss of the two adrenal medullae does not stop action; rather it is slowed.[30] On the other hand, destruction of the sympathetic pathway to the organs does not stop action because norepinephrine and epinephrine are still released into the circulating fluids and indirectly cause stimulation.[187]

Activity of the adrenal medulla is important in the "fight or flight" mechanism. There is stimulation to functions that are needed to meet stress, whether one fights it or runs from it. Epinephrine's effect of increasing glycogenolysis provides more sugar for energy. The medulla stimulates the cortex, which additionally provides more sugar and releases the pro- and anti-inflammatory hormones for repair should one be injured. Heart rate and blood pressure increase, and digestive processes are inhibited. Digesting food just eaten is for energy at a later time; it is not necessary to meet this emergency. It is the depression of the digestive system that gives an individual the feeling of a knot in the stomach at an emotionally upsetting time.

Epinephrine excites the radiating muscle of the iris, which in turn dilates the pupil of the eye during stress.[30] As will be noted later during the examination discussion, a common finding in relative adrenal insufficiency is dilation of the eye pupil. Because of the innervation, one would think that the pupil would constrict rather than dilate. The reason for the dilation in some cases of relative hypoadrenia is the increased intracellular level of potassium. Potassium inhibits the cholinergic nerve fibers. Since the sphincter muscle that constricts the pupil of the eye is cholinergic, there may be an inhibition of its action causing the dilation during relative adrenal insufficiency. In other cases the response of the pupil to light stimulation may be a sign of sympathetic stress response. Because of the mixed response the body may have to adrenal stress, the pupil response to light must be interpreted with all factors being considered.

Another finding that will be discussed in the examination section relates to the drop in blood pressure on standing, observed in relative hypoadrenia. Normally the splanchnic vessels are constricted to maintain or raise blood pressure in the upright position.

In general, epinephrine is the body's tool for alerting and mobilizing the circulatory, nervous, and muscle systems to combat stress. Fantastic feats of strength have been recorded, such as a woman lifting a car off her child. This, of course, is an extreme example of emotional stress resulting in adrenal gland function. Many activities we perform throughout the day that may be emotional or physical require some adrenal action. The simple process of standing from a lying position depends on effective adrenal function to raise the blood pressure slightly, combating the effect of gravity. Most of the problems we deal with from day to day are not severe adrenal crises; rather, they are mild stresses to which the body must react.

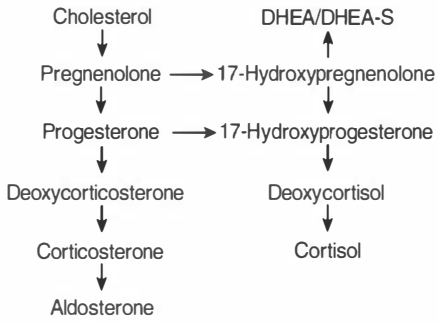
The most common clinically observed entity is mild to moderate adrenal insufficiency. If an individual has not reached the stage of frank adrenal exhaustion, he will be able to react to stress; however, it will take longer and will be less efficient. The individual with poor adrenal function needs to be made aware of the difficulty his body has in adjusting to stress. The four types of stress previously discussed have their cumulative effects. As many of these stresses as possible must be eliminated while treatment is administered for adrenal rehabilitation.

Cortex

The hypothalamic-pituitary-adrenal axis (HPA), together with the arousal and autonomic nervous systems, constitutes the stress system.[80] The principle activator is corticotrophin releasing factor (CRF) from the hypothalamus that causes release of adrenocorticotrophic hormone (ACTH) from the pituitary, in turn releasing the corticosteroids from the adrenal cortex.

The corticosteroids are derived from cholesterol from which pregnenolone is formed. From pregnenolone there are steps to finally form the major corticosteroids of aldosterone, cortisol, and dehydroepiandrosterone (DHEA,) which are the primary mineralocorticoids, glu-

cocorticoids, and sex hormones, respectively. The hallmark of adrenal stress disorder is increased production for one of the corticosteroids and a deficiency of one or more of the other corticosteroids. The deficiency develops because there is not enough pregnenolone available to meet all demands. The common problem is an increase of cortisol and decrease of DHEA.



12—6. Pregnenolone is the precursor for both cortisol and DHEA. After Hechter, et al.[192]

Since the plasma half-life of DHEA is less than 30 minutes, over 95% of circulating DHEA is in sulfate form (DHEA-S).[343,344] DHEA-S provides a readily available source of DHEA for the production of estrogens and androgens in the adrenal glands, ovaries, and testes. Throughout this discussion the acronym DHEA refers to both DHEA and DHEA-S unless referring to a specific reference to DHEA-S.

Many of the symptoms that develop in adrenal stress disorder relate to disturbance of the immune system. DHEA is a precursor of androgens and estrogens and has multiple immunomodulating effects.[106,286,302,394] In addition to immune problems there are many health problems that arise from adrenal stress disorder that must be recognized. Common symptoms are structural instability, decreased immune function, digestive problems, dysbiosis, blood sugar handling stress, sleep disturbances, headaches, liver inactivity, fatigue, and asthma. Often the symptoms are treated rather than the cause, when treatment should be directed to balancing production of the corticosteroids. Treatment to improve adrenal function should not create further imbalance just to obtain relief of the patient's symptoms.

Mineralocorticoids. The mineralocorticoids consist mainly of aldosterone, corticosterone, and desoxycorticosterone. Aldosterone is of primary importance. It is produced in the zona glomerulosa, which is a thin superficial layer of the adrenal cortex. This layer functions almost entirely independently of the deeper cells. Aldosterone secretion is based on electrolyte concentrations, extracellular fluid volume, blood volume, arterial pressure, and special aspects of renal function. ACTH has little control on the rate of aldosterone secretion, but it is necessary for secretion.[187]

Aldosterone is responsible for the reabsorption of sodium in the distal convoluted tubules of the kidneys and the renal excretion of potassium. Loss of this function causes death within three days to one week, unless the person receives extensive salt therapy or mineralocorticoid therapy. This occurs because of a marked loss of water from the extracellular fluid and eventually from the blood volume, which in turn leads to a decreased cardiac output and then shock. A loss of one-fourth of the body's water is usually fatal.[30] These effects of frank Addison's disease or adrenalectomy without salt replacement therapy indicate the importance of mineralocorticoid production. The adrenal stress disorder highlighted in this text may produce a reduction of mineralocorticoids, causing a persistently low blood pressure and ion imbalance.

Aldosterone, which accounts for 95% of the action of the mineralocorticoids, increases the reabsorption of chloride ions, as well as sodium ions. Regulation of aldosterone's secretion depends on potassium and sodium concentrations, renin-angiotensin system, and ACTH. When potassium ion concentration in the extracellular fluid increases, aldosterone production increases to return the potassium ion concentration to normal. After several days of a sodium-deficient diet, an individual's aldosterone secretion increases markedly, although the sodium ion concentration of the body fluids does not fall significantly. The exact reason for this is unknown.

When renin is released from the kidney, there is an increased rate of angiotensin-II, which stimulates the adrenal cortex to produce aldosterone. Aldosterone, in turn, increases the extracellular fluid level, which inhibits continued renin release.[428]

In adrenal stress disorder or other causes of corticosteroid imbalance, there may be an inadequate production of mineralocorticoids. In the case of adrenal stress disorder it is because there is not enough pregnenolone to satisfy the production of all corticosteroids needed. The distal convoluted tubules reabsorb less water than is needed by the body. The blood pressure may drop with decreased volume. In more severe cases, the skin and other tissues requiring constant fluid bathing may be depleted. The symptoms of dehydration may occur with a rise in temperature, hot, dry skin, flushing, and excessive thirst. The electrolyte imbalance that occurs varies in degree.

Acidosis may occur when a deficiency of aldosterone secretion exists. Too little sodium ion reabsorption causes a deficient amount of ion secretion in the bicarbonate buffer system, which in turn causes spillage

of bicarbonate ions into the urine, resulting in acidosis. An elevated potassium level creates the same effect. The major effect of acidosis is depression of the central nervous system. Metabolic acidosis in this case causes an increased rate and depth of respiration. If the adrenal glands are not thoroughly depleted, the heart rate may tend to increase in an attempt to compensate for the decreased blood volume and resultant decreased oxygen supply to the tissues. This takes place in response to the stress of acidosis, which causes adrenalin to be released from the adrenal medulla. If this compensating action does not take place, as in the exhaustion stage, the acidosis will not be corrected.

Potassium is an intracellular ion; sodium is an extracellular ion. Under normal conditions, the intracellular fluid has a high concentration of potassium and a low concentration of sodium, whereas the extracellular fluid contains a high sodium level and low potassium level.[30] The retained potassium and loss of sodium in hypoadrenia result in an osmotic pressure shift from the normal isotonic condition to a hypertonic intracellular condition. Water is then drawn into the cells from the hypotonic extracellular fluid, resulting in intracellular edema.[187] If this edematous condition is clinically observed, the doctor (or patient) is tempted to restrict the individual's salt intake; unfortunately, salt restriction in this condition promotes further intracellular edema. The patient edematous from this condition should have increased salt intake in addition to correction of the adrenal stress disorder. The applied kinesiology examination technique described later helps determine when salt or other factors are needed.

Glucocorticoids. The main glucocorticoid is cortisol, which provides 95% of glucocorticoid activity. Corticosterone provides about 4% of the activity and is much less potent than cortisol. Cortisone is synthetic and almost as potent as cortisol. Prednisone, methylprednisone, and dexamethasone are also synthetic and are four, five, and thirty times more potent than cortisol, respectively.[187]

The glucocorticoids stimulate gluconeogenesis in the liver by increasing the transportation of amino acids from the extracellular fluid into the liver for conversion into glucose. They also mobilize amino acids from tissues, especially muscle. Finally, they mobilize the enzymes necessary for gluconeogenesis in the liver.

Adequate cortisol production is necessary to maintain adequate blood sugar levels through conversion of protein and fatty acids to sugar in the absence of recent sugar ingestion. Cortisol also helps raise the blood sugar level by decreasing the rate of glucose use by the cells. The decreased use is accomplished by depressed oxidation of some of the mechanisms important in glycolysis. Cortisol also slightly decreases glucose transport into the cells.

Gluconeogenesis and glycogenolysis occur during fasting periods, usually beginning approximately four hours after eating. This is when the blood sugar usually drops to a low level and new stores of glucose are needed. The adrenal cortex is stimulated by ACTH from the anterior pituitary gland, which in turn is stimulated by the corticotrophin-releasing hormone from the hypothalamus. Dietary sugar is absorbed through the walls of the small intestine to trigger the pancreas to secrete insulin. Insulin transports the glucose into liver, muscle, and other tissue cells, where it is raw material for energy production. When the cells have received all the glucose they need, the remaining glucose enters the liver and muscles to be stored as glycogen. The remaining carbohydrate is rapidly converted into triglycerides for storage in this form. When the blood sugar level decreases, it is the responsibility of the glucocorticoids to break down triglycerides into blood sugar.

A person with adrenal stress disorder either fails to return the blood sugar level to normal, or it is delayed. Because of the waning blood sugar level, the individual wants to eat something, especially carbohydrates. The ingestion of carbohydrates, particularly refined ones, will cause a roller coaster effect with further depletion of the blood sugar level. (This is discussed in the section on blood sugar handling stress.) The constant effort of the body to stimulate the adrenals seems to put them further into the third stage of exhaustion.

Sex hormones. The third category of hormones secreted by the adrenal cortex is the sex hormones, which are given much less coverage in the literature. Male and female hormones are secreted in both men and women. Both testosterone and estrogen are secreted in minute quantities as compared with the production from the testes and ovaries.

It is of interest that these hormones are secreted by the adrenal cortex. Some have commented in the literature that they have no influence on health unless a tumor develops and causes excessive production. Those in natural health care generally recognize that the body does not have unimportant organs, glands, or functions.

One of Selye's[376] first observations in the general adaptation syndrome was that animals under prolonged stress developed sexual derangements. The ovaries and testicles shrank and became less active in proportion to the enlargement and increased activities of the adrenals. Selye speculates that as ACTH production increases for adrenal function, there is a reduction of the gonadotrophic hormones by the pituitary. This seems reasonable, because intense stress causes young animals to cease to grow and lactating females to produce no milk. The stress of training in elite female athletes causes amenorrhea.[349]

Pottenger and Simonsen[323] observed a high frequency of undescended testicles in children being treated for asthma. They found a history of thyroid or so-called "ovarian trouble" in well over 50% of the mothers of these children. When the boys with cryptorchidism were treated with full adrenal gland extract given orally, the

testes descended and there was improvement in the asthma. The average time for testicle descent following treatment was about nine months.

Prolonged stress may be partially or totally responsible for amenorrhea in female athletes who are under intense training.[50] There appears to be a reduction of female hormones in combination with the stress of training,[94] which may relate with what Selye calls the "shift in pituitary hormone production."

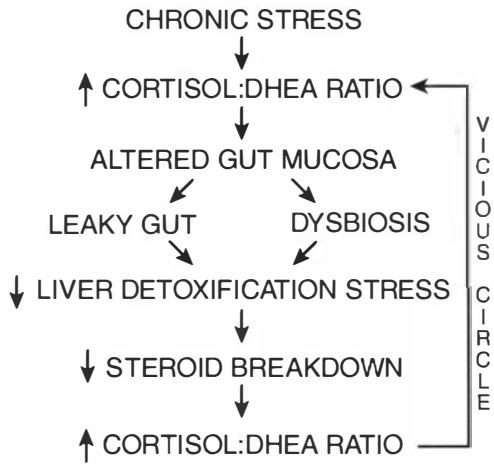
Roberts[340] attributes 85% of estrogen and progesterone production to the ovaries and 15% to the adrenal cortex in women of child-bearing age. He points out the importance of adrenal production of these hormones when the ovaries cease their production with menopause. Clinically it is observed that women with hypoadrenia have much more difficulty with menopause than those with optimal adrenal function. In many cases, treatment toward improving the adrenal function eliminates the symptoms of menopause.

Pro- and Anti-inflammatory Hormones. The mineralocorticoids have a pro-inflammatory effect, and the glucocorticoids have an anti-inflammatory effect.[376] The body regulates the ability of tissues to undergo inflammation in response to local injury and to contain it properly by maintaining a proper balance of these hormones. The mineralocorticoids promote local inflammation as part of the healing process, while the glucocorticoids build a wall around the problem area to separate it from the rest of the body. When there is poor adrenal gland function, the body may not be able to maintain a proper balance between these hormones. Any susceptible area of the body may suffer. The manifestation may be seen as rheumatoid arthritis, colitis, duodenal or gastric ulcers, rhinitis, sinusitis, bronchitis, hay fever, asthma, chronic upper respiratory infections, skin rashes, and/or any other inflammatory disorder that does not serve a useful function.

Vicious Circles

Adrenal stress disorder often starts a series of vicious circles that continue on to blood sugar handling stress and interaction between the liver, adrenal, joints, and sulfation, discussed later. Initially stress causes an increase in cortisol production as the alarm reaction, a normal reaction to respond to the stress. When the stress is no longer present because it was effectively dealt with, the hypothalamus-pituitary-adrenal axis returns to homeostasis, ready for the next time a response to stress is needed. If the stress becomes chronic, the response is to move into the resistance stage with continued increased production of cortisol. With greater chronicity a point is reached where there is not enough pregnenolone to produce cortisol and DHEA, and cortisol:DHEA ratio increases.

Reduced DHEA causes the immune system to become inefficient[106,286,302,394] and bowel conditions develop, leading to intestinal permeability and dysbiosis. The toxicity from these conditions puts a heavy load on the liver's detoxification systems. The resulting liver overload reduces its ability to detoxify the steroid hormones, resulting in an even greater increase of the cortisol:DHEA ratio.



12—7. The beginning of vicious circles that develop from chronic stress. After Schmitt, with permission.[366]

Symptoms and Conditions

Adrenal stress disorder displays itself in a variety of ways, such as asthma, chronic upper respiratory infections, hay fever, skin rash, ulcerative colitis,[393] Crohn's disease,[393] gastric or duodenal ulcers, rheumatoid arthritis,[110,306] immune problems,[309] insomnia, headaches, fatigue, fainting spells, female reproductive problems,[80] obesity,[12] heart palpitations, edema, learning difficulties[232] — the list could go on and on. Emotions are often involved, manifesting from mild depression to suicidal tendencies.[313] Numerous female conditions are the result of interactions between the hypothalamic-pituitary-adrenal axis, including amenorrhea, postpartum depression, premenstrual syndrome, anovulation, and climacteric depression, among others.[80] Many children's conditions are rooted in adrenal stress disorder, including attention deficit hyperactivity disorder (ADHD),[232] autoimmune conditions, asthma, and depression.[313] Early detection of adrenal stress syndrome may eliminate or stop the destructive progress of rheumatoid arthritis[306] and other inflammatory diseases. Does successfully treating adrenal stress disorder sound like a panacea? It certainly may, but remember — the triad of stress described by Selye is a condition that accompanies many other health problems.

Sometimes symptomatic treatment of the primary health problem contributes to further adrenal stress. Many who suffer from adrenal stress disorder have sought help for their problems, and have been given tranquilizers or mood elevators, analgesics for pain, sedatives for insomnia, amphetamines and diuretics for obesity, anti-cholinergics and a bland diet for ulcers, a

bland diet for colitis, antihistamines and bronchial dilators for asthma, as well as a suggestion to move to a drier climate. They are often individuals who have had extensive examination work-ups with no cause found for their symptoms; therefore, the victims of adrenal stress disorder are often given treatment to diminish the symptoms rather than to eliminate the cause. These people are, as Selye said, "just being sick."

Examination

A good practice is to examine all new patients for adrenal stress disorder because of its prevalence in people seeking health care. The condition may be masked on the first few visits to a new doctor because of the tension and stress often associated with experiencing a new doctor, and anxiety about the examination and new procedures. Under these conditions the hypothalamus-pituitary-adrenal axis will probably meet this crisis unless it has advanced into exhaustion. The applied kinesiology examination will find the problem even though it is in a covert stage.

Postural Hypotension. The terms "postural hypotension" and "orthostatic hypotension" are used interchangeably. The choice used here is the one used by the authors of the various texts cited. Ragland's sign is an abnormal drop in the systolic blood pressure when a patient rises from a supine to a standing position. Ideally, there is a rise of approximately 8 mm mercury. In our modern society, few individuals have this rise. A drop in blood pressure upon standing has become so common that some persons working in the health care field think it is normal. Certainly, it may be close to average, but not normal.

The cause of postural hypotension can be adrenal stress, malnutrition, or a pathologic condition. Birchfield[34] made an extensive study of postural hypotension in Wernicke's disease (most often caused by chronic alcoholism). In the study, a normal response from recumbent to 45° tilt was considered a plus or minus 5 mm Hg change in mean blood pressure, and an increase of 6-16 beats per minutes in the heart rate. This extensive study of postural hypotension found it to be "...due to defective neural regulation of vasoadaptation and that the site of neural involvement is in the efferent sympathetic nervous system, most probably either in peripheral nerves, sympathetic ganglia, or pre-ganglionic neurons of the intermedial lateral cell column." For normal sympathetic activity to raise blood pressure from the recumbent to the standing position, it is necessary to have a proper release of norepinephrine.[432]

Kontos et al.[244] compared patients with idiopathic orthostatic hypotension to normal controls. The cause of the drop in blood pressure was a marked depletion of norepinephrine in the blood vessels normally innervated by sympathetic nerves. In control subjects, applying ice to the forehead caused significant vasoconstriction in the forearm, but it did not in patients with orthostatic hypotension. The Valsalva maneuver in control subjects caused an increase in vasoconstriction in the forearm, but vasodilation in the patients with orthostatic hypotension. Tyramine is a drug that produces vasoconstriction by release of norepinephrine from nerve terminals. Its administration failed to produce vasoconstriction in the patients with orthostatic hypotension because of lack of available norepinephrine, while infusion of norepinephrine caused pronounced vasoconstriction, resulting in more reduction in blood flow in these patients than in the control subjects.

The effect of vasoconstriction to help raise blood pressure is indicated by treatment sometimes proposed for postural hypotension. In one approach, the "...first line of defense consists of anti-gravity stockings, which prevent pooling of blood in the lower extremities on standing."[35] Sewall[378] found that providing the patient with an abdominal belt stopped the blood pressure drop upon standing.

Although many patients with adrenal stress also have hypotension, it is not a universal correlation. The important factor in the Ragland sign is the amount the systolic blood pressure drops when the individual stands. A person may have high blood pressure with a considerable drop on standing. It is the rate and amount of fall relative to the patient's usual blood pressure that causes the symptoms.[160]

When there is a drop in blood pressure upon standing, one must differentially diagnose the cause. Postural hypotension is often present in Wernicke's disease. The condition usually develops from chronic alcoholism and responds rapidly to thiamine supplementation.[125] Many drugs — especially the stronger anti-hypertensives — may cause orthostatic hypotension.[266]

Orthostatic hypotension from adrenal stress is present in many conditions, probably because the general adaptation syndrome is present with many disease processes. Emphasizing this, Ziegler[432] states, "Many diseases cause postural hypotension and evaluating them all in every patient whose blood pressure falls upon standing is impossible." He goes on to relate that when a person stands, there is pooling of 300-800 ml blood in the legs. After standing for ten minutes, water moves out of the vessels into interstitial spaces, causing a 10% hemoconcentration. This reduces blood volume and filling pressure to the heart. To compensate for this, normal people have a powerful compensatory mechanism to maintain blood pressure. This is accomplished by an increased heart rate within two seconds of standing. The

sympathetic nervous system increases peripheral vascular resistance to maintain blood pressure while pulse pressure decreases.

Several mechanisms are necessary for blood vascular response upon standing; failure of any of them leads to postural hypotension. The mechanisms are (1) input of baroreceptors located in the aortic arch and carotid bifurcation to brainstem centers. This input inhibits vagal tone while stimulating spinal nerve tracts synapsing with sympathetic ganglia resulting in the release of norepinephrine from sympathetic nerves; (2) venous and arteriolar vasoconstriction in response to norepinephrine; (3) cardiac response to increased norepinephrine and decreased vagal effects; and (4) adequate blood volume to supply the heart and blood vessels.

The most important neurogenic factor is the increase of norepinephrine to cause a diffuse increase in sympathetic activity. Norepinephrine increases by 50% within two minutes of standing and doubles after five minutes. Mild exercise continues the increase to triple the quantity.[432]

Combining with adrenal stress, there are several mechanisms that decrease the activity of the sympathetic system. Vitamin deficiency, alcohol, and many drugs cause failure of proper function. Ziegler[432] states, "The most common cause [of postural hypotension] is iatrogenic when a combination of volume depleting and sympatholytic drugs are given to treat hypertension."

Conditions that affect the brainstem and basal ganglia, such as Huntington's chorea and Parkinson's disease, can cause a drop in blood pressure on standing. Central nervous system disease that affects the sympathetic system is associated with other signs of a cord lesion, such as paralysis and spasticity. Patients should be examined for these conditions when recumbent because hypotension can cause transient neurologic defects. Diseases of the central and peripheral vasoregulatory nerves may cause postural hypotension, affecting both sympathetic and parasympathetic nerves. Diabetes is a common cause of autonomic neuropathy.

Patients with multiple central nervous system defects, as well as peripheral autonomic nervous dysfunction, have normal plasma levels of norepinephrine that fail to increase normally after standing or exertion. Recumbent patients with peripheral autonomic insufficiency without signs of central nervous system defects have low levels of plasma norepinephrine that also fail to increase normally after standing or exercising. Both groups have low levels of plasma dopamine-B-hydroxylase (the enzyme that converts dopamine to norepinephrine).[433]

Rogoff's Sign. Rogoff's sign is definite tenderness at the lower rib junction with the erector spinae muscles. This appears to be a reflex tenderness from adrenal dysfunction and is located at the location of the posterior neurolymphatic reflex for the adrenal associated muscles.

Pupil Dilation. Normally the pupil holds a good constriction to sustained light stimulation. When there is adrenal insufficiency the pupil will usually dilate after light stimulation. This appears to be due to sympathetic stress response. The elevated intracellular potassium level may cause poor function of the cholinergic nerves, which are responsible for activation of the sphincter muscle of the eye. Test by shining a light in the patient's eye and observing the constriction. Continue stimulating the eye with the light, observing for an undulating dilation that develops after approximately thirty seconds.

Heart Auscultation. There is an association of an increased second heart sound (S_2) over the first one (S_1) in adrenal insufficiency. This is heard best over the pulmonary area but can be heard at other or all four heart auscultation areas. When measured by a phonocardiograph, the normal S_2 sound will be close to one-third the height of S_1; greater than that indicates functional hypoadrenia, although there are other possible causes.[361] The increase of S_2 is due to increased blood pressure in the lungs. In a general AK practice, 63% of 160 randomly selected patients had an increased S_2 over the one-third height of S_1.[315]

Ligament Stretch Reaction. In the presence of adrenal stress, associated muscles to a joint will test weak after the joint ligaments are stretched. This is discussed on page 200.

Muscle Association. There will often be dysfunction of some or all of the sartorius, gracilis, soleus, gastrocnemius, and tibialis posterior muscles when there is adrenal insufficiency. The muscle(s) will often test strong after the patient tastes an adrenal gland supplement, but that does not necessarily mean that it is the proper nutritional treatment. All five factors of the IVF and other regulating factors should be evaluated and corrected, if necessary, as explained later.

Sacroiliac Subluxation. There is often a category II posterior ilium subluxation as a result of sartorius and/or gracilis weakness.

Blood Laboratory. The usual laboratory tests to evaluate hypoadrenia are basically designed to test for Addison's disease. The primary one is to evaluate the adrenals' response to ACTH administered intravenously or intramuscularly.[263]

In standard blood screening one would expect to see sodium decreased and potassium increased with hypoadrenia. The sodium deficiency may be seen in a blood test, but potassium increase is rarely observed because it is an intracellular ion. The ECG shows a tall, narrow, peaked T-wave that is indicative of hypokalemia.[212,265] This may be due to the intracellular nature of potassium.

Adrenal Stress Index Test (ASI). Recently salivary assays of dehydroepiandrosterone (DHEA) and its sulfated form (DHEA-S) and cortisol have become available, providing an improved evaluation of the hypothalamus-pituitary-adrenal axis. A laboratory-pro-

vided cotton roll is soaked with saliva at four prescribed times from early morning to near midnight, providing the circadian levels of cortisol and the cortisol:DHEA ratio. The saliva test has several advantages over blood or urine tests. The steroid hormone bioactive levels in the concentration the cells are exposed to are determined. The samples are taken during real life exposure, i.e., during a regular workday. The sample taking is non-invasive, eliminating the patient's possible adrenal stimulation that might occur with serum tests that must be taken in the clinic. Urine tests are not time-specific.

The adrenal stress index provides help in determining how far the patient is progressing through the stages of stress. The laboratory will plot the four cortisol levels on a graph indicating the "Temporal Adrenal Profile" (TAP). The cortisol and DHEA will be calculated as a ratio (cortisol:DHEA). As the stress response moves into the resistance stage, the DHEA level drops and the cortisol level increases. When the DHEA level drops below the cortisol level, there is not enough pregnenolone for full steroid production. As the condition nears exhaustion there will be an effort to increase DHEA just before exhaustion, making the results seem nearly normal.

Some laboratories plot the cortisol:DHEA ratio on a graph with boxes numbered 2 through 7.[116] The first stage of stress is the alarm phase, indicated by box number 2. As the condition moves into the resistance stage, the cortisol:DHEA ratio moves into boxes 3 and 4. The small spurt of DHEA as a last ditch effort is at the number 6 box. Finally, at box 7, there is adrenal failure.

AK Examination and Treatment

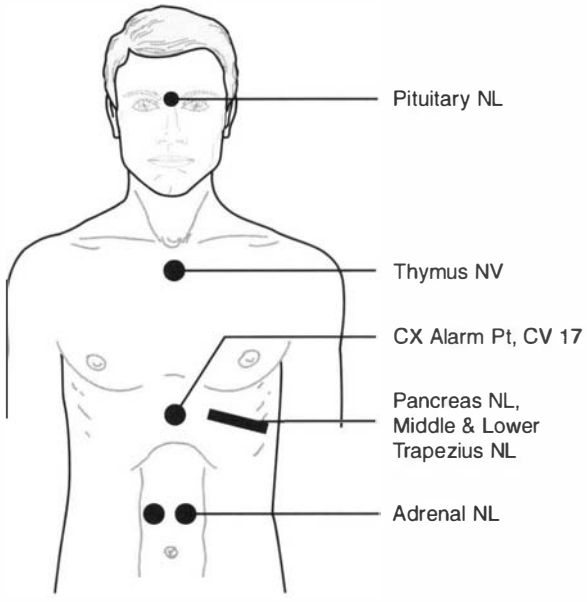
Successful treatment of adrenal stress disorder requires finding the dysfunction within the hypothalamus-pituitary-adrenal axis and determining which of the three sides of the triad of health is involved. In most chronic cases of adrenal stress disorder, all three sides of the triad will be involved to more or less degree and must be addressed for optimal correction.

The interplay in this disorder is great, and treatment must be directed to balance the interactions. At some stages of adrenal stress there is an abundance of cortisol and low DHEA that will inhibit the immune system,[393] affecting the mucous membranes in areas such as the nasopharynx, lungs, and digestive tract. In this case treatment to enhance the adrenal cortex can cause greater imbalance. The increased cortisol will result in even more immune inhibition, causing that aspect of the patient's condition to worsen. Some symptoms caused by the adrenal disorder may improve, making one think the treatment is successful when in reality the patient's overall condition is being made worse. The key is to determine what treatment to the adrenal will enhance its function without being detrimental to the immune system.

Prior to examining the cause and treatment of adrenal stress disorder, examine the patient structurally and clear any involvement in the spine, cranial-sacral primary respiratory system, and any other major factors that may be involved with this patient. Determine the pituitary and immune system status by AK indicators and make corrections, if indicated. The pituitary therapy localization (TL) point is located at the glabella.[170] Remember that therapy localization only tells something is active at that point, not what is active. Keep in mind that TL at the glabella may be indicating a cranial fault. As noted, these should be cleared before this stage of investigation; however, the patient's status can change at any time. Direct pituitary involvement is often caused by cranial faults[269] and will usually be cleared by correcting all aspects of the stomatognathic system.

The immune system muscle associations are the middle and lower trapezius with the spleen, and the infraspinatus with the thymus. If these muscles test weak, strengthen them with the five factors of the IVF before proceeding.

Muscles associated with the adrenal are the sartorius, gracilis, gastrocnemius, soleus, and tibialis posterior. The muscles may test weak or strong in the presence of adrenal stress. If a muscle tests weak, find and make note of the factors that will strengthen the muscle but do not treat. At this point it is important to discover the interrelation within the hypothalamus-pituitary-adrenal axis and other factors, which helps determine the type of treatment to the adrenal glands that will enhance their function and not be detrimental to or create other problems.



12—8. Adrenal NV at lambda, medial & lower trapezius NV 1" above lambda.

Chemical. Usually the chemical side of the triad of health is considered first. Included is nutrition and diet change to enhance function. Toxic factors in the diet or environment may be part or all of the adrenal stress etiology. Investigate this in the initial patient consultation.

In the past, nutrition directed specifically to the adrenal glands has been emphasized in AK. Now types of nutrition and treatment have been broadened. This has come about because of better hypothalamus-pituitary-adrenal axis understanding made available by the saliva test of cortisol:DHEA ratio.

Nutrition that may be needed in adrenal stress includes adrenal, pituitary, and/or thymus substance, vitamin C, pantothenic acid, choline, niacinamide, phosphorylated serine, and essential fatty acids. Some of the nutritional substances may be detrimental in relation to the patient's overall status. When a specific nutritional substance strengthens an adrenal muscle, simultaneously TL to the pituitary NL reflex to determine if it now becomes positive. Do the same to the thymus and spleen NL reflexes. If either becomes positive from the nutrition, it is contraindicated because it will push the adrenal to produce even more cortisol, increasing the imbalanced cortisol:DHEA ratio. Examine the other treatment factors to the adrenal, such as the NL and NV reflexes and alarm point (CX 17), in the same manner to determine what enhances the adrenal and is not detrimental to pituitary or immune function. For example, if TL to the adrenal NL reflex strengthens the sartorius, but combining that TL with TL to the pituitary causes the sartorius or an indicator muscle to weaken, treatment to the adrenal NL reflex is contraindicated at this time.

Treatment is directed to the items that did not cause a positive TL at the pituitary, thymus, or spleen reflex. On subsequent visits, re-evaluate the findings. As the condition is corrected, nutrition that was previously contraindicated may become applicable as the adrenal function improves. Conversely, previously indicated nutrition may become contraindicated since it now causes the pituitary or thymus points to become positive. The changing nature of the condition usually settles down within the first few weeks.

Often adrenal tissue in the form of a concentrate or nucleoprotein extract is contraindicated in the beginning treatment of adrenal stress, especially when there are problems with the immune system. As with all nutritional products, when adrenal substance is needed consider carefully the product to use. There are many adrenal products on the market; no particular one seems to be universally the best. Some are adrenal tissue only, while others are combined with additional nutritional factors to support the adrenal. Clinical experience shows that the best product for improving a patient's condition is that which optimally strengthens the adrenal-associated muscles when the product is chewed and is not contradicted with the pituitary, thymus, or spleen simultaneous tests.

If treatment to the adrenal neurolymphatic reflex is indicated, it often needs prolonged stimulation. Its effectiveness in changing adrenal function is emphasized in a study by Mannino,[273] which found that stimulation of the posterior reflex causes a lowering of aldosterone in low renin-high aldosterone hypertension.

Pottenger found that orally administered adrenal tissue is effective in treating various types of the common cold,[317] and he even determined that it will improve tubercular patients.[316] Pottenger and Pottenger[321] found that orally administered adrenal cortex extract is an important adjunct in the treatment of asthma. Pottenger and Krohn[320] observed that the most outstanding manifestation of asthma is adrenal cortical exhaustion. In addition to the adrenal supplementation, they added 1-3 tsp. of sodium chloride.

If a patient also has functional hypoglycemia, which is often the case with adrenal stress, it is necessary that a blood sugar handling diet be closely followed. Lebowitz[253] recommends a very strict diet for one to two months as an adrenal recovery program. The diet often causes the individual to feel worse for a week or two, because he is losing the dietary stimulants that people in adrenal stress so often desire and use.

The aldosterone pathway can be tested by tasting sodium chloride and potassium. If sodium chloride weakens potassium will strengthen, and vice versa. Potassium is high in many fruits that are often included in a sugar handling stress diet; it may need to be regulated until there is better adrenal function to balance the mineralocorticoids. Schmitt treats the imbalance with injury recall technique while the patient holds the offending substance in his mouth.[367]

There are some substances used for adrenal problems that are stimulants rather than nutrition. Licorice is an example; although it often provides symptomatic improvement, it is not a factor in rebuilding the system. In higher and prolonged dosage the licorice side effects are sodium retention and potassium loss,[39] which may make it worthwhile for short-term usage if there are continued AK findings of increased potassium and decreased sodium. Other stimulants such as coffee, tea, chocolate, and over-the-counter medications containing caffeine should be restricted.

DHEA found in its natural form in wild yam may test positive for improving adrenal stress. This type of replacement therapy is discouraged. Schmitt[367] recommends that other approaches be applied first; if further testing still indicates the replacement therapy, use it only for a short time.

If the adrenal-associated muscles are strong in the clear, determine what weakens them. For example, the sartorius may test strong but weakens with TL to the neurolymphatic reflex. In this case, TL the pituitary, thymus, and spleen points in conjunction with the adrenal neurolymphatic reflex. If the two-point TL again causes the adrenal-associated muscle to weaken, it indicates

that stimulating the adrenal NL reflex would be detrimental to the interaction, causing the adrenal to produce more cortisol and increasing the cortisol:DHEA ratio imbalance.

Tasting refined sugar may cause a strong muscle to test weak or make a weak muscle strong in adrenal stress disorder. In either case refined sugar is detrimental to the condition. If there is no reaction to sugar, i.e., strong muscles are not weakened, retest with TL to the pancreas NL reflex. A positive finding indicates sugar will cause the pancreas to secrete more insulin, which is contraindicated in insulin resistance (discussed with blood sugar).

Structural. Obviously the presence of adrenal stress disorder is due to stress. One of the best methods for reducing stress is to correct the patient's structure as emphasized throughout this text. Discuss how applied kinesiology examination and treatment reduce structural stress so patients will appreciate the importance of this aspect of their treatment.

There is often a category II posterior ilium subluxation associated with sartorius and/or gracilis weakness. It is frequently difficult to determine whether the category II involvement is primary or secondary. In any event, adrenal stress is typically not effectively corrected until the category II posterior ilium is stabilized.

Foot problems, usually extended pronation, are common with adrenal stress. This is probably due to the tibialis posterior relationship with the adrenal and its important role in stabilizing the ankle.

A thorough investigation of the stomatognathic system, and treatment when necessary, is routinely indicated with adrenal stress disorder. In a study of fifty patients with hypoadrenia, Seugling[377] found thirty-seven with a sphenobasilar cranial fault. It is theorized that failure of proper sphenobasilar motion is detrimental to proper pituitary function.[269]

Mental. The triad of health mental side is usually involved if adrenal stress disorder has been present for very long. Test the emotional neurovascular reflex (NV) by therapy localization. The emotional NV reflex may or may not strengthen an adrenal-associated muscle. There does not appear to be a correlation where treating the emotional NV reflex is detrimental to the cortisol:DHEA ratio imbalance.

If therapy localization to the emotional NV reflex is positive, combine that TL with TL to the adrenal NL reflex. If the two-hand TL is positive, it indicates the patient's emotional status is pushing the adrenal glands into an imbalanced state. Treat the emotional neurolymphatic reflex of the pectoralis (clavicular division) muscle when this condition exists.

Sometimes the stress involved with adrenal stress disorder is obvious; other times it is covert, and a thorough investigation must be done to identify it. Consider all four categories of stress. The patient may work or reside in an environment of chemical toxicity, push himself too hard with minimal sleep, or be emotionally stressed. The techniques discussed in Chapter 10 often reveal covert areas of emotional stress and help bring them under control.

Blood Sugar Handling Stress

There has been a major controversy in the healing arts over hypoglycemia. Some practitioners still go so far as to say there is no such thing as hypoglycemia unless there is a pancreatic tumor or over-administration of insulin. On the other side are those who think refined sugar is a poison to everyone, and they find an extremely high percentage of their patients to be hypoglycemic. In a natural health care practice the answer is somewhere in between the two ideologies.

The term "hypoglycemia" should be qualified. Many use it to refer only to a condition that develops as a result of a pancreatic tumor, or one induced by the over-administration of insulin or oral hypoglycemic medications. Reactive hypoglycemia has been related to hyperinsulinism, which occurs when insulin is released in too great an amount for the body's needs or its release is delayed past the optimal time. There has been some question that such a condition exists.

To add to the confusion insulin resistance — also called syndrome X — has been described.[332] In this condition the cells become resistant to insulin, and a higher level of insulin is necessary for glucose to enter the cells. As the condition advances the blood sugar stays at a normal level, but the amount of insulin keeps increasing in order to use the sugar. Eventually the amount of insulin needed is more than the pancreas can supply, and type 2 diabetes develops.

This section is titled "Blood Sugar Handling Stress" to encompass the many variables of disordered blood sugar levels. The proper diagnosis and treatment of the many types of blood sugar handling stress provide a significant challenge to the physician. Blood sugar handling stress is a multi-system condition that includes the various aspects of the endocrine system, digestive system and balance of the autonomic nervous system. Referring to low blood sugar, the term "functional hypoglycemia" is used here to indicate a broad classification of sugar handling stress.

Controversy is not limited to whether there is a condition of functional hypoglycemia; it extends to the

proper methods of diagnosis and treatment. Some consider the five- or six-hour glucose tolerance test the only proper method for diagnosing the condition. The test may be negative, yet the individual has symptoms consistent with the body's efforts or inability to regulate blood sugar properly. A six-hour glucose tolerance test may show that an individual has a normal rise in blood sugar after ingesting the glucose, followed by a drop to a hypoglycemic level. Finally, the blood sugar rises to a normal level from glycogenolysis and gluconeogenesis resulting from adrenal activity. When the blood sugar rises again many physicians analyze this as normal, yet the patient may have had exacerbations of all the condition's symptoms during the glucose tolerance test. Many of these individuals have adrenal stress that causes the delayed rise of the blood sugar level. In this case their symptoms are probably due to adrenal stress, not the short periods of hypoglycemia. Many symptoms during a glucose tolerance test may be from imbalance of the autonomic nervous system as the body attempts to regulate the blood sugar.

Patients with adrenal stress are often unable to handle stress effectively, as discussed in the previous section. The stress may be emotional, chemical, physical, thermal, or a combination of some or all the factors. Some who have studied and written about functional hypoglycemia discount its existence, indicating that the symptoms are anxiety reactions. In 1973, the American Diabetes Association, the Endocrine Society, and the American Medical Association[8] issued a "Statement on Hypoglycemia" in which they say "...it is often attended by symptoms of sweating, shakiness, trembling, anxiety, fast heart action, headache, hunger sensations, brief feelings of weakness, and occasionally seizures and coma." They go on to relate that these symptoms are probably not due to hypoglycemia; rather, they are anxiety reactions. They further state, "...there is no good evidence that hypoglycemia causes depression, chronic fatigue, allergies, nervous breakdowns, alcoholism, juvenile delinquency, childhood behavioral problems, drug addiction, or inadequate sexual performance." The reader must put these statements in perspective, relying on information available in the literature as well as the clinical examination findings the patient presents when all factors are taken into consideration. The "Statement on Hypoglycemia" gives the criteria that must be present before hypoglycemia is treated. "...occurrence of low blood sugar [must] be documented, that the particular symptoms of which the patient complains be shown to be due to hypoglycemia, that the symptoms be relieved by ingestion of food or sugar, and that the particular kind of hypoglycemia which is producing the symptoms be established." It is this writer's opinion that all these factors should be considered; although they may not all be present in a particular patient, a proper diagnosis of the patient's condition can still be made. For example, the patient's symptoms may not be relieved by ingestion of refined sugar, since it may cause an additional imbalance of the nervous system.

There are many causes and degrees of hypoglycemia. The physician must be familiar with all aspects of the condition to relate with the physical, chemical, and mental perspective of a patient's particular situation. Both functional and pathological conditions must be considered.

Recently a new problem has developed in evaluating blood sugar handling stress. Some patients come to the office with the self-diagnosis of hypoglycemia from reading about it in the popular press. Some will even support their conclusion by self-administered glucose tests from capillary blood with or without a photoelectric measuring device. The glucose readings may or may not be accurate. They may be abnormally low because the drop of blood was spread too thin for the purpose of covering the chemically impregnated test strip.[151] When patients come in with this self-diagnosis, it is usually best to agree that, "Yes, it certainly does sound as though you have a blood sugar problem," and then proceed with your usual evaluation without deviation. Patients with a self-diagnosis of hypoglycemia may be difficult to deal with. The self-diagnosis is often an acceptable way of explaining to friends and relatives the individual's bizarre complaints, signs, and symptoms.[427]

Both the overdiagnosis and underdiagnosis of hypoglycemia should be kept in mind as one pursues the subject in the study and evaluation of patients. There are many conditions with symptoms similar to relative hypoglycemia. Each must be differentially diagnosed from proper work-up. On one hand there is the tendency to overdiagnose hypoglycemia as the "in" condition, and — on the other hand — to consider it a "non-condition." One should avoid the extremes of the former, considering refined sugar almost as a poison, and the latter, which takes the position that hypoglycemia cannot be present unless there is a pancreatic tumor, excessive insulin administration, or some frank pathology.

My introduction to hypoglycemia as a working tool had a major impact on the lives of many individuals. I was familiar with hypoglycemia as a textbook entity from my college experience; however, in my first five years of practice I found no cases of hypoglycemia, simply because I was not aware enough of the condition to recognize it. Goodheart[159] first wrote on relative hypoglycemia in 1965, shortly after he introduced applied kinesiology to the profession. After reading his article, I suddenly recognized the possibility of relative hypoglycemia in about fifteen patients who were not responding to chiropractic care as well as they should. Although they had shown enough improvement to continue treatment with me, there were persistent health problems of many varieties. All but one of the patients proved positive for hypoglycemia on a six-hour glucose tolerance test. It was a revelation to observe the rapid

change that took place in these chronic, possibly (I thought) psychosomatic patients whose health care had been a perplexing problem. As I look back now, even the one who did not have a positive glucose tolerance test may have had a sugar handling stress not found by the test.

One particular patient in this group comes to mind — a 38-year-old female working as an officer in a savings and loan association. She had been my patient for several years, and had made moderate improvement under chiropractic care. Prior to chiropractic, she had severe headaches most of the time, a recurring sacroiliac pain that developed with the slightest twist, and general migrating pains and complaints without correlating objective findings. The migration of symptoms indicated to me that this individual might be somewhat emotionally involved with her illness. About the time I became interested in hypoglycemia, this patient approached me with an urgent look on her face. She told me of a problem that had been plaguing her, but she had been unable to discuss it with anyone; now she had to talk. For many months she had been experiencing shakiness and an inability to think in the mid-afternoon. She found it almost impossible to articulate sentences and make sense when communicating with customers. Because of her management status, she was able to leave the office on the pretense of an errand when the problem developed. Soon she found that a double vodka martini almost immediately stopped the shakiness and returned a semblance of proper thought pattern. Her problem worsened to the point that the martini became necessary during mid-morning as well as mid-afternoon. She was almost panic-stricken at the thought of becoming an alcoholic and being unable to control it. I asked her to come to my office when symptoms developed, instead of going to a cocktail lounge. A blood sugar test revealed severe hypoglycemia during that period. Dietary correction and applied kinesiology treatment made an immediate change; she no longer needed or desired the alcohol. What most amazed me at the time was that her sacroiliac subluxation quit recurring. She had no more headaches, and her cervical spine — almost impossible to keep corrected — no longer had recurring subluxations. This patient had previously been treated approximately once a week, at her request; the frequency of treatments changed almost immediately to about every three months. She is still seen occasionally, thirty-four years later. She still must maintain proper dietary control, because failure to do so causes some of the original symptoms, such as the sacroiliac subluxation, to recur. I now recognize her as an individual with hereditarily weak adrenals. If a correct diagnosis had not been made, where would she be today?

In my practice I have many patients in whom I diagnosed hypoglycemia long ago. One such patient required gallbladder surgery for numerous stones. Before surgery, she contacted me to determine what she should do about her hypoglycemia. I told her to inform the surgeon and her internist about the condition. During her hospital stay her blood sugar was monitored regularly, but her post-surgical diet consisted primarily of Jell-O®. She informed the nurse that she could not eat it because of her blood sugar handling problem, and another meal was substituted. The nurse informed the patient's doctor of the requested diet change. During his next round, he told the patient that the food she refused was ideal for her if she had hypoglycemia, because if her blood sugar was low she needed more sugar. She stated with emphasis, "That may seem right to you, but I know how bad I will feel if I eat that."

The appropriate treatment for relative hypoglycemia varies greatly among individuals. The two patients discussed here are among those who will have to regulate their diets for the remainder of their lives because of inherited weak adrenal glands. In some cases there is no glandular imbalance, but the diet is so poor that relative hypoglycemia develops. I examined a teen-age girl who had been in an auto accident eighteen months previously. Her chief complaint was that she awoke every morning with a severe headache and neck pain. She and her family attributed this to the auto accident. Thorough examination found that she ate no breakfast and subsisted basically on junk food, sporadically eaten. Random blood samples were taken, both to confirm the diagnosis of relative hypoglycemia and to emphasize to her the necessity of eating properly. Once the diet was changed, the headaches and neck pain subsided and did not return. Extended treatment to her cervical spine and possibly stomatognathic system would not have improved the neck pain and headaches.

Although dramatic changes are being made in the general health, emotional status, and thinking ability of patients treated for relative hypoglycemia, the controversy about it rages on, even within natural health care. I once heard a chiropractic leader who stresses chiropractic philosophy say, "The current factor of no value is hypoglycemia, used by those practitioners who do not understand the innate wisdom of the body."

The final analysis and answer to the controversy must remain with the practitioner. Those who believe that hypoglycemia is a "non-condition" should study the principles and apply examination and AK treatment procedures to their patients. Evaluate those treated after a period of six months, and then decide what change has been made in the lives of the people involved.

Physiology

A brief review of carbohydrate metabolism helps understand the various types of hypoglycemia.

Blood sugar is derived from three major sources:

Diet. Digestion of ingested carbohydrates begins in the mouth with mastication. The enzyme ptyalin hydrolyzes maltose from starch. There is further breakdown of starch to maltose in the stomach, with very little other digestion occurring there. Digestion of carbohydrates is completed in the small intestine by pancreatic amylase breaking down the remaining sugars. The digested carbohydrates are then absorbed through the walls of the small intestine. From there some are used to produce pyruvic acid, which enters into the Krebs cycle to produce energy. The remaining amounts are stored in the liver and muscle tissues as glycogen. After the glycogen stores are full, the rest is converted into triglycerides and stored in the adipose tissues. Storage of glycogen and triglycerides is done after all tissues have received sufficient energy.

Glycogenolysis. Glycogenolysis is the breakdown of glycogen to glucose. The glucose molecule is broken away from the glycogen polymer by phosphorylation, catalyzed by the enzyme phosphorylase. The release of epinephrine from the adrenal medulla and glucagon from the pancreas activates phosphorylase, causing rapid glycogenolysis. Stimulation of the sympathetic nervous system releases the epinephrine to ready the body for action. This may take place whether or not the blood sugar is low. The mechanism to raise the blood sugar level when it drops too low is release of glucagon from the pancreas.

Both hormones — epinephrine and glucagon — cause an increase in the formation of the cyclic adenosine monophosphate (cAMP), which initiates the chemical reaction that ultimately activates the phosphorylase. The phosphorylase is present at all times, but it is inactive. The liver, on the average, stores 100 gm of glycogen,[30] which can maintain blood sugar levels for about four hours before the store is depleted. Gluconeogenesis then takes over with breakdown of proteins and fats.

Gluconeogenesis. New glucose is formed from amino acids and from the glycerol portion of fat. About 60% of the amino acids in the body proteins have the chemical configuration enabling them to be converted into carbohydrates. The rate of processing is stimulated by a decreased amount of carbohydrates in the cells and a decreased blood sugar level. In the system of checks and balances, the thyrotrophin-releasing factor of the hypothalamus causes release of ACTH from the anterior pituitary. ACTH, in turn, causes the release of the glucocorticoid hormones, especially cortisol, from the adrenal cortex. Cortisol first mobilizes proteins and then fats from the body depots. The conversion of these substances into energy is by the Krebs cycle.

Mechanisms and Effects of Hypoglycemia

The different types of dietary sugar and other carbohydrates react differently in the body. The speed with which the sugar enters the bloodstream depends on the type of carbohydrate food, and the speed and frequency of its ingestion.[220,262] This is seen in the numerous studies that have been done to determine the optimal feeding of athletes for endurance performance.[293,299,301]

The speed with which blood sugar rises after the ingestion of a single food is called the glycemic index. It is assumed by many that simple sugars, such as glucose, sucrose, and fructose, are immediately absorbed by the gut and cause a rapid rise in blood sugar and blood insulin, while complex carbohydrates, such as starches, rice, and potatoes, take longer to absorb and result in a slower, more moderate rise in blood glucose and blood insulin; however, this is not the case, especially when sugars are evaluated as single items of ingestion. Rice gives a flat glucose response, and potatoes give a rapid rise, as much as would be expected with pure glucose. The blood glucose responses to complex carbohydrates are, in increasing order: rice, bread, corn, and potatoes. In general, foods that require little mastication have a faster glucose response, such as apple puree over the whole apple, and rice slurry over the whole-grain product. Legumes eaten as a single item cause half the rise of their cereal counterparts. There is no difference in blood glucose rise between white and whole wheat pasta, or white and whole wheat bread, or white and brown rice. It should be recognized, however, that the whole product has vitamins, minerals, and roughage in it that the refined product does not.

O'Dea[297] points out that there is more to consider than the glycemic index when evaluating the effect of a food on blood sugar levels. Fat eaten in combination with carbohydrates delays gastric emptying, causing a flatter blood glucose response, but there may not be a corresponding reduction of insulin response. "On a longer time scale, high fat, low carbohydrate diets have been shown to impair glucose tolerance and insulin sensitivity...." The glycemic index refers to the subject eating a specific food, such as a potato. Rarely do people eat a single food; they consume foods in various combinations. The glycemic index for carbohydrate-containing foods can be highly misleading when used as the sole criterion for dietary guide.

Another important factor in how carbohydrates are used in the body is whether the necessary vitamins and minerals are present for metabolism. Natural foods high in carbohydrates usually have the proper balance of nutrition for carbohydrate metabolism. Refined substances, such as white sugar and white flour, have lost these necessary vitamins and minerals.

Large quantities of sugars satiate an individual, limiting the desire to eat foods containing the protein, fat, vitamins, and minerals necessary for health. Royal

Lee,[257] an early advocate of reducing refined sugar in the diet, comments that glucose, in comparison, has very little sweet taste. It is used by manufacturers to add bulk to their product at a low cost, and its low level of sweetness causes one to eat more of the product before the craving for sweetness is satisfied. In commenting on general nutritional deficiency, Lee states, "The problem exists because so many people unconsciously satiate themselves on these refined sugar products, soft drinks, ice cream, pastry made from white flour and sugar, white bread, toast, donuts, pie, and candy. They are constantly trying to defeat one of the fundamental laws of the physical world. They are trying to make something out of nothing. You can no more build or repair your body with white flour, white sugar, glucose, or corn syrup, than you can repair your automobile with gasoline or build a locomotive out of coal."

Cleave, a surgeon with the Royal Navy,[82] points out that nature, from a practical point of view, is never wrong. He states that dietary problems result from changes in the food supply that are made too rapidly for the species to adapt to it. The human self-selection studies of natural food, discussed in Chapter 4, are excellent examples of how the body will control itself if allowed to do so. In his strong statement against refined carbohydrates in 1956, Cleave commented that the English annual consumption of sugar jumped from 15 pounds per head in 1815 to 85 pounds in 1900, and to 104 pounds in 1954.

Snow[386] also comments on the substantial increase in sugar consumption over the past 100 years. He points out, "Throughout the world wherever unrefined or natural sugars are found, an all-wise Creator has always accompanied them by vitamins and minerals which are of importance to the body in the digestion and metabolism of those sugars." Men used sugars in their unrefined state for thousands of years without the health problems that are developing as a result of the high level of refined use. Snow, who considers refined sugar a drug, states, "Some parents who are horrified at the idea of having a drunkard in the family think nothing of allowing their children to become habituated excessively to candy, chewing gum, soft drinks, ice cream, pie, cake, jam, and jellies. Many of these children grow up to transfer their sugar habit over to that other carbohydrate, alcohol, which has most of sugar's bad effects in addition to its own peculiar ability to cause drunkenness and insanity. Children are not benefited by eating sweets."

Children are often taught by adults to like excessive amounts of sugar. One commonly hears, "Be good at Aunt Judy's, and I'll get you an ice cream cone on the way home." When sugar is used as a reward, it is only natural to develop a craving for it.

Morgan and Zabik[291] studied the total sugar intake of children five to twelve years of age (n657). It varied from 44 gm per day to a high of 280 gm per day, with an average of 134 gm per day. This is equivalent to 35 lbs/yr, 224 lbs/yr, and 107 lbs/yr, respectively. The greatest percentage of the total sugars came from milk (20.4%); second, from sweetened beverages (17.9%); and third, from fruits (17.1%). The children with the highest total intake of sugar had the highest intake of cakes, cookies, pies, and other types of desserts. The children with the lowest total sugar intake had the lowest intake of dessert-type foods.

There is extensive documentation in the literature of the detrimental effects of excessive refined sugar use. Cleave[83] has made an extensive review of the literature, indicating problems in the colon, blood vascular system, periodontal disease, obesity, diabetes, ulcers, and other conditions. He studied many primitive cultures that developed disease processes after white sugar became a part of their diet.

Refined sugar almost immediately affects the nervous system. In a study done at the Massachusetts Institute of Technology,[157] "normal" children were evaluated on separate mornings. One morning they received orange juice sweetened with sucrose, and another morning the juice contained aspartame. The children were evaluated on a blind basis for efficiency in performing structured tasks and during free-play sessions. There were significantly more errors in performing the structured tasks when the children drank the sucrose-sweetened orange juice. The largest number of errors occurred 60 minutes after having the drink. The free-play sessions were graded on appropriate and inappropriate actions during 10-second coding intervals. During the 15-minute free-play session, 45 to 55 minutes into the study, the children who ingested the sucrose drinks had inappropriate action 29% of the time, as compared with only 10% for those who had the control drink. One wonders what the results would have been if the control drink had not contained aspartame. Some adverse reactions to its use are coming to light.[124,222,423]

The increase of refined sugar intake has almost immediate effects on a person's health, as indicated by the many studies of Cheraskin et al. A three-day increased diet of glucose caused a significant decrease in the height of the T-wave in lead 1 of an ECG.[76] Cheraskin and Ringsdorf[78] cite "...evidence linking sucrose consumption to widespread derangements in metabolism such as increased dental plaque, decreased phagocytosis, increased blood cholesterol, increased blood triglycerides, increased blood sugar, increased blood insulin, increased blood uric acid, increased platelet stickiness, increased body fat, increased urinary calcium excretion, increased urine pH, increased gastric acidity, increased blood pressure, and the encouragement of malnutrition."

Within four days of increased dietary sucrose, gingival health — as graded by gingivitis, hyperemia, swelling, loss of gingival stippling, bleeding, and the symptomatic picture — deteriorated significantly.[73] During a similar period of time, with an increased diet of

glucose, the sulcus depth increased,[71,72,74,337] and there was a significant increase in clinical tooth mobility.[72,75]

Examination Considerations

Proper examination for relative hypoglycemia or a blood sugar handling problem presents a challenge to the physician. One of the time-honored testing procedures is the five- or six-hour glucose tolerance test. Some believe the condition cannot be diagnosed without the lengthy test, and berate those who use a three-hour test to evaluate blood sugar. On the other hand, an article directed to the public points out that the five-hour glucose tolerance test may, in fact, cause hypoglycemia because of an overreaction of insulin to the high charge of carbohydrates.[250] No wonder doctors and the general population are confused and/or in disagreement about hypoglycemia and blood sugar handling stress.

In my early years of diagnosing this condition, I firmly believed that the five- or six-hour glucose tolerance test was necessary. In order to prevent the glucose challenge from creating the condition, patients were instructed to eat high carbohydrate meals for three days prior to the test so their bodies would be accustomed to the large amount of sugar. Some patients had an increase of symptoms from the increased sugar prior to the test. During and after the six hours many patients were observed to have severe reactions. Some had their symptoms greatly exacerbated during the test, and some even had the symptoms continue for days afterward. Some practitioners who have done the procedure report patients who felt like they were going to die during the test.[69] After using the glucose tolerance test for a few years, I concluded that it does not test normal physiology. Rarely does an individual drink 100 gm of glucose after fasting for about twelve hours; this, in itself, is a shock to the body, even if high carbohydrate meals are eaten for three days prior to the test. Because of the epinephrine role in controlling blood sugar, the emotional conditions of the test are important. Consider a patient being brought to a clinic or laboratory knowing that he will have seven blood tests over the next six hours. What is the patient's emotional reaction to the situation? If the blood is taken by venipuncture, the situation may be made worse if the technician misses the vein and has to repeat the blood draw. Between tests the patient has an hour to think about the next one, how he feels, and the meaning of all this procedure. Depending on the patient's endocrine balance, the adrenal gland may or may not react to these unusual circumstances. In any event, this is not examining the patient the way he lives. An ideal test to evaluate the levels of blood sugar would be an instrument that continually samples the blood sugar and insulin levels as the patient arises in the morning, eats his usual breakfast, goes to work, interacts with fellow employees, eats his snacks and lunch, and goes home to fight with the kids — all the time having his blood sugar continuously recorded, unaware of the instrument's action. Obviously, this is not available. We must analyze the studies reported in the literature, combining the meaningful ones with a thorough examination and applicable laboratory evaluation to arrive at a proper diagnosis.

During my early period of running numerous six-hour glucose tolerance tests, many patients developed severe symptoms during the test that paralleled the chief complaint for which they sought help, yet the glucose tolerance test was within normal ranges. The symptoms varied widely, from severe headaches and shakiness to frank tremors, depression to the point of crying, paresthesia, and difficulty in thinking, such as forming proper sentences. In an attempt to understand the enigma of severe symptoms and negative tests, the patients were advised to inform the technician immediately at the onset of symptoms, at which time another blood sample was drawn. Sometimes the routine test would be taken on the hour, and the patient complained of symptoms within five minutes after the blood draw. Often one would see a considerable difference between the previous test and the one taken at the time of symptoms, although there might only be a few minutes between them. The test at the time of symptoms would be considerably lower or sometimes higher than the scheduled test. If the six-hour blood glucose was plotted without the additional test(s), the results were sometimes normal; with inclusion of the additional tests, the results fit the criteria of a sugar handling problem.

The discussion here is primarily directed toward functional hypoglycemia, yet it has just been indicated that sometimes a patient's symptoms would develop with a sharp rise of blood sugar levels, usually from the third to sixth hour. It is generally considered that glucagon is the mechanism that activates phosphorylase for glycogenolysis when the blood sugar drops low, and that epinephrine is the mechanism called upon when increased blood sugar is needed for body action.[187] It was often observed that the patient's symptomatic pattern with elevated blood sugar correlated with strong sympathetic stimulation symptoms, such as increased heart rate, clammy sweating, general shakiness, and nervous agitation. Some indicate that people who have symptoms of hypoglycemia have them as a result of epinephrine release touched off by emotional stress.[8] It is true that emotional stress does initiate the pattern of relative hypoglycemia, but is it not related to relative adrenal insufficiency and an inability to handle the stress? Under these circumstances, the emotional reaction may be secondary to the physiologic deficit, rather than the emotion causing the dysfunction. This seems to be the case, because when the endocrine system and other involved mechanisms are properly treated, the patient ceases to have what might have been considered a primary anxiety reaction, and the symptoms are no longer present.

Glucose Tolerance Test

With mixed emotions I read the literature of the early 1980s confirming that the glucose tolerance test is of little or no value in diagnosing "reactive hypoglycemia."[58,151,223] Although they conclude that the glucose tolerance test has little value, there are also those who, in general, do not support dysfunction secondary to a blood sugar handling stress, considering it necessary to have a pancreatic tumor or excessive administration of insulin to have hypoglycemia. Often the symptoms of patients being examined for hypoglycemia were attributed to an emotional etiology. This was often based on a poor timing correlation between symptoms supposedly associated with hypoglycemia, and the low point of the plasma glucose level during an oral glucose tolerance test. Johnson et al.,[223] on the basis of the Minnesota Multi-phasic Personality Inventory (MMPI), concluded, "Several results taken together suggest that no cause-and-effect relationship between hypoglycemia and personality disturbance can be made." They "...conclude that there is a high incidence of emotional disturbance in patients referred for the evaluation of reactive hypoglycemia but that there is no support for the contention that emotional disturbance can be ascribed to reactive hypoglycemia." The last statement is difficult to accept because consistently patients who have emotional disturbances improve with no counseling when treated by the applied kinesiology method discussed in this chapter. Rarely is it necessary to use the applied kinesiology emotional/mental techniques with them.

Hogan et al.[199] compared the oral glucose tolerance test with a mixed meal containing the same amount of carbohydrate. Nineteen of the thirty-three patients experienced symptoms during the glucose tolerance test, and thirteen of those had the low point of the plasma glucose level drop below 60 mg/dl. In those patients who did not develop symptoms during their glucose tolerance test, eleven of fourteen also had the plasma glucose low point below this level. There was poor correlation between symptom development and the low point of glucose level. When the same patients were subjected to the mixed meal, none developed a plasma glucose level less than 75 mg/dl. Nine became symptomatic during the meal test, at which time blood glucose concentrations were within normal levels.

Studies done in applied kinesiology also indicate that natural foods react differently than glucose in a glucose tolerance test. Achilly[4] challenged seventeen subjects with oranges, grapes, and a medium banana calculated to provide 100 gm of glucose and found that they produced a flat curve in all subjects. Markham[274] found that ten normal subjects tolerated honey better than glucose on the six-hour tolerance test.

Although it is recognized that there are problems in making a proper diagnosis from a five- or six-hour glucose tolerance test, many still consider it the appropriate method for diagnosing diabetes, reactive hypoglycemia, and relative hypoglycemia. If one chooses to subject a patient to a glucose tolerance test, several precautions should be taken. The analysis of the test should be combined with other clinical findings and, possibly, laboratory findings.

If the physician is unaware of the patient's general blood sugar status, a test should be done before loading the patient with glucose to be certain that a high blood sugar is not already present. It is recommended that glucose tolerance tests be done where the physician in charge can evaluate the patient during symptomatic changes, such as in a clinic setting. The blood should be analyzed as the test progresses, rather than accumulating the samples to send to an outside laboratory at a later time. Only by doing this can the patient be monitored for the extreme changes in blood sugar that would require the test to be abandoned. Fortunately, this is easy to do with fingertip blood and a glucometer available today.

Fruit, fruit juice, and Tupelo honey should be available in case the patient's blood sugar drops to extremely low levels. It is very unusual for the blood sugar to rise to extremely high levels; however, if the physician is not one who administers medication, arrangements should be made with a nearby physician to manage high blood sugar, should it occur. Also, have the patient arrange for a friend or relative to drive him home after the glucose tolerance test. An acute crisis could occur, even several hours after the test is completed.

In cases where a glucose tolerance test is indicated, the patient is often on a self-imposed, low carbohydrate diet prior to seeking help in managing his blood sugar levels. Under these circumstances, the body will be very sensitive to the glucose charge administered in the test. To eliminate this possibility, the patient should be put on a high carbohydrate diet for three days prior to the test. Most authorities suggest 300 gm per day to ensure that the patient is not sensitized to carbohydrates. If the patient has been able to somewhat improve his symptoms by adhering to a low carbohydrate diet, he may object to this preparatory procedure; in fact, the three days of high carbohydrates may exacerbate his symptoms. As already indicated, the test itself can put the patient into an acute crisis. These are only a few of the problems in administering and evaluating the glucose tolerance test.

There are many methods for analyzing the glucose tolerance test. Some use hard and fast rules to determine a diagnosis. One can find support in the literature for almost any criteria chosen. It is important to understand the mechanisms regulating blood glucose and treat each patient on an individual basis, taking into consideration all three factors of the triad of health along with clinical and laboratory findings. Applied kinesiology testing adds an additional dimension to the evaluation.

Some have indicated that the type of glandular dysfunction an individual has can be determined by the change in the glucose tolerance at a particular hour. One

such approach indicates the following associated dysfunction: 1/2 hour — liver; first hour — pancreas; second hour — adrenal; third hour — gonad; fourth hour — thyroid; fifth and sixth hours — spleen. Although these relationships can be considered, I have found them mostly random in relating with the patient's clinical status.

There are four characteristics of the glucose tolerance curves that are applicable as a beginning point for patient evaluation.

Diabetic Curve. The patient's blood sugar rises above normal level and does not return.

Hyperinsulinism. The blood sugar rises and peaks at or before the first hour, at which time it begins to drop. It continues to drop throughout the test, possibly rising by the fifth hour. If the blood sugar does elevate toward the end of the test, it is usually a result of glucocorticoid activity from the adrenal cortex.

Flat Curve. The glucose level never rises to an adequate level, and it usually drops to a hypoglycemic level toward the end of the test. The mechanism of the flat curve is controversial. Some authorities believe the flat curve is due to a rapid release of insulin, lowering the blood sugar even before the half-hour test is taken. Some consider this to be caused by such a zestless life that there is no mental stimulation to the adrenal gland; consequently, insulin continues to act, with a failure of counterbalancing adrenal activity.

Another potential cause of the flat curve is a malabsorption syndrome in which the intestinal villi are clogged with mucus, lowering the amount of surface for absorption in the small intestine. In this case the patient will have additional signs and symptoms, such as chronic gas, low-grade diarrhea, undigested food in the stool, and a feeling of hunger even after eating.

Dysinsulinism. This is a paradoxical condition in which the patient has diabetes during one portion of the day and hypoglycemia at another time. In this condition, the blood sugar peaks at approximately the first hour and then drops close to a normal level around the third hour or later. The blood sugar then moves into a hypoglycemic level. If a three-hour glucose tolerance test is done — which is often considered standard for determining diabetes — the later or hypoglycemic portion of the test will be missed, and the patient will receive treatment for diabetes. This is not much of a problem if the treatment is strictly dietary. Attempts to treat this type of diabetes by medication usually create problems, such as that in the case discussion on page 429.

In addition to the four types of curves, the amount of change between time periods and high and low levels may indicate disturbed function, although authorities disagree on what constitutes a problem. A frequently used criterion is that the glucose level should never drop more than 20% below fasting. Other studies indicate the blood sugar should never fall below the fasting state.[144] Further considerations are that the blood sugar should increase by about 50% over the fasting level, but no more than 100%. It should not drop more than 25 mg/dl in any hour, and there should not be more than 100 mg/dl between the high and the low levels. An initial consideration is that the fasting blood sugar should not be below normal. Although all of these criteria have been published, one should take great care in applying them. Many healthy individuals would be classified as hypoglycemic should these criteria be strictly applied.

Adrenal stress disorder can cause hypoglycemia, or it can develop secondarily as a result of the adrenal glands having to frequently bring the blood sugar levels up by glycogenolysis and gluconeogenesis. Testing for adrenal stress can help interpret the glucose tolerance test. Achilly[3] did a six-hour glucose tolerance test on 284 individuals considered candidates for relative hypoglycemia. He considered that 163 had a positive glucose tolerance test for hypoglycemia. Using the postural blood pressure and pupillary dilation tests, he found 65 negative, 22 borderline, and 76 with relative hypoadrenia.

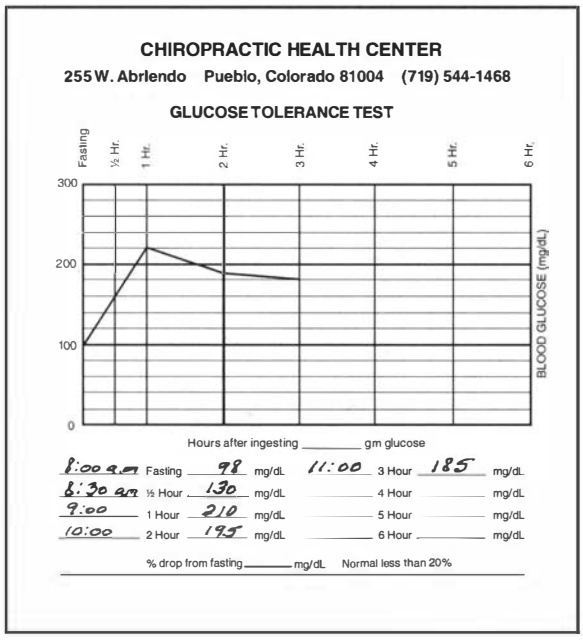
An alternative to the glucose tolerance test is spot testing blood sugar levels during the patient's normal physical activities. The patient can self-administer the tests with a glucometer. The tests can be planned, such as early in the morning before breakfast to obtain a fasting level, two hours after a usual meal, or just prior to eating. Another approach is to obtain blood samples during the symptomatic periods. Both of these test plans are more physiologic than the glucose tolerance test, and when the patient brings in the results the physician can diagnose the condition objectively without subjecting the patient to the stress and errors of the glucose tolerance test.

Applied Kinesiology Testing

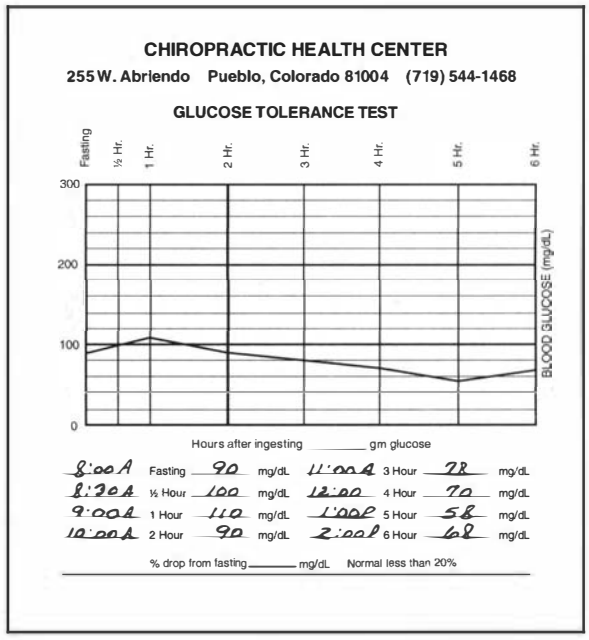
Evaluation of a patient's sugar handling status can be enhanced by evaluating muscle function after stimulating the patient's gustatory receptors with refined sugar. One must take care to use quality muscle testing and avoid a preconceived test outcome. Some examiners have a bias against sugar, i.e., they feel that sugar weakens all individuals. This is not a fact. A double-blind study by Jacobs[217] determined a random muscle weakening on gustatory stimulation with refined sugar. In some cases sugar will cause improved muscle function, depending on the patient's physiologic status at the time. Some research has indicated that applied kinesiology has no value because the study failed to show a statistically significant weakening to refined sugar in "normal" subjects.[145] None of the literature by applied kinesiology diplomates, to my knowledge, indicates that all individuals weaken on oral stimulation with refined sugars.[163,392,413]

The glands most commonly involved with hypoglycemia are the pancreas, adrenal, liver, and thyroid.

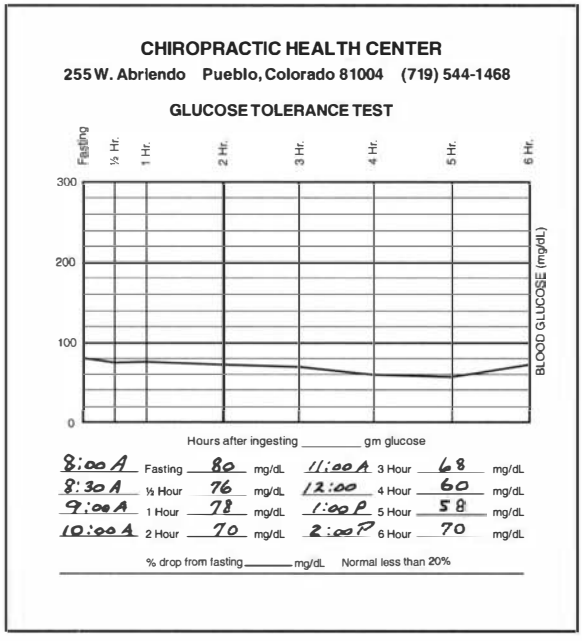
Pancreas. Normal pancreatic function is necessary for regulation of high and low glucose levels. Glucose absorption into the bloodstream causes a rapid



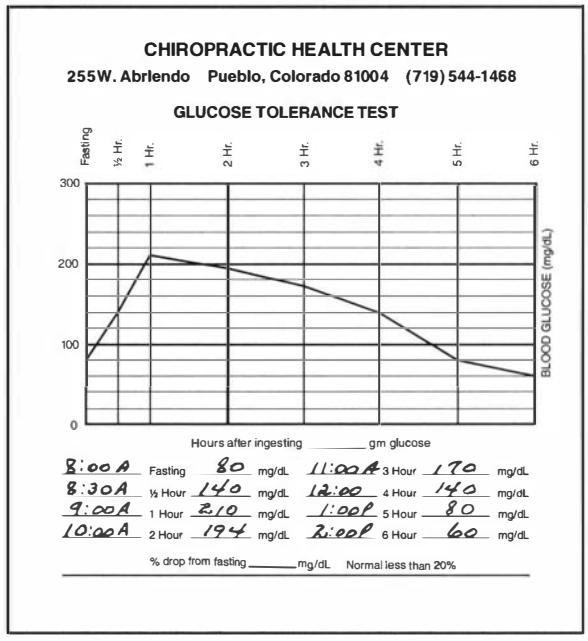
12—9. Diabetic curve.



12—10. Hyperinsulinism.



12—11. Flat curve.



12—12. Dysinsulinism.

release of insulin that allows the body to use and store glucose in the liver and muscles as glycogen, keeping the blood sugar level within normal ranges. As the glucose level falls from use, insulin production is reduced and glucagon is released from the islets of Langerhans alpha cells. Glucagon has an effect opposite that of in-

sulin. It is secreted when the blood sugar drops, and raises the blood sugar by glycogenolysis. A third hormone from the islets of Langerhans is somatostatin, which inhibits the secretion of insulin and glucagon. Although its action is not well understood, it is considered a regulator of the other two hormones from the islets of Langerhans.

Insulin secretion is affected by the type of food people eat. Healthy subjects were given four different types of snacks on different days.[298] These snacks consisted of a Mars® bar with tea; a can of Coca-Cola® with salted potato chips; seedless raisins, raw peanuts, and tea; and ripe bananas, peanuts, and tea. All snacks were proportioned to be similar in fat and total energy content. Glucose and insulin were measured at 10-minute intervals for the first hour, 15-minute intervals for the second, and 30-minute intervals for the third hour. The manufactured snacks (cola and candy bar) produced appreciably higher peaks and lower nadirs than the natural food snacks. In evaluating the plasma insulin, one subject was eliminated from the study because she developed pathologically high insulin levels with the manufactured snacks. Her peak values were 5.6 and 3.4 times higher than the mean peak value for the rest of the group, and 2-3 times greater than the next higher value. Her insulin response was the same as the rest of the group for natural food snacks. Her reaction certainly shows biochemical individuality! Even with the single case excluded from the study, there was a significant increase in insulin production from the manufactured snacks over the natural ones. The increased insulin production showed that the body's homeostatic mechanisms worked harder but coped less successfully with the manufactured snacks containing fiber-depleted sugars than with the whole-food snacks containing fibers and intact sugars (refined vs. natural sugars).

Liver. The liver's role in regulating blood sugar by storage and release of glycogen is very important in the regulation of sugar. With a sugar handling problem, quite often there is an elevation of triglycerides that can cause the early stages of liver congestion. Life-style changes may be needed along with dietary and nutritional support to the liver. The usual physical signs of jaundice in the eyes, liver enlargement or tenderness, congested veins in the legs, chest, and hemorrhoids indicate that further work-up is needed. General examination and laboratory tests in combination with applied kinesiology examination are presented in applied kinesiology seminars, and further writings are intended in Volume V of this applied kinesiology series.

Adrenal. The role of the adrenal gland in regulating blood sugar was discussed in the previous section. Evaluate for adrenal stress disorder, which is often present with relative hypoglycemia, and treat accordingly.

The pancreas, liver, and adrenal glands should be routinely evaluated in any case of blood sugar handling stress. The muscle associations in applied kinesiology help determine methods of treatment, which include the five factors of the IVF, nutritional supplementation, and dietary management.

Diet

Developing the proper diet for a hypoglycemic is very important in obtaining optimal improvement and rehabilitation. If one reads the many "authorities" on the subject, confusion will reign. Some recommend frequent feedings[1] or avoidance of frequent feedings.[285] Some recommend high protein and low carbohydrate,[128,210] while others recommend higher complex carbohydrates and lower protein. The very high protein diet had a rationale of providing a steady source of glucose via gluconeogenesis. This is somewhat self-defeating because protein is a potent stimulus for the release of insulin.[151] The wide range of dietary recommendations is exemplified by a survey that included thirty-five teaching hospitals.[27] There was no recommendation regarding alcohol and caffeine by a high percentage of the respondents. Most institutions made no recommendation concerning fiber. Most adjusted the caloric intake to maintain ideal body weight, and there was wide consensus on limiting simple sugars and eating more than three times per day. Sixty-six percent indicated good or excellent compliance with the diet by patients. Sixty-three percent believed the success of the diet in relieving the symptoms of the disorder was good or excellent.

In this writer's experience, the success rate is much higher than the 63% indicated in the study above when applied kinesiology techniques are used. The major difficulty lies in changing a person's desire for sweets, which is a learned response. Rats develop a learned response for sugar preference that is so strong the animal will maintain an improper diet to the point of death when a life-saving diet is present. If the rat develops a sugar preference before an adrenalectomy, it will maintain that diet after surgery, avoiding a sodium chloride solution that would preserve its health. On the other hand, if the choice between sugar and sodium chloride solutions is not given until after the adrenalectomy, the rat will choose the sodium chloride solution and survive, with no weight loss.[189]

When a proper diet is supplied, the learned preference for sugar lessens. Another study cited by Williams[421] showed that rats on a deficient diet self-select more sugar. On a good diet, they reduce their sugar intake. When initially placed on an adequate diet, there is less desire for a high sugar intake.

After the physician has put many patients on a proper diet to correct blood sugar handling stress, he will frequently hear, "Doctor, I've been feeling so good that I didn't think it would matter if I slipped off my diet. I was at the restaurant the other day, and just couldn't resist the pecan pie a la mode. It didn't taste nearly as good as I remembered, and I got a headache and was nauseous about a half hour after eating it."

Many artificial sweeteners have come and gone. Some have been forcefully removed from the marketplace because of a carcinogenic linkage and other reasons. Currently the popular artificial sweetener is aspartame. Negative reports on its use are seen in the literature.[124,222,339,423] It is unknown what effects may develop as a result of its increased use. Experience shows that allowing patients with sugar handling stress to shift to artificial sweeteners just maintains the desire for sweets.

The diet presented on the next page was originally developed by Seale Harris and popularized by Abrahamson.[1] It is a relatively easy diet to follow, and its components are usually available in a general grocery store.

Whatever diet is chosen as a base, one must evaluate the patient and modify the diet to fit his needs. For example, the amount of fat in the Harris diet may need to be reduced to meet caloric requirements, or for reduction of hyperlipoproteinemia.

In the dietary management of hypoglycemia, it is very important to evaluate for coexistent hypoadrenia. Many hypoglycemic diets like the Harris diet may be high in potassium. If the adrenal is functioning well, potassium stimulates it; mineralocorticoids are produced to eliminate excessive potassium. If, however, adrenal stress disorder is present and the adrenals are advanced into the second stage of resistance, there is already an increased potassium level. Additional potassium will cause a hyperirritability reaction in the patient.

Elimination of refined sugar is possibly the most important dietary factor in treating hypoglycemia. It is sometimes difficult to comprehend how much sugar some people actually use. This is often associated with a high intake of caffeine. The United States has the highest intake of caffeine in the civilized world. Sources of caffeine are chocolate, cocoa, coffee, colas and tea. Note that often these items are drunk with added sugar. Other sources of caffeine are over-the-counter and prescription medications, often those used in attempts to control the symptoms of hypoglycemia. A one-ounce chocolate bar contains 20 mg of caffeine, over-the-counter aspirin contains 15-30 mg caffeine, and prescriptions for pain or to keep awake contain 100 mg or more.[77]

Withdrawing refined sugar and caffeine plus other stimulants is sometimes as difficult as drying out an alcoholic. There are many parallels in the withdrawal of alcohol and sugar from the alcoholic and sugarholic.

An extreme example is an individual sent to me by a psychologist for hypoglycemia examination and treatment. This young man had been under psychotherapy for several years. He weighed over 300 pounds and consumed six to twelve colas per day. He wanted to withdraw from the colas and other refined sugars, but would periodically drop off the wagon. He told me he would get so desperate for a cola that he would run two blocks to the neighborhood grocery store, get two six-packs of 16 oz. colas, and consume one of the six-packs within fifteen minutes. By that time he would stop shaking.

In consultation with patients, it is often good to draw a parallel between withdrawing from alcohol and sugar and the symptoms that may develop temporarily. The patient must recognize that he is losing stimulants in his diet. There is a basic law in physiology that artificial stimulation ultimately results in depression. In the alcohol withdrawal parallel, when an individual develops a headache or begins to shake, there is one thing that will "straighten him out" — another shot of booze — but it is the worst thing he can do. Sometimes patients will develop headaches or other symptoms during the withdrawal of sugar. In these cases, ingestion of some sugar may temporarily improve the symptoms. If one fails to explain this in advance, the patient may think the diet is exactly wrong for him because it causes him to feel worse.

Diet is the common method used to treat hypoglycemia by those not using applied kinesiology. It is important to stress that all aspects of AK examination and treatment enhance the results obtained. During my early use of the glucose tolerance test, other chiropractors referred patients for the test. This usually happened as a result of my patients talking to their patients and recommending a transfer of doctors. I suggested that I would do the test for their doctor, and they could continue with him for treatment procedures. Eventually the patients came back, stating that they had followed the diet but did not seem to be improving. When I accepted the patient for treatment and used applied kinesiology procedures, limited as they were at that time, the patients began to experience higher levels of improvement. Treating the person in addition to diet and use of applicable nutrition supplementation provides the best results.

Application of meridian therapy to specific conditions is outside the scope of this text; it will be presented in others. I cannot, however, refrain from giving an example originally brought to my attention by Khoe.[231] A common finding in hypoglycemia of the hyperinsulinism type is the two-hour postprandial low blood sugar following breakfast. In the meridian system's general circulation of energy, this is the high-energy time of the spleen/pancreas meridian, from 9-11 a.m. The spleen/pancreas meridian is associated with sweet in the five-element law. In addition the circulation sex (CX) meridian associated with the adrenal gland is the mother of the spleen/pancreas meridian in the five-element law. If energy is low in the CX meridian, as it often is with relative hypoadrenia, it cannot give energy to its child (spleen/pancreas). Balancing these meridians, as indicated in Chapter 7, is very helpful to the person with sugar handling stress.

Harris Diet

On arising: medium orange, half grapefruit, or 4 oz. juice.

Breakfast: fruit or 4 oz. juice; 1 egg with or without two slices of ham or bacon; ONLY ONE slice of bread or toast with plenty of butter; beverage.

2 hours after breakfast: 4 oz. juice.

Lunch: meat, fish, cheese, or eggs; salad (large serving of lettuce, tomato, or Waldorf salad with mayonnaise or French dressing); vegetables if desired; ONLY ONE slice of bread or toast with plenty of butter; dessert; beverage.

3 hours after lunch: 8 oz. milk.

1 hour before dinner: 4 oz. juice.

Dinner: soup if desired (not thickened with flour); vegetables; liberal portion of meat, fish, or poultry; ONLY ONE slice of bread if desired, with butter; dessert; beverage.

2-3 hours after dinner: 8 oz. milk.

Every 2 hours until bedtime: 4 oz. milk or a small handful of nuts.

Permitted vegetables: asparagus, avocado, beets, broccoli, brussels sprouts, cabbage, cauliflower, carrots, celery, corn, cucumber, eggplant, lima beans, onions, peas, radishes, sauerkraut, squash, string beans, tomatoes, turnips.

Permitted fruits: apples, apricots, berries, grapefruit, melons, oranges, peaches, pears, pineapples, tangerines. May be cooked or raw, with or without cream but without sugar. Canned fruits should be packed in water, not syrup. Lettuce, mushrooms, and nuts may be taken as freely as desired.

Juice: any unsweetened fruit or vegetable juice except grape or prune juice. Beverages: weak tea (tea ball, not brewed); decaffeinated coffee, coffee substitutes.

Desserts: fruit, unsweetened gelatin, junket (made from tablets, not mix).

Avoid Absolutely

Refined carbohydrates: sugar, candy, and other sweets such as cake, pie, pastries, sweet custards, puddings, and ice cream.

High carbohydrate foods: potatoes, rice, spaghetti, macaroni, noodles, grapes, raisins, plums, figs, dates, bananas, and any dried fruit.

Caffeine: ordinary coffee, strong brewed tea, beverages containing caffeine.

Alcoholic beverages: wines, cordials, cocktails, and beer.

Insulin Resistance and Syndrome X

Physicians who have been working with and watching patients with adrenal stress disorder and blood sugar handling stress since the 60s have observed a group of body language signs and symptoms that are readily recognized. It is easy to see that many patients who have developed Type II diabetes have a history of health problems, indicating they probably went through stages of adrenal stress and periods of blood sugar handling stress, as previously discussed, before the diabetes developed. Do these people go through stages during which blood sugar regulation is being bounced back and forth from low to high blood sugar? What happens to blood sugar regulation when diabetes II ultimately develops?

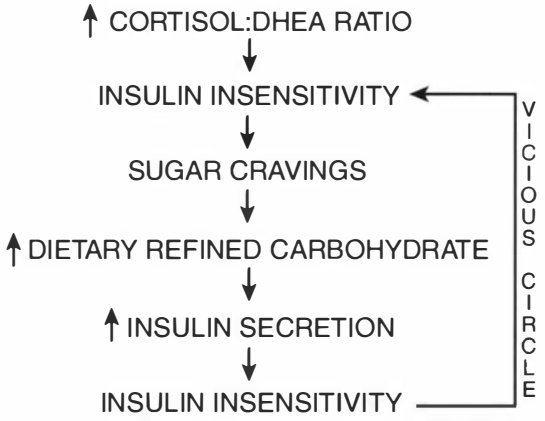
Insulin resistance is an abnormal condition in which the tissues become progressively insensitive to insulin-mediated glucose uptake. In order to maintain glucose tolerance the body produces more and more insulin, keeping the glucose levels within a normal range. Eventually the ß cells can no longer produce adequate insulin and the person's glucose tolerance deteriorates, creating Type II diabetes mellitus, i.e., non-insulin-dependent diabetes mellitus (NIDDM).[224]

Reaven[332] provides a history of the developing understanding of insulin resistance. In 1939 Himsworth presented lectures on the mechanisms of diabetes mellitus to the Royal College of Physicians of London. He questioned the current general belief that all cases of human diabetes could be explained by insulin deficiency. He proposed that diabetes mellitus is a disease in which the essential lesion is diminished ability of the tissue to utilize glucose. He indicated that the increase of blood sugar is the body's controlled and compensatory phenomenon to facilitate tissue use of glucose. After this explanation he subdivided diabetes into two categories in which it results from inefficient action of insulin, as well as from a lack of insulin; the former is a less severe case, the latter more severe. There followed a rather long period until the National Diabetes Data Group in 1979 gave the official approval of dividing patients with diabetes mellitus into two major sub-types. In insulin deficiency (Type I) there is no plasma insulin activity with weight loss and ketosis. In adult onset diabetes (Type II), there is plasma insulin activity and no ketosis. Reaven goes on to report that studies done during the early 1970s using new techniques demonstrated that patients with impaired glucose tolerance and Type II diabetes were, as a group, insulin resistant; i.e., the tissues were incapable of using the available insulin.

Reaven closes his discussion on the development of understanding insulin resistance with a quote from Himsworth and Kerr[197] that illustrates how long it takes for a new thought process to take hold in the "scientific community." "On the whole the insulin-sensitive diabetics tend to be younger, thin, to have a normal blood

pressure and healthy arteries; in them the disease is sudden and severe at onset; they easily develop ketosis and react to a slight excess of insulin with a hypoglycaemic attack. The insulin-insensitive diabetics on the other hand tend to be older, obese, to have hypertension and to exhibit arteriosclerosis; in them the onset of the disease is insidious; they rarely develop ketosis and can tolerate over-dosage of insulin without showing symptoms of hypoglycaemia."

The propensity for insulin resistance is genetically determined,[224] predicting the probable development of NIDDM, but it is not sufficient to cause the disease.[108,328,408] Experimentally, rats are made to be insulin resistant by feeding them a fructose-enriched diet.[328] In the section on adrenal stress disorder we saw the beginning of a series of vicious circles that lead a person into continuing decline of health. Here we see another step in this downward spiral. Insulin insensitivity causes decreased glucose use for energy, causing increased sugar cravings — and the condition goes on.



12—13. After Schmitt,[366] with permission.

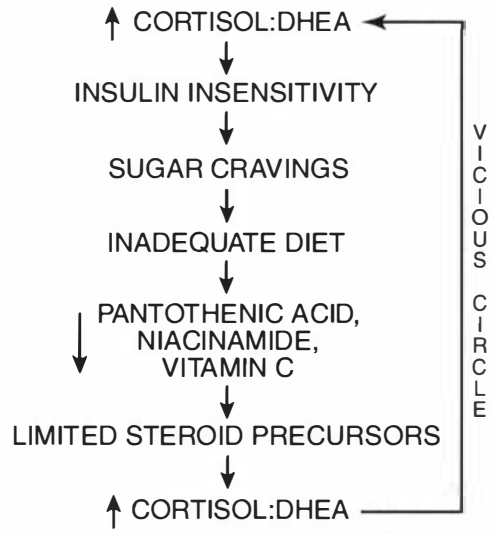
There is a great variance in blood sugar handling among supposedly normal, healthy people. The ability of insulin to stimulate glucose uptake varies ten-fold among the non-diabetic population.[201] Approximately 25% of a "normal" population has insulin resistance to the same level as patients who have impaired glucose tolerance and/or Type II diabetes.[201,331] This means that the ß cells are producing enough insulin to keep these people from developing impaired glucose tolerance or type II diabetes. Unfortunately, even though they do not become diabetic it is not without consequences. The high amount of insulin increases the likelihood that these people will develop the group of symptoms called syndrome X, placing them at increased risk for coronary heart disease.[431]

The term "syndrome X" was coined by Reaven in 1988[328] and then updated in 1993[330] to group the conditions associated with insulin resistance leading to increased risk of coronary heart disease. Syndrome X starts with insulin resistance and eventually includes an increase in plasma triglyceride, decrease in high density lipoprotein-cholesterol concentration,[208,329,397] high blood pressure,[138] microvascular angina, hyperuricemia, and plasminogen activator inhibitor.[330]

Increased abdominal fat indicates probable hyperinsulinemia. Besides inherited genes, controllable life-style factors have been shown to contribute to excessive fat deposits in the middle. These include alcohol, smoking, stress, lack of exercise, and excessive fat or simple sugars in the diet. Why is abdominal fat risky? Fat cells located in the abdomen release fat into the blood more easily than fat cells found elsewhere. Release of fat begins 3 to 4 hours after the last meal compared to many more hours for other fat cells. This easy release shows up as higher triglyceride and free fatty acid levels. Free fatty acids themselves cause insulin resistance

To determine the waist-to-hip ratio, measure around the waist an inch above the navel. Then measure the hips at their widest point. Divide the waist measurement by the hip measurement. A ratio over 0.8 for women or 1.0 for men suggests an unhealthy accumulation of fat in the middle.

Even in youth and young adults, hyperinsulinemia in the presence of normal glucose tolerance predicts later type II diabetes development.[434] Reduced glucose clearance and hyperinsulinemia can be demonstrated one to two decades before diabetes is actually diagnosed. Many of the people with insulin resistance, especially the young, desire an inadequate diet that is high in refined carbohydrates and trans fatty acids resulting in nutritional deficiency. Pantothenic acid, niacinamide and vitamin C are necessary in steroid synthesis, liver detoxication, and cartilage repair. It is just one more step in the resulting series of vicious circles.

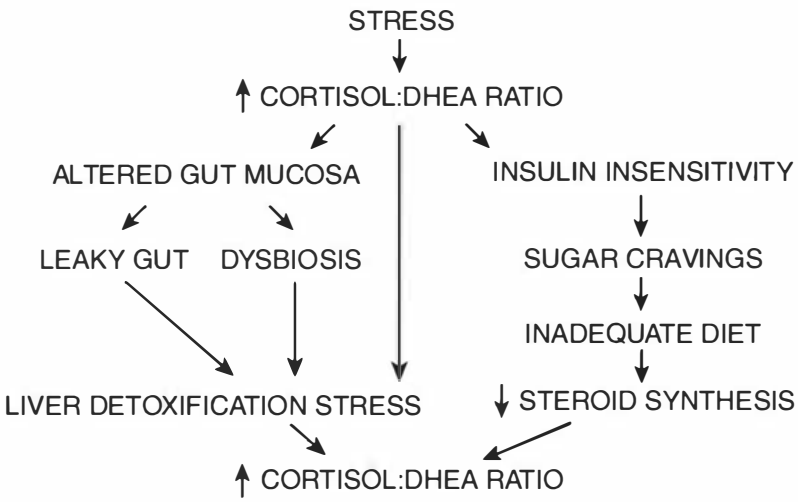


12—14. After Schmitt,[366] with permission.

Insulin resistance and syndrome X are a part of major interaction within the body. If the patient has adrenal stress disorder, which is often present, treat it as previously discussed. Correcting an increased cortisol:DHEA ratio is a firm step in improving the altered gut mucosa that develops from the increased dietary refined carbohydrate.

Since the conditions in syndrome X appear in a cluster, treatment to any one of them tends to help all. The same general examination, treatment, and recommendations that are applicable to people who have blood sugar handling stress, high blood pressure, and hyperlipoproteinemia are applied. Weight loss, maintenance of ideal body weight, and regular exercise are important. Brown et al.[51] placed eleven obese, hypertensive, sedentary, insulin resistant African American women on seven days of aerobic exercise. Following the week, just six of the eleven women remained insulin-resistant.

Laboratory analysis for insulin resistance is available via the glucose and insulin tolerance test. The test is classic oral glucose loading; in addition to measuring glucose levels, insulin levels are measured in response to the glucose challenge. The response of insulin to the challenge provides information to differentiate between insulin insensitivity and insulin deficiency.



12—15. Summary of vicious circles of stress, increased cortisol:DHEA ratio, and insulin insensitivity. After Schmitt, with permission.[366]

Adrenals, Sugar, Liver, Sulfate, and Joints†

This discussion is directed toward the maintenance and repair of joint cartilage, including the intervertebral discs. How vicious circles maintain and increase dysfunction is discussed in the adrenal and sugar handling sections and is primary to this discussion. Here we will see more vicious circles that contribute to joint degeneration.

The ground substance of connective tissue is made up of large complexes of negatively charged carbohydrate chains called glycosaminoglycans (GAGS) that are usually associated with a small amount of protein. There are six major classes of GAGS, most of which depend on sulfate for their production. Chondroitin sulfate is the most abundant GAG in the body and is found in cartilage, bone, and the heart valves. Keratan sulfate is in cartilage proteoglycan aggregates with chondroitin sulfate, and in the cornea and bone. Dermatan sulfate is in the skin, blood vessels, and heart valves. Heparin is an anticoagulant and is the only GAG that is intracellular as a component of mast cells that line arteries. Heparin sulfate is in basement membrane and a ubiquitous component of cell surfaces. Hyaluronic acid is the only GAG that is unsulfated and not covalently attached to protein. It has lubricating and shock-absorbing characteristics and is found in the synovial fluid of joints.

The ground substance of cartilage attracts a large amount of water because of its negative charge, producing a gel-like matrix. The negative charge of GAGS results from the sulfate necessary for their production. To a large extent the concentrations of protein, carbohydrate, and water define the connective tissue characteristics. Tendons have a high amount of protein, cartilage is rich in ground substance, and synovial fluid is an aqueous solution.

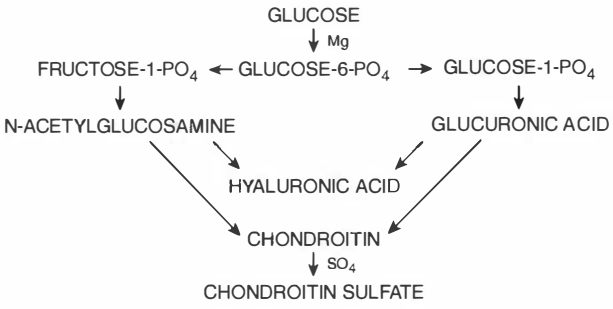
Production of GAGS depends on available glucose and cell utilization of glucose made possible by insulin. Maintenance of adequate glucose depends on proper adrenal function. For glucose to be used, the tissues must be insulin-sensitive with adequate insulin present. (See the Adrenal Stress Disorder and Blood Sugar Handling sections in this chapter.)

Formation and repair of cartilage depend on adequate chondroitin sulfate, hyaluronic acid, and similar substances (GAGS). Nutrition for production of these

† For additional information on Adrenals, Sugar, Liver, Sulfate, and Joints, see the video education programs by Walter H. Schmitt, Jr., D.C.[364,366, 367]

substances must be constantly present because most of the GAGS have a relatively short half-life, ranging from about three days for hyaluronic acid to ten days for chondroitin sulfate.[68]

The first step is the conversion of glucose to glucose-6-phosphate, which is magnesium dependent. From here glucosamine and glucuronic acid are formed; both are necessary for the production of hyaluronic acid and chondroitin sulfate, which in turn are necessary for cartilage synthesis.

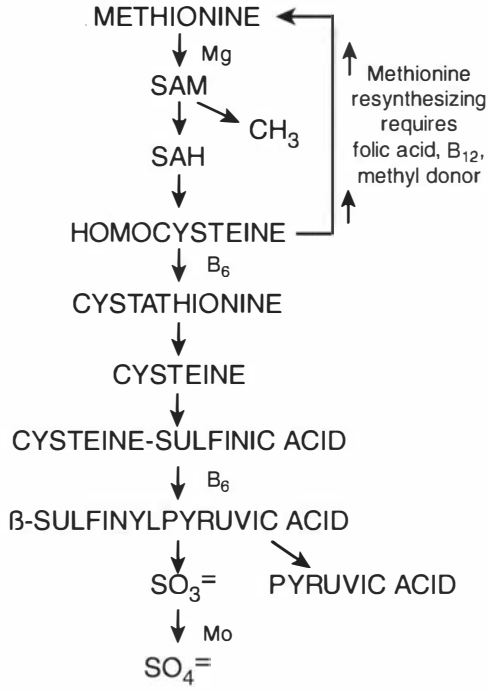


12—16. After Schmitt, with permission.[366]

Sulfate is essential in the production of GAGS, which must be in adequate supply for cartilage maintenance and repair. One of the first events in the pathogenesis of osteoarthritis is a reduction of GAGS in the affected articular cartilage.[270,271] Reduction of sulfate concentration from 0.3 m*M* (physiological) to 0.2 m*M* causes GAGS' synthesis to be reduced by 33%.[368]

The role of sulfur in arthritis was observed long ago. Lenoch[261] comments that healing of degenerated articular cartilage is highly unlikely from bathing in a spa, but he notes that a course of thermal sulfuric waters may be of benefit. In this older text he notes, "Sulfur in any form can be incorporated into the chondroitin sulfate A and C of the articular cartilage but it is doubtful that the fact is responsible for the improvement so often observed during and after spa treatment." There may have been more results from the sulfur than Lenoch recognized. Werbach[417] cites an older observation from Osterberg (1929) that following sulfuric baths, there was an increase in the patient's blood sulfur levels. In another citation by Werbach, he notes that Sullivan and Hess (1935) found normal fingernail cystine content is 12%, but in arthritics only 8.9%.

Sulfur is available in the diet in protein-containing foods such as meat, poultry, eggs, fish, legumes, and milk. The vegetable sources are cauliflower, broccoli, onions, and garlic. Most of the sulfur in the body is found in the sulfur-containing amino acids methionine, cystine, and cysteine. The dietary source of sulfate is important, but most of the sulfate is from the sulfoxidation of cysteine (CYS). In addition to the CYS in the dietary protein, it can be taken as a supplement as the essential amino acid. CYS can also be produced from the essential amino acid methionine (MET). MET condenses with ATP, forming S-adenosylmethionine (SAM). Removal of the methyl group forms S-adenosylhomocysteine (SAH). SAH is hydrolyzed to homocysteine, which can either re-synthesize MET or synthesize cysteine. Re-synthesizing MET requires B_{12}, folic acid, and a methyl donor. Homocysteine combines with serine to form cystathionine, which is B_6 dependent. Cystathionine → cysteine → cysteine-sulfinic acid → ß-sulfinylpyruvic acid, which splits off to sulfite and pyruvic acid. Conversion of sulfite to sulfate requires molybdenum.



12—17. Pathways for methionine to cysteine to sulfate. After Schmitt, with permission.[366]

AK Testing for Adequate Sulfate.[365] When applicable general history, physical examination, imaging studies, and laboratory tests indicate deficiency of GAGS, the first AK test is to determine if CYS strengthens muscles. If so, then MET is tested to determine if it also strengthens the weak muscles. When MET strengthens the muscles, it indicates that either the essential amino acid MET should be supplied as nutritional support or the digestive system needs improvement to breakdown polypeptide chains to free up MET. This is done by improving pancreatic function or supplementation with chymotrypsin. Be certain the patient has adequate HCl and is not taking antacids, because the stomach's acid medium dumping into the duodenum triggers release of pancreatic enzymes. If only CYS strengthens weak muscles, evaluate the need for the cofactors necessary in the conversion of Met to CYS and the recycling of MET (Mg, folic acid, B_{12}, methyl donors, Mo, and B_6).

Homocysteine is produced as an intermediate in the pathway to CYS and must be used toward that end or be re-synthesized to MET. When homocysteine accumulates because of deficiency of either vitamin B_{12}, folic acid, or

other causes, it is neurotoxic and vasculotoxic, promoting heart attacks, thrombotic strokes, and peripheral vascular occlusions,[195] and neurodegenerative conditions such as Alzheimer's disease. This condition may be much more common than previously appreciated. Adequate cofactors for re-synthesizing homocysteine to MET should be routinely evaluated for and supplied as nutritional supplementation when indicated, along with evaluation of the patient's diet.

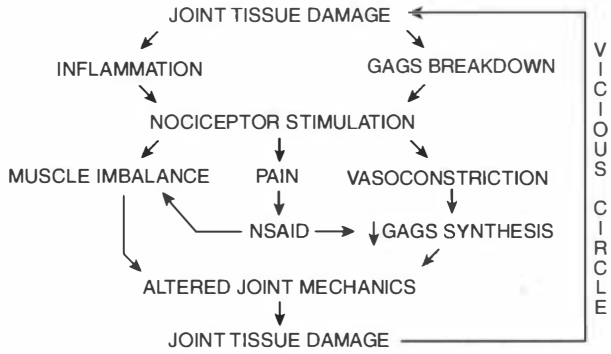
When the patient is supplemented with CYS, be certain to determine if molybdenum (Mo), an overlooked mineral, is available because it is needed for conversion of CYS to sulfate. If molybdenum is deficient there may be failure in the sulfite-to-sulfate production,[296] which could worsen the patient's condition. Schmitt describes methods of testing for Mo.[362,363] The methods depend on muscles weakening from stimulation by certain substances and Mo canceling the weakness.

Sniffing weakens muscle	Cancels weakness
Aldehyde	Molybdenum
Hypochlorite (Bleach)	Molybdenum
Ammonia	Molybdenum

Joint Trauma or Degeneration. Joint trauma, chronic structural stress, or degenerative joint disease stimulates the joint nociceptors. As the condition progresses, the inflammatory response and proteoglycan breakdown products from cartilage damage enhance the nociceptive barrage, resulting in vasoconstriction and further muscle imbalance from the flexor withdrawal reflex responses. The large nerve supply of the joints keeps driving the process while the poor blood supply of the ligaments and cartilage, enhanced by the vasoconstriction, slows GAGS synthesis and tissue healing. The discussed examination and diet modification and/or nutritional supplementation help insure that the production of GAGS will be adequate. Nothing should be done to interfere with GAGS production, but unfortunately that is exactly what often happens. Doctors prescribe non-steroidal anti-inflammatory drugs (NSAIDS) or the patient self-medicates with them, causing several detrimental effects.

NSAIDS block the anti-inflammatory and pro-inflammatory prostaglandins (PG) and also enhance leukotriene synthesis. Increased leukotriene can cancel the anti-inflammatory effect of the PG. In these patients the drug helps for a week to ten days and then has the opposite effect, increasing pain and inflammation. Typically the patient will increase the drug dosage, which never works. Either the drug will be changed or, often, continued even without symptomatic improvement.

It seems strange that many people continue to take NSAIDS and other medication when it is not making much difference in their condition. Sometimes a person may find there is no increase of pain from simply quitting the drug, which should be evaluated. One has to consider the placebo effect of NSAIDS, often enhanced by the constant barrage of television and other advertising that embellishes the drug's value. In a placebo-controlled two-year study, 20 of 44 patients who changed from an NSAID to a placebo had no symptomatic increase.[118] There are natural approaches of dealing with pain such as those discussed by Maffetone.[268]

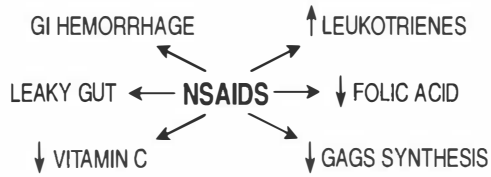


12—18. Joint damage starts a process that can develop a vicious circle that is self-perpetuating. After Schmitt, with permission.[366]

One of the most common reasons to take NSAIDS is to relieve joint pain. Unfortunately the very thing that is expected to gain relief often makes the condition worse by blocking GAGS production necessary for repair,[111,115,207a] in one *in vitro* study by as much as 60-70%.[430] Therapeutic levels of aspirin *in vivo* have effects of suppressing proteoglycan biosynthesis in normal and degenerating articular cartilage similar to several other NSAIDS[44] and permeate osteoarthritic cartilage 35% more than in normal cartilage.[43,305]

NSAIDS cause problems in the entire gastrointestinal tract,[345] ranging from peptic ulcers[149,182,249] to small intestine[287] and colon problems. In a study to determine the safety of low-dose daily aspirin therapy in the gastrointestinal tract, it was concluded that the safety of even 10 mg of daily aspirin is questionable,[102] which is way below the typical baby aspirin dosage of 80 mg. The problems do not stop in the gut; the breakdown of the gut mucous membranes leads to leaky gut syndrome with all of its ramifications, including liver toxicity.

In addition to the liver problems that develop from leaky gut syndrome, NSAIDS themselves cause hepatotoxicity.[326] There is risk of kidney failure from NSAIDS,[308] and in some patients even aseptic meningi-



12—19. The ramifications of NSAIDS. After Schmitt, with permission.[366]

tis, psychosis, and cognitive dysfunction may result from the drugs.[204] The list can go on and on.

Rochon et al.[341] studied fifty-two publications representing fifty-six trials of NSAIDS associated with the drugs' manufacturers. Because of the scarcity of non-manufacturer-associated articles, they did not study any. They concluded, "The manufacturer-associated NSAID is almost always reported as being equal or superior in efficacy and toxicity to the comparison drug. These claims of superiority, especially in regard to side effect profiles, are often not supported by trial data. These data raise concerns about selective publication or biased interpretation of results in manufacturer-associated trials."

Allergies and Hypersensitivity

Much research has been devoted to the autoimmune system in recent years. Three widely differing subjects have accelerated this activity: organ transplant technology, cancer research, and the autoimmune deficiency syndrome (AIDS). The basic purpose of the autoimmune system is to recognize and clear foreign substances from the body. Some consider cancer an autoimmune disease in which the body fails to distinguish immature cancer cells from healthy body cells. Ideal autoimmune function should be able to destroy the cancer cells and maintain homeostasis. Research directed toward AIDS is also, in general, looking for a method by which the autoimmune system can be enhanced to recognize the various types of AIDS virus for their isolation and destruction. On the other hand, organ transplant technology is directed toward reducing the effectiveness of the autoimmune system so it will not cause transplant rejection.

One can see how rapidly information is developing on the subject by realizing that the thymus gland's function has only been recognized for a little over forty years[409]; knowledge of its involvement continues to develop. Many severe health problems result from improper function of the autoimmune system. In some cases, such as AIDS, the system fails to recognize the foreign substance in order to isolate and remove it from the body. On the other hand, sometimes the system overreacts and destroys or interferes with proper function of the body's own tissues, such as in allergic and hypersensitive reactions.

There is considerable controversy regarding the diagnosis and treatment of allergies. The healing arts' deficiency in dealing with the autoimmune system is obvious from the numerous types of examination and treatment procedures that have been and currently are in vogue. Patients are tested for allergic reaction and hypersensitivity with a wide variety of *in vivo* and *in vitro* tests, none of which are all-encompassing. Allergies and hypersensitivity are multifactorial conditions, as evidenced by the fact that people react to allergens and other noxious substances in a variety of ways. In some cases, many of the methods used to examine for reactions to these substances appear to be ineffective.

Better delineation of the mechanisms behind allergy and hypersensitivity is needed to establish improved methods of examination and desensitization. Bland[37] points out major themes in research as differentiation between allergy and hypersensitivity disorders, immediate and delayed hypersensitivity reactions, and investigation into food, airborne, and contact alarm substances. This discussion will be limited to a brief look at some of the different methods of examination, and an introduction to applied kinesiology testing and treatment methods. The AK method of testing and determining methods of treatment has added another dimension to diagnosis. It helps in understanding how the body reacts to protect itself from foreign substances, and what is happening when it fails to react appropriately.

Grieco,[181] a traditional allergist, reviews methods of testing for allergy. He concludes that the Rinkel end point skin titration method appears to provide a valid measure of allergen sensitivity, but Feingold[136] points out the limits of skin testing for certain types of allergy and hypersensitivity.

Some strongly recommend skin testing as the best method of examination, while others condemn it as being limited in dependability and comprehensiveness, sometimes indicating non-reactive substances as reactive and other times failing to identify reactive substances. To maintain confusion in the general population, the popular press abounds with articles on allergy. Mirkin and Morton[289] quote Dean Metcalfe of the National Institute of Allergy and Infectious Diseases as commenting that skin-scratch testing is dependable in diagnosing allergies to pollens and other inhalants. However, it can't be depended on to diagnose food allergies, probably because food is changed in the intestines to a chemical form different from the food injected into the skin. "Selected food extracts including nut, egg, milk, soy and fish correlate fairly reliably with allergic manifestations, but patients should never be advised that they are allergic to certain foods solely on the basis of positive skin tests. Individuals may have positive skin tests in the absence of food allergy (false positive). However, allergic reactions to foods are unusual in the face of negative skin tests (false negative)."

The radioallergosorbent (RAST) assay for IgE antibody is an *in vitro* test that evaluates for IgE mediated allergies. The test is strongly supported in the chiropractic literature by some who are critical of applied

kinesiology testing methods.[389,390,391] The test lacks universality because only 25-30% of food allergies are IgE mediated. Foods to be tested by RAST should be eaten within forty-eight hours of the test. There is controversy over the general effectiveness of the RAST method.[28] It is an expensive test and, according to Grieco,[181] "Potential therapeutic applications of the modified RAST remain speculative." "In view of the technical factors such as IgG blocking antibody, which interferes with interpretation of RAST results and the relative cost compared with skin testing, it does not appear reasonable to replace skin testing with the RAST assay for routine diagnosis."

The leukocytotoxicity test is based on a specific allergen added *in vitro* to whole blood, or to a serum leukocyte suspension. Observation is made for a reduction in white cell count, or the death of leukocytes. Grieco[181] cites several studies that fail to show correlation with positive skin tests, and concludes that the leukocytotoxicity test is not supported by objective evidence.

Sublingual provocative testing and desensitization have not gained wide approval in traditional allergy treatment, although there are many in the medical profession practicing the procedures. This is usually done under the general name of "clinical ecology." Randolph, the father of clinical ecology, indicates in an interview[59] that he finds the incidence of allergy increasing because environmental pollution is creating a toxic buildup. He states, "Thirty years ago when I first became aware of this chemical susceptibility problem, it was the third most important factor in my practice. Pollens, dust, molds and spores were first. Foods were second, and the chemical thing was a weak third. Now chemical hypersensitivity dominates my practice above and beyond the importance of foods, and especially beyond pollens, dust, animal danders and so on." Grieco[181] fails to find objective support for most of the methods practiced by the clinical ecologists. It is pointed out that two-thirds of clinical ecologists' patients have psychoneurotic components,[60] yet many proponents of natural health care attribute some types of psychoneurosis to allergic reactions.[77,144,312]

Harris is pictured as a moderate on the term "allergy" by the *Complementary Medicine* staff.[61] He limits his discussion on allergy to those mediated by IgE. He considers other factors often described as allergy to be hypersensitive phenomena, such as those involving IgG antibodies or other mediators. For these Harris uses the term "allergy-like." He describes traditional allergists as highly-trained individuals with strong academic ties, basing examination and treatment on scientific principles supported by valid laboratory studies. On the other hand, the ecologist is more a clinician and less a researcher. He feels the ecologist "...has devised some intriguing techniques and technologies, but there is not a lot of science to back them up. It doesn't mean they don't work; it's just that the appropriate double-blind clinical studies have not been done." Harris' opinion parallels that of applied kinesiology. He indicates that, "Any test should be viewed simply as a diagnostic tool; it is from a combination of such tools — the patient's history, physical signs, symptoms and various test results — that the diagnosis is made. 'No testing tool is the gold standard; that patient is the gold standard.'"

Much effort in improving patients with allergic reactions or hypersensitivity is directed toward establishing what the patient is allergic or hypersensitive to and then eliminating those items from his diet or environment. This may be accomplished by some of the tests previously indicated. One method is to eliminate suspect foods from the diet based on the case history or the results of other tests. The patient's reaction is observed as foods are re-introduced into the diet.[28]

Applied Kinesiology Testing

The applied kinesiology approach to allergy and hypersensitivity is to identify items to which the patient is allergic or sensitive by how the nervous system reacts, as observed by its control of muscles evaluated by manual muscle testing. Ideally, by this method of testing one finds why the autoimmune system is incapable of coping with the noxious substance. This is accomplished by combining the reactive substance test with another factor such as therapy localization, nutritional substance, or challenge to find what abolishes the reaction to the offensive substance. In this manner, one can often identify the therapeutic approach that will improve the autoimmune system's ability to cope with the substance. It is not always possible to improve this function within the body. For various reasons, one may have a genetic allergy or hypersensitivity that is not correctable. In this case, applied kinesiology, combined with other methods of analyzing and testing the patient's reaction to various substances, can determine what should be avoided in the diet and/or environment. Obviously, the ideal method is to improve the body's ability to maintain homeostasis rather than avoid substances.

The genetic factor in some allergies is well-established. Pottenger[318] sheds some light on one cause of inheriting allergies. In a long genetic line of healthy cats, he caused allergies to develop as a result of nutritional deficiency. Moreover, the second generation of the nutritionally deficient cats developed allergies, and in the third generation the incidence of allergies was almost 100%. The cats with inherited allergy were fed a diet adequate in nutrition; allergy in their generation diminished, and their offspring had fewer allergies. At the end of the fourth generation of regenerated animals, some of the offspring were normal, showing no evidence of allergy.

Genetic change from nutritional deficiency causes major change in body structure. Normal cats in Pottenger's study had a gastrointestinal tract of 48" measured linearly from the epiglottis to the rectum. Some of the allergic cats had digestive tracts as long as

72-80", with a lack of intestinal tone.

Applied kinesiology testing for reaction to potentially noxious substances is done by simply stimulating the gustatory, olfactory, or — sometimes — the cutaneous nerves with the substance, then testing a previously strong muscle for weakening. The substance to be tested can be a food, such as wheat flour, a dairy product, or any other item that might be suspect. Kits that provide a wide range of substances, such as animal dander, various types of weeds and grasses, and chemical substances, are available from suppliers to the profession. Any substance being tested for an allergic or hypersensitive reaction should come directly into contact with the nerve receptors, such as in the mouth or by inhaling, to stimulate the gustatory or olfactory receptors. In testing for contact dermatitis, one can rub detergent, cosmetics, or other suspect substances onto the skin and evaluate by muscle testing in the same way as when the gustatory or olfactory receptors are stimulated. In other words, the tested substance should come into contact with the body in the same way it does in normal day-to-day function. Holding a bottle or vial that contains the substance is inappropriate.

As noted previously, there has been minimal objective support for the various types of allergy testing. There is great controversy over the skin-scratch test, which is the one most widely accepted by the traditional allergists. Likewise, there is controversy about applied kinesiology methods of testing substances for allergic and hypersensitive reactions. We are understanding manual muscle testing better as the result of research,[259,260] but more objective research is needed. For this reason, one must take great care to determine the objectivity of his applied kinesiology testing. One method is to randomly repeat the tests on a blind basis. Have a support person supply the substance to the patient while you remain blind to the item being tested. Failure to obtain the same response on a repeated basis usually indicates poor quality muscle testing, or neurologic disorganization in the patient. Another method of judging validity of the testing is to compare applied kinesiology results with other test results such as RAST laboratory methods. Schmitt and Leisman report the results of AK food testing compared with IgE (RAST test), IgG (RAST test), IgE-immune complexes, and IgG-immune complexes blood testing methods.[368,369] The AK method of testing used is described by Schmitt[357,358] and Lebowitz.[256] Seventeen subjects were tested for allergy by the AK method, and there were twenty-one foods that showed positive findings. Some subjects were found to test positive to two foods. Nineteen of the foods that tested positive to the AK method also showed positive (reactive) blood tests.

There are several types of provocative tests. These are usually indicated as positive when symptoms develop as a result of specific exposure. Another, but less used, provocative test is elimination of symptoms by exposure abstinence.[117]

Coca's pulse test[89] (described later) is yet another method of provocative test in which the pulse rate is observed rather than the symptoms. The applied kinesiology method can narrow down the substances to which the patient appears reactive, limiting the items that need to be tested with other more time-consuming methods. All testing methods appear to have their limitations, as does applied kinesiology. Clinical judgment of all the contributing information is necessary to make a proper diagnosis.

Scopp[374] compared manual muscle testing identification of allergens with the Philpott-type fast[311] of progressive re-introduction of foods (provocative food testing). He states, "Correlation between foods identified as provocative by muscle testing and by the fast was .81." He goes on to state, "Observation of clinical results obtained with muscle testing suggests the method has substantial clinical utility."

Callahan[64] has observed that under certain circumstances some people can become psychologically reversed (page 427) when being tested for nutritional support or allergic reactions. An individual may test negative for psychological reversal, but when an allergen stimulates the system, psychological reversal may develop, i.e., the patient will weaken when saying, "I want to be happy and healthy," and strengthen when saying, "I want to be sick." In addition, when testing a nutritional substance to strengthen a previously weak muscle, one may become psychologically reversed from the nutritional product; this strengthens the muscle when, in reality, the substance is bad for the individual. This may be the reason some nutritional substances that seem to be indicated for improvement cause adverse reactions in the patient. Test for psychological reversal while the muscle is strengthened by the substance. If it is now present and was not in the clear, what appears to be a good nutritional support is actually detrimental to the patient.

Substances being tested are often made up of compounds. One may think a particular item is being tested when, in reality, another portion of the compound is causing a reaction. An example of this situation is when a person reacts to the fillers and binders of a nutritional product rather than the major component on the product label. Lebowitz[255] obtained samples of fillers and binders from nutritional companies and found that in some cases his patients were sensitive to them. This may be another reason why patients adversely react to nutritional substances that appear necessary for improved health.

When testing for reaction to substances, it is best to test a muscle associated with the patient's symptoms. For example, in food allergies one might think of the stomach, pancreas, and small intestine. With reactions to airborne pollens, the systems involved may be the eyes, nasal passages, and sinuses.

Muscles	Organs
Upper trapezius	Eyes and ears
Deep neck flexors	Sinuses and nasal passages
Sternocleidomastoid	
Bilateral pectoralis major (clavicular division)	Stomach
Pectoralis major (sternal division)	Liver
Latissimus dorsi	Pancreas
Infraspinatus	Thymus
Rectus femoris	Small intestine
Sartorius	Adrenal

In addition to specific muscle association, evaluate any chronically weak muscle. In a bad allergic or hypersensitive reaction, all muscles will test weak.

The list of foods to which people react may be very large, or it may be limited to only a few items. Common foods that cause reactions are:

1. Coffee, colas, chocolate, and black tea.
2. Nightshade family of foods — tomatoes, green peppers, eggplant, white potatoes, tobacco, and paprika (these often cause joint pain and arthritis).
3. Dairy products — cheese, milk, and eggs. Patient may react to one kind of dairy product but not to another.
4. Spices
5. Salt
6. Mayonnaise (kind and type may have an effect).
7. Meat. It is necessary to test different kinds of meat to determine reactivity.
8. Bread and grains of various types.
9. Sugar in any form.

Pulse Method Testing†

Coca, an immunologist of international reputation, developed the "pulse test" for identifying substances to which a patient is reactive. The method was described in the literature[88] but was never accepted as a standard method of testing by orthodox immunologists,[156] probably because it is time-consuming for both the patient and the physician, and has several variables that must be taken into consideration. It is difficult to objectively study a testing method and compare it with the "standard" methods of testing, which are in such a state of disarray, when the totality of the autoimmune system is unknown. Some in clinical ecology have commented on Coca's method. Corwin[97] states, "Coca pointed out...that a large group of abnormal reactions existed for which no serological method of diagnosis had been found. He called these non-reaginic. The mechanism of these abnormal reactions is still unknown and there is not reliable evidence to link them to any known immunological reaction. Nevertheless, they are easily demonstrated." Boxer[41] comments about some investigators questioning "...the accuracy of pulse rate acceleration as a consistently valid test for food intolerance or allergy. However, it is difficult to debate the significance of objective measurements of this magnitude, especially when the change in pulse rate is either absent or of a far lower magnitude when the same patient is exposed to a variety of other foods by the same testing technique. Establishment of a significant pulse response does not insure an allergic or immunologic mechanism, and Randolph prefers to consider the problem one of food intolerance rather than necessarily of food allergy. The implication is that regardless of the mechanism involved, the effect of the food upon the patient is the aspect which needs recognition."

Although Coca's method has been published in a lay edition, it should be managed by someone knowledgeable about the autoimmune system for optimal effectiveness. The method is relatively simple. It is based on the pulse rate rising when one is subjected to allergens. Coca states that any resting pulse rate over 84 beats per minute is due to an allergic reaction. There are two types of pulse testing. One is to count the pulse during the day while the patient eats his usual meals and has his usual contact with his environment. During this testing procedure, the pulse is counted on a specific schedule fourteen times per day and recorded along with the foods eaten. The other method is to evaluate the pulse rate change as a result of eating single foods. The basic procedures are as follows:

1. If the patient smokes, he must discontinue doing so in advance of and throughout the testing procedure.
2. The pulse is counted for a full minute (a) just after waking before rising in the morning, (b) just before each meal, (c) three times after each meal at half-hour intervals, (d) and just before retiring. All pulse counts are made sitting, except the important one on waking; this is made before sitting up.
3. Record all items eaten at each meal. This includes ingredients in combination foods such as soups, goulash, or salads. It may also be necessary to record brand names of prepared foods since one may be sensitive to one particular brand of bean soup and not another. It is best to avoid eating between meals, including chewing gum, soft drinks, and other snacks. Some people do not understand the impor-

†Modified from *The Pulse Test*, revised edition, by Arthur F. Coca (New York: Lyle Stuart Publisher, 1958). Used by special arrangement with Lyle Stuart, publisher.

tance of recording all substances that are ingested or with which they come in contact.

4. Continue the pulse dietary records for two or three days with the usual three meals.
5. Single food tests are made for two or more full days in this manner. Beginning at approximately breakfast time, eat a small portion of a single food every hour. For example, slice of bread, glass of milk, orange, two tablespoons of sugar in water, dried prunes, fresh peach, egg, potato, black coffee, various plain meats, apple, banana, raw carrot, celery, raw cabbage, onion, tea, date, cucumber, nuts, sweet chocolate, grape (or raisin), frozen corn, or any other food that is in the usual dietary regimen, but not those known to cause reaction. Each single food must be eaten by itself to avoid confusion.

One can often see a pattern of pulse elevation with certain foods or food groups. When there is an elevation with a meal but the cause of the reaction cannot be determined, one can test the foods that were in that meal by the individual food test method.

The pulse reaction to some foods can be long-lasting, with a carry-over of even up to six or seven days. This may require postponing further testing until the pulse returns to its base rate. The return may not be linear, but drop to rise again without any further stimulus.

One may have powerful sensitivities that cover up mild and weak reactions. Eliminating the food that caused the strong reaction may gain improvement, but symptoms return and further testing must be done to determine the weaker allergic reactions currently causing symptoms. These lesser reactions are not new; they have been present all along. They may now produce symptoms because of increased ingestion of the weaker allergens. "Wheat is usually major over rice, rye and oat, but corn is independent of the other cereals; that is, many individuals must avoid all cereals except corn.... It is the minor allergen that can be eaten at intervals of a week or more with impunity."[89]

Consider other environmental factors and contact substances as possible allergens. If the pulse rate upon arising is considerably higher than before retiring, there is a probability that "house dust" is responsible. "House dust" is in quotation marks because it does not refer to the particles that settle on furniture; rather, it is that which accumulates in furniture, rugs, mattresses, and other items. If the pulse is in its normal range upon arising but then elevates before breakfast, consider toilet articles such as perfumes, makeup, or shaving lotion.

Other environmental factors could include such items as auto exhaust, fumes from a leaky furnace, pesticides, animal dander, or airborne pollen. Aluminum cooking utensils or food wrapped in aluminum foil can be the inciting factor.

Most people are sensitive to tobacco, whether by smoke or oral use. To test for this sensitivity, one must abstain from its use until the normal base pulse has been maintained for several days. The examination consists of smoking, chewing tobacco, or taking snuff, followed by measuring the pulse every three minutes. No elevation within fifteen minutes indicates the person is not allergic to the substance, according to Coca.[89]

Coca presents eight rules of technique and interpretation for the pulse dietary record. They are modified only slightly here. He emphasizes that there are occasional exceptions to the rules.

---

1. If at least 14 pulse counts are taken each day and the daily maximum pulse rate is constant (within 1 or 2 two beats) for three days in succession, it indicates that all "food allergens" have been avoided on those days.
2. If the daily maximum pulse rate varies more than 2 beats — e.g., Monday, 72; Tuesday, 78; Wednesday, 76; Thursday, 71 — there is probably an allergic reaction taking place, provided there is no infection.
3. If the ingestion of a frequently eaten food causes no acceleration of the pulse (at least 6 beats above the estimated normal maximum), that food can tentatively be considered non-allergic to the patient.
4. If exposure to "house dust" causes irregularity of the pulse, this usually excludes the commonly eaten foods as allergens. "House dust" is usually a minor allergen; it does not affect persons who are affected by the stronger reactions caused by foods.
5. Pulse reaction to an inhaled allergen (particularly "house dust") is more likely to be of short duration than that to a major food allergen.
6. Pulse rates that are not more than 6 beats above the estimated normal daily maximum should not be blamed on a recently eaten food, but on an inhalant or a recurrent reaction.
7. If the minimum pulse rate does not regularly occur before rising after the night's rest but at some other time of the day, this usually indicates sensitivity to the "house dust" in mattresses or pillows.
8. If one is not susceptible to common colds, he is probably allergic to only a few, if any, commonly eaten foods. There may be an allergy to some inhaled substances, e.g., "house dust," which may even cause respiratory symptoms.

Success is indicated by the highest count of the routine 14-count not varying more than 1 or 2 beats from the same figure every day, and by no symptoms.

Treatment

A common method of dealing with allergies and hypersensitivity is simply avoiding the noxious substance. Avoiding the reactive substances identified with applied kinesiology testing is an effective way of reducing symptoms, but it does not get to the cause of the problem. In some cases, such as inherited IgE allergies, avoidance and desensitization may be necessary.

Many patients have received dramatic relief from allergies by spinal adjustments designed to improve neurologic function. These, along with other structural corrections done in applied kinesiology, are the basic and first approaches to treatment for allergic and hypersensitive reactions. The importance of the nervous system in allergic reaction is emphasized by the effects some of Coca's[89] patients obtained from a limited sympathectomy (1 or 2 lumbar ganglia on one side only), which he recommended for those with a massive number of allergic reactions.

The first approach is a complete structural examination and correction where indicated. When there is positive sensitivity or allergic reaction observed by muscle testing, attempt to find a factor that cancels the positive reaction. Involved factors may be found by examination, such as therapy localizing to the NL reflexes for the autoimmune system, liver, and digestive system, or finding a nutritional substance that cancels the positive reaction. Treat the positive findings with the five factors of the IVF.

In addition to treatment using the five factors of the IVF, there are several approaches unique to applied kinesiology to help treat the basic underlying cause of allergy or hypersensitivity.

Calcium and Hydrochloric Acid

A time-honored approach to dealing with allergies in applied kinesiology is improving hydrochloric acid and calcium balance in the body, presented by Goodheart in 1969.[165] Prior literature indicated the use of calcium and hydrochloric acid supplementation for the treatment of allergies, but the methods had been abandoned. There were sanitariums that treated allergies by calcium injection, with effective results. Unfortunately, the results didn't last after the injections were discontinued. Roberts[340] states, "Calcium is thought to be synergistic with adrenalin; hence, its use in allergic states." The value of calcium therapy for allergies was discredited because there were no abnormal blood levels for calcium, phosphorus, potassium, and protein. Furthermore, studies of calcium, phosphorus, and potassium levels were normal in the spinal fluid of asthmatic patients. It is interesting that the injected calcium in the sanitariums relieved the allergies, but oral calcium did not.

A consistent finding in patients with allergies or hypersensitivity that responds to the calcium hypothesis is bilateral pectoralis major (clavicular division) weakness. This has been associated in applied kinesiology with hydrochloric acid disturbance. It may be hypochlorhydria or achlorhydria caused by poor stomach production of hydrochloric acid, or it may be a systemic lack of hydrochloric acid that causes the problem. When a deficiency of calcium and hydrochloric acid is relative, it will probably not be apparent in blood tests because the body's compensatory mechanism takes over; the blood calcium is returned to normal by drawing on bone storage by way of parathyroid activity.[91] When there is gastric hypochlorhydria or achlorhydria, calcium absorption is impaired. This appears to be why oral administration did not help but calcium injection did.

Adequate gastric hydrochloric acid is important in many aspects of the digestive process. It is the first step in protein breakdown. Acid entering the small intestine is the trigger for pancreatic enzyme release. The proteolytic enzymes further break down the proteins into amino acids. Partially split proteins can cross the gut wall to cause inflammation and antigen reaction.[193]

It appears that both schools of thought regarding hydrochloric acid and calcium deficiency were correct as far as they went. Injections of calcium and hydrochloric acid are simply short-term, symptomatic approaches. What needs to be accomplished is better body function to produce hydrochloric acid and properly metabolize proteins and calcium.

The bilateral pectoralis major (clavicular division) weakness found with hydrochloric acid deficiency becomes strong when HCl is placed in the patient's mouth. This is the basis for associating this bilateral muscle test with hydrochloric acid disturbance. A temporal bulge cranial fault will consistently be present with the weakness. The cranial fault appears to interfere with normal vagus nerve function possibly due to entrapment at the jugular foramen. The cranium is corrected as indicated in Chapter 9. Be certain to evaluate the total stomatognathic system and the pelvis.

Supplementation with betaine hydrochloride can be of value with allergies. One must put in perspective that hydrochloric acid supplementation is **not** nutritional support; rather, it is an allopathic approach.

At one time I routinely supplemented allergy patients with hydrochloric acid. I treated a severely asthmatic child that way, along with correction of cranial faults and some of the other methods indicated in this section. The child improved to the point that he no longer had to be rushed to the hospital emergency room for adrenalin. He reached a plateau where he continued to have asthmatic attacks, but they were not severe. When it appeared that the initial corrections were holding, he was put on a maintenance-type schedule of one visit per month. After nine months of treatment, I was evaluating him on a routine visit and was surprised to find a temporal bulge cranial fault; it had not been necessary to correct that fault after the third visit in his initial treatment phase. I questioned the mother and child about a fall or some other injury that might have caused

the cranial fault to recur, with negative response. After making the correction, I was ready to dismiss him for the day when his mother asked, "Should he still be taking hydrochloric acid? We ran out about two weeks ago, and I haven't replaced it." Suddenly the light dawned, and I realized the hydrochloric acid was covering up the bilateral pectoralis major (clavicular division) muscle weakness, which is the indicator for a probable temporal bulge. I told him to stay off hydrochloric acid and scheduled him for more frequent evaluation. It took several visits to obtain lasting cranial correction. From that point on, the child recovered completely from his asthma and had no more allergies of any kind.

Several other allergy patients who had reached plateaus and were on routine hydrochloric acid supplementation were recalled and taken off the substance; sure enough, temporal bulge cranial faults appeared when I thought they had been corrected on a lasting basis. Consistently, when the temporal bulge was corrected with no recidivism, the allergic and hypersensitive reactions of these patients improved to a much higher level. One of the interesting findings from this experience was that lasting correction of the temporal bulge cranial fault was much more difficult to obtain than I had previously thought. Of course, this was because I was not really correcting the temporal bulge. Effective treatment required much more extensive correction to the total stomatognathic system and other remote structures such as the pelvis. Since then I have not used hydrochloric acid on a routine basis, reserving it instead for people who appear unable to produce their own. These are usually older patients.

The hydrochloric acid-calcium approach is often effective in lessening reaction to airborne allergens, such as the pollen that causes hay fever. Adrenal support is less effective in these conditions. In asthma the reverse is applicable.[322]

Hypoadrenia

Quite often with an allergy or hypersensitivity complex, there is adrenal stress disorder. The pro- and anti-inflammatory hormones previously discussed are important in containing an inflammatory reaction and participating in the healing process.

Supporting the adrenal gland to treat asthma was done by Pottenger et al.[322] as far back as 1935. The procedure was to treat the asthmatic with whole adrenal gland, or adrenal gland extract and sodium chloride. Sodium chloride was used because of the observed loss of sodium in hypoadrenia. The sodium chloride was administered as table salt in the amount of 3-6 gms in 8 oz. water, thirty minutes before meals. They report that sodium chloride by mouth produced the following effects: "(1) Improvement in the feeling of well-being; (2) improved bowel function, the stools containing a larger amount of moisture though formed; (3) some lessening of the frequency of asthmatic attacks, and (4) fortification of the effect of the cortical extract." Salt alone did not control asthma, but it lessened the amount of adrenal substance necessary to relieve the patient. The adrenal and sodium chloride treatment was effective in freeing the patient from symptoms, not merely giving temporary relief. In some cases, the asthma was precipitated again by colds, other acute infections, or overexertion, but it responded again to subsequent treatment.

One of the structural corrections often needed for allergies is a posterior inferior ilium.[165] This may be secondary to allergies and hypersensitivity associated with adrenal stress disorder in which the sartorius and/or gracilis weaken, allowing the sacroiliac subluxation to develop. On the other hand, an individual may traumatically slip the sacroiliac and create the category II, which cannot be corrected by the body's muscular efforts. In this case, the neurolymphatic-neurovascular reflexes and other associated factors may become disturbed from the constant efforts of the sartorius and gracilis to correct the posterior ilium. The adrenal eventually dysfunctions and sets up the pattern for an allergic or hypersensitive reaction. Both the structural fault and all five factors of the IVF of the adrenals should be treated and supported, if necessary.

Mental

Lang[248] has developed an applied kinesiology system to test allergies correlated with mental processing. A parallel can be drawn between how the body cannot properly counteract an irritating substance or chemical that enters or contacts it with how the mental processes fail to deal with a situation that causes a phobia. Callahan[64] points out that a phobia is present when the body's psychological defense system fails to defend against a situation that would not cause fear in an average person. Likewise, an allergy or hypersensitivity is present when the body's autoimmune system fails to defend against a substance that would not cause a problem in an average person.

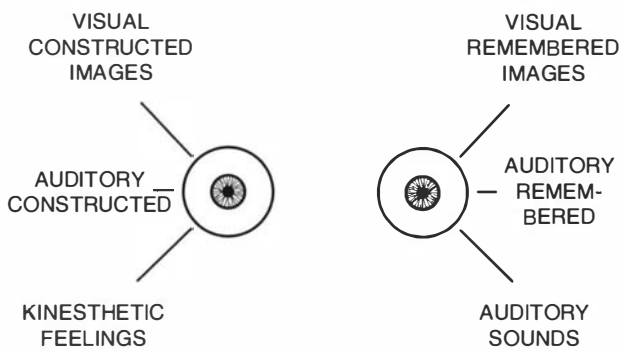
Obviously, phobias are associated with the mental side of the triad of health. Nearly all health problems have been found, at one time or another, to be associated with any side of the triad of health. With Lang's technique it appears that there is a breakthrough to accessing the mental association of allergies and treating them.

The mental process originally creating an allergy may be unknown; there may be a recollection of when the allergy started from a particular problem, such as a childhood illness that temporarily made the digestive system function poorly. For example, if the child eats a large portion of potatoes that the digestive system cannot handle, it may be recorded in his nervous system as a negative experience. From that point on, the child may have an allergic reaction every time he is exposed to anything from the nightshade group. The original reaction of the body during illness may have been to remove

the potatoes by emesis or diarrhea, or to be unable to cope with the substance because of poor general function during the illness. The nervous system's memory may associate any future contact with items of the nightshade group with the way the body functioned at the time of the original illness. As long as that encoded memory association is present, the body adversely reacts to foods in the nightshade group. Under these circumstances, the principle of association is present behind phobias and allergies.

Lang's approach is to first find a food or some other substance that causes an allergic reaction, as observed by a previously strong muscle weakening on manual muscle testing. Possible food allergies are tested by placing the substance in the mouth; airborne allergens are tested by inhalation. Testing substances by holding them or having other contact with the skin is not generally accepted in applied kinesiology. An exception to this is allergens that cause contact dermatitis. These can be effectively tested by rubbing them onto the skin. This would apply to substances such as detergents and cosmetics.

The mental connection with the allergy is found by accessing the brain with the eye positions of neurolinguistic programming (NLP), described by Bandler and Grinder.[16] Each of the six positions (Figure 12—20) relates to how a person processes his thoughts. For example, if you ask a person the color of his house, he will move his eyes up and to the left as he visually remembers or recalls it. If one is thinking creatively about a house he would like to build, the eyes move up and to the right as he constructs the image. Eyes moving to the left indicate that the person is accessing remembered sounds or words, such as a poem once memorized. When the eyes move to the right, the person is constructing what he wants to tell you. Eye movement down and to the left indicates accessing auditory sounds or words, as if having a conversation with himself. Eyes down and to the right indicate kinesthetic feelings, which include smell and taste. A practical example of communicating with a normally organized individual is telling him a joke. If he has heard the joke before, his eyes will move to the left as he remembers his previous experience with it. If, as you tell the joke, the individual is constructing a joke to tell you rather than really listening to your joke, his eyes will move to his right. Although in-depth knowledge of neurolinguistic programming is of great value in communication, it is not necessary to know all its intricacies to evaluate how allergens are being accessed by the nervous system.



12—20. Visual accessing cues for a "normally organized" right-handed person. After Bandler and Grinder.[16]

The first step is to stimulate the patient with the allergen in the manner that causes symptoms. This may be gustatory, olfactory, or perhaps cutaneous. While the muscle being tested is weak from the stimulation, have the patient turn his eyes up and to his right, and re-test the muscle for strengthening. In this manner, progressively test all six eye positions. One or more directions will cause the muscle weakened from the allergen stimulation to now test strong. Next, remove the allergen stimulation so that the patient's muscle again tests strong. After making certain that the patient is not wearing contact lenses, gently contact the patient's eyelid and push in the direction that abolished the muscle weakness resulting from allergen stimulation; the muscle will again test weak while you hold the eye in that position. Retest the muscle while the patient takes a deep inspiration or expiration. One phase, usually inspiration, will abolish the weakness that developed from holding the eye in the previously positive position.

Treatment is by gently contacting the patient's eye and pushing it in the direction associated with the allergy, while the patient takes the phase of respiration that abolished the weakness. Repeat this with 4-8 oz. pressure, four or five times, on both eyes. After all directions of eye movement associated with the allergen have been treated, re-test the patient by re-stimulating with the allergen; it should now cause no muscle weakening. Effectiveness of treatment can be confirmed by continuing allergen contact while the patient looks straight down, which is the neutral position in NLP. This helps prevent the patient from moving the eyes into one of the cardinal positions to cover up muscle weakness that may still be induced by the allergen stimulation.

One should not attempt to treat allergies that are for the patient's benefit. For example, one may have an intolerance to milk that is considered an allergy; instead it is a genetic lack of lactase, causing inability to break down lactose. When treating allergies, one should first consider if the reaction has a beneficial purpose for that individual.

Retrograde Lymphatic Technique

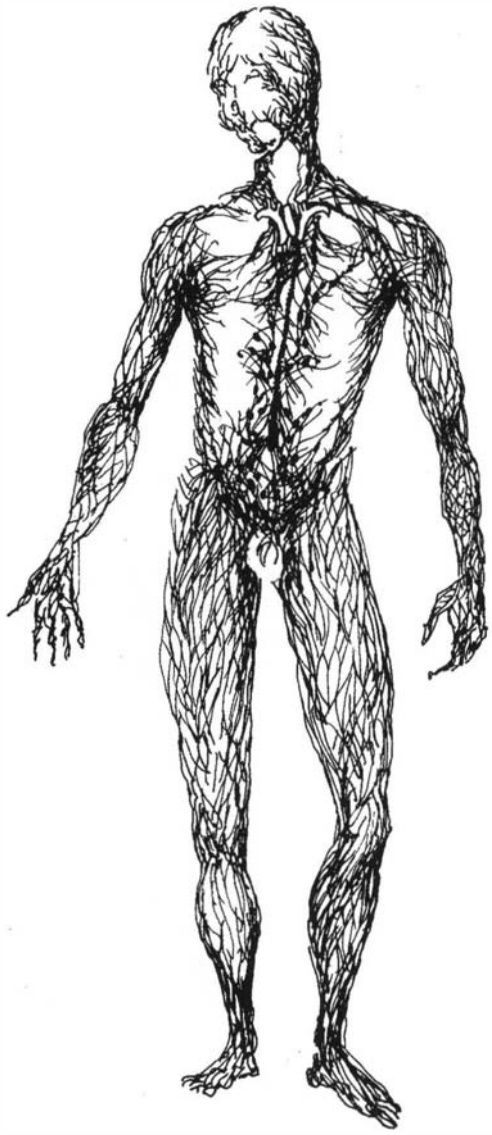
Poor lymphatic drainage is a relatively common finding in functional health problems. In general, members of the healing arts have paid little attention to poor lymphatic drainage considering the contribution it makes to many common health problems.

The lymphatic system collects lymph throughout the body. The larger vessels have smooth muscle fibers that contract with increased pressure within the lymphatic system. Their contractions propel the lymph through the one-way valves located within the lymphatic system toward its ultimate drainage into the venous system. The myoendothelial fibers in the endothelial cells of the vessel also contract to add to the propulsion of lymph fluid.[187] In addition to this "lymphatic pump," Yoffey and Courtice[429] list six extrinsic factors for the movement of lymph: (1) muscular activity, (2) passive movement, (3) pulsation of blood vessels, (4) motility of the intestinal tract, (5) venous pressure, and (6) gravity. Lymph is moved to progressively larger vessels, finally ending in the thoracic duct or right lymphatic duct. The thoracic duct drains all the body except the right side of the head, neck, and thorax, the right upper limb, the right lung, right side of the heart, and the diaphragmatic surface of the liver. These areas are drained by the smaller right lymphatic duct. The thoracic duct passes into the root of the neck. It has a rather tortuous route as it forms a sharp arch that ends by opening into the angle of the junction of the left subclavian vein, with the left internal jugular vein. There is a pair of semilunar valves at the termination that prevent the passage of venous blood into the lymphatic system.

The right lymphatic duct is short, about 1.2 cm in length. It receives collection from several lymphatic vessels to drain the areas not drained by the thoracic duct.

Zink and Lawson[435] attribute poor lymphatic drainage in part to the restriction of free flow through the thoracic duct and right lymphatic duct as they turn to drain into the venous system. There is continuity of the fascia throughout the body. Postural distortions, especially in the shoulder girdle area, put tension on the fascia that appears to cause this restriction. Their method for improving lymphatic flow is stretch on the pectoral muscles. This is applied by contacting the anterior axillary fold and slowly and gently applying traction in an upward and backward direction to stretch the pectoralis muscles. Goodheart has developed an applied kinesiology method to test for poor lymphatic drainage and additional therapeutic approaches to correct the condition when present.[169]

Indications to evaluate for poor lymphatic drainage are numerous. Obviously, pitting edema and congested lymph nodes indicate the problem. When these are present, the condition is often well advanced. Although there may be no gross evidence of poor lymphatic drainage, all patients with conditions in which the lymphatic system is an important part of the healing process should be evaluated for efficient lymphatic drainage. These include, among others, upper respiratory infection; sinus, ear, and eustachian tube infections; nose and throat problems; common colds and tonsillitis; and lower respiratory infections such as bronchitis and pneumonia. Conditions in the extremities, such as recurrent joint problems, numbness and tingling, and joint trauma that is slow to heal, are indications for examination.



12—21. General lymphatic system.

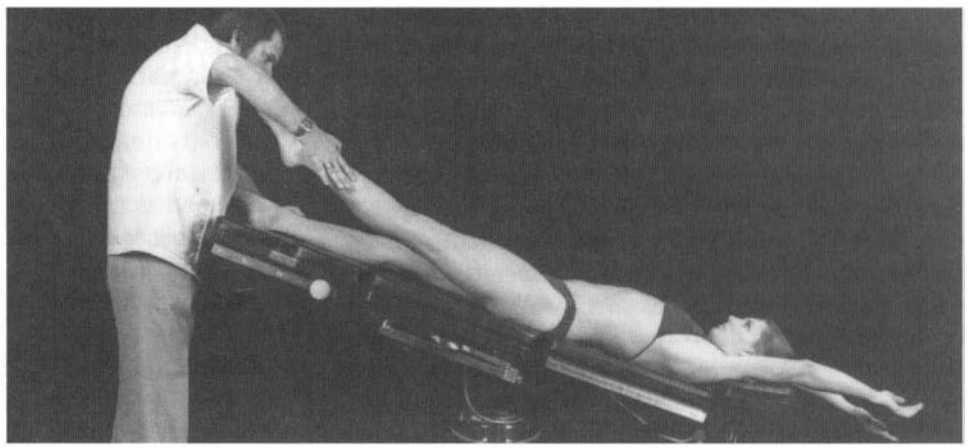
Conditions that develop during sleep often improve with improved lymphatic drainage. This appears to result from the inactivity of sleep, causing increased

lymphatic congestion. There is minimum muscle activity to squeeze the lymph through the vessels. Goodheart observed that nocturnal bruxism and dyspnea that occur at night but not during the day are improved by correcting poor lymphatic drainage. Zink and Lawson[435] even found that frequent nocturnal urination is helped by improving lymphatic flow.

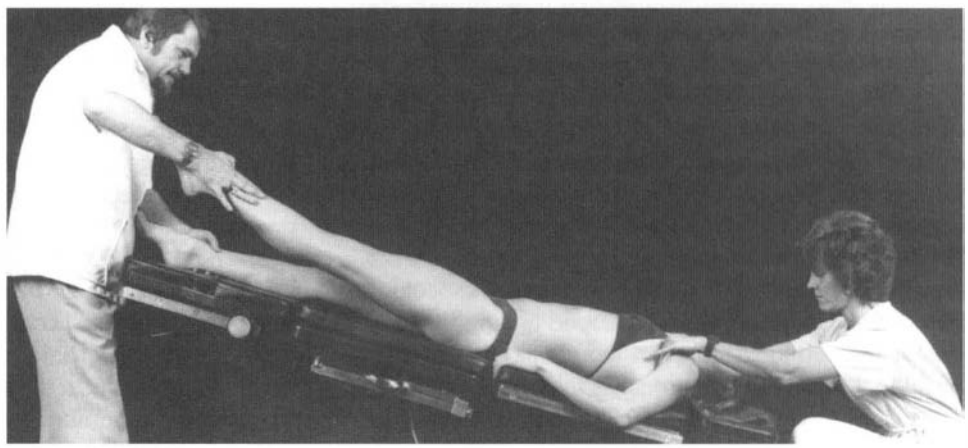
A consistent finding when there is lymphatic congestion is weakness in general indicator muscles when a patient is put into a 20° retrograde position, that is, with his head lower than the rest of his body. This appears to be a result of gravity draining the lymphatic system into the thoracic duct and the right lymphatic duct, which then cannot drain effectively into the venous system. This is confirmed by having the patient raise his arms above his head, which stretches the pectoral girdle and allows drainage to take place. Immediately after lifting the arms into this position, the weakened indicator muscle becomes strong. One can have an assistant perform the pectoral stretch in the manner of Zink and Lawson, previously discussed; this will also cause the weakened indicator muscle to strengthen. The exact neurologic mechanism causing an indicator muscle to weaken is unknown. It may be the result of pressure applied to baroreceptors in the lymphatic ducts with the impulses integrating into the nervous system, ultimately causing poor control of the muscle so that it appears weak.

The retrograde position is usually obtained by putting the patient's head at the foot end of a hi-lo table. The table is raised partway and stopped, and the test is performed. If one does not have a hi-lo table, pillows can be used to put the patient into the retrograde position. The retrograde position is recommended as the best one for retrograde lymphatic testing. Two modified tests have been described.

Schmitt[355] describes challenging the pectoral girdle by contacting the axillary spaces bilaterally and stretching in a cephalad and lateral direction to separate the axillae from each other and elevate them. Immediately



12—22. Retrograde position with shoulder flexed as shown. Positive test should be abolished (see text).



12—23. Pectoral stretch being applied in retrograde position.

afterward a previously strong indicator muscle is tested for weakening. This, in essence, tests for muscle stretch reaction of the pectoral muscles. It will not find all positive retrograde lymphatic conditions.

Blaich[36] found that a modified bilateral pectoralis major (clavicular division) muscle test often parallels the retrograde lymphatic test. The test is done with the patient supine, arms flexed at the shoulders to approximately 170°. Care should be taken that the patient does not flex the elbows as the wrists are separated during the test. Like Schmitt's test, this one will not find all retrograde lymphatic problems, but it is an asset in the absence of facilities to put the patient into a retrograde position.

When the retrograde lymphatic test is positive, there is consistent dysfunction of the pectoralis minor muscle. The pectoralis minor passes over major lymph vessels, the subclavian lymph trunks, and those from the lateral and anterior thoracic wall. In addition, the pectoralis minor — when hypertonic and shortened — rolls the shoulders forward, increasing postural tension on the fascia of the shoulder girdle.

Pectoralis minor muscles will sometimes test weak in the clear, but more often they have a muscle stretch response, i.e., they are strong in the clear but test weak after being stretched to their full length. This is easily done by abducting and extending the arm and immediately bringing the patient into position for testing the pectoralis minor. The pectoralis minor muscles can also be stretched by having the patient rapidly contract the rhomboid and middle trapezius muscles, bringing the scapular vertebral borders together. This moves the coracoid process posteriorly and slightly superiorly, stretching the pectoralis minor. The disadvantage of this method is that the patient must be seated to perform the stretch, and then move into the supine position for testing of the pectoralis minor muscle.

When the pectoralis minor has a muscle stretch reaction, the treatment usually required is fascial release. The massaging pressure begins at the coracoid process and sweeps down over the length of the muscle. Several passes over the muscle are made, being careful to cover the entire width of the muscle to its origin on the ribs. The muscle will usually be quite tender, and treatment is uncomfortable for the patient. A lubricating massage lotion helps decrease skin irritation.

If the muscle is weak in the clear, the most common treatment is to the Golgi tendon organs. Sometimes neuromuscular spindle cell treatment is necessary.

Although the pectoralis minor muscle is frequently the primary muscle involved in the pectoral girdle, test the pectoralis major (clavicular and sternal divisions) for muscle stretch reaction; treat if indicated.

Tight pectoralis minor muscles are often secondary to weak lower trapezius muscles, which are antagonists. Correction of a dorsolumbar fixation usually corrects the lower trapezius muscles. Failure to return them to normal causes the pectoralis minor muscles to shorten again, even though local treatment to them was effective.

The neurolymphatic reflex for the pectoralis minor muscles is located just above the xiphoid process on the manubrium. It is usually very tender and requires vigorous manipulation for approximately two minutes to achieve a good response. Subsequent negative therapy localization to the neurolymphatic reflex indicates that the treatment was effective.

The thoracic spine and ribs should be evaluated for subluxations or fixations. Pelvic dysfunction may be responsible for the shoulder girdle distortion. The problem in the shoulder girdle will return if it is secondary to pelvic distortion.

To a certain extent, lymphatic drainage from the lower portion of the body can be challenged by having the patient elevate his legs by flexing the hips to 45°. This, in effect, only challenges the thoracic duct since there is no change in trunk position.

Brea[48] uses the leg elevation method in the absence of a table that tilts. As a result, he has found that muscles of the legs and left upper extremity weaken but the muscles of the right upper extremity do not, because of failure to challenge the right lymphatic duct since there is no retrograde position of the trunk and arm. He has found that the right lymphatic duct can be challenged by having the patient elevate the right arm and head. The arm is flexed to 90°, with the chin approximated to the chest and held for approximately five seconds. When positive, this produces a selective weakness in the muscles of the right arm and right neck. When the head or arm is individually elevated, there is usually no weakening. Apparently it does not produce enough drainage to stress the system.

Elevation of the head and arm to challenge the right lymphatic duct will find some lymphatic drainage problems that are not apparent with the retrograde technique. When that is positive and the retrograde technique is not, the problem usually relates with a lymphatic condition in the arm, right head, or right side of the neck, such as sinusitis, ear and eustachian tube infection, or nose and throat problems. Usually all the muscles drained by the right lymphatic duct will weaken, but occasionally only selective muscles do. Treatment is directed to the pectoralis minor, lower trapezius, thoracic spine, and rib cage in the same manner as for the usual retrograde lymphatic condition.

If all structural corrections have been made and there is a recurrence of positive retrograde lymphatic, nutritional supplementation is indicated. Goodheart[169] has found low-potency vitamin A or emulsified vitamin A to be effective in eliminating the recurrence. Schmitt[355] recommends iron, sometimes in combination with manganese, for recurring retrograde lymphatic problems. The iron or both items are indicated when the positive retrograde lymphatic challenge is eliminated with insalivation of the nutrition.

Chapter 12

Thyroid

The thyroid hormones are the general regulators of the body's metabolic rate. They directly increase the metabolic rate of all tissues, with the exception of the brain, retina, spleen, testes, and lungs. Even these are indirectly affected by the thyroid hormones' influence on blood circulation and other remote factors. Although the wide range of thyroid hormone action is well-known, often the basic mechanism (or mechanisms) by which they act is somewhat of a mystery.[186] Since thyroid hormones regulate nearly all the body cells, symptoms of dysfunction are widespread when there is thyroid dysfunction. Frank hypo- or hyperthyroidism, such as in myxedema or Grave's disease, respectively, is relatively rare compared to the subclinical or functional thyroid problems encountered in everyday practice. The endocrinologist has relied primarily on laboratory tests to diagnose and monitor the treatment of thyroid problems. Although these tests are effective in diagnosing the reason for frank disease states, they do not evaluate the continuum from complete failure to optimal function. The subclinical problem is often not revealed by these tests.

The two significant thyroid hormones secreted by the thyroid are thyroxine (T_4) and triiodothyronine (T_3). In addition, the thyroid secretes calcitonin, important in calcium metabolism. Thyroid-stimulating hormone (TSH), also known as thyrotrophin, from the anterior pituitary gland stimulates the release of T_4 and T_3 by the thyroid gland. TSH is in turn stimulated by thyrotrophin-releasing hormone (TRH) from the hypothalamus. TRH is transported from the hypothalamus to the anterior pituitary by the hypothalamic-hypophyseal portal blood. The hypothalamus can also inhibit anterior pituitary secretion of the TSH by secreting somatostatin, which also inhibits growth hormone secretion. A negative feedback system controls the amount of TSH secreted by the anterior pituitary. With the rise of thyroid hormone secretion, TSH secretion essentially stops.

The function of T_3 and T_4 is essentially the same, although T_3 is about four times as potent as T_4; on the other hand, it is present in the blood in a smaller quantity. The thyroid hormones are formed in the follicles of the thyroid. A large glycoprotein molecule called thyroglobulin, which contains 140 tyrosine amino acids, combines with iodine to form the thyroid hormones. In the formation of the thyroid hormones, the iodide ions are oxidized to combine directly with the amino acid tyrosine. The enzyme peroxidase and its accompanying hydrogen peroxide promote the oxidation of iodine. When this system is blocked or hereditarily absent from the cells, the rate of formation of thyroid hormones falls to zero. Thyroid hormones remain in the follicles until needed. They can be stored for several months, making a sufficient reserve to supply the body with its normal requirements for one to three months. If thyroid hormone production ceases, no symptoms may be observed for several months. Over one-half of the iodinated tyrosine in the thyroglobulin never becomes thyroid hormones. An iodase enzyme releases this iodine to recycle and combine again with tyrosine in the thyroglobulin molecule. If there is a congenital absence of iodase enzyme, the person will often become iodine deficient.

As noted, the effect of thyroid hormones is widespread throughout the body. They are responsible for decreasing the quantity of cholesterol, phospholipids, and triglycerides in the blood. In hypothyroidism there is almost always an excessive deposition of fat in the liver. Generally one associates hypothyroidism with weight gain, and hyperthyroidism with weight loss. This does not always occur, because a change of thyroid levels influences the appetite, which may have a counterbalancing effect.

Thought processing is in direct ratio to thyroid levels. Excessive thyroid ultimately becomes instrumental in extreme nervousness and psychoneurotic tendencies, such as anxiety complexes, extreme worry, or paranoia.

As indicated in the section on the adrenal glands, people tend to inherit their endocrine balance, which gives characteristic body features and personalities. The strong-thyroid person can be likened to a racehorse in body features and quickness of thought and action. Unless thyroid dysfunction develops in the strong-thyroid type, they are slim and have fine body features. When the thyroid is overstressed and they gain weight, it is around the middle; the calves, ankles, feet, wrists, and hands continue to be slim, looking like they belong to someone else.[2]

Bieler[31] lists the typical thyroid type, which is modified slightly here.

Hair:	On head, it is fine and silky. Body hair, except that of the pubis and axillae, is scarcely noticeable because it is so fine and thinly distributed.
Features:	Delicate and streamlined. Great beauty is the rule.
Eyes:	Large and often slightly prominent — the type called "soulful."
Teeth:	Narrowly spaced, of moderate size; pearly white, soft and not resistant to caries; dental arches usually V-shaped rather than round, partially erupted and unerupted third molars.
Tongue:	Moderately thin and long, with fine papillae and sensitivity.

Palate:	High; more V-shaped than arched.
Neck:	Graceful, thin, and long.
Chest:	Long and thin, usually smaller than so-called normal. Exquisitely shaped breasts in female. Nipples more sensitive than in adrenal types.
Abdomen:	Long and usually thin.
Genitals:	Medium in size. Their increased sensitivity makes up for their lack of size.
Extremities:	Finely molded, graceful hands; beautiful, shapely fingers that are neither stubby nor markedly elongated.

Laboratory Examination

More tests are available for evaluating thyroid function and its effect on the body than for any other endocrine gland. The numerous tests have been developed because each procedure has inherent limitations. Each procedure can, in some way, be altered by endogenous or exogenous factors, complicating the interpretation and — in some cases — causing the various tests to give conflicting information.

There are several categories of laboratory tests for evaluating thyroid function, including: (1) direct thyroid function tests determining how iodine is handled; (2) tests that establish the status of the hypothalamus-pituitary-thyroid axis; (3) tests of the thyroid hormones' concentration and binding in the blood; (4) evaluation of body activity influenced by thyroid hormones; and (5) miscellaneous tests.[252] Within these categories, the endocrinologist has a large battery of tests available to assess pathological states of thyroid function. A brief discussion of some tests is presented here. Laboratory procedures for thyroid evaluation become more sophisticated every year. There are many diverse combinations of changes from normal, and factors that cause the changes. The interplay of laboratory test results must be considered in conjunction with the clinical presentation. Laboratory analysis of thyroid function is best left to the endocrinologist.

Radioiodine Uptake

Radioiodine Uptake (RAIU) is the only direct measure of thyroid function. These are *in vivo* tests that evaluate the thyroid metabolism of radioiodine. Their use has diminished for two reasons. The first is because of improved methods for specific measurement of thyroid hormones in the blood and of the regulating mechanisms of the thyroid gland. The second factor "...is the decrease in normal values for thyroid RAIU consequent to the widespread increase in daily dietary iodine intake, reducing the usefulness of the test in the diagnosis of hypothyroidism."[252] They continue to be used by endocrinologists for evaluation of several pathologic states and the evaluation of thyroid suppressibility.

Tracer quantities of inorganic radioiodine are administered orally or intravenously to mix with the endogenous stable iodide in the extracellular fluid. The isotope is removed from the extracellular fluid primarily by the thyroid and kidneys. Among the factors that cause an increased thyroid iodide uptake are hyperthyroidism, nephrotic syndrome, chronic diarrhea, and iodine deficiency. Decreased uptake can be caused by primary hypothyrodism, Hashimoto's disease, anti-thyroid argents, increased availability of iodine, and diet or drugs.

Thyroid Stimulation

The endocrinologist has several tests that measure the regulating mechanism of the thyroid. These are designed to suppress the thyroid, or influence the levels of thyroid-stimulating hormone (TSH) or thyrotrophin-releasing hormone (TRH) from the anterior pituitary. Commonly used are TSH stimulation tests, which are *in vivo* radioimmunoassay tests to determine the levels of thyroid-stimulating hormone. Recently an ultrasensitive immunoradiometric assay (IRMA) has been developed, making it possible to distinguish suppressed from normal TSH values with confidence. Braverman[46] states that this "...may prove to be the best single test to evaluate thyroid function, with low values found in thyrotoxic patients, normal values in euthyroid patients, and elevated values in those with primary hypothyroidism."

Direct Hormone Measurement

The PBI measurement to estimate thyroid level in the blood was replaced by measuring thyroxine (T_4), which was augmented several years later by the ability to measure triiodothyronine (T_3). There are several methods of evaluating T_4 and T_3. The currently popular methods are *in vitro* serum tests by radioimmunoassay. They include the following[6,348]:

T_4 (RIA), total thyroxine by radioimmunoassay. This test measures the total thyroxine extracted from a serum sample.

RT_3U, resin triiodothyronine uptake. This test is sometimes listed as T_3 uptake. It is an indirect test measuring the binding capacity of the thyroid-binding protein in the serum, or the percent saturation of thyroxine-binding proteins. This is not a measurement of absolute-serum T_3 concentration.

T_3 (RIA), triiodothyronine by radioimmunoassay, measures the total triiodothyronine in the patient's serum. T_3 is most useful in helping diagnose hyperthyroidism.[348]

T_4 and T_3 are altered by changes in protein concentration and drugs, and certain physiological states, e.g., pregnancy, and by some pathologic states, e.g., kidney conditions. When there is an abnormal level of thyroid hormones in the blood with thyroid disorders, T_4 and T_3 usually diverge from normal in the same direction, indicating either hyper- or hypothyroidism.[213]

Usual Testing Procedures[6]

Hyperthyroidism suspected: order T_4 (RIA), RT_3U.

T_4 and RT_3U high indicates hyperthyroidism.

T_4 high and RT_3U low indicates probable protein-binding abnormality.

T_4 normal and RT_3U normal or low, order T_3 (RIA).

Hypothyroidism suspected: order T_4 (RIA), RT_3U, and TSH.

T_4 low and RT_3U normal or low and TSH high, primary hypothyroidism is confirmed.

T_3 normal, RT_3U normal and TSH high, patient may be hypothyroid but definitely has "dying gland."

T_4 low, RT_3U high and TSH normal, patient has euthyroid and protein-binding abnormality.

T_4 low, RT_3U normal or low and TSH normal, patient has secondary hypothyroidism.

Thyroid Physiologic Activity

Several tests measure the metabolic function of the body apparently influenced by thyroid hormones. These include the early basal metabolic rate (BMR), serum cholesterol concentration, Achilles reflex time, and temperature measurement. With the advent of measuring thyroid hormones in the blood, the metabolic tests have lost favor. This is partly justified by their lack of specificity. The problem of thyroid diagnosis at the time was put in perspective by Ingbar and Woeber.[213] "Conventional metabolic indices such as the BMR or serum cholesterol concentration are neglected, while more recently described metabolic or physiologic indices, which may be equally nonspecific, enjoy a vogue of popularity." After discussing the lack of specificity and questionable normal ranges, they go on to say, "For the foregoing reasons, great value would devolve on any test which could clearly differentiate between the normal state and instances of mild disease difficult to recognize on clinical grounds. It is most unlikely that one will discover an all-or-none metabolic response to slight excess or deficiency of a normal bodily constituent, i.e., thyroid hormone. By the same token, it is probably a forlorn hope to seek clear-cut abnormalities of any metabolic index in a patient whose metabolic disturbance is only mild."

Basal Metabolic Rate (BMR). Consideration of the abandoned BMR test for thyroid function reveals the dramatic change that has taken place in endocrine diagnosis. One of the main criticisms of the BMR was the difficulty and uncertainty of obtaining a basal measurement. The test was done first thing in the morning, often after an overnight stay in the hospital. Hospital confinement is not exactly conducive to relaxation, in most cases. On the other hand, some patients were asked to get up slowly, dress, and go to the physician's office and relax for thirty minutes prior to the test. The test might be done with the patient breathing into a large tube with a clamp over his nose, or alternatively with a tight mask over both nose and mouth. Again, this is not conducive to the relaxation necessary to be at a basal rate. A high percentage of patients are anxious about any examination procedure, adding still more problems in obtaining the basal rate.

Recently a new technique has been developed for evaluating thyroid function by basal oxygen uptake. Using modern equipment, oxygen uptake is quantified to determine the metabolic impact of thyroid hormones. It is expected that this measurement may be especially useful when thyroid hormone estimates are ambiguous, and in evaluating patients with non-thyroidal illness who have low levels of T_4 and T_3. Braverman[46] states, "Finally we have a potentially accurate test of the whole body metabolic rate."

Cholesterol and Total Lipid Levels. Routine evaluation of thyroid function should be done when cholesterol levels are elevated. Barnes[19,20,21] makes a strong case for hypothyroidism being a major cause of coronary heart disease. Schwartz[372] states, "Although thyroid antibodies are not documented risk factors for coronary heart disease, it is possible that they cause minor degrees of thyroid failure, resulting in hypercholesterolemia and coronary disease."

When cholesterol levels are elevated in conjunction with hypothyroidism, serial measurements of cholesterol can help evaluate the effectiveness of treatment.

Achilles Reflex Time. Deep tendon reflex muscle contraction is slower in hypothyroidism and more rapid in thyrotoxicosis.[252] Instruments have been developed to measure the time from the point of tendon stimulation to one-half muscle relaxation. The most popular of these measures the foot movement from Achilles tendon reflex stimulation with a photoelectric cell. The instrument is called a photomotogram (PMG)[53]; it is no longer manufactured. The change in muscle contraction and relaxation speed is not due to differences in the neural component of the reflex arc, but to differences in the speed of both muscular contraction and relaxation, particularly the latter.

Fogel et al.[143] evaluated thyroid function of 270 subjects by Achilles tendon reflex time (photomotogram), and compared the results with PBI, cholesterol and I^{131}, which were the laboratory tests available at that time. The laboratory method and Achilles reflex time were statistically comparable in diagnosing both hypo- and hyperthyroidism. More important, the Achilles reflex time reflected the improvement of the patients' symptoms when the abnormal subjects were put on medication. Once the reliability and validity of the Achilles reflex test was demonstrated, they proceeded to test people under various physiologic and pathologic states. They found the test was not adversely affected by pregnancy, obesity, malignancy, agitated emotional state, or neurologic or neuromuscular disorders, as long as the Achilles reflex could be measured. They note that the Achilles reflex test measures but one parameter of thyroid function; to thoroughly evaluate the thyroid, a battery of tests is needed. This test has value as a screening

tool and for following patients under therapy; "...it has been shown to be a specific measure of the peripheral activity of thyroid hormones."

In another study, Sherman et al.[380] found no significant difference between the I^{131} test and the Achilles reflex time in detecting either hypo- or hyperthyroidism. Initially there were many favorable reports similar to these on the accuracy of the photomotogram in evaluating the thyroid state. Other conditions can also influence the muscle contraction, as further studies have indicated[130,382]; consequently, there is dispute regarding its effectiveness as a diagnostic tool. Additional factors negative to its use are that an electrocardiogram is necessary to act as the recording device, and the test is limited to those who have an Achilles reflex that can be measured. When there is bilateral absence of the reflex, one should suspect diabetes mellitus even in the absence of fasting hyperglycemia and glycosuria.[130]

Advantages of the photomotogram, when its limitations are taken into consideration, are that it gives immediate information for analysis, is not influenced by drugs the individual may be taking for thyroid regulation, it measures body function rather than the thyroid hormones available in the bloodstream, and radioactivity need not be used in pregnant women. The best advantage in a natural health care practice is that it provides an excellent method to follow up with serial determination the effectiveness of thyroid treatment.

This instrument has been in use in my office since it was introduced around 1965. At that time applied kinesiology had just been introduced, and my practice consisted mostly of spinal manipulation and exercise; it was very limited as far as recommending nutritional supplementation. I had numerous patients who were taking thyroid medication at the time they came to me for additional assistance with their health problems. As chiropractic corrections were obtained, patients often told me that they were feeling so much better they wondered if they should discontinue taking their thyroid medication. As is my usual practice, I recommended that they consult the physician who originally prescribed the medication. Many of them decided to discontinue the medication on their own; they continued to feel better, had more energy, and often there was some weight loss. Naturally, I believed my chiropractic approach was allowing the patient's body to improve his thyroid function. When the photomotogram became available, I quickly bought one to "prove" that chiropractic was "curing" hypothyroidism. This young doctor had much to learn!

Many of the patients were originally given thyroid medication as a therapeutic trial by their physician because of fatigue and weight gain. When the patient improved hypothyroidism was diagnosed, whether or not there was objective laboratory evidence that it was responsible for the patient's fatigue. The apparent improvement from taking thyroxine was simply from artificially speeding up the metabolism when, actually, no hypothyroidism was present. This is analogous to driving an automobile in second gear, then when wanting to go faster, stepping on the accelerator harder and harder instead of shifting to high gear. A car can travel faster by this method, but it is hard on the engine and obviously not the proper thing to do. I found that when a new patient on thyroxine tested within the normal range by the photomotogram, my treatment was usually ineffective in improving the condition. Only those patients who tested hyperthyroid when taking thyroxine were able to withdraw from the medication coincident with my treatment. It was not until years later that my ability to improve true hypothyroidism increased. The use of applied kinesiology techniques presented here allowed some patients who tested normal on the photomotogram while taking thyroxine to improve and be able to reduce or eliminate their medication.

Basal Axillary Temperature. An effective method of evaluating an individual's metabolic rate is monitoring basal temperature. The method is described by Barnes[18,21] for the routine evaluation of thyroid activity. He originally observed the correlation of low temperature with low basal metabolic rate while doing BMR studies when they were the only method of diagnosing hypothyroidism by laboratory methods. In 1942, he reported on his study of 1,000 college students, which indicated subnormal body temperature is a better index of hypothyroidism and the need for thyroid treatment than the basal metabolic rate.[18] During his WWII tour of duty, he and another doctor evaluated 1,000 soldiers and found the normal axillary temperature to be 97.8-98.2° F (36.5-36.7° C) when taken first thing in the morning before arising. The axillary area was standardized as the optimal place to take the temperature because it eliminates the variables of oral infection, such as periodontitis.

The patient is instructed to shake the thermometer down before going to bed, and place it on a nightstand within easy reach without getting out of bed. Immediately upon awakening, the patient puts the thermometer in the axilla and lies quietly for ten minutes, after which the thermometer is read. (Figure 12—24 shows a chart with these instructions, and a graph on which the temperatures can be recorded to bring to the physician's office for evaluation.)

Thyroid disturbance is a potential problem at any age. It is often difficult to get young children to lie quietly while the axillary temperature is taken. In this case, the temperature can be taken rectally for two minutes. Be certain to tell the parent to use a rectal thermometer with its thicker glass and round bulb. Normal rectal temperature is 1° higher (98.8-99.2° F) than axillary.

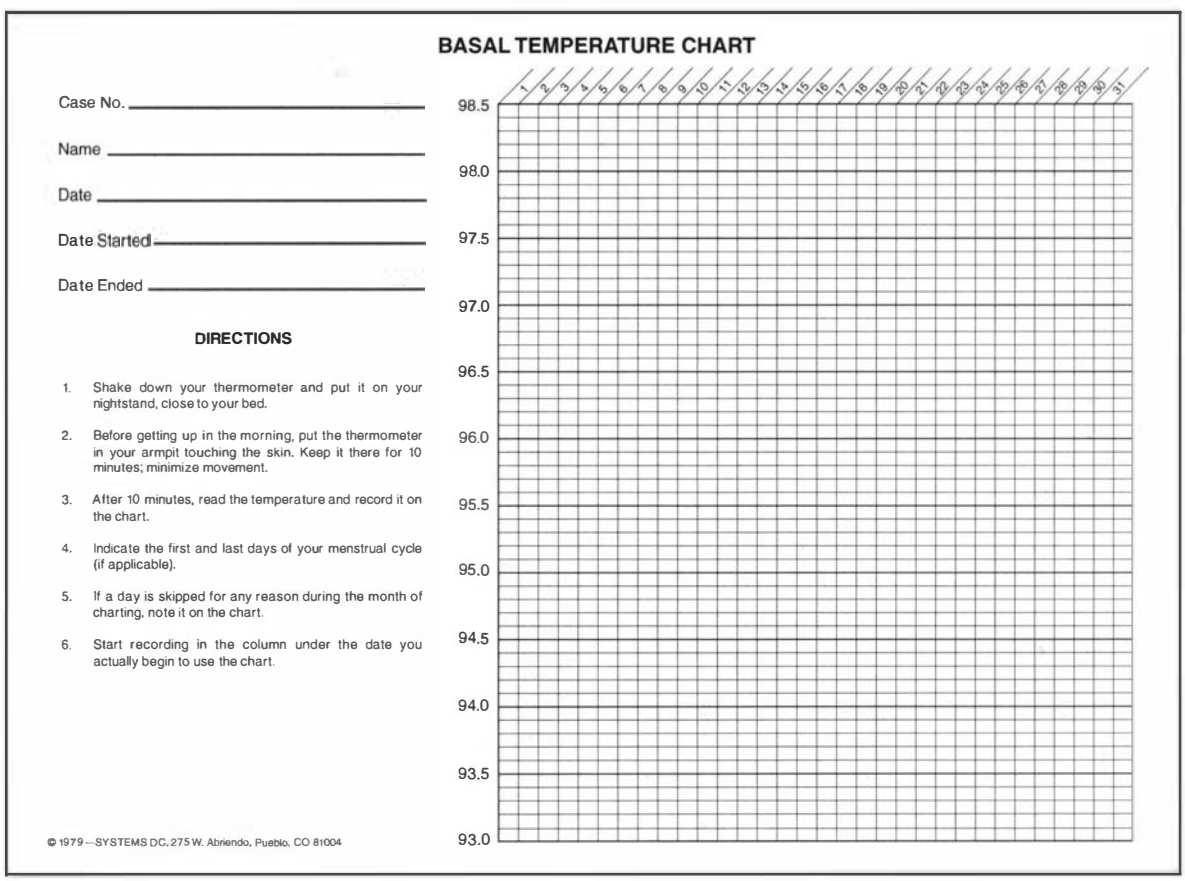
In males it is generally adequate to record the temperature for one week. There will be some variance from day to day, but it will normally be minimal. In females of child-bearing age, there is a normal fluctuation in

temperature with the menstrual cycle. The temperature is lowest during ovulation; it is highest shortly before the start of the menstrual flow. Barnes[21] suggests that the ideal time to evaluate the female's temperature is the second and third days after menstruation begins. There is no fluctuation before menarche or after menopause. It is recommended that menstruating females take their temperature for a full month, indicating on the chart the beginning and ending dates. The changes occurring throughout the cycle help analyze female endocrine patterns.

As the day progresses, an individual's temperature will increase from the basal level. When examining a patient in the office, one can generally consider that an axillary temperature below 97.8° F reflects low thyroid function. This should be followed up with the basal temperature taken at home, as indicated above. Many who take temperatures in the office use an electronic thermometer for a more rapid evaluation. The time needed to take the temperature electronically varies with the instrument. If the patient has axillary perspiration, the electronic transducer — or, for that matter, a regular thermometer — should be covered with a plastic sleeve to isolate it from the perspiration. These are available from health care supply houses.

Basal axillary temperature above 98.2° without infection is strongly indicative of hyperthyroidism. This is not found nearly as often as the lower temperature. When a patient is taking thyroid medication, temperature elevation is an indication that the dose is too high.

Axillary temperature is not specific for thyroid function. Starvation, infection, and adrenal and pituitary deficiency, among other factors, can cause a deviation from the normal level. Barnes,[19] the primary proponent of axillary temperature as a monitor of thyroid function, recognizes this by stating, "It is apparent that basal temperature is not a specific test for thyroid function, but it is a very useful tool for diagnosing and treating cases of hypothyroidism. Frequently, subnormal basal temperatures will be found in patients who are apparently in excellent health. They do not require treatment at that time, but subsequently they often develop symptoms that usually respond to therapy when needed. Furthermore, if too much hormone is administered, the basal temperature is quickly elevated above the normal range." Barnes primarily treats his patients with desiccated thyroid U.S.P., and has found that the temperature will rise slowly (two months) with the treatment. The temperature may still be subnormal when all symptoms are gone.



12—24. Basal temperature chart.

Subclinical Hypothyroid

In an applied kinesiology practice, one sees many cases where it appears there are undiagnosed subclinical hypothyroid conditions although a battery of examination procedures has been done. The endocrinologist is to be applauded for his in-depth ability to study pathology. It appears many patients are overlooked that do not have optimal function. As mentioned in the introduction to this section, there is a continuum between optimal thyroid function and frank disease processes. Those who have symptoms from poor thyroid function deserve the same effort to gain optimal function as those with frank disease processes.

Barnes[21] recognizes the difficulty in obtaining accurate information regarding a patient's thyroid hormone level and how his body is using it. "What we ideally need to measure is the amount of thyroid hormone on the inside of each cell in the body where it controls the rate of oxidation of fuel burning within the cell." Obviously, with the billions of cells in the body this is impossible, and the exact role played by thyroid hormones in cell metabolism is unknown.

Occasionally one finds information in the literature pertaining to subclinical hypothyroidism. In a study by Ridgway et al.,[335] the cardiac systolic time intervals and serum cholesterol levels were studied in patients (n20) who had normal serum levels of thyroxine (T_4) and triiodothyronine (T_3) and elevated levels of thyroid-stimulating hormone (TSH). They were treated with medication (L-T_4) to normalize the TSH secretion; it significantly changed the systolic time interval, indicating that such patients apparently have a mild form of primary hypothyroidism that benefits from appropriate therapy. Schwartz[336] indicates, "This important study provides additional justification for the treatment of patients with 'asymptomatic' hypothyroidism. 'Asymptomatic' is the proper term here because the patient is unaware of subtle changes that have taken place." Commenting on another study, Schwartz[372] states, "...I am becoming convinced that there is a sub-population of patients with coronary artery disease in whom subclinical hypothyroidism is a risk factor."

Barnes,[20] who diagnoses thyroid deficiency primarily by clinical symptoms and findings and the basal temperature test, was asked by a pharmaceutical firm to compare a new thyroid product with the natural desiccated product he had been using. Before the patients changed to the new product, the pharmaceutical house requested that their blood be evaluated by three different thyroid-function tests. In 20% of the cases, the three tests gave three different diagnoses — one indicating too much thyroid hormone present, one revealing the concentration was just right, and the third showing too little present. In another experience, Barnes' laboratory included the then new radioimmunoassay test for T_4, along with other tests being run, at no additional charge. Forty-eight of his new patients with classical symptoms and findings of thyroid deficiency were tested. None were hypothyroid according to the test; one was found to be hyperthyroid. In addition, thirty-two symptom-free patients being treated with desiccated thyroid were evaluated. All showed values within the normal range by the new method. The average for the treated group was almost identical to that of the untreated group.

Hypothyroid Symptoms

When reviewing the symptoms of hypothyroidism, it is necessary to remember the continuum between optimal function and frank disease processes. The symptoms and the function discussed vary widely among patients. Severe conditions, such as myxedema, are rare in the general practice; however, "...lesser degrees of hypothyroidism are very common and undoubtedly often overlooked."[212] The symptoms listed here should be put in perspective with the continuum of dysfunction, with greater involvement relating with greater symptoms and milder symptoms with milder involvement.

Rubel[346] describes the body build of the hypothyroid individual as quite typical, in that weight and bulk are carried in the middle. Waist and abdomen are large, the trunk is full, and waist/hip measurements differ by less than 10".

The hypothyroid patient will generally be sluggish, with low ambition and motivation. Once the individual gets up in the morning and begins daily activity he feels better, especially with active exercise. The problem is that the individual has difficulty motivating himself to do any physical activity. Often these people are considered lazy and apathetic, but this may be reversed with proper treatment.

The general sluggishness extends to the mental processes, including poor thought processing, memory loss, confusion, and depression; these people are often stoic or morose. There may be a sudden change in the person's personality. The patient tends to go to pieces easily under only slight pressure, does not like to be watched, and tends to cry easily. The latter is more prevalent in women because of the training men receive early in life: "Boys don't cry." Depression may or may not be associated with the crying, which often occurs for no apparent reason. Both learning disabilities and attention deficit disorder can be caused by hypothyroidism.[19] This is also frequently associated with blood sugar handling stress and neurologic disorganization.

The slowness of metabolism results in frequent complaints of cold hands and feet. The pattern for the hypothyroid person is to be sensitive to cold, and he likes warm weather. The hypoadrenic is sensitive to both cold and hot, humid weather. Lowered tissue metabolism and circulation cause conditions to develop in the skin and its appendages, including dry, chapped, and flaky skin. Cracking around the heels and hands may be severe enough to bleed. Lotions used by these people usually provide little or only temporary relief, but people con-

tinue to use them in great quantity. "Because of the skin's slow rate of growth, wounds of the skin tend to heal slowly. A bruising tendency is common in hypothyroidism and results from an increase in capillary fragility."[213] Poor quality nails are associated with thyroid deficiency. These can be recognized in examination by brittleness, leading to cracking or softness. They fail to have a sheen and a healthy look, and grow slowly. Hair also grows slowly, so that haircuts and shaving are required less frequently. There may also be hair loss associated with thyroid deficiency. It may be spotty alopecia, loss at an early age, or change in hair loss rate. The classic loss of hair in hypothyroidism is at the lateral third of the eyebrow.

Fat metabolism, in general, is a problem. Fatty congestion of the liver often develops, and there is usually an elevated cholesterol level. Barnes[20] attributes the abnormal accumulation of mucopolysaccharides in hypothyroidism as a major factor in the development of atherosclerosis. "The mucinous material consists of protein complexed with two mucopolysaccharides, hyaluronic acid, and chondroitin sulfate B, especially the former. It is mobilized early during treatment with thyroid hormone, leading to an increase in the urinary excretion of nitrogen and hexosamines."[213] This accumulation in the skin and under tissues is responsible for the thickened features and puffy appearance of hypothyroidism.

Kountz[245] studied the effects of thyroid treatment on the cardiovascular system in persons with low basal metabolic rates. His subjects were divided into three groups. Some were treated with thyroid substance, and others were not treated to serve as controls. The patients were followed for a period of five to seven years.

The first group consisted of office patients who were business executives; they were the youngest of the three groups. None were under forty years of age; most were in their late fifties, with an average age of fifty-five. This group had very little evidence of atherosclerosis. In the treated group there were no deaths; in the untreated group there was 15% cardiovascular deaths.

Group two was made up of general office patients with thyroid deficiency. In general, the group had mild to moderate cardiovascular degenerative changes. People with advanced disease were excluded from the study. There was a striking difference between those treated and untreated for their thyroid deficiency, which was 3% and 20% cardiovascular deaths, respectively.

The third group consisted of patients who, for one reason or another, had to be confined to the St. Louis City Infirmary and its hospital. These all showed an advanced degree of vascular degeneration, both on clinical and laboratory examination. The death rate in this group during the study period was considerably greater, but there was still a great difference between those treated and untreated for their thyroid deficiency, i.e., 38% and 75% deaths from cardiovascular disease, respectively. The study by Kountz vividly demonstrates that the earlier people with hypothyroidism are effectively treated, the less chance they have of developing coronary heart disease.

Weight control is often a problem; however, all hypothyroid persons are not overweight, nor are all hyperthyroid persons underweight. In hyperthyroidism there is an increase in the appetite and ultimate food intake. The body can typically use the extra calories, because the thyroid hormones increase the rate of secretion of digestive juices and the motility of the gastrointestinal tract. Conversely, people who have hypothyroidism tend to have a reduced energy level with a reduced appetite. If they consume more calories than their bodies need, they rapidly gain weight and have a difficult time losing it. Their digestive systems tend to be sluggish with hypo- or achlorhydria, hypoproteinemia, and constipation.

Swelling is a classic sign in myxedema. In subclinical thyroid deficiency the swelling may be limited to a few areas, e.g., ankles, elbows, or wrists, leading to a carpal tunnel syndrome. The swelling may be most easily observed in the eyelids, or as a thick, swollen tongue.

It is common for females with hypothyroidism to have menstrual irregularities and miscarriages. Rubel[346] attributes the miscarriages to a lack of thyroid secretion preventing or decreasing efficiency of the corpora lutea to form progesterone. There is often a loss of libido in both males and females.

An unusual correlation found with thyroid deficiency is extreme tenderness of the costal cartilages when palpated. This was originally reported by Rubel.[346] It has been found in applied kinesiology that chewing iodine will often immediately relieve the pain from 20% to as much as 80%.[356]

Hyperthyroid Symptoms

In a natural health care practice, it is not necessary to treat hyperthyroidism as frequently as hypothyroidism. These people tend to have thin skins and fine features. They tend to have occasional erratic, flighty behavior. This includes nervousness, restlessness, irritability, temper tantrums, weeping, and depression. There is an increased appetite but little tendency to gain weight.

With hyperthyroidism there is a tendency toward muscle hyperactivity, with poor balance when standing on one leg. Muscle tremors of a fine and rapid frequency — 10 to 15 times per second — are present. Symptomatically, the tremors are often described as shakiness. They are most easily observed in the tongue and fingers. An easy method of observing the tremor is to place a piece of paper on the patient's outstretched hand. The vibrations are exaggerated in the paper and are easily recognized.

Objectively, tachycardia is often present and the patient complains of heart consciousness and shortness of breath. He is sensitive to heat and tends to like cold weather.

Increased thyroid hormone increases the secretion rate of most of the other endocrine glands, but not necessarily by direct action. Examples are increased metabolic rate causing higher glucose metabolism, with an increased need for insulin secretion by the pancreas; increased bone formation, requiring more parathyroid hormone; and, finally, the liver inactivates adrenal glucocorticoids more rapidly, leading to ACTH production by the anterior pituitary and, therefore, increased adrenal cortex activity.

Diagnosis and Treatment

All methods of examination should be considered in evaluating thyroid function. The laboratory tests previously discussed put in perspective the frank disease processes of hypo- or hyperthyroidism. The Achilles reflex time and basal temperature help put in perspective subclinical hypothyroidism, and — finally — applied kinesiology methods augment the investigation. A study by Jacobs et al.[218] found that, "AK enhanced but did not replace clinical/laboratory diagnosis of thyroid dysfunction."

The teres minor muscle is associated with the thyroid gland in applied kinesiology. A high percentage of patients with subnormal axillary temperature will have a standing posture of one or both arms internally rotated so that the palms face more posteriorly because of weakness of the teres minor muscle(s).[168] About one-third of the patients with low axillary temperature will test weak on one or both teres minor muscles in the clear, or with therapy localization to the neurolymphatic or neurovascular reflex. Therapy localization to the reflexes is augmented by simultaneous right or left brain activity.[218] As with other factors in applied kinesiology, it is of value to test the individual in various positions, such as standing or sitting as well as supine and prone.[353] Other factors, such as running and walking, can also have an influence on the meridian system and other patterns of the thyroid gland. The teres minor muscle that does not test weak in the clear or with therapy localization will frequently have a muscle stretch reaction, indicating the need for fascial release technique.

Nutrition

Nutritional supplementation can be an asset in improving thyroid dysfunction; in some cases, it may be the cause of the dysfunction. Nutritional products commonly involved are iodine, thyroid nucleoprotein extract or concentrate, and vitamin A. Some patients will require thyroid hormones, preferably desiccated thyroid U.S.P., discussed later.

The teres minor and sternocleidomastoid muscles frequently respond to iodine supplementation when they test weak. Occasionally the pectoralis major (sternal division) is involved. When the sternocleidomastoid muscle tests weak and is strengthened with iodine in the mouth, it is very often associated with thick mucous secretions. The thick mucus may appear as postnasal discharge, which gags and irritates the patient during the day or night. There may also be thick secretions elsewhere in the body that are only observed in external orifices, such as the vagina.

Paradoxically, iodine may improve hypothyroidism; too much may cause hypothyroidism. Iodine therapy was used to treat hyperthyroidism before the currently used drugs, which are more effective.[103] In hypothyroidism, a higher dose of iodine can be given for approximately one week; then it should be tapered down to close to the recommended daily allowance, which is 0.1 mg.

Some patients respond well to iodine supplementation, and then it seems to lose its effect, even when the proper dosage is taken. These patients may have a parotid, or possibly thymus, gland involvement. If therapy localization to the suspected gland causes the teres minor muscle to strengthen, it is indication that supplementation with that glandular substance will be of value. Have the patient taste the nutrition to determine if it, too, strengthens the teres minor muscle.

It is necessary that the patient receive the proper amount of vitamin A. "The rate of thyroidal uptake of iodine is markedly reduced in A-deficient animals."[221] Goodheart finds that excessive vitamin A is sometimes the cause for hypothyroidism.[158]

When supplementation with iodine and iron is necessary, the two must be given at different times since they are antagonistic. For example, have the patient take iodine in the morning and iron at night. Giving iron to an individual who needs iodine will make him worse.

Occasionally one will find an individual who has the symptomatic pattern of hyperthyroidism, yet he has a low basal temperature and photomotogram reading. Goodheart[158] proposes that ribonucleic acid (RNA) unlocks the cell door for thyroid hormone to control metabolism. These patients also tend to have a poor memory, which will be helped by RNA supplementation. Sometimes numerous tablets per day are necessary to obtain results.

In the mildly hyperthyroid patient, higher dosages of iodine may be of value. "Clinically, hyperthyroidism increases the need for vitamin A and lowers the serum level of the vitamin and its precursor."[221] Vitamin A helps with the increased load of fat metabolism caused by the rapid metabolic rate. Hyperthyroidism increases the requirements for vitamin B_6.[221] This is indicated by the decreased uptake of radioiodine when the thyroid is deficient in B_6. This suggests that vitamin B_6 has a role in thyroid metabolism.

Examine for and treat all five factors of the IVF associated with the thyroid gland. Because there is considerable interaction within the endocrine system, other glands should be evaluated for their possible correlation with thyroid dysfunction by two-handed therapy localization. For example, after clearing the

thyroid neurolymphatic reflex, continue having the patient therapy localize it while his other hand touches the adrenal, spleen, gonad, or other glandular reflex. If there is again positive therapy localization, the second gland being therapy localized is probably involved in some way with the thyroid dysfunction. Evaluate the five factors of the IVF for that gland and correct as indicated.

After treating all five factors of the IVF for the teres minor and for any associated organ involvement, remeasure the patient's axillary temperature. If it is still low, test the teres minor for a positive muscle stretch reaction (page 192), which will be present in most cases. Application of the fascial release technique will cause the axillary temperature to rise to normal, or close to normal, in some of the patients. If the temperature fails to rise adequately, have the patient chew thyroid concentrate or nucleoprotein extract, combined with iodine. Re-apply the fascial release massage; in most cases, the temperature will rise. When it is necessary to chew nutrition in combination with the fascial release technique to obtain results, daily supplementation will be necessary to maintain correction. If there is a failure to obtain temperature elevation with the fascial release, re-evaluate all of the muscles associated with the gonads, because something may have been missed. The gonads are inhibitory to thyroid function.[167]

Thyroid Medication

Some patients will not respond to the thyroid treatment described. If prescription medication is not within the treating doctor's scope of practice, the patient should be referred for appropriate examination and treatment. The type of thyroid medication given may be important in the patient's response. Barnes[20] strongly recommends the use of desiccated thyroid powder U.S.P. in tablet form. He points out that the balance between T_4 and T_3 is important, and they must be given together. "Each of the synthetics will relieve some of the symptoms but not all of them." Barnes goes on to state, "No one can guarantee that these two active synthetic compounds represent all of the physiological activity present in the natural gland. I have tried the various new preparations on patients who had been on the natural gland for years and no one has preferred the synthetic. I repeatedly see patients who have been on one or the other preparations and still have some symptoms of physical findings which disappear on whole gland therapy." All endocrinologists do not agree with this. One study indicates that "...treatment of thyroid disorders with desiccated thyroid results in an elevated T_3 level that may cause thyrotoxic symptoms and a low thyroxine level that could lead to an injudicious increase in dosage."[371]

Thyroid supplementation immediately after a heart attack may be detrimental. The thyroid causes the heart to work harder; thus it could cause another crisis. Hutton[212] states, "...One should remember that thyroid increases the heart's work faster than its strength." Barnes[19] recommends not giving thyroid for two months after a heart attack, then starting with 1/2 grain. Treating the thyroid with natural methods, as indicated here, does not appear to present the same level of potential problem as supplementing the patient with thyroid hormones; however, the physician should be cautious in post-coronary occlusion treatment.

There may be times when you have treated a person with hypothyroidism that withdrawal from thyroid medication will be indicated. Schwartz[336] comments on a study in which thyroid medication was withdrawn for three weeks from patients (n10) who had been on long-term thyroid medication to determine if they needed it. None developed symptoms during this period; only four were documented as hypothyroid by both an elevated thyrotrophin and a low T_4 level. This justifies a three-week period of withdrawal of thyroid hormone if it is felt there is a possibility of normal thyroid function. Schwartz goes on to comment on the overuse of thyroid for obesity and fatigue, or "...to regularize menstrual periods (a 'double blind' ploy of 'practical docs,' (double blind in the sense that neither physician nor patient knows that it is a placebo that has been prescribed), or simply as a 'tonic.'"

Hyperlipoproteinemia

It is well-established in the literature by epidemiological studies that the risk of coronary heart disease increases with elevated serum cholesterol levels. Although this information is not new, documentation of the relationship continues to strengthen. That seems to be about the only point of general agreement, and there are even those who disagree on that!

Journalist Meg Greenfield[180] of *Newsweek* comments on how "they" keep changing their advice on how to control cholesterol, and even whether it should be controlled. By "they" she means those indicated in "'They don't think you should eat eggs. They don't think bacon is as bad now as they thought for a while.' The references are to those medical researchers at what we all must have figured out by now is the prestigious They Institute of America. Of the They Institute, shrouded in mystery and old cholesterol recipes, we know only this; it is an adjunct of two teaching hospitals in a big university somewhere, and its scientists, who are affiliated with various other institutions around the coun-

try, are charged with producing 'new findings.' These do not overturn 'old findings,' as you might expect, because, thanks to the ceaseless efforts of the They Institute, there are no 'old findings' any more. What new findings overturn are other new findings that had just barely got established in the understandably addled public mind." She goes on to state, "By now [the public] suspects that what is banned today is likely to be administered intravenously in the best clinics tomorrow."

Over thirty-five years ago I was interested in the cholesterol levels of my patients. When the family/patient history or examination revealed high blood pressure, coronary heart disease, atherosclerosis, arteriosclerosis, cerebral vascular accident, or liver congestion, I would recommend a blood test to evaluate the patient's cholesterol and triglyceride levels. Often I would hear, "That's not necessary, I'm on a low cholesterol diet." I would ask, "When did your doctor last do a blood test?" Often the answer was, "Oh, he never checked it again after he found the high cholesterol." I then strongly recommended that it be re-tested to determine if the diet was effective. Very often the cholesterol level was still high and often higher than before going on the diet.

In those early days of practice, I emphasized to the patient the importance of eliminating eggs, saturated fats, and dairy and meat products from his diet. This is what I had been taught, and what I read in the literature. Often the patient told me that's what he was doing. I emphasized how important it was to follow the recommendation rigidly. When I re-tested the patient's cholesterol after about two months, it had rarely gone down; often it had increased. I tended to blame the patient's lack of compliance. Eventually I quit actively looking for patients with an elevated cholesterol level because it wouldn't come down anyway.

Over the years, many factors regarding hyperlipoproteinemia have come to my attention in the natural health literature. In the final analysis, much of the answer is — as usual — that when we get away from natural health processes, problems develop.

During my early years of practice, I considered normal cholesterol levels to be 150-250 mg/dl. I was amazed and somewhat amused to see the laboratory that I referred to continue to elevate its "normal" levels. At one point, the normal level for persons over 65 was considered 325 mg/dl. It appeared that as the population gained higher average cholesterol levels with aging, the accepted norm was elevated because the average was considered "normal." In 1983 the Council on Scientific Affairs of the AMA[98] stated, "There is good reason to believe that the average cholesterol levels in persons in the United States may be higher than optimal due in part to the typical US diet."

In 1972, Wynder and Hill[426] polled 35 eminent cholesterol researchers regarding their opinions on optimal cholesterol levels. They recommended the following for various ages: 10 years, 146 mg/dl; 30 years, 174 mg/dl; 50 years and above, 185 mg/dl. Their general consensus was that the 1972 accepted levels were too high. In 1979, cholesterol researchers at a workshop concluded that the ideal level for adults is in the range of 130-190 mg/dl.[388]

Today the dietary approach generally recommended (but not totally agreed with here) is from the 1982 recommendations of the American Heart Association (AHA),[185] i.e., to reduce saturated fatty acids to less than 10% of total calories by substituting unsaturated fats, also not to exceed 10% of total calories. The recommended total fat intake by the AHA, including monounsaturated fatty acids, is 30%. If overweight, reduce the percentage of fat in the diet and increase carbohydrates to compensate for the loss of fat. The carbohydrate recommended is complex carbohydrate, to make up 55% of total calories. Cholesterol reduction to 300 mg per day is recommended. Adjust caloric intake to maintain optimal weight.

The literature abounds with numerous studies testing the lipid hypothesis and its relation to coronary heart disease. Although research continues on a massive scale, the hypothesis still has clearly not been accepted by all who test it.[25,132] Prominent investigators and groups have criticized the diet recommendations generally given to prevent coronary heart disease.[20,25,188,272,284,403] Ahrens[5] outlines many of the problems in developing properly designed tests.

Animal experiments should play a secondary role in the evaluation of lipoproteins. Their metabolism differs in many ways from that of man, and research results may not be reproducible in man. Rabbits are herbivores; consequently, they have no cholesterol in their natural diet. It is thus reasonable that they would have a great sensitivity to dietary cholesterol that is not reproducible in carnivores. Rabbits are the animals most sensitive to dietary cholesterol,[403] yet many conclusions have been drawn by studying them.

The source of cholesterol in research is important. Dietary cholesterol is combined with phospholipids, causing them to be large molecules. Pure cholesterol, on the other hand, is much smaller and may be absorbed at a different rate.[338] Crystalline cholesterol added to a low-fat diet is poorly absorbed, and in some experiments it may be recovered quantitatively in the feces.[120]

Klevay[240] cites German scientists who began in 1924 to increase rabbits' absorption of cholesterol by adding bile acids to the cholesterol diet. One must question extrapolating data from animal experiments that require the animal to consume food and chemicals outside its normal diet, and applying that data to man. This is compounded by the difference in cholesterol metabolism between an animal and man.[403] Dietschy and Wilson[120] state, "...It is clear that man absorbs less than 10% of that [cholesterol] fed, whereas in the other species 30-70% of the fed amount is absorbed." "Although it is clear

that this variation in cholesterol absorption is a major cause of the differences among the species in the response of both the serum concentration and overall balance to cholesterol feeding and, as such, is the major reason that the serum concentration of man is altered very little by cholesterol feeding, the reasons for the differences among species are totally obscure."

In the discussion here, much literature that evaluated crystalline cholesterol in animals has been ignored. When a study is cited, it will be indicated if it is an animal study that should be put in proper perspective by the reader.

Research should be conducted on humans living their usual life-styles and eating their usual foods. When research data is applied out of context, one may come to erroneous conclusions. Truswell[403] points out that the analysis of milk, with its highly saturated fat content and only 2% linoleic acid, indicates it should raise cholesterol levels, but he cites research indicating that it may slightly lower them. He points out the importance of studying foods in their total complex and in the manner in which they are eaten, rather than in laboratory-prepared concentrations. The substances that neutralize the effects of the saturated fats in milk may be calcium, lactose, or hydroxymethylglutarate. He states, "It is less scientific to say 'milk contains more saturated than polyunsaturated fat, therefore it will raise LDL' [low density lipoproteins] than to measure the effect of milk, with all its constituents, on plasma lipids and find that it does not. We should think in terms of whole foods and not try to generalize from partial knowledge of their biochemical composition. What really matters too is the effect of foods in the range that people normally consume."

The method by which many have studied the effect of egg consumption on serum cholesterol levels is an example of taking food out of its natural supply in the research design. Many studies have been done with egg yolk powder. Kummerow et al.[247] point out, "Even though two medium sized fresh whole eggs contain only 11.7 g of protein, they contain, except for methionine and phenylalanine, the approximate total daily requirement for all eight of the essential amino acids. Two fresh egg yolks do not provide enough protein to satisfy the total daily amino acid need for any of the eight essential amino acids, although two fresh egg yolks contain as much cholesterol as two fresh whole eggs." They go on to point out that amino acids are essential for building the apolipoproteins that "carry" the cholesterol in the blood. A balanced dietary amino acid level results in a lower serum cholesterol level than does an imbalanced one. Studies done using the whole egg[142,247,314] resulted in findings different from those using powdered egg yolk. (Eggs and their relationship to diet will be discussed later.)

If one simply watches television commercials and reads popular press magazine articles and advertisements, one has no question that dietary cholesterol and saturated fats should be reduced and polyunsaturated fats should be increased; however, confusion reigns if one looks deeper into the subject. On one hand, the American Heart Association[185] recommends the increase of polyunsaturated fat consumption to prevent coronary heart disease; on the other hand, the National Academy of Science[184] recommends decreasing it to prevent cancer. What is one to do? There is no simple answer.

This writer and most practitioners of applied kinesiology are adamantly opposed to the application of the American Heart Association's dietary recommendations to the masses. For a large percentage of the population, the recommendations are not necessary; for others, they are inadequate. In addition, their recommendations do not take into consideration important factors such as reducing the use of trans fats to a minimum, and the importance of dietary fiber, which will be discussed later. Our position has been unpopular in the past among the "scientific community"; however, there is currently support for the position applied kinesiology has taken for over twenty- five years. Commenting on the "Rationale of the Diet-Heart Statement of the American Heart Association," in which they list ten references as the best available evidence to support the recommendation that the amount of dietary cholesterol be limited to 300 mg/dl per day, Becker[25] cites Reiser as discovering that "...almost all of [the references] were either irrelevant or actually antithetical to the recommendation — and after analyzing further the cited basis for additional recommendations relating to total fat, saturated fat, and substitution of unsaturated fats and carbohydrates, he concluded that 'the Rationale is not a logical explanation of the dietary recommendations but an assemblage of obsolete and misquoted references. Since rational explanations for the recommendations are essential for their acceptance, the public to whom they are addressed is justified in remaining skeptical of them.'"

In the early and mid-1950s, many papers contributed to the diet-heart hypothesis. Looking back at these studies and what has happened in the food market, such as the increased promotion of artificial foods and trans fats, one interested in natural health has to be concerned with where the trend is taking the modern dietary pattern. Mann[272] critically looks at the trend, stating, "The diet-heart hypothesis was inflated by Keys in 1953. He used a selection of data from the World Health tabulations to conclude that in six countries, experience with coronary heart disease was correlated with available food fat. The naïveté of such an interpretation of associated attributes is now a classroom demonstration. Nevertheless, in a few years some combination of the urgent needs of health agencies, oil-food companies and ambitious scientists have transformed that fragile hypothesis into treatment dogma." As one who is interested in natural health evaluates the food supply available at the typical supermarket, it becomes obvious that a major portion of the problem with the

food supply is economic. Processed foods are adulterated for longer shelf life. Fats are hydrogenated to avoid rancidity; they are colored and refined to provide more eye and palate appeal. Mann[272] goes on to state, "The dietary dogma was a money-maker for segments of the food industry, a fund raiser for the Heart Association, and busy work for thousands of fat chemists."

Rather than making an across-the-board dietary recommendation, the population should be educated regarding proper food selection. Individuals with specific health problems should consult a physician knowledgeable in natural health when manipulation of the diet is necessary for the prevention or treatment of a condition. Conditions such as coronary heart disease and cancer are multifactorial, and biochemical individuality[421] must be considered for each person. It is important to consider sensitivity and resistance to dietary items.[188] Some people may be more sensitive and develop alcoholic-type hypertriglyceridemia, or have a large increase in plasma cholesterol when they eat eggs. The latter may result from genetic hypercholesterolemia.[403]

Cholesterol Metabolism

Total body cholesterol comes from endogenous and exogenous sources. "With the possible exception of mature nerve tissue, every animal tissue adequately investigated seems capable of incorporating labeled acetate into cholesterol."[146] The primary sources of endogenous cholesterol are the small intestine (jejunum) and the liver.[119] There is considerable interaction in the regulatory systems by which cholesterol synthesis and degradation interact, causing a remarkable homeostasis in which the steady state concentration of body cholesterol tends to readjust to a constant norm. A change that tends to deplete either cholesterol or bile acids causes an increased synthesis. On the other hand, expansion of the cholesterol pool causes an inhibition of cholesterol synthesis and promotes cholesterol metabolism to bile acids. Approaches to regulate cholesterol by dietary restrictions deal with only one aspect of the cholesterol metabolism; an ideal approach is consideration of both.[121] In addition to diet and nutritional supplementation, the applied kinesiologist should evaluate and correct function, when necessary, of the organs and systems involved in exogenous absorption and endogenous production of cholesterol.

Potentially limiting factors in cholesterol absorption are the physical form of cholesterol in the diet, the size of the bile acid pool, the permeability characteristics of the lumen mucosal membrane, the relative activity of cholesterol esterase, and the rate of chylomicron formation. Cholesterol is best absorbed in conjunction with a high fat diet.[120] Total body cholesterol is regulated by three factors: absorption, synthesis, and excretion. When dietary cholesterol is increased, two mechanisms help maintain a steady state of body cholesterol levels: "(a) Increased reexcretion of cholesterol (but not of bile acids), and (b) decrease in total body synthesis." There is a great variability between individuals regarding the decrease in synthesis.[325]

Hepatic cholesterol synthesis is suppressed by exogenous cholesterol in the diet, or by feeding exogenous bile acid, which facilitates the absorption of endogenous and exogenous cholesterol.[119] One of the mechanisms for maintaining a proper cholesterol level is its enhanced excretion through the biliary tract when there is greater absorption. Vitamins C and B_6 are necessary for cholesterol to be converted into bile acids in the liver.[338] In addition, "...when the enterohepatic circulation is intact, synthesis of cholesterol is inhibited by continual reabsorption of endogenous cholesterol."[325] When endogenous cholesterol production is not suppressed, there are greater accumulations of cholesterol. Hepatic synthesis is increased by any activity that interferes with the absorption of cholesterol reaching the liver from the intestine in chylomicrons. For this reason, it is important that the lymphatic system function in an optimal manner.[161]

Examination and treatment of the pancreas, small intestine, liver, and gallbladder, when indicated, are important in cholesterol-regulating problems. It is particularly important to have optimal lymphatic function, especially of the small intestine. Cholesterol absorption takes place in the small intestine, and it is transported by the lymphatic system. The colon plays no part in cholesterol absorption. When cholesterol is put into the large bowel, there is no observed increase in cholesterol absorption.[146]

There are six steps in the absorption of cholesterol[120]:

1. Cholesterol esters are hydrolyzed to free cholesterol in the presence of pancreatic cholesterol esterase and bile acid.
2. Free cholesterol in the intestine is solubilized in mixed micelles by bile acids and other amphipathic substances, such as monoglycerides and fatty acids.
3. In contact with the brush border, it moves across the cell membrane, probably by passive diffusion.
4. Cholesterol then mixes with the intracellular pool of unesterified cholesterol.
5. A major portion of this pool is then esterified with long-chain fatty acids.
6. Cholesterol is then incorporated into the intestinal lymph as chylomicrons.

LDL-HDL

The low-density lipoproteins (LDL) are richest in cholesterol, and the high-density lipoproteins (HDL) appear richest in phospholipids. The very low-density lipoproteins (VLDL) consist primarily of triglycerides. The cholesterol in LDL is that responsible for atherosclerosis, important in coronary heart disease. The cholesterol in HDL is considered the "good" cholesterol. HDL appears to remove deposited cholesterol from the tissues.[403]

The ratio between LDL and HDL is important in determining the risk of coronary heart disease. A practical approach done by most laboratories is to obtain the ratio of total cholesterol to HDL, rather than the LDL:HDL ratio. If one's total cholesterol is below 150 mg/dl, the HDL level is not as important since there is not enough LDL to be a problem. On the other hand, when cholesterol is very high (over 350 mg/dl), the HDL will not be high enough to protect against the atherogenic level of cholesterol. The ratio of total:HDL cholesterol below 4.5 is a reasonable goal. This is the standard risk of women, who have fewer heart attacks than men. An ideal ratio is 3.5, which is half the standard risk for men.[226] The inclusion of the total:HDL cholesterol ratio in the laboratory report helps evaluate the risk for coronary heart disease. The impact of HDL level appears greater than that of LDL beyond the age of 50.[226,324]

The best established dietary factor that changes the total:HDL cholesterol ratio is weight reduction of the obese. HDL tends to be subnormal in any case of severe hyperlipidemia, and it may also be decreased in diabetes. Alcoholics and women tend to have a higher HDL level. Estrogen raises HDL, even in men, and androgens lower it. Women tend to have a higher HDL level even after menopause, persisting beyond the age of 60. Middle-aged men who do leisurely long-distance running have a higher HDL level.[403] Tangier's disease is a familial condition in which there is absence of or an extremely low HDL level.[190]

Types of Fats

As noted previously, the scientific community has recommended a reduction in total fat intake, concentrating on reducing the saturated fats. Applied kinesiology's position is that cholesterol and natural fats are not the problem in coronary heart disease. In fact, it is often found that health problems are due to a deficiency of essential fatty acids. The metabolic pathways of essential fatty acid breakdown to the prostaglandin series and the effect of prostaglandins on health provide a fascinating study outside the scope of this discussion and textbook. The reader is encouraged to independently study the applied kinesiology literature on essential fatty acids.[171,254,268,359,360]

In 1965 Goodheart indicated that meat fat is not a major problem in cholesterol metabolism.[161] Stearic acid contributes substantially to the composition of beef fat and other animal fats. In a recent study[40] comparing the effect of dietary stearic acid, oleic acid, and palmitic acid on plasma cholesterol, it was found that stearic acid lowered the total cholesterol level by 14%. The LDL cholesterol was lowered by 22%; there was no change in HDL cholesterol. The ratio of LDL:HDL cholesterol was 19% lower. On the high oleic acid diet, the total cholesterol and LDL cholesterol levels were 10% and 15% lower, respectively, than when on the high palmitic acid diet. This study was designed to compare stearic-acid-rich fat directly with palm oil, which is high in palmitic acid. Although this study seems to indicate that beef fat and other fats containing high stearic acid are not the major problem, an editorial in the same journal[342] recommends not changing the basic dietary message to the American public.

In a crossover study, Flynn[141] had subjects eat five ounces of raw red meat and one egg daily for three months, and then five ounces of fish or poultry and one egg daily for three months. Although the beef fat provided an average of 24 gm of total fat compared to 10 gm of fat from the poultry or fish, there was no change in total serum cholesterol.

Gamma linolenic acid has been shown to lower cholesterol in both animal and human studies. Animal studies can determine the viability of an approach prior to human studies that are more important in the final determination of effectiveness. In a comparative rat study, Huang et al.[207] first made the animals deficient in essential fatty acids for eight weeks. They developed symptoms of essential fatty acid deficiency, such as growth retardation, scaly skin, and rough fur. The animals were then divided into three groups and fed 20% of their energy as fat from hydrogenated coconut oil, safflower oil, or evening primrose oil (EPO), a common source of gamma linolenic acid. Half of each of those groups was also supplemented with cholesterol as 1% of the diet by weight. The cholesterol-fed groups having hydrogenated coconut oil and safflower oil had substantially elevated plasma cholesterol, but the EPO group had only a slight elevation. The plasma cholesterol concentration was similar in all three of the groups that did not have cholesterol supplementation. Total liver cholesterol was significantly lower in the non-cholesterol-fed EPO group, indicating a reduction of endogenous cholesterol synthesis. "In the cholesterol-fed groups, in contrast, total liver cholesterol was highest and plasma cholesterol lowest in the EPO group. This suggests that EPO is able to stimulate the transport of cholesterol from the plasma into the liver. Thus there appears to be two separate effects, a suppression of cholesterol synthesis and an enhancement of clearance of cholesterol from the plasma."

When the rats in the above study were provided fat after the fat deprivation period, the ones on safflower oil and EPO recovered from the essential fatty acid

deficiency symptoms; the ones on hydrogenated coconut oil did not.

The effect of gamma linolenic acid compared to cis-linoleic acid on cholesterol was evaluated in a human double-blind crossover study by Horrobin and Manku.[206] The gamma linolenic acid was supplied by EPO, which was about 100 times as effective at lowering cholesterol than the cis-linoleic acid.[205] An effective dosage was 3 or 4 gm per day for twelve weeks. The EPO had no effect on HDL cholesterol levels. All lowering was attributed to a fall in the LDL cholesterol. There was no lowering of cholesterol in those who started the study with normal levels, suggesting that EPO acts physiologically to regulate cholesterol metabolism and does not have a pharmacological effect. The effectiveness of gamma linolenic acid in lowering cholesterol increases with higher starting levels of cholesterol.

Another source of gamma linolenic acid is black currant seed oil.[412] In addition, it supplies linolethic, alpha linolenic, and stearidonic acid. In both EPO and black currant seed oil, the fats are provided in their "cis" form, which is the natural unprocessed polyunsaturated fatty acid. When fatty acids are processed to be the "trans" form, discussed next, essential fatty acid metabolism is blocked.

The use of polyunsaturated fats in lowering cholesterol should not be abused. There has been considerable publicity about their effectiveness in efforts to obtain general population diet modification. Exuberance about developing health may cause some to overreact in diet change. Under certain conditions, polyunsaturated fats can be harmful. There is considerable agreement that diets rich in polyunsaturated fatty acids depress the cell-mediated immune response. Evidence is strong that the immune response is sensitive to both the quantity of dietary fat and its degree of unsaturation.[383] In addition, polyunsaturated fats enhance tumor growth.[184] "The conclusion at the present time is that diets rich in polyunsaturated fat are more effective than diets rich in saturated fat in enhancing tumorigenesis in animals, and this argues that administration of polyunsaturated fatty acids to humans should always be on a replacement basis."[383]

Hydrogenated Fats

The hydrogenation of fats began around the turn of the century as a method to prolong storage and the shelf life of fats in grocery stores. "When products based on partially hydrogenated vegetable oils were being established as competitors of dairy products, a plethora of publications on both sides of the political controversy shed more heat than light upon the subject."[202] The hydrogenation of fats has found its strongest niche in making corn oil solid to serve as margarine. Hydrogenation is the process of adding hydrogen atoms to the double bonds of unsaturated fatty acids, which convert the molecule from its natural cis configuration to the "trans" configuration. Cis and trans fats metabolize differently. Each isomer must be recognized as an entity because biological systems do just that.[202] Hydrogenated fats inhibit desaturation and elongation of normal polyunsaturated fatty acids.[233]

Most diet recommendations to the general public strongly emphasize the reduction of saturated fat. Emphasis is on reducing animal fats, such as meat fats, butter, and milk, and increasing polyunsaturated vegetable fats. Inadequate public reference is made to the detrimental effects of hydrogenated fats. Americans consume 600 million pounds of hydrogenated fats each year, more than all the corn oil manufactured in the fifty states.[150] With the emphasis on decreasing saturated and increasing polyunsaturated fat, there was an increase in vegetable fat consumption from 24.0 to 43.1 pounds per person per year between 1950 and 1975. Unfortunately, two-thirds of that fat was hydrogenated to make margarines and shortenings.[247] "These fats contain more saturated fats than butter, whole milk, and meat, and few — if any — vitamins and minerals."[150]

Even when a fat starts out as a quality one, it can be transformed by oxygenation. This can happen from exposure to air, making the fat rancid, or by overheating. Oils used in frying foods are particularly subject to rancidity, especially in restaurants where french-frying oil is heated continuously for long periods. The total amount of oxidized products from heating oil and other material that was in the unused oil, such as free fatty acids and mono- and diglycerides, is called "polar material." In general, polar material not exceeding 25% is considered acceptable as far as odor and taste are concerned; above 30%, the fat has deteriorated.[32]

Both the length of time and amount of heat are important in the oxidation process. Shallow or pan-frying is accomplished quickly, and it is not common to re-use the fat; therefore, the polar material usually does not reach the objectionable level. In deep-fat frying in the home or restaurant, the fat is kept hot for prolonged periods with only occasional use for frying. In commercial factories there is constant frying, and a great amount of fat is removed along with the fried food. Since fat is continually being added, it does not develop a high amount of polar material. It is with the continued re-use of oil for deep-fat frying in the home or restaurant that the polar material may go over the "acceptable" level.[32]

To demonstrate the amount of fat in a potato chip, light one and watch the oil drip out as the chip burns. Be careful! Once when I did this, the flaming oil dripping from the potato chip burned a hole in the carpet.

With the increased consumption of overheated fats and trans fatty acids, there is an increased need for essential fatty acids, such as linoleic acid. Hydrogenated fats contain little linoleic acid. Trans fatty acids also reduce prostaglandin production and interfere with the conversion of linoleic acid to arachidonic acid in

the formation of prostaglandins.[150]

Many people are misled by food labels when seeking to reduce their intake of saturated fatty acids. One should look for polyunsaturated vegetable oil, such as safflower, corn, sesame, soybean, or sunflower. Highly monosaturated oils — such as olive or peanut — are also good. When one looks for vegetable oils, coconut, palm, and palm kernel oil may be considered good when actually they are highly saturated and should be avoided. As previously indicated, hydrogenated oil should be avoided. The term "partially hydrogenated" tells little; it can mean anything from 5% to about 60% saturated. Food processors may simply list "vegetable oil" rather than giving the specific type, or there may be a list, such as, "Contains one or more of the following: soybean, corn, safflower, hydrogenated cottonseed, and/or palm kernel oil." By listing the oils in this manner, the manufacturer is free to choose the oil that has the lowest market price at the time of manufacture. The choice is usually the highly saturated oils because of price, and they also provide a more stable product for longer shelf life.[416]

Many who recommend using margarine instead of butter fail to take into consideration that an appreciable portion of vegetable oil used to make margarine is partially hydrogenated. This increases the need for the four essential fatty acids, such as linoleic acid, which unfortunately is in low supply in many margarines.[23] In a study of fifty margarines, there tended to be high concentrations of trans fatty acids accompanied by low concentrations of linoleic acid. The amount of trans fat in margarine varies from 8% to 70%, depending on the brand.[23] The rule for labeling margarine is that the first item listed must represent at least 50% of the product.[310]

Processed and Artificial Foods

In my opinion, the major contributing problem to elevated cholesterol levels and coronary heart disease and other conditions included in syndrome X is the increased use of artificial and processed foods, not natural foods such as meat, dairy products, and eggs. Hunter, in her book *The Great Nutrition Robbery*,[209] quotes frequently from food industry journals with regard to increasing profit levels by adulterating foods for improved storage, appearance, palatability, and changed texture. In 1970 the United States Department of Agriculture estimated the percentage of retail cost for ingredients in coffee cream at 45%, as opposed to 27% in non-dairy cream; whipping cream, 59%, non-dairy whipping cream 15%; and sour cream, 32%, imitation sour cream 15%.

Advertising for non-dairy coffee creamers directed to restaurateurs emphasizes another economic advantage of the artificial food. It can be left on a restaurant table unrefrigerated for prolonged periods without spoilage.

Hunter[209] comments on the hydrogenation of polyunsaturated fats to trans fats, "The alteration makes them suitable for the manufacture of margarine and other plastic shortenings, as well as for use in various other processed foods including breads, cakes, pies, and cookies. The raised melting point improves the fats' consistency and color in french frying of foods and protects the fats from developing off-flavors or becoming rancid quickly."

When non-dairy coffee creamers first hit the market, they had to be labeled as a product of non-dairy origin. Hunter[209] comments that the action was daring because it was thought that consumers would shy away from the imitation food. She goes on to say, "But the new products were launched at the time of the cholesterol scare. Although the fat level of the coffee creamers and nondairy creamers was basically the same, people inferred wrongly that the new products were low calorie, low fat, and low cholesterol. The new products gained in popularity." "Nondairy creamers have at least as many calories as whole milk and they contain *more* total saturated fat than is found in the butterfat of milk." Non-dairy creamers — in addition to water — contain some hydrogenated oil, which usually consists of coconut or palm kernel oil. Soybean oil is used less frequently. There are often sodium caseinate solids (from milk) or, less frequently, "vegetable protein" (probably from the soybean). Sweetening agents usually consist of sucrose, corn syrup solids, and — less frequently — lactose or sorbitol. In addition, there are a number of chemicals, "...including sodium and calcium phosphate or di-potassium, sodium citrate; propylene glycol monostearate, polysorbate 60, mono- and di-glycerides, sorbitan monostearate, or sodium stearoyl 2-lactylate; carrageen and guar gum; sodium silico aluminate; salt; lecithin; artificial color and flavor, and at times a preservative, benzoate of soda."[209]

Non-dairy whipped toppings control a large percentage of the market. "By 1974, nondairy whipped toppings controlled about 85% of the whipped topping market."[209] These products are judged to have excellent flavor and texture, and — possibly more important — they have long shelf life and look good on desserts. Toppings made from real cream are perishable, have bacterial problems, and, in general, will not hold up in restaurant and institutional situations. The early success of frozen cream pies was due almost entirely to the development of a coconut oil based non-dairy product. "Another synthetic cream was created by combining about 15 percent sodium alginate and 5 percent methyl ethyl cellulose. The product had very satisfactory whipping properties."

Examples of food adulteration and synthetic foods could go on and on. One more example will suffice to emphasize the seriousness of this problem. "Imitation cheeses and cheese products are usually made of vegetable oils (frequently highly saturated coconut oil), protein, hydrolyzed cereal solids, buffer salts, color, and flavor." "Lack of mold growth on the replacer cheeses

during storage is an asset for processors. For consumers, the speed with which perishable foods mold may serve as an indicator of their high biological quality."[209] Hunter quotes an eminent nutritional researcher, Dr. Elmer V. McCollum, who advised, "Eat *only* foods that rot, spoil or decay — but eat them before they do!"

"A new margarine substitute: free from chemical additives, based on old family recipe passed down from cow to cow."
— Sign in grocery

Cholesterol Ingestion

After reading well over one hundred articles on the effects of cholesterol ingestion on serum cholesterol levels, it seems to this writer that almost any conclusion can be drawn; it just depends on the observer's attitude about natural and adulterated foods, and the interpretation of laboratory experiments done on animals and on humans. In my opinion, free-living human studies are best. For example, in one human experiment[81] daily food intake was recorded for a group of twenty-nine healthy subjects over a one-year period. They did not consume any nutritional supplements and were on their own self-selected diets. Their blood was monitored five times during the one-year study. Serum cholesterol did not correlate significantly with dietary cholesterol, saturated fat, or linoleic acid.

In another human study (n12),[9] crystalline cholesterol was added to a cholesterol-free diet. During one study period, the subjects were given saturated fats to compare with another study period of unsaturated fats. In both cases, the crystalline cholesterol and other diet factors remained the same. Almost all animal products were eliminated from the diet to maintain the low-cholesterol level, with the exception of the crystalline additive. The serum cholesterol rose the same in the subjects, whether the diet contained saturated or unsaturated fatty acid combinations. One wonders what the results of this study would have been if the cholesterol had been supplied as found in natural foods.

The literature abounds with animal studies indicating that cholesterol intake raises serum cholesterol. As has been previously mentioned, it is questionable whether this data can properly be extrapolated to man. Since many authorities use animal studies to evaluate cholesterol consumption and its relation to serum cholesterol, it is interesting to compare how animals prosper on food products designed to reduce cholesterol consumption in man. Navidi and Kummerow[295] fed mother rats and their two- to three-day-old pups egg substitute (Egg Beaters™) and compared them with mother rats and pups that ate whole natural eggs. As a control, another group was fed a stock commercial laboratory chow. The pups on the egg substitute averaged only 31.6 gm in weight at three weeks of age. Those fed whole eggs achieved 66.5 gm, or more than double. Animals fed the stock diet did slightly better, with a weight of 70 gm. Both the mothers and pups fed the substitute egg product developed diarrhea within one week; those fed whole eggs did not develop diarrhea. The pups were weaned at five weeks of age. All those fed the egg substitute died within three to four weeks after weaning, with an appearance of gross nutritional deficiency. The rats eating the natural egg diet appeared to be in good health.

In applied kinesiology it is consistently emphasized that it is necessary to examine patients in the way in which they live. Structurally, this means standing, after walking and running, in postural positions (BID), and other methods that simulate the way one lives. Similarly, it seems reasonable that research on cholesterol should be done with foods available from grocery stores, and with the subjects maintaining their normal daily activities. Porter et al.[314] conducted a crossover study (n55) of free-living men who ate a diet that included one egg per day for three months, then they ate no eggs for three months. During the egg period, the dietary records indicated an increase in dietary cholesterol of approximately the amount of one egg over that when eating no eggs. There was no significant change in serum cholesterol levels with the dietary intake of one whole egg per day compared to no eggs. In a similar study, Flynn et al.[142] increased the eggs to two per day and found no significant increase in serum cholesterol or triglycerides during the egg period as compared with the no-egg period. Vegetarians differ greatly in their egg consumption. In a study to evaluate the cholesterol levels among those who consume considerable eggs and those who do not, there was no difference in cholesterol levels.[264]

Experiments in three separate countries indicated that most patients' cholesterol did not rise at five hours or up to fifty-four days after two whole eggs per day were included in their diets. In all three countries, the serum cholesterol level of some subjects increased while others decreased on the egg diet. In two individuals, the serum cholesterol raised more than 100 mg/dl, showing the individuality of response to dietary intake of cholesterol.[247]

Pfeiffer[310] gives strong support for eating two eggs per day for their nutritive value. He emphasizes that it has not been shown that the cholesterol in eggs is a primary reason for elevated serum cholesterol, or that the elimination of them (unless one's cholesterol is extremely high) helps reduce cholesterol levels. Pfeiffer indicates that one of the main nutritive needs supplied by eggs is sulfur, which is difficult to get in the usual diet. In addition to sulfur, he lists the following nutritional advantages. "The egg has the amino acid pattern in the proportion closest to the needs of your body. The egg is low in fat, rich in protein and vitamin A, low

in calories and economical (much cheaper and higher quality protein than meat). It is, furthermore, a good source of vitamin B_{12} if you are a vegetarian. (B_{12} is found only in animal products.) It also contains (some in larger amounts than others) choline, tryptophan (precursor of niacin), pyridoxine (B_6), biotin, folic acid (a B vitamin), riboflavin (B_2), thiamine (B_1), pantothenic acid (a B vitamin), selenium, zinc, phosphorus, calcium, and sulfur." Truswell[403] supports this view with the comment, "What now appears to be a doubtful disadvantage to eating moderate amounts of egg has to be balanced against the valuable nutrients in this inexpensive and convenient food...."

Another value of eggs is the ideally designed shell container provided by the hen. Once the egg is laid, it cannot be adulterated. Granted, eggs are probably not of the quality they once were because hens are confined and bred for higher egg production. Cages are usually 14" x 24", with five to seven hens per cage. Their food supply is manipulated for maximum egg production, and the lights stay on twenty-three hours a day to keep them feeding and laying.[93] Eggs from chickens raised in a natural environment are probably more healthful, if one can obtain them.

Egg consumption can be positive or negative, and the literature can be cited to support either view. It appears that most individuals can properly process eggs in a natural diet. There are some who, because of hereditary factors, cannot. Kummerow et al.[247] suggest there is value in determining the effect of whole eggs on patients. It is necessary to run more than one cholesterol determination to obtain reliable information on a specific subject's response to the source of dietary cholesterol. If an individual's cholesterol does not rise with the dietary intake of eggs, it provides a wider choice of food items than does the present recommended protocol given to the general population.

The National Research Council[294] cites several studies in which diet modification was employed to lower the incidence of coronary heart disease. The studies comprised about 20,000 man hours of observation, and there was no overall reduction in mortality. For five years other studies on hypocholesterolemic drugs involving 18,000 men were carried out. In addition to an unimpressive reduction of coronary artery disease, some unpredicted toxicities were observed.

Reflecting on the dietary recommendations of the American Heart Association and the Senate Select Committee on Nutrition and Human Needs, the National Research Council states, "Unfortunately, the benefit of altering the diet to this extent has not been established. As noted, other studies employing diets containing 35-40 percent of calories from fat and higher P:S ratios (polyunsaturated to saturated) have shown equivocal effects on coronary disease and have been accompanied by a somewhat greater incidence of gastrointestinal disease." "No significant correlation between cholesterol intake and serum cholesterol concentration has been shown in free-living persons in this country. For these reasons, the Board makes no specific recommendations about dietary cholesterol for the healthy person."[294]

Mass modification of the general population's diet appears to be ill-advised. The Canadian Committee on Diet and Cardiovascular Disease[113] states in their recommendation, "Evidence is mounting that dietary cholesterol may not be important to the great majority of people and that severe restrictions of cholesterol need only be applied to those genetically disposed toward hypercholesterolemia. Thus, a diet restricted in cholesterol would not be necessary for the general population."

The Associated Press recently reported that the American Heart Association plans to identify and endorse processed foods that meet the group's dietary quidelines. It went on to state, "The first group of foods that will be examined by an independent testing group will be margarines, cooking oils and salad dressings. The association does not expect to endorse meats, fruits and other non-processed food items."[14]

Hunter[209] quotes Kummerow of the Burnsides Research Laboratory: "What I have considered strange in the diet recommendations of the AHA (American Heart Association) is the slight concern towards the consumption of potato chips, french fried potatoes, doughnuts, snack foods and soft drinks. These are all high calorie foods that can be converted to cholesterol in the body. Yet, none of these items have been vilified in the same manner as the egg."

One of the problems with the dietary recommendations generally given for cholesterol reduction is the replacement of natural food rich in nutrition with processed foods containing trans fats and with nutritional products refined out. "Principal sources of cholesterol in the American diet are meats, poultry, fish, shellfish, eggs, and dairy products."[135] These are food products that are valuable nutritional sources. Even if one does desire to eliminate fat and cholesterol from meat, the information being provided is misleading. Feeley[135] points out that trimming fat from meat does not cause a proportionate reduction in cholesterol, as is commonly believed.

The basis on which applied kinesiologists and most others in natural health care deal with the diet-heart question is to educate the patient in choosing an optimal diet. This includes reduction of processed foods containing trans fats, ultra-refined foods (such as white sugar and white flour), obtaining adequate roughage, and other general principles of good diet. In addition to not generally recommending cholesterol reduction, the Canadian Committee on Diet and Cardiovascular Disease gives this summary of dietary recommendations[113]:

"a) The consumption of a nutritionally adequate diet as outlined in Canada's Food Guide.

"b) A reduction in calories from fat to 35% of total calories. Include a source of polyunsaturated fatty

acid (linoleic acid) in the diet.

"c) The consumption of a diet which emphasizes whole grain products and fruits and vegetables and minimizes alcohol, salt, and refined sugars.

"d) The prevention and control of obesity through reducing excess consumption of calories and increasing physical activity. Precautions should be taken that no deficiency of vitamins and minerals occurs when total calories are reduced."

As indicated in the early portion of this section, patients should be evaluated for hyperlipoproteinemia when the family/patient history or examination reveals high blood pressure, coronary heart disease, atherosclerosis, arteriosclerosis, cerebral vascular accident, or liver congestion. In addition, some recommend that cholesterol measuring become a routine of all health maintenance physical examinations.[7] Hyperlipidemia in an individual suggests that family members may be at risk; since dietary management is a family affair, consideration should be given to extending the dietary recommendations to the patient's entire family.[98]

In general, the lipoproteins should be evaluated after a 12- or 14-hour overnight fast because triglyceride levels increase shortly after ingestion of any fats. There is little diurnal variation in the plasma cholesterol levels. Before beginning treatment of hyperlipoproteinemia, it is necessary to have at least two serial determinations, approximately one week apart, for a baseline value.[98]

Treatment

In addition to the factors already discussed, the generalized dietary recommendations currently being given to the masses by the American Heart Association have a relatively low success rate in greatly lowering cholesterol levels. Dietary reduction in the free-living subjects of the Diet-Heart Feasibility Study averaged only 10-12%; in the Multiple Risk Factor Intervention Trial, there was only a 6.5% reduction, despite special attention to patient motivation and dietary instruction.

Many of the factors to aid in reducing hyperlipoproteinemia that follow are basic health recommendations that are usually given by applied kinesiologists. When dietary change is made solely to lower cholesterol, one must consider the patient's total condition, including age. Major efforts to lower the cholesterol may not be in the patient's best interest. Taylor et al.[395] have recently constructed a mathematical model to predict the value of cholesterol, blood pressure, and smoking cessation on life expectancy increase. The model is based on the findings in the Framingham Study. According to their model, the increased life expectancy by lowering cholesterol is quite low for people at "low risk," and it is calculated to be from eighteen days to twelve months for those at high risk in the age range of 20-60 years. The cessation of smoking and lowering of high blood pressure give a much higher increase of life expectancy. Both of these factors contribute to other diseases besides coronary heart disease and, consequently, reduce morbidity.

Whenever there is hyperlipoproteinemia, a complete physical examination is necessary. Any condition primary to hyperlipoproteinemia, such as diabetes mellitus, hypothyroidism, nephrosis, obesity, excessive alcohol consumption, the use of contraceptive pills, biliary obstruction, or genetic hyperlipidemia should be considered and treated, if necessary, in their relationship to hyperlipoproteinemia.[98]

Phospholipids. Lecithin, a common form of phospholipids, is effective in lowering cholesterol.[150] Phospholipids increase fecal excretion of neutral sterols, helping to reduce the absorption of both exogenous and endogenous cholesterol.

Pottenger and Krohn[319] studied 122 high-cholesterol patients who were fed a high-fat diet consisting of over 4,000 Kcal daily. It included at least one tablespoon of raw liver and one of raw brains daily — foods rich in cholesterol. The diet also included B vitamin concentrate from rice bran. The treated group (n91) received, in addition, one teaspoon of soybean phospholipids, generally known as lecithin. The other group eating the same diet without the lecithin served as controls (n31). Seventy-nine percent of the patients who took the phospholipids had a cholesterol reduction of 15 mg/dl or more. Thirteen patients had an increase of cholesterol up to 15 mg/dl, and six had an increase of 15 mg/dl or more. In the control subjects, about half had a lowering of cholesterol and half had an increase. This again shows the biochemical individuality[421] of people.

Rinse[338] reports on a study comparing blood from men with atherosclerosis with those not having the disease. The cholesterol level did not correlate with the occurrence of atherosclerosis, whereas the quantity of phospholipids contained in the blood fat did. If a blood sample contained 36% or more phospholipids in its blood fats, there was no atherosclerosis; when there was 34% or less, everybody had the disease.

Zinc/copper ratio. There is considerable data to support the hypothesis by Klevay[238] that a copper deficiency increases serum cholesterol levels. Initial studies were done on rats, where it was shown there was a decrease in liver copper as a result of feeding cholesterol plus cholic acid. When copper was added late in the experiment, the elevated serum cholesterol returned to normal, along with liver copper.[240]

In the initial human study of one man, depletion of copper in the diet caused an increase in plasma cholesterol. Repletion of copper brought the cholesterol back down.[241]

In a more extensive study by Reiser et al.,[333] twenty-four subjects were fed a copper-deficient diet of 0.36 mg copper per day per 1000 Kcal for eleven weeks. During this time, there was a significant increase in LDL cholesterol and significant decrease in HDL

cholesterol when compared with the pre-experiment level of 0.57 mg copper per day per 1000 Kcal. Upon repletion of copper to their diet (1.41 mg copper per day per 1000 Kcal), the subjects' HDL and LDL levels improved to better than in pre-study levels. There was no significant change in total cholesterol levels. During the copper depletion stage, four individuals had to discontinue the study because they had cardiac-related symptoms.

Klevay documents that a large percentage of the population does not consume an adequate amount of copper per day.[239] In addition, high levels of zinc compete with copper absorption for binding to metallothein, which can cause copper deficiency.[139] When rats are fed an increased zinc to copper ratio (40/1), there is an increase of serum cholesterol in comparison with a lower zinc to copper ratio (5/1).[235]

To further study his hypothesis, Klevay[236] investigated the relationship of zinc to copper in high- and low-fat foods with the suspicion that higher fat-containing foods might have a higher zinc to copper ratio, interfering with copper absorption. In general, and statistically significant, the high-fat foods have a higher zinc to copper ratio. This may indicate that the increase of cholesterol is actually due to the decrease in copper absorption in high-fat diets. Another interpretation that Klevay places on the data is "...that both amount of fat and ratio of zinc to copper are important determinants of risk. Consideration of both factors may improve the utility of dietary analysis in the prediction of risk."

HDL cholesterol is increased with active exercise, such as running,[403] or long-duration, low-intensity physical activity, such as mail carriers walking in their occupation.[94a]

Two hundred seventy healthy volunteers over sixty years of age were graded as to physical exercise.[177] In the early phase of the study, it was surprising to find that there was not higher HDL cholesterol in the more physically active people. When the individuals taking over 15 mg of zinc per day were eliminated from the analysis, the active group had a significantly higher level of HDL cholesterol. Zinc supplementation was eliminated from twenty-two individuals; in eight weeks there was a significant increase in their exercise-related HDL levels.

Another study indicating the adverse effect of a high zinc to copper ratio on HDL levels was done by Hooper et al.[203a] Twelve healthy adult men ingested 440 mg of zinc sulfate per day for five weeks. Their HDL cholesterol concentration decreased 25% below baseline levels (40.5 to 30.1 mg/dl). Total cholesterol, triglyceride, and LDL cholesterol levels did not change throughout the study. To determine if a low dosage supplement of zinc lowers HDL levels like a high dosage, a study of sedentary and endurance-trained men was done.[101] Twenty-one men engaged in endurance exercises, and twenty-three sedentary men took 50 mg per day of zinc sulfate. After eight weeks, while plasma zinc increased 15%, there was no significant change in fasting HDL cholesterol, LDL cholesterol, total cholesterol, or triglycerides.

Zinc has received considerable publicity as a treatment for colds and other conditions. When a patient self-administers zinc, one should evaluate the amount being taken. When zinc and copper are kept within realistic levels, there is no significant effect on cholesterol metabolism.[139]

Klevay[242] studied drugs and chemicals known to affect cholesterol levels and their relation to copper metabolism. "Some of these chemicals are hypercholesterolemic and copper inhibiting: ascorbic acid, cadmium, cholesterol plus cholic acid, fructose, glucose, histidine, sucrose, and zinc. Others are hypocholesterolemic and copper enhancing: calcium, clofibrate, guaran (the principal polysaccharide from guar seeds), and sodium phytate." He proposes that a main effect on cholesterol may be due indirectly to their action on copper metabolism. Clofibrate (Antromid-S®), a main drug used to lower cholesterol, enhances the utilization of copper in rats fed a diet low in copper, concomitantly lowering cholesterol. Klevay[242] states, "The change in cholesterolemia from copper seems to be approximately one hundred times the response to clofibrate."

Recently Brea[47,49] has proposed an applied kinesiology test for copper that may prove of value in diagnosing copper deficiency. He hypothesizes that pressure applied to bend a long bone causes a piezoelectric release of electrons in a copper deficiency, causing a previously strong indicator muscle to test weak. This effect is eliminated by gustatory stimulation with copper.

Vitamin C. There is much controversy over the vitamin C effect on serum cholesterol levels. Cheraskin et al.[79] discuss several studies indicating that vitamin C deficiency can cause abnormal cholesterol levels. A one-year study[81] showed a significant positive correlation between vitamin C intake and HDL cholesterol levels in women; however, the same correlation was not observed in men. In the two cases of men with vitamin C intake in excess of 200 mg per day, there were very high HDL cholesterol levels, approximately 70 mg/dl, which were within the high normal range for women.

Truswell[403] reviewed several studies of vitamin C's effect on cholesterol levels that show numerous inconsistencies. Even proponents of vitamin C disagree with each other. In a rat study,[237] the cholesterol level was significantly higher with a vitamin C supplementation of 630 mg per day. It is proposed that the elevated cholesterol may be due to the ascorbic acid depressing copper absorption in the intestine, thus throwing off the zinc to copper ratio. One wonders why research is designed using this high level of synthetic vitamin C in animals that do not require the nutrient in the first place.

Thyroid. The thyroid should be routinely evaluated in hypercholesterolemia. Thyroid hormones increase bile steroid secretion in man.[288] In addition,

mucopolysaccharides are increased in thyroid deficiency. They are deposited in the artery wall, even in infants, to start atherosclerosis.

When either hypothyroidism or hypercholesterolemia is found, there is indication to evaluate for the other. Before the more sophisticated thyroid tests of today were developed, cholesterol levels were used to evaluate the possibility of hypo- or hyperthyroidism.[154,211] In 1932,Simonds[381] described the effect of the thyroid on serum cholesterol. When dogs were made hyperthyroid by feeding them excessive amounts of desiccated thyroid, there was no noteworthy change in the serum cholesterol level; however, when the thyroid was withdrawn, the dogs became hypothyroid as a result of marked thyroid atrophy that developed during the hyperthyroid period. During this period of recovery, there was a marked increase in blood cholesterol.

Barnes[20] cites numerous German scientists who show that thyroid deficiency causes atherosclerosis. Barnes had a practice high in thyroid-supported patients with a low incidence of heart disease — far below the national average.

Fiber. In general, increased dietary fiber is needed by a high percentage of people in the United States. The subject is discussed more thoroughly earlier, with colon function. The effects of a high-fiber diet may alter serum cholesterol in three ways[11]: (1) alteration of intestinal absorption, metabolism, and the release of cholesterol, (2) alteration of hepatic metabolism and release of cholesterol stored in the liver, and (3) alteration of peripheral metabolism of various lipoproteins.

The particular type of fiber most noted for reducing cholesterol levels is oat bran. Oat gum is a fraction of oat bran and constitutes 33% of the oat bran fiber. It is the apparent factor that lowers both triglycerides and total cholesterol, while increasing the HDL levels.[70] When oat bran is added to the American Heart Association's fat-modified diet, there is a further reduction in cholesterol level.[410]

When bread made with rolled oats replaced the usual bread of an experimental group (n21), the cholesterol level of the subjects lowered from 251 to 239 mg/dl after seven days, reaching a value of 223 mg/dl after only three weeks. When the rolled oat bread was replaced with the volunteers' normal bread, the serum cholesterol mean value rose to 246 mg/dl after two weeks.[109] Kirby et al.[234] found that 100 gm of oat bran caused a total cholesterol reduction of 15%, with an LDL reduction of 14%; HDL was unchanged. Fecal excretion of total bile acids was 54% higher on oat bran diets than on the control diets. They currently use 50 gm/day in outpatient treatment, with good patient acceptance and compliance.

An excellent method of consuming bran is in homemade muffins. Kowalski,[246] author of a recent popular book on lowering cholesterol, gives many excellent recipes for oat bran muffins, varied with different types of fruits and nuts to provide a palatable approach to consuming oat bran. In his book, he continues the popular recommendation of limited cholesterol intake, especially by eliminating eggs and changing fat levels to lower saturated fats and increasing polyunsaturated ones. He gives little consideration to the effect of trans fats on essential fatty acid metabolism.

Most studies indicate that fiber, in general, lowers cholesterol. Truswell[403] states, "Wheat fiber does not lower plasma cholesterol but pectin does and so does the pulp of 2 apples thrice daily." When Bengal gram (chick-peas) was added to the diet of volunteers in the amount of 247 gm per day to provide 16 grams of fiber daily, there was a marked reduction of mean serum cholesterol (from 206 mg to 160 mg/dl), even though the diet was high in fat (156 gms of butter fat per day). It was necessary to consume the diet for two weeks to produce the maximum lowering of cholesterol. Excretion of all bile salts was significantly increased on the high-fiber diet.[277] Increased fiber causes bile to become unsaturated with cholesterol within four weeks in subjects with gallstones.[282]

Trowell[401] emphasizes the loss of fiber from milling wheat and other grains. As a result, Western man has increased his incidence of colon and coronary disease. Trowell practiced in Africa for thirteen years and observed that Africans still eat approximately two-thirds of their calories in unprocessed foods or lightly milled maize. They have a low incidence of heart and diverticular disease. During his time in Africa, Trowell saw only one person with coronary heart disease — an African judge who ate Western low-fiber diets.[400]

An excellent method to obtain additional fiber is with leguminous seeds (beans, peas), which contain about 50% more fiber than cereal seeds for an equal number of calories. In addition, they are usually consumed whole; they are not processed into fiber-poor flour, as occurs in the case of cereals. They are therefore the richest source of fiber in the diet of Western man.[400]

Generally it is best to obtain fiber from natural food products. When this creates problems, it is possible to use psyllium as obtained in Metamucil® to provide bulk. Anderson et al.[10] demonstrated in a well-controlled study (n26) that packets of 3.4 gm psyllium three times per day lowered total cholesterol 14.8% with 20.2% reduction of LDL, making a reduction of the LDL:HDL ratio of 14.8%. Triglycerides were also lowered to 12.7% over the eight-week study.

Drugs. An article in *U.S. News and World Report*[405] briefly discusses drugs to increase HDL and lower LDL cholesterol levels. The importance of changing one's life-style to accomplish the same end is emphasized, indicating that the annual cost of the drug approach is from $500 to $3,000. This is only part of the story; the drugs have side effects.[13]

The Coronary Drug Project[96] was a massive study

to evaluate five drug approaches to lowering cholesterol. It started with 8,341 patients who were randomly assigned to six treatment groups: clofibrate (Antromid-S®), niacin, dextrothyroxine sodium, two dosage levels of conjugated estrogen, and a lactose placebo. The study of the two estrogen and dextrothyroxine sodium treatments was discontinued before the project was completed because of excess mortality, non-fatal cardiovascular events, thromboembolism, and cancer, as compared with the placebo group. The study of clofibrate and niacin was done for a minimum of five years; it did not support the use of clofibrate in reducing total mortality or cause-specific mortality from coronary heart disease. Clofibrate had "...a statistically significant excess incidence of thromboembolism, angina pectoris, intermittent claudication, and cardiac arrhythmia...as well as an excess incidence of the endpoint of any definite or suspected non-fatal cardiovascular event as compared with the placebo group. A non-cardiovascular finding of importance was a more than two-fold incidence in gallstones in patients treated with clofibrate."

In the Coronary Drug Project,[96] they also found no evidence of the efficacy of niacin with regard to total mortality or cause-specific mortality. Niacin did significantly lower the incidence of definite non-fatal myocardial infarction, but "...because of the excess incidence of arrhythmias, gastrointestinal problems, and abnormal chemistry findings in the niacin group, great care and caution must be exercised if this drug is to be used for treatment of persons with coronary heart disease."

Anabolic steroids increase LDL and decrease HDL cholesterol levels in rats. A study further showed anerobic exercise increases HDL and decreases LDL levels, and exercise removes the effect of steroids.[258] Nicotine is not only detrimental when obtained through smoking cigarettes; long-term use of oral nicotine, such as chewing tobacco and nicotine gum, induces an atherogenic lipoprotein profile of increased LDL and decreased HDL cholesterol levels.[87,200]

Hypertension

There are numerous ways applied kinesiology can reduce hypertension. Essential hypertension accounts for 90% of high blood pressure; the remaining 10% is secondary hypertension due to a variety of conditions, such as renal, endocrine, vascular, and neurologic disorders. Differential diagnosis must include evaluating for these conditions, since certain types of hypertension do not respond to the natural approaches indicated here. The natural methods to return blood pressure to normal discussed in this section are not intended to indicate that pharmacological methods should not be used. They serve to highlight corrective and preventive methods that can be used in applicable cases, eliminating the side effects that often parallel medication given to regulate high blood pressure.

An example is high blood pressure secondary to arteriosclerosis. With inflexibility of the blood vessels and increased resistance to circulation, the body's innate response is to raise the blood pressure to provide adequate blood to the brain and other structures. A high blood pressure is what the body wants, because this is its natural adaptive process to cope with the pathology. Attempting to lower blood pressure by examining for interference with normal nerve function and eliminating it is fruitless, because the nervous system is doing what it is supposed to do. Unfortunately, the adaptive process of elevating blood pressure for improved circulation to the brain makes the patient vulnerable to cerebral vascular accident. When blood pressure is elevated because of irreversible pathology, it is important that it be lowered by allopathic measures. Unfortunately, when this is accomplished many patients begin to have symptoms such as poor thought processes, depression, and fatigue, because their circulation is inadequate under the circumstances.

Studies have shown that patients with mild essential hypertension (diastolic pressure 90-109 mm Hg) have not derived any cardiac benefit from drug therapy.[144a,284a] "In fact health risk of some drugs used to treat mild hypertension probably outweighs the marginal benefit that many patients in this group would obtain, particularly since up to one-third of these individuals have been found to be normotensive when away from the physician's office."[242a]

There are many advantages to obtaining and maintaining normal blood pressure through natural means. Martin and Dubbert[275] write, "...Pharmacotherapy for mild hypertension, as powerful as it has proven to be, may not be the best first line of defense in combating hypertension and its serious health sequelae for all patients. For example, the known short-term undesirable side effects and the unknown long-term side effects of these potent medications have resulted in problems in medication adherence in up to half the treated patients. The financial implications of treating as much as one-fourth the adult population are also problematic. In light of these concerns and research findings, the 1984 recommendations of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure placed increased emphasis on nondrug approaches in primary and adjunctive treatment of hypertensive patients." In addition, the

natural approaches to lowering high blood pressure have their beneficial effects on general health, such as possibly reducing the overall cardiovascular risk profile. Often the patient must make changes in his lifestyle, improving the appearance and/or quality of life.

Control of blood pressure, hyper- and hypotension, is multifactorial. It is a complex integration of neural, endocrine, renal, and cardiovascular factors. By definition, essential hypertension is a diagnosis of exclusion and most likely comprised of heterogeneous etiologies. Each contributing factor, in itself, may not be enough to affect the blood pressure sufficiently to create an abnormality. The challenge is to determine the factor or combination of factors causing or contributing to the problem. Weight reduction has been shown to be effective in reducing blood pressure, but obviously, one would not put a thin hypertensive person on a weight-reduction program as treatment.[67] This analogy is applicable to all the treatment suggestions in this section. Correcting vertebral subluxations and fixations is often important in lowering hypertension; however, not all hypertensives have these spinal conditions. As Goodheart states, "Diagnose the need, supply the need, and observe the results."

Greater attention should be given to the natural approaches to maintain normal blood pressure levels because of the enormous number of hypertensive persons in the world. The pharmacological approach to reducing blood pressure on such a wide scale is impractical because of the attendant side effects of medications currently available for this type of treatment.[38] Side effects are of increasing concern when one deals with milder and milder levels of blood pressure elevation. In some instances, a person may feel well with elevated blood pressure and ill when taking blood pressure medication, even though his blood pressure returns to normal with the medication.

Neurologic Control

Regulation of blood pressure is by the sympathetic and parasympathetic nervous systems, which originate from the hypothalamic and medullary regions of the brain. They control the heart, kidneys, and peripheral vascular components of blood pressure regulation. The sympathetic nervous system increases cardiac output, and increases blood flow to muscular and nerve tissue components. It causes vasoconstriction in non-muscular tissues, including the kidneys, skin, and gut, causing an overall increase in systemic blood pressure. The parasympathetic system reduces cardiac output and rate, causing a decreased blood pressure. There is also an increase in digestive system activity from the parasympathetic nervous system. Guyton and Hall[187] suggest that the sympathetic and parasympathetic nervous systems fire continuously at a base rate to establish an equalizing vasomotor tone.

The effect of the sympathetic nervous system in raising blood pressure is well-known. One simply needs to observe the elevation that accompanies any exposure to stressful or threatening stimuli. Caudill et al.[67] cite many studies in which animals were chronically stressed by conditions such as overcrowding and conflicts that raised their blood pressure. When stress is combined with other hypertensinogenic stimuli, the resulting blood pressure elevation is exaggerated over that from each stimulus alone.

Many cases of essential hypertension respond well to structural correction, dietary management, and nutritional support. White and Baldt[419] point out, "With the advent and acceptance of diuretics and beta-blockers for hypertension therapy, the manipulative approach apparently has fallen to disuse. This is due to the ease that accompanies prescribing medication. It is obviously faster and easier to prescribe a medication to treat a symptom (high blood pressure) than it is to treat the actual biomechanical/structural cause of the disease (hypertension)." Applied kinesiology offers additional methods of treatment for hypertension besides the "biomechanical/structural" cause they mention. When effective, the natural approach eliminates the side effects of drug therapy, which are often unpleasant and may potentially create health problems. Grimm and Prineas[183] refer to the natural approach in lowering blood pressure as "a hygienic approach." They state, "An effective hygienic approach is the only conceivable way that will save millions of hypertensives decades of drug treatment with its unknown long-term consequences. Hygienic methods are also the only rational approach to the ultimate solution, the primary prevention of hypertension."

An overview of the type of treatment that might be given in natural health care is presented by Hood[203] in a study of seventy-five blood pressure cases, some elevated and some low. He found that most of the subjects consumed highly processed foods, limiting their fat consumption to 2% in skim milk and margarine. Cream was totally avoided, and egg consumption strictly limited. Foods were thoroughly cooked, and no attention was paid to limiting food additives.

The group's ability to obtain restful sleep was limited. Poor mattress and pillow quality was evident, as was frequent stomach sleeping. Exercise, in general, was limited.

Spinal examination and treatment were administered with the Gonstead technique. The diet was corrected to eliminate the refined food, including refined sugar and flour. Emphasis was placed on eating fresh fruits and vegetables, organ meat, and generally foods as unprocessed as possible. Whole milk replaced skim and 2%, and the use of cream was encouraged in the diet. Butter was suggested to replace margarine. Eggs were added to the diet, and raw, unrefined honey was the only sweetener allowed. Coffee, soft drinks, beer, and whiskey were eliminated; wine was allowed in strict

moderation. The nutritional support given included organic minerals, wheat germ oil, and vitamins A, C, and P. To help improve structure, sleeping habits were changed by improving the mattresses, changing high pillows to low, and sleeping on the side or back. Spinal stretching exercises were initiated, along with walking outside for four to twelve blocks daily.

The patients in this group were on medication when this program was initiated, but they still had hypertensive disease. They decided to eliminate the medication since it had not improved their condition. Of the group (n75), sixty-seven were hypertensive, with an average blood pressure of 163/94 mm Hg. The average blood pressure of the group was reduced to 130/82 in 9.8 treatments in seventy-one days. The eight hypotensive patients had an average blood pressure of 100/67 mm Hg. This changed to 114/76 in 5.5 treatments in seventy-two days. The post-treatment blood pressure remained stable unless further spinal injury occurred.

Hood states, "I am convinced that there is a cause for the 28,800,000 Americans who have hypertension from 'an unknown origin.' I am also convinced that many of the 3,200,000 cases of 'secondary' high blood pressure are misdiagnosed and would benefit greatly from this form of treatment."

The details of this study are presented to indicate how different the dietary recommendations and overall case management in natural health care are from ordinary treatment procedures. In the applied kinesiology regimen, some of the nutritional recommendations would differ from Hood's, based on individual patient findings. In addition, the stomatognathic system would be examined and treated, when necessary.

Before determining that a patient is hypertensive, blood pressure determination should be made on several visits. Often a patient new to a doctor's office is anxious about the examination, and the increased sympathetic activity raises the blood pressure above its normal range. Before examining for the cause of high blood pressure, determine if the patient is taking any medications that might elevate it. These include nasal sprays or inhalants containing potent fluorinated steroids, decongestants, thyroid hormone, glucocorticoids, mineralocorticoids, appetite suppressants, progestins (e.g., oral contraceptives), and licorice or chewing tobacco containing licorice.[95]

Cranial Manipulation

The effectiveness of treating essential hypertension with a combination of cranial, facial, and total body manipulation was demonstrated in the early 1950s by some osteopaths, and earlier on an anecdotal basis by chiropractors using spinal manipulation. Goodheart[164] points out that the dural envelope is inelastic and nonexpandable. If cranial dysfunction causes poor cerebrospinal fluid motion, he suggests there may be an elevation of blood pressure to increase the venous pressure within the spinal column, in turn increasing the pressure of the cerebrospinal fluid. He has found that when hypertension is associated with an elevation of systolic pressure on standing, cranial techniques are often needed. There will frequently be a spinal fixation in the area of C4 to T2.

White and Baldt[419] have found that relaxing the musculature of the head, neck, and face, and correcting temporomandibular joint dysfunction are important aspects in correcting essential hypertension. They comment on the dental error of equilibrating an occlusion into a retruded mandibular condyle position. "This position causes the blood pressure to increase. Usually it does not increase instantaneously, but is diagnosed gradually, after several visits to the physician. Most patients and dentists do not make the correlation between the TMJ-recessed position and the new finding of hypertension in previously asymptomatic persons!"

The first step in essential hypertension is to completely evaluate the cranium and correct any cranial faults. A common cranial fault found with high blood pressure in applied kinesiology is the glabella fault. Of course, as previously pointed out, when the cranium is treated, the entire stomatognathic system and pelvis should also be evaluated and corrected, if necessary. After this correction, re-evaluate the blood pressure. If reduced, do no more at this time; if not, proceed to spinal evaluation and correction.

Spinal Correction

On a repeated anecdotal basis, most chiropractors have observed improvement in blood pressure from vertebral adjusting. The term "vertebral adjustment" here indicates the correction of vertebral subluxations by manipulation. Manipulation itself indicates just that — manipulation of a vertebra, whether or not a subluxation or fixation is present. There is an effect on blood pressure from manipulation of the spine in normal subjects when evaluation for a subluxation has not been made. Tran and Kirby[398] manipulated the spine at the T1-2 level of normal subjects, and caused a marked drop in systolic blood pressure in 91.6% of persons in the study (n24). There was a drop in diastolic pressure in 70.8%. There was no significant change in the pulse rate or on the electrocardiogram.

Sato and Swenson[350] demonstrated in animals that stimulation of the lower thoracic vertebrae and upper lumbar vertebrae causes decreased renal and increased adrenal nerve activities, resulting in a reduction of overall blood pressure. Acute spinalization at the C1-2 level revealed that this inhibitory activity was mediated at supraspinal levels. After the C1-2 spinalization, the same spinal column mechanical stimulation produced small increases in blood pressure and increases in adrenal and renal activity.

The three major areas observed to correlate with

hypertensive disease are the upper six thoracic levels, with particular involvement of the T2-3 motion segment, the occipital-atlas articulation, and the lower thoracic region of T11-12.[99]

If there is inadequate blood pressure reduction from cranial correction, evaluate the cervical and dorsal segments for fixations, subluxations, or intraosseous subluxations. Make corrections and re-evaluate the blood pressure. Apply no further treatment if it has reduced satisfactorily. If there is still inadequate improvement, Goodheart[164] recommends applying equal pressure bilaterally along the spinal column from C1 to the apex of the sacrum. After one minute, observe the paraspinal areas for blanching or redness. When there is blanching, evaluate for a subluxation, fixation, or intraosseous subluxation at that level; correct as indicated. Another method is to apply local heat limited to the blanched area. Block any non-blanched areas with toweling so that the application of heat is specific. Maintain heat for approximately ten minutes, and then re-evaluate the blood pressure. A good response is systolic reduction of 20-40 mm Hg. If still unreduced, the heat may be maintained for a longer period.

Kidney and Adrenal

When the diastolic blood pressure is high, the cause is often found to be kidney dysfunction or pathology. Goodheart[164] recommends testing the blood pressure in three positions. Begin with the patient seated and use this as a base to compare standing and recumbent systolic blood pressures. In adrenal depletion, there is a drop in systolic blood pressure as one stands. Often when hypertension is due to renal or liver dysfunction, the systolic pressure is higher in the recumbent position and lower in the seated position. This often relates with subluxations or fixations in the lower thoracic area. Crawford et al.[99] point out, "The splanchnic nerves terminate to form the renal plexus and thereby indirectly influence blood volume and flow pressure through the kidney. Therefore, the splanchnic area, where signs of visceral involvement may appear before any chemical determinations become significant, may be one of the most important spinal levels to be evaluated."

High blood pressure may be caused by an allergic reaction that restricts blood flow to the kidney by internal swelling pressure. This type of localized swelling due to allergy is called an Arthus allergic reaction or phenomenon.[136] In many cases, there is other evidence of allergic reaction. When appropriately treated or the allergic substance is avoided, the hypertension often improves. Coca[89] first observed allergenic hypertension when it improved as he was treating people for allergies causing other symptoms.

Renin-sodium profiling is done with a serum potassium measurement as a primary screening device for the cause of hypertension.[251] In general, elevated renin indicates vasoconstriction of a neurogenic etiology, while low renin is non-neurogenic with volume expansion. The renin profile has three major sub-groups with percentages of low renin (30%), normal renin (55%), and high renin (15%). Patients with low renin are subject to strikingly fewer heart attacks and strokes than those with normal or high renin. The results of the testing procedure provide a starting point for allopathic treatment rather than an empiric approach. In general, patients with low renin are treated with diuretics, low renin and low potassium are considered for a primary aldosteronism, and high renin is considered for possible renovascular hypertension and surgery; both the high-renin patient (not applicable to surgery) and the normal-renin patient are treated with a beta-blocker.

The overactivity of the sympathetic nervous system in the patient with elevated plasma renin is evidenced by a higher level of norepinephrine than that found in normal or low-renin patients. "The elevation of blood pressure appears to be sustained predominantly by neuromechanisms and not the renin-angiotensin system."[133] There appears to be a psychological component to the high-renin essential hypertension patients. As a group they are guilt-prone, controlled, and submissive, with a high level of unexpressed anger.[133] Caudill et al.[67] state, "Psychological characteristics which tend to be manifest in the high-renin hypertensive patient are precisely those which have traditionally been linked to sympathetic nervous system arousal."

Aldosterone from the adrenal cortex increases the rate of reabsorption of salt and water in the distal tubercles of the kidney, thereby greatly reducing the rate of water and salt excretion. In a crossover control study of forty-five patients with low renin-high aldosterone hypertension, Mannino[273] stimulated the posterior Chapman reflex for the adrenal gland (neurolymphatic reflex between the 11th and 12th thoracic vertebrae midway between the spinous process and the tips of the transverse processes). For a sham treatment, the intertransverse spaces between the 8th and 9th thoracic vertebrae were stimulated. In normal controls, there was no significant change in aldosterone levels or blood pressure throughout the investigation. In the hypertensive group, there was a significant drop ($p < 0.01$) in aldosterone levels within thirty-six hours of manipulation. When a sham Chapman reflex manipulation was performed, the aldosterone reverted to pre-treatment levels, but it again lowered significantly when the appropriate level was treated. The decrease in aldosterone levels again followed the manipulation by approximately thirty-six hours. Within the time limit of the study, there was no reduction in blood pressure. When aldosterone is lowered by spirononlactone (in full therapeutic doses), its effect on blood pressure is not observed for five to seven days.

Nutrition

Many factors must be taken into considereation when evaluating research studies on the effects of nutrition causing or correcting hypertension.[242a] First, the measurement of blood pressure must be accurate. In order for this to take place, the study subjects must be habituated to blood pressure measurements for up to six weeks. It has been shown that in a hospital randomized crossover study, several weeks of placebo treatment caused decreases in urinary catecholamine excretion and 10 mm Hg drops in blood pressure.[206a] If dietary change is responsible for blood pressure reduction, then a post-treatment period of going back to the original diet or off the nutritional supplementation should return the blood pressure to pre-study status, unless a permanent cure is expected. The post-study reversal part of the study is rarely done.

In applied kinesiology literature it is often noted that the patient should be evaluated in the manner in which he lives. For example, this includes examining the patient weight-bearing, sitting, and in various gait positions for structural problems. Earlier in this chapter the glucose tolerance test is compared with taking blood sugar samples during the patient's daily working and living habits showing how the two often do not correlate. Likewise, the patient's blood pressure may vary greatly during his work or play day in comparison to findings in the doctor's office. Knapp[242a] points out this problem with doing metabolic-ward-type studies. Improved understanding of a person's blood pressure levels in the everyday world is available with the advent of ambulatory blood pressure monitoring now available. This method has revealed considerable differences between subjects' blood pressures obtained in a clinic, at work, and at home.[312a]

The only nutritional approaches recommended by the Joint National Committee on the Detection, Evaluation and Treatment of High Blood Pressure in their 1984 report[126] are weight loss — if the patient is greater than 1.15 of ideal weight — low sodium intake, and moderate alcohol intake. Caggiula et al.[63] recommend, in addition, that two other nutrients — potassium and calcium — be considered. They point out that both nutrients encourage more positive diet. High potassium foods are generally lower in sodium and fat. Increased calcium intake may help prevent osteoporosis in women. Also, supplementing calcium helps prevent the compounding of the hypertension due to calcium loss from weight-control diets.

When dietary change is necessary for control of hypertension, the patient must get involved in the program; simply giving dietary advice is usually inadequate. The patient should be required to calculate his own food intake by looking up data on charts, reading labels, and maintaining a diary of food consumption. Within a relatively short period, one becomes aware of food contents, adulteration, and the quantity required to maintain proper intake. Goals should be set that are relatively easy to meet, and modified as success is obtained.

The general approach to nutrition is that of a quality diet. The approach discussed in the section on hyperlipoproteinemia is applicable to most people with hypertension. In fact, a large percentage of hypertensives have elevated cholesterol and/or triglyceride levels.

The application of a quality diet is evidenced by vegetarians who are on a lacto-ovo-vegetarian diet. They have lower blood pressure independent of obesity than do those who eat meat.[26] Considerable research has been done in an effort to determine the difference in their diets responsible for the lower blood pressure. Factors studied have been the polyunsaturated to saturated fat ratio, sodium, calcium, magnesium, and fiber ingestion. None of these factors independently have been found to relate with the lower blood pressure. There may be a combination of factors in the food of vegetarians that is responsible, or there may be some factor as yet not identified in the food of meat eaters that causes a tendency toward hypertension. In general, vegetarians tend to be people who pay great attention to their health. In most of the studies in the literature, there is little control for alcohol, exercise, total caloric intake and life-style in general, leaving one to speculate. It would appear that the general attention to health plays a major role.

Sodium. Reduction of hypertension by sodium restriction has been done since shortly after the first reliable indirect method of measuring blood pressure was discovered in 1896 by Riva-Rocci. Throughout this period, enthusiasm for its effectiveness has waxed and waned. With the introduction of pharmacological methods, interest in sodium restriction lessened considerably; it has again become an important area of investigation in the past ten to fifteen years. Grimm and Prineas[183] point out, "The renewed interest stems largely from well-established adverse effects which have been observed with many of the commonly used antihypertensive agents, such as the thiazide diuretics. Thiazide diuretics frequently cause hypokalemia, increased blood lipids and increased uric acid and these effects have become a major concern." There is probably considerable biochemical individuality between people regarding sodium. It has been shown in rat studies that strains of rats can be developed that are sodium sensitive, and strains that are sodium insensitive. "Overall the evidence from clinical trials is suggestive but not conclusive that sodium reduction will lower blood pressure in hypertensives."[183] Care should be taken regarding indiscriminate sodium restriction, as discussed in the section on adrenal stress disorder. Practical methods to reduce salt intake are as follows:

1. Add no salt during food preparation. Flavor may be added with garlic, wine, herbs (e.g., basil,

oregano), and spices (e.g., cinnamon, nutmeg).
2. Add no salt at the table.
3. Avoid heavily or visibly salted items, such as potato chips, nuts, and saltine crackers.
4. Avoid most processed foods, such as bacon, corned beef, and peanut butter.
5. Replace salted sauces, condiments, monosodium glutamate, and relishes — such as pickles — with vinegar, lemon juice, pepper, or dry mustard. Various types of herbs and seasonings are available with little or no salt.
6. Labels of all items should be read thoroughly. Many vitamin tablets have high sodium levels.

Within approximately two months, the patient's taste should adapt to the low-salt diet. After becoming accustomed to it, returning to a high-salt diet is unpalatable and often leaves the patient excessively thirsty shortly after a meal.

Calcium. Epidemiological studies indicate that hypertension parallels low calcium intake.[280] In a placebo-controlled double-blind crossover trial, McCarron and Morris[281] found 44% of hypertensives to have at least a 10 mm blood pressure reduction with calcium supplementation. Since only a sub-set of the hypertensive people had the reduction in blood pressure, it is evident that calcium is only one aspect of the multifactorial problem of hypertension. Calcium supplementation was carried out over an eight-week period. They point out that if it had not been done for that length of time, the beneficial effects of calcium supplementation might have been missed. Calcium deficiency is more prevalent among the elderly, who also have greater frequency of essential hypertension. Morris[62] points out that there is a similarity between the percentage of people and the amount of reduction of blood pressure between calcium supplementation and sodium restriction.

Fatty acids and prostaglandins. By studying subcutaneous adipose tissue by needle aspiration from 399 free-living male subjects, Berry and Hirsch[29] found that with a 1% increase of linolenic acid there was an associated decrease of 5 mm Hg in systolic, diastolic, and composite mean arterial blood pressure.

It has been suggested that the effect of unsaturated fatty acids on blood pressure is mediated through prostaglandin production and/or effects of the renin-angiotensin system. Berry and Hirsch found that linolenic acid affects the diastolic more than the systolic blood pressure, indicating that prostaglandins may therefore be expected to act more as vasoregulators than as producers of structural arterial changes.

Weight reduction. Among the many nutritional approaches to treating hypertension, weight reduction appears to be the best validated approach. Again, there is variability among individuals, with some weight-sensitive and some weight-insensitive obese hypertensives. This difference among people may be due to the balance of the autonomic nervous system, a factor often treated by applied kinesiologists. Maxwell and Waks[278] state, "Catecholamines may affect blood pressure by directly increasing peripheral resistance, by increasing renal sodium reabsorption and by stimulating renin secretion. The increased cardiac output in obesity may relate to increased sympathetic activity. Thus, it is possible that the primary abnormality in obesity hypertension is increased sympathetic nervous system activity with associated hemodynamic and hormonal changes." Weight reduction of the obese improves cardiovascular factors in addition to elevated blood pressure, including hyperlipidemia, hyperuricemia, and impaired glucose tolerance.

Exercise. Martin and Dubbert[275] reviewed the literature on lowering blood pressure by exercise and found some evidence of moderate reduction. They point out the need for further controlled studies to determine the effectiveness and mechanisms of pressure reduction. They performed a study in which two groups were assigned to aerobic training or, as a control, stretching in easy calisthenics for non-aerobic activity (less than 60% aerobic capacity). Diastolic pressure decreased 9.6 mm Hg, and systolic decreased 6.4 mm Hg in the aerobic exercise group. In the control group, the diastolic and systolic pressure increased .8 and .9 mm Hg, respectively. There were no significant changes in weight, body fat, urinary electrolytes, resting heart rate, or catecholamines. The control group then performed the aerobic exercise, and their diastolic and systolic pressures dropped 7.2 and 8.1 mm Hg, respectively.

When each patient is analyzed as an individual and the appropriate factors of nutrition, exercise, weight reduction, spinal, and stomatognathic system correction are applied, hypertension can be brought under control in many patients. When applicable, the natural approach has the benefit of no side effects, and it generally causes an improvement in other non-blood-pressure-related aspects of health. When blood pressure is brought under control by natural means, the patient should be followed systematically to determine that it is continuously controlled. Hypertensives are notorious for non-compliance, whether treatment is medication, nutrition, or structural correction, because they do not "feel bad."

Pineal Gland

When one does manual muscle tests on numerous patients daily, it is inevitable that unusual and unexplainable findings will be observed, although this is not as common today as it was in the early development of applied kinesiology. Electromagnetism, various types of light, and other environmental factors can influence the results of a manual muscle test.

As knowledge of the meridian system's influence on body function has been integrated into applied kinesiology, electromagnetic factors influencing function have been better understood. Understanding the effect of light on health has been pioneered by Ott[300] and Wurtman,[424] among others. A full discussion of electromagnetism and light is outside the scope of this text. A brief discussion of the effect of light on muscle testing and its apparent association with the pineal gland is of value here because of its influence on certain types of health problems.

The human pineal gland is located at the base of the brain behind and above the pituitary gland. It is shaped like a small pine cone, thus its name, and is about 1/2" in length. There is a considerable difference in pineal gland function between amphibians and mammals. In the amphibian, it is a photoreceptor that sends direct information to the brain. The mammalian pineal has no direct connection to the central nervous system; rather, it functions as an endocrine gland controlled by environmental lighting via an indirect pathway involving its peripheral sympathetic nerves. In the mammal, the pinealocytes are neuroendocrine transducers that respond to norepinephrine released from their synaptic neurons, ultimately producing melatonin, the major hormone of the pineal gland.[425] "Tryptophan is a precursor for serotonin, which is converted to melatonin by N-acetyltransferase in the pineal gland."[104] The pineal gland is the regulator of circadian rhythm.[33] It, in turn, is regulated by bright sunlight or artificial light and the earth's electromagnetic field. The concentration of melatonin in human blood cycles through the twenty-four-hour period, with maximum values during darkness.

Darnall[104] promotes the hypothesis that the pineal gland is important in the aging process. "Perhaps literally and symbolically, our longevity is related to our ability to integrate and synchronize ourselves with the planetary and solar-stellar energies that surround us." "Within the pineal gland and its interdependence with the pituitary and medulla lie the mysteries of our aging as well as our agelessness." The range of melatonin's effect on the body is unknown. Wurtman and Cardinali[425] state, "Melatonin injections inhibit various aspects of gonadal, thyroid, and adrenal function and, in particular, decrease ovarian weight, inhibit pregnant mares' serum gonadotropin induced by ovulation, and delay pubescence in immature rats; melatonin also reduced the frequency of vaginal estrus in mature rats." In applied kinesiology, treatment attributed to improving pineal function is primarily associated with various types of endocrine system imbalance.

Goodheart[169] first observed the apparent effect of the pineal gland on body function when a patient who had been treated for deficient thyroid and adrenal function was allowed to rest on the treatment table before leaving the office. As Goodheart left the room and turned out the light, he observed that the patient's foot rotated laterally. He turned the light back on, returned the foot to its original position, and once more turned the light out; the patient's foot again involuntarily rotated laterally. Pursuing this further, Goodheart tested the sartorius with the light on, and again with it off. When the light was off, the sartorius muscle weakened; it was strong with the light on. Further investigation indicated that the eyes did not appear to be involved with the weakening. When the patient closed her eyes with the room light on, the muscle remained strong, as it did when the eyes were covered and no light could be seen. Goodheart had the patient taste pineal substance because of the pineal gland's association with light. While the substance was in the patient's mouth, the muscle did not weaken when the light was turned off. Other nutritional substances will also cause the muscle to remain strong with the light off, but only temporarily. For example, if the patient chews adrenal substance, the sartorius will no longer weaken when the light is turned off; however, when the test is repeated several times, even though the patient continues to have the adrenal substance in his mouth, the muscle will again be weak when the light is off. If the light is cycled on and off several times, the effect of all nutrition — other than pineal — is only temporary.

Usually a specific type of cranial and mandibular challenge is present when a patient has muscles that weaken in darkness. Compression of the greater wings of the sphenoid or the rami of the mandible — or both — causes a general indicator muscle to temporarily test weak. Challenge is done by simply pressing from lateral to medial on the bilateral greater wings of the sphenoid or the rami of the mandible. The challenge determines whether correction is to be applied. If both the mandible and sphenoid challenge are positive, both are corrected. If only one is positive, it alone is corrected. Correction is made by contacting the medial aspects of the pterygoid processes or the internal aspect of the mandibular rami and spreading the structures. The amount of pressure used is approximately 4-5 pounds. When the pterygoid processes are contacted, one should consider the tenderness of the area to avoid excessive pain to the patient. The pterygoid hamulus is a delicate structure; care should be taken not to fracture it. No respiratory correlation has been found to enhance these corrections.

It is unknown exactly what mechanism is taking place when a patient's muscle tests weak in darkness but not in light. The neural pathway to stimulate the pineal gland has been shown to be light stimulation to the eyes, which reaches the pineal by postganglionic sympathetic neurons.[425] Light stimulation to the eyes, or lack of it, is not a necessary factor in the applied kinesiology test since, as noted, closing the eyes or covering them does not influence the outcome. It does, however, seem that the pineal is intricately involved with the applied kinesiology test, since stimulation of the gustatory receptors with pineal substance abolishes the muscle weakening when the light is turned out.

The time interval between the patient's last major exposure to sunlight or ultraviolet light has a bearing on the outcome of the test. To demonstrate this, one can take a patient who tests weak when the light is turned out and expose him to bright sunlight outdoors; then repeat the test in darkness. Approximately 50% of those who previously tested positive will now test negative.

In the usual clinical examination, prior exposure to sunlight or ultraviolet light is inconsequential since there is usually enough time between the patient entering the clinic and being tested. The interval between bright sunlight exposure and testing for pineal involvement varies with the degree of pineal dysfunction. Those most involved will test weak in the dark even though there was strong exposure to outdoor sunlight immediately prior to the test.

It is not necessary, in most cases, to have the examination room totally dark to obtain a positive response. Again, this varies, depending on the patient's involvement with the condition.

Treatment consists of supplementation with pineal substance, usually one tablet three times per day, and correcting the characteristic cranial or mandibular fault if the challenge is positive. All other aspects of the stomatognathic system should also be examined and corrected, if involved. There is often an improvement in endocrine dysfunction when pineal correction is obtained. This is most valuable when there is recidivism of endocrine dysfunction.

Male/Female Conditions

Examination using applied kinesiology methods is exceptionally effective in finding the cause of functional conditions unique to the male or female. The answer to the problem will often be found with the techniques discussed earlier in this text and in other applied kinesiology sources. The cause of a male's impotence may be adrenal stress disorder and a sugar handling problem, found with evaluation of the endocrine system, or it may be mental, found and corrected by the techniques of mental/emotional examination presented in Chapter 10. The key is that often the disturbance is in a remote system or function. To be effective in applied kinesiology examination, one should be thoroughly familiar with the interaction of the endocrine system. This brief overview of reproductive system hormones should be thoroughly understood. A thorough study in a more in-depth text is indicated if one has been away from this subject for a prolonged period.

Hormones

Estrogen. The estrogens are synthesized in the body from cholesterol, and to a slight extent from acetyl coenzyme A. This is the same as the production of adrenal cortical hormones. The estrogen present in males and females is the same chemical compound; the only variance is that the amount of estrogen in females is higher.

The liver is of prime importance in the metabolism of estrogen, as well as other steroids. It inactivates estrogen with the by-products excreted in the bile, to be ultimately eliminated from the body through the bowel and in the urine.[187] Gynecomastia, loss of chest hair, testicular atrophy, and loss of libido have all been described in men with cirrhosis of the liver and can be tied to the failure of the liver to inactivate estrogens.

Estrogen has many effects in the body, primarily in the genital tract and accessory sexual organs, as well as general effects on the skeleton and on the metabolism and deposition of proteins and fats. Estrogen causes hypertrophy of the uterus, which is particularly marked during pregnancy. Prolonged estrogen administration will induce ovarian atrophy in the female, as well as testicular atrophy in the male. This occurs because estrogen is a strong inhibitor of the pituitary and its release of gonadotrophin.

Estrogen is responsible for the development of secondary sexual characteristics. It produces water retention by increased retention of sodium and chlorides. This is most evident at the time of ovulation and just before menstruation, when the blood level of estrogen is high.[30] Rubel[346] states that fluid retention prior to menstruation is due to the protein anabolic effect of excess estrogen, and that it also accounts for premenstrual tension and general weight gain prior to menstruation. The estrogen-progesterone ratio is exceedingly important. If there is any liver involvement causing an excess of estrogen, there will be a pituitary drive to produce more progesterone, possibly causing an increase of estrogen at the same time.

Progesterone. Progesterone is produced by luteal tissue, the adrenal cortex, and the placenta. Much

of the progesterone is degraded to other steroids by the liver and eliminated via the bile.

The effects of progesterone are on the uterus, maintenance of pregnancy, participation in control of the mammary glands, and suppression of ovulation. Progesterone also has an influence on electrolyte balance and protein catabolism.

For progesterone to be effective in preparing the uterus for pregnancy, it must be in correct ratio with estrogen. Too much estrogen in the estrogen-progesterone ratio inhibits the action of progesterone; however, the effects of estrogen are blocked by progesterone. Depending on the relative amounts of estrogen and progesterone, a variety of effects can be produced by interaction.

Progesterone is responsible for the maintenance of pregnancy. It is not necessary to have active ovary function for the continuation of pregnancy, since progesterone will be produced by the placenta or from the corpora lutea. Suppression of ovulation and the menstrual cycle during pregnancy is accomplished by progesterone.

Follicle-Stimulating Hormone (FSH). FSH is a gonadotrophic hormone from the anterior pituitary that activates the ovarian follicle to mature and for the ovum to enlarge. The tissues around the ovum form estrogen. During this time the uterus is prepared to receive the developing ovum.

Luteinizing Hormone (LH). On the fourteenth day of ovum development, the luteinizing hormone is released by the anterior pituitary and the ripe follicle ruptures, releasing the ovum. A corpus luteum develops. Progesterone is secreted from the corpus luteum, contributing to the continued secretory activity of the endometrium and changing the uterine muscle. During the next fourteen days, estrogen decreases while progesterone increases. The increased progesterone level in the estrogen-progesterone ratio curbs the anterior pituitary in its production of the luteinizing hormone. If fertilization of the ovum does not occur, the corpus luteum does not persist; its influence on the endometrium wanes and menstrual flow starts, ending the cycle. Balance between estrogen and progesterone is necessary for normal control of the anterior pituitary.

Relaxin. The hormone secreted from the sixth week of pregnancy until termination is called relaxin. It is responsible for softening the ligaments of the pelvis for easier delivery. It accomplishes this by dissolving and splitting collagenous fibers into thin threads. There are changes following progesterone treatment similar to those produced by relaxin. Guyton and Hall indicate that estrogen is more responsible for relaxing the ligaments than relaxin.[187]

Following parturition pelvic distortion has been noted on x-ray[352] and often women will comment, "I never had a backache until I had Billy." This may result from pelvic distortion easily produced because of ligament laxity compounded by the structural strain of childbirth. Relaxin is not detectable in the serum twenty-four hours after parturition; thus, if the pelvic articulations tighten when there is still distortion, a more permanent problem develops. Many future problems can be avoided by examining and correcting the pelvis and other structure as soon after parturition as possible.

Androgens. Androgens are the masculinizing hormones and they, too, are found in both males and females. The most important hormone in this role is testosterone. The hormone is the same in both the female and male, but the quantity is higher in the male. The androgens promote the growth of accessory sexual organs, such as the seminal vesicles and prostate gland. In addition, they cause a general growth of muscle, kidneys, and bones. The androgens are protein anabolic. By directly acting on the nervous system, they change an individual's sexual behavior. A lack of testosterone causes a pituitary push of gonadotrophins attempting to bring up the testosterone level. This effect is primarily directed toward the testes; however, other glands affected by the pituitary are also involved.

Dysmenorrhea

Dysmenorrhea always requires an evaluation of functional hormone balance. One common cause of imbalance is blood sugar handling stress. As the blood sugar level drops from hyperinsulinism, poor diet, or some other cause, a pituitary drive develops for the production of glucocorticoids or gluconeogenesis. As the pituitary produces ACTH, it also affects the gonads by the production of gonadotrophins. If progesterone does not keep pace with estrogen, dysmenorrhea occurs. Liver congestion may also be involved, which is common in sugar handling stress. There may be a failure to adequately destroy estrogen, throwing the estrogen-progesterone ratio off-balance. The gonad-hormone imbalance may affect secondary sexual characteristics, giving masculine characteristics to females and feminine characteristics to males.

The pituitary push may affect other aspects of the endocrine system, such as the mineralocorticoids, causing excessive retention of sodium and chlorides and excessive potassium secretion. There may be symptoms of weakness, fluid retention, exhaustion, and hypertension.

A thyroid deficiency can cause a similar type of pituitary push. Hyperpituitarism may result from the pituitary's attempt to raise the level of thyroid function with thyroid-stimulating hormone (TSH). The elimination of dysmenorrhea in this case depends on regaining normal thyroid function, if possible; if not, thyroid medication — preferably with a natural substance — may be necessary.

When the hyperpituitarism is returned to normal by treating the primary cause, there may be a change in the usual menstrual cycle. Because of the long-term pituitary push, there is an overabundance of gonado-

trophins. The reproductive organs become accustomed to the higher levels of estrogen and progesterone. As pituitary balance is re-established, a period of time is needed for the reproductive organs to adjust to the normal hormone levels. The patient should be informed of this possible change to forestall any anxiety, especially if the patient is using the rhythm method of birth control. Vertebral subluxations and fixations should be evaluated for and corrected, if present. The spinal association with dysmenorrhea is often marked by back pain with menstruation. The pelvic categories are often involved. Sacral correction is of primary importance in dysmenorrhea. Structural faults may be primary in causing dysmenorrhea, or they may be secondary as a result of imbalance of muscles such as the piriformis, gluteus medius and maximus, and adductors. Structural disturbance of the pelvis may be part of a modular interaction, such as equilibrium reflex synchronization, PRYT, or dural tension.

There may be uterine ptosis, which causes tension on the uterosacral ligament. This will usually be accompanied by symptoms simulating an anterior 5th lumbar subluxation. Typically there will be pain of a cramping nature and very profuse menstrual flow. The pelvic diaphragm, made up primarily of the levator ani muscle, should be examined and corrected, and the uterine lift technique administered. Follow up with examination of the thoracic and cranial diaphragms.

Cramps with dysmenorrhea may be due to calcium deficiency. A practical test for this is to place a blood pressure cuff on an arm or leg. Raise the cuff pressure to diastolic level and hold it for three or four minutes. If cramping occurs, calcium administration will often eliminate menstrual cramping. In the presence of cramps, heavy dosage of four 41 mg calcium tablets chewed every fifteen minutes for one hour may be necessary; it is then reduced to three tablets per hour throughout the daytime period. For prevention of menstrual cramps, calcium administration should start approximately one week prior to the onset of menstruation. As with any calcium deficiency, the patient should be evaluated for adequate hydrochloric acid and for balance of vitamins F and D. Vitamin E is often helpful in eliminating cramping.

Menstrual cramps can be secondary to either an open or closed ileocecal valve syndrome. In either condition, the body becomes toxic and other organs attempt to take over the elimination process. Almost any area responsible for elimination of material from the body in an excretory or secretory function may become involved. A patient with an ileocecal valve syndrome may have symptoms of excessive nasal discharge, sweaty, odorous feet, or acne. The uterus also acts as an elimination organ when the patient is toxic. When dysmenorrhea is secondary to an ileocecal valve syndrome, there will usually be abdominal pain radiations. This particular type of dysmenorrhea is very disabling, often causing the patient to stay home from work or school while trying many types of medication to overcome the pain.

Amenorrhea

A general physical examination is the first consideration in either primary or secondary amenorrhea. The patient should particularly be evaluated for anemia and thyroid dysfunction. A gynecologic examination should be included for neoplasms and hyperplasia. If amenorrhea persists after the termination of a pregnancy, particularly an abortion or miscarriage, persistent lutein tissue of the ovary must be suspected.

Either hypo- or hyperthyroidism can cause amenorrhea. When thyroid dysfunction is found, one should suspect that other areas of dysfunction might also be present.

Hypoproteinemia can be the cause of scanty menstruation or amenorrhea. There may be a natural effort by the body to conserve protein by shutting down menstruation as a protective device for its protein stores. All factors of protein metabolism should be evaluated, as well as the patient's diet.

The prognosis for amenorrhea is excellent in the absence of tumors, using the applied kinesiology approach and related factors. It is difficult to predict the exact length of time it will take to regain a normal menstrual cycle. It depends on when treatment is begun in the normal cycle. Time is also necessary to regain glandular balance. Sometimes menstruation starts immediately after treatment; at other times, it may take up to three months for a normal menstrual pattern to develop.

Climacteric

The climacteric period develops in both males and females, but it is usually associated with female symptoms; however, there may be symptoms in the male. In females the age of menopause is usually between 42 and 52; in males the climacteric age is later, between the ages of 60 and 65.

Today it is almost taken for granted that a female will have a difficult time with menopause. The reason that so many women have menopausal problems is that most people, by the time they reach the fourth and fifth decades of life, have developed health problems.

Under normal conditions, the adrenal cortex is expected to mature in its estrogen-androgen production at approximately the same time in life that gonad function decreases. Normally the adrenal cortex takes over gonad activity, including protein anabolism, without undue symptoms or stress to the individual. Unfortunately, many people have relative adrenal insufficiency, and the cortex fails to produce the necessary androgen or estrogen hormones. Because these sex hormones are powerful pituitary inhibitors, their absence causes a pituitary push that produces large

amounts of gonadotrophins in an attempt to bring up the level of androgen or estrogen. The pituitary push simultaneously produces ACTH, which in effect tries to "whip a tired horse," and further adrenal exhaustion develops. There is a spillover effect through the hypothalamus into the autonomic nervous system, promoting symptoms of hot flashes, cold chills, sweats, nervousness, headaches, insomnia, and mental depression. Any nutritional imbalance that affects neurohumeral balance will complicate the condition.

When a hysterectomy with bilateral oophorectomy is done, it is quite often necessary to supplement the patient with hormones. If this is not done, the above-mentioned pituitary push develops with its consequences. In earlier life, the adrenals are not ready to take over the function of increased estrogen and androgen production.

Breast Soreness

A common correlation with breast soreness in the female, especially lateral breast soreness, is colon or general digestive dysfunction. The neurolymphatic reflex for the tensor fascia lata and colon is usually very active, often requiring prolonged digital stimulation along the full length of the tensor fascia lata bilaterally. One can monitor the lateral breast soreness by digital pressure as the neurolymphatic reflex is stimulated. It is not unusual that four, five, or even more minutes of stimulation are required. Therapy localization to the reflex helps determine when it has been adequately stimulated. Often the breast tenderness will be markedly reduced or eliminated.

Examination and treatment of colon dysfunction are indicated, as discussed on page 500. While normal colon function is being obtained, it may be of value to teach the patient to treat her own neurolymphatic reflex.

Post-Parturition Backache

The pelvis is more vulnerable to structural distortion during the latter portion of pregnancy and delivery because of relaxin hormone presence. Often one sees major misalignment of the symphysis pubis in women who complain of backache that developed with a pregnancy and continued thereafter. Schleyer-Saunders[352] describes the increased movement of pelvic articulations due to the hormone relaxin. During labor the symphysis pubis stretches and may become misaligned. As the hormone is absent from the urine within twenty-four hours, the pelvic joints tighten again and may do so in a misaligned state.

The major pelvic distortion may be at the sacroiliac(s) or the symphysis pubis, or two or three of the articulations. Ideally, one corrects the pelvic distortion while the articulations still have increased movement. If the pelvis has returned to its more stable state, one must manipulate the articulations as best possible. A good practice is to advise pregnant patients to be examined structurally by a chiropractor as soon as possible after delivery.

Vaginitis

The predominant organism in many cases of vaginitis is E. coli, which develops from improper flora in the bowel. It is important first to make sure that the female is using good hygiene and cleansing after a bowel movement, taking care to always wipe to the posterior. If she is prone to vaginitis, she should routinely take showers instead of baths until the system improves to reduce possible contamination. These precautions are equally applicable with chronic cystitis.

A patient should be warned against using douches to avoid washing out the normal protective secretion of the vagina. Orificial ultraviolet can be administered. Treatment to return normal colon function is important to prevent recurrence.

Yeast infection often relates with candida albicans, which is outside the scope of this text. One should study the subject in the extensive literature available.[100,292,399,402]

Vaginal molds tend to develop only when there is a fertile field in which to grow. There is a high level of glycogen released during ovulation, and most molds correlate with a sugar metabolism problem. The patient should be examined thoroughly for any sugar handling stress.

Extensive neurolymphatic treatment is often required in any case of vaginitis or cystitis. The active reflex may require extensive stimulation. Even after proper stimulation, the reflex may again become active within a day or two, until the basic underlying cause of the infection is corrected.

Libido

The estrus of different animal species is controlled by the gonadal hormones affecting the upper midbrain or hypothalamus. This makes the mating desire neurologic and controlled by specific sensory stimuli, such as visual, auditory, and olfactory, which are derived from a possible mate. In the human, this basic neuromechanism has become largely dominated by cortical factors. Learning and conditioning play an increasingly important role. Methods used in applied kinesiology to penetrate the subconscious mind are often effective in finding the cause of and correcting loss of libido.

Before considering psychological factors, a complete physical examination should be done. A common cause of loss of libido is loss of general energy. Special attention should be given to sugar handling stress. A low blood sugar level with accompanying adrenal stress disorder is a common cause of libido loss. When there is general low energy in the meridian system, correction of the diaphragm will often improve an individual's energy level.

Prostate

Prostate massage is usually done in a manner that pulls it inferiorly. In actuality, it should be lifted. Lift the prostate in a cephalad direction until the patient has the desire to urinate. Hold that position for one minute.

The dropped prostate is frequently correlated with a weak levator ani muscle, which is the diaphragm of the pelvis. The levator ani should be examined and treated, if indicated, prior to lifting the prostate. The adductors are often involved with the levator ani; they should be checked and treated, if involved.

The organism frequently involved with prostatitis is E. coli; consequently, the bowel should be evaluated for normal flora. As a routine measure it is a good practice to prescribe an enema when prostatitis is present.

Vitamin F, which is linoleic and linolenic acid, produces good results. Oil of calabash or Mexican pumpkin is also of assistance. Oil of calabash can be placed directly into the rectum for a topical treatment. Zinc is indicated in many prostate problems.

Impotence

Impotence can be psychological or physiological. Again, the psychologic methods of applied kinesiology may be of help, combined with a complete physical examination.

The nerve endings in the skin of the penis can be involved, causing the impotence. The skin is challenged by surface traction on the dorsal surface in both the distal and proximal directions; the ventral surface is challenged the same way. If the nerve endings of the skin are involved, a previously strong indicator muscle will weaken. The usual treatment is prolonged neurolymphatic activity to the gluteus medius. This treatment is especially effective in the diabetic.

The neurotransmitter nitric oxide has been shown to be important in impotence[56] and high blood pressure.[290] Goodheart has described an applied kinesiology method to examine for an adequate supply of the precursors for nitric oxide.[173]

Levator Ani Muscle

Neurolymphatic:

Anterior: medial fascia of the thigh on the upper 1/3.

Posterior: adjacent to the transverse process of L5, and posteromedial fascia of the thigh on the upper 1/3.

Neurovascular: on the posterior aspect of the parietal eminence.

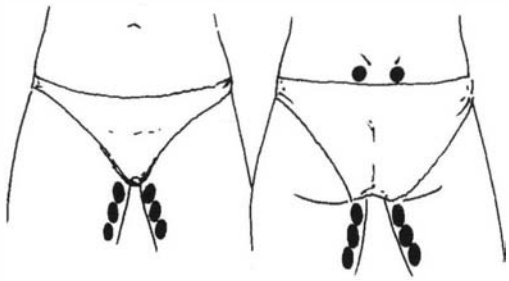
Meridian: large intestine.

Origin: two bony origins, one from the pelvic surface of the pubis and the other from the inner surface of the ischial spine. Between these bony origins it arises from the obturator fascia.

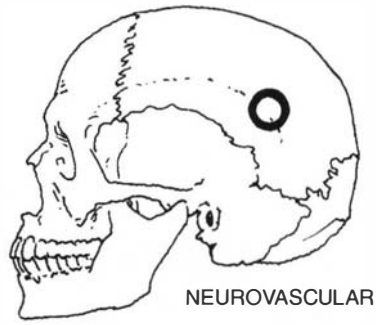
Insertion: To bone, it inserts on the front and sides of the coccyx. It inserts into the anal coccygeal raphe, which is a narrow fibrous band extending from the coccyx to the posterior margin of the anus. It also inserts into the external sphincter ani and the central tendinous point of the perineum.

Function: forms the floor of the pelvic cavity and is referred to as the pelvic diaphragm. It constricts the lower end of the rectum and vagina, retains viscera in position, and is a muscle of forced expiration.

Muscle testing precautions: As with any other test where a patient's privacy is of concern, several precautionary measures must be taken. First the procedure is explained thoroughly to the patient and informed consent obtained. A support person of the same gender as the patient should be in the room and witness the informed consent. The patient should be in a gown or clothed for this procedure; skin contact is not necessary.

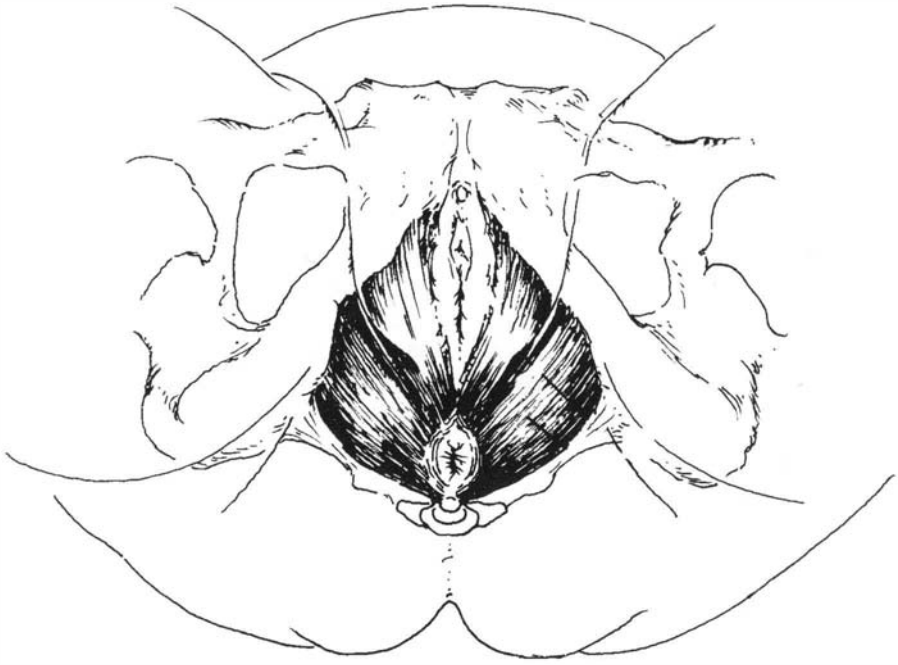


NEUROLYMPHATIC



NEUROVASCULAR

12—25.



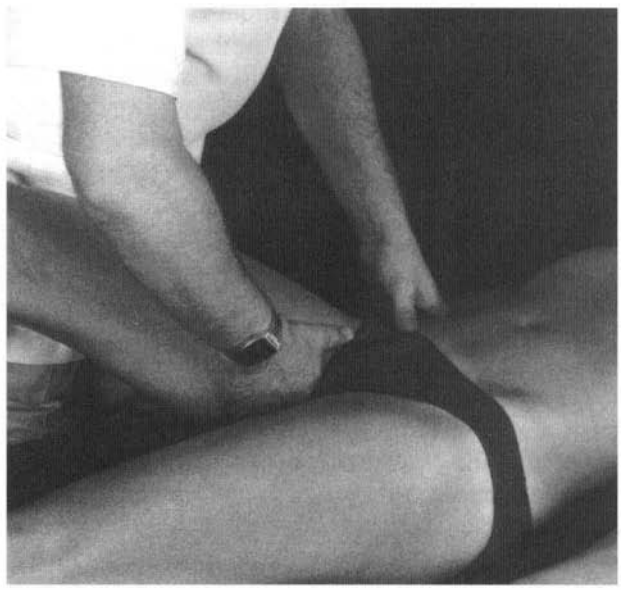
12—26. Levator ani muscle.

Test: Direct tests cannot be done on the levator ani muscle. One must observe for its ability to contract against abdominal pressure. The patient is supine, with the hip flexed and abducted. The examiner places the knife edge of his hand lateral and slightly anterior to the anus. Fingers should press lightly back into the ischial rectal fossa. The patient is instructed to bear down, as in the Valsava maneuver, and the examiner observes for bulging of the levator ani muscle, which indicates weakness.

When there is a dropped perineum, the levator ani is weak. The levator ani weakness is sometimes in conjunction with problems of the adductors, especially the pectineus. Often the adductors will be weak on one side and hypertonic on the other. If the condition is the result of injury at parturition, the prognosis is guarded; otherwise, the prognosis for correcting the muscle function is excellent.

The pelvic diaphragm functions in conjunction with the thoracic and cranial diaphragms. This is discussed with the thoracic diaphragm after the uterine lift technique.

12—27. Contact point for evaluating levator ani muscle bulge.



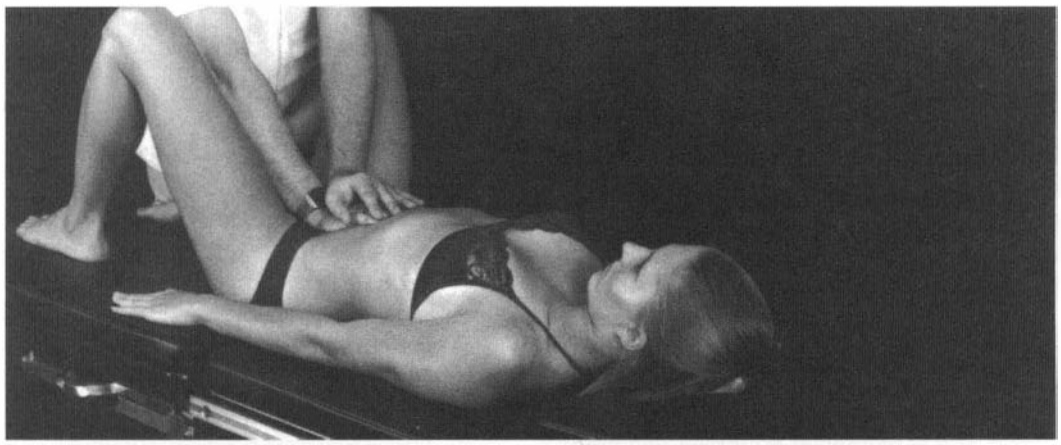
12—28. To avoid contact with the labia, slide hand along leg to the contact with the levator ani muscle.

Uterine Lift Technique

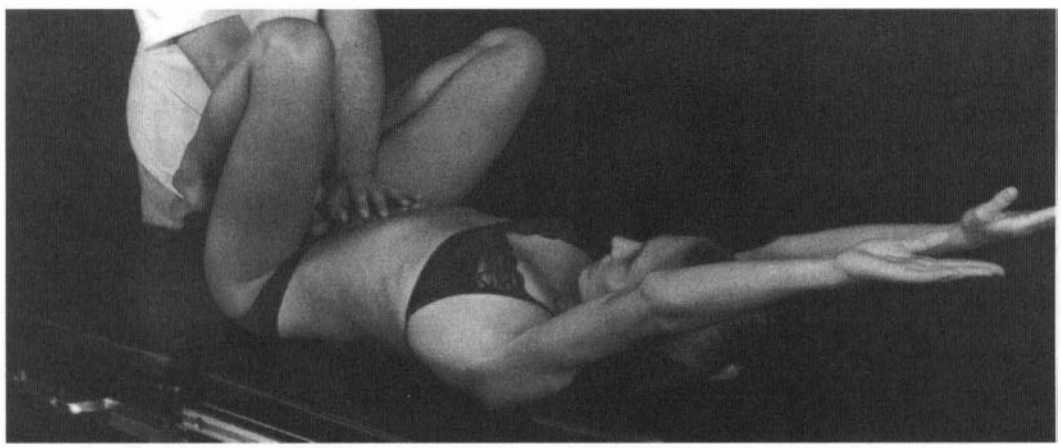
The uterine lift technique is often required after correction of a weak levator ani and in cases of uterine ptosis. Before using this technique, test the piriformis on both sides and correct, if necessary. Examine for other pelvic muscular imbalance and pelvic dysfunctions, such as categories I and II. Pay special attention to the gluteus medius and other muscles associated with the reproductive organs.

Goodheart says that body language indication for the need of the uterine lift technique is a female with exceptionally wide-open eyes. Need for the uterine lift technique is indicated by a positive challenge. Pressure is applied to the lower central abdominal area in a caudal direction. In the presence of a positive challenge, a previously strong indicator muscle will weaken.

The physician's first contact for correction is midway from the umbilicus to the pubis. The patient is requested to raise her arms and legs at the same time and forcefully exhale. As the patient brings her arms and legs up, the physician directs pressure with his fingertips in a posterior superior manner to lift the viscera. The maneuver is repeated three times. The second time the maneuver is begun more inferiorly than at first, and the last maneuver is done with the physician's hands starting just above the pubis.



12—29.



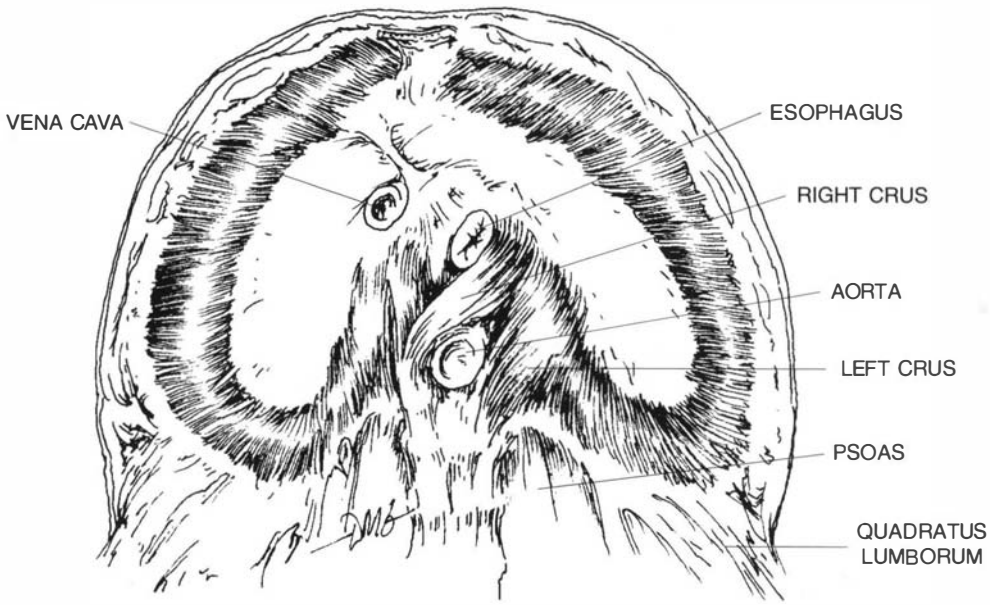
12—30.

Diaphragm

It is said that the heart and the diaphragm are the only two muscles that must function continuously or death will ensue. Another diaphragm whose function is necessary for life is the tentorium cerebelli in conjunction with the cranial-sacral primary respiratory system. The cranial diaphragm functions in synchronization with the pelvic diaphragm.

With inspiration, the cranial diaphragm descends and flattens in the same way the thoracic diaphragm does with inspiration. The pelvic diaphragm forms the floor of the abdominal cavity and is associated with sacral motion. It, too, descends and flattens on inspiration.

Each diaphragm is attached at its periphery to a potentially mobile part of the bony skeleton and is subject to rhythmic motion related to the phases of respi-



12—31. Inferior view of the diaphragm.

ration.[147] Frymann[148] objectively demonstrated the enhanced motion of the cranium with thoracic inspiration. This activity is observed daily in an AK practice as we see muscles return to strength with certain phases of held respiration. Respiratory assist is used in many phases of AK patient treatment.

Frymann[147] and Goodheart[176] describe the importance of activity and synchronization of the body's three diaphragms. Motion of the cranial diaphragm continues involuntarily and is enhanced by forced voluntary thoracic diaphragmatic motion. The thoracic diaphragm is under voluntary control but is maintained involuntarily. The pelvic diaphragm correlates with the cranial diaphragmatic motion as the cranial-sacral primary respiratory system, connected and synchronized by the core link (dura mater).

Motion of the cranium and pelvis and their importance to the cranial-sacral primary respiratory system have been discussed in Chapters 3 and 9. Likewise the thoracic cage and spine must be freely moving for the thoracic diaphragm to function normally. The interplay between the three diaphragms requires that each be functioning properly or the capacity of the others will be adversely affected.

Anatomy

Although not directly connected to the core link between the cranium and sacrum, the thoracic diaphragm has a major role in the organization between the three diaphragms. Frymann[147] quotes Still, "All parts of the body have direct or indirect connection with the diaphragm."

The diaphragm is a dome-shaped musculo-aponeurotic septum separating the abdominal and thoracic cavities. It is muscular at its periphery, with fibers converging into a central tendon. The muscular fibers are grouped into three parts — sternal, costal, and lumbar.

The sternal origin is by two short, fleshy strips arising from the posterior aspect of the xiphoid process. These fibers do not ascend toward the central tendon as sharply as do fibers from the other two areas of origin. In some diaphragmatic positions, the fibers may even angle down toward the central tendon. With this fiber angle, there is minimal lifting of the sternum by diaphragmatic contraction in comparison with other muscular fibers of the diaphragm lifting the ribs.

The costal origin is primarily from the inner surfaces of the costal cartilages and the adjacent parts of the lower six ribs on both sides. There is interdigitation of these fibers with the transverse abdominal muscle.

The lumbar origin is from the medial and lateral lumbocostal arches, sometimes called the medial and lateral arcuate ligaments, and from two crura from the lumbar vertebrae. The medial lumbosacral arch is tendinous, blending with the fascia over the upper part of the psoas major muscle. The lateral lumbocostal arch is a thickening of the fascia over the quadratus lumborum muscle. The crura originate as tendinous structures from the fronts and sides of the upper lumbar vertebrae. The right crus is broader and longer than the left, arising from the anterolateral surfaces of the bodies and intervertebral discs of the upper three lumbar vertebrae. The medial fibers of the right crus diverge to ascend on the left side of the esophageal opening; thus, the hiatus is surrounded by muscular fibers from the crura. The muscular fibers from the three origins converge to insert into the central tendon, which is a thin, strong aponeurosis located closer to the sternal origin than the costal and lumbar origins.

The esophageal hiatus is located in the muscular portion posterior to the central tendon at about the level of T10. The vagus nerves are transmitted through this opening. In applied kinesiology it is important to note the muscular envelopment of the esophageal hiatus.

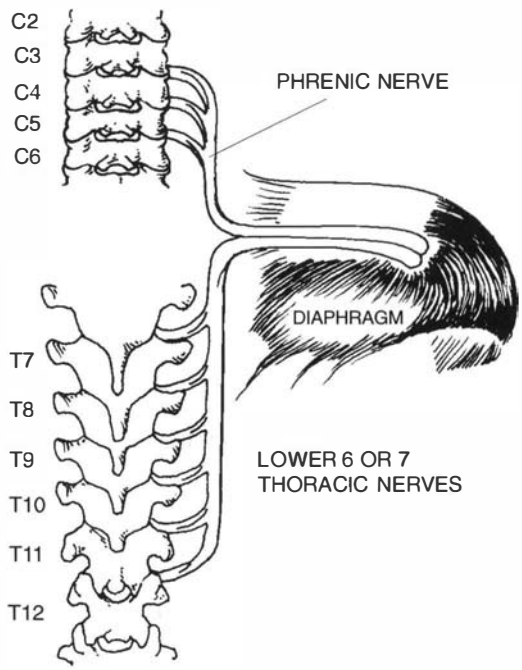
Diaphragm Apertures. The diaphragm has three large openings for passage of the aorta, esophagus, and vena cava. There are also a number of smaller openings. Anterior is the vena caval hiatus, located at the intervertebral level of T8 and T9. The phrenic nerve is often transmitted through this opening.

The aortic aperture, rather than being a hiatus in the diaphragm, is more of a notch, being an osseo-aponeurotic opening located between the diaphragmatic crura laterally, the diaphragm anteriorly, and the vertebral column posteriorly. It is located at the level of the lower border of the 12th thoracic vertebra, slightly left of the median plane.

12—32. Superior view of the diaphragmatic apertures.

Nerve Supply. Efferent supply to the diaphragm is by the phrenic nerves, arising from C3, **4**, 5.[65] Very often when this nerve appears to be disturbed a subluxation is found at C3, although it can be located at C2, 4, or 5. The crura are supplied ipsilaterally by the phrenic nerves. The right crus, passing fibers to the left of the esophagus to insert into the central tendon, receives innervation from the right phrenic nerve at its origin, and from the left phrenic nerve for the fibers on the left of the esophagus; thus the fibers of both crura, which insert into the left portion of the central tendon, receive left phrenic nerve supply, and those inserting on the right from the right phrenic nerve.[92,379] The lower six or seven intercostal nerves distribute sensory fibers to the peripheral part of the muscle. The solely sensory nature of these nerves is supported by complete muscular atrophy on the side of phrenic nerve destruction.[351]

Neurolymphatic: The neurolymphatic reflex for the diaphragm is located along the entire length of the sternum. When this reflex is active the entire area is usually extremely tender, and prolonged stimulation may be necessary to eliminate positive therapy localization.

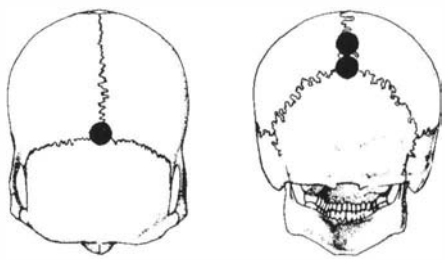


12—33. Schematic of diaphragmatic nerve supply.

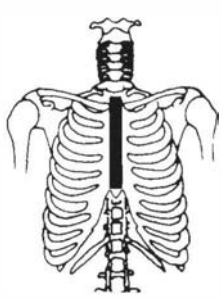
Neurovascular: Three neurovascular points are associated with the diaphragm. These are located at the bregma, lambda, and approximately 1" above and anterior to the lambda on the sagittal suture.

Stress receptor: The stress receptor is located on the frontal bone in the transverse plane, approximately 1-1/2" superior to the glabella.

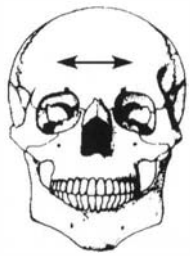
Meridian: conception vessel.



NEUROVASCULAR

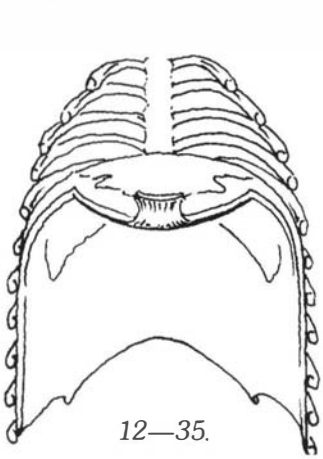


NEUROLYMPHATIC

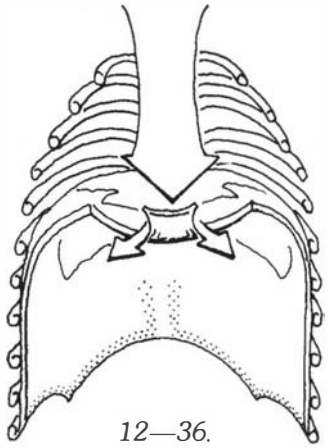


STRESS RECEPTOR

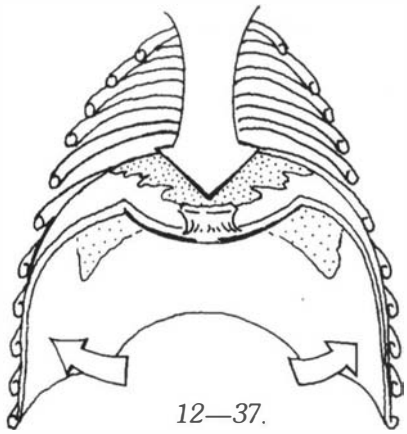
12—34.



12—35.



12—36.



12—37.

Figure 12—35 represents the neutral position of the diaphragm. In Figure 12—36 the dots represent the fixed point of diaphragmatic origin. The central tendon is moving inferiorly, pushing the organs ahead of it. In Figure 12—37 the dots represent the central tendon as the fixed point of origin for the diaphragm. It moves no further because of the organ compression. The ribs elevate with continued diaphragm contraction.

Action. The diaphragm is the major muscle of inspiration. The first stage of inspiration is traction of the muscles located at the circumference of the central tendon. These pull from the fixed base of the lower ribs, bringing the dome of the diaphragm inferiorly and pushing the abdominal viscera along with it. The 12th rib must be adequately fixed by the quadratus lumborum so the diaphragm can provide maximum downward thrust on the abdominal viscera in forced inspiration. Electromyography shows the quadratus lumborum is most active in the latter half of inspiration.[42] When the movement of the viscera reaches its limit, the central tendon becomes the fixed point; the action of the diaphragm elevates first the lower ribs and then the upper ribs to expand the size of the thoracic cage. The balance between abdominal and thoracic movement varies with different individuals and with the depth of respiration. The thoracic element is usually more marked in females.

Diaphragmatic Electromagnetic Activity

An additional concept of diaphragmatic activity presented by Goodheart[166] relates to the electromagnetic characteristics of the body. Investigators in this field relate to the anterior right and posterior left portions of the body being electrically positive, and the anterior left and posterior right portions being negative.[105] There is also the concept that energy in the meridian system of acupuncture is electromagnetic. Goodheart compares the diaphragm to an armature in a dynamo that activates these energy patterns of the body. These concepts are supported by the clinical changes that occur after treatment is administered to improve clinically determined poor function of the diaphragmatic muscle. The meridian system evaluated as low in energy improves considerably with therapeutic efforts to help diaphragmatic function. In many cases where the meridians are imbalanced, normal function will return with diaphragmatic treatment alone.

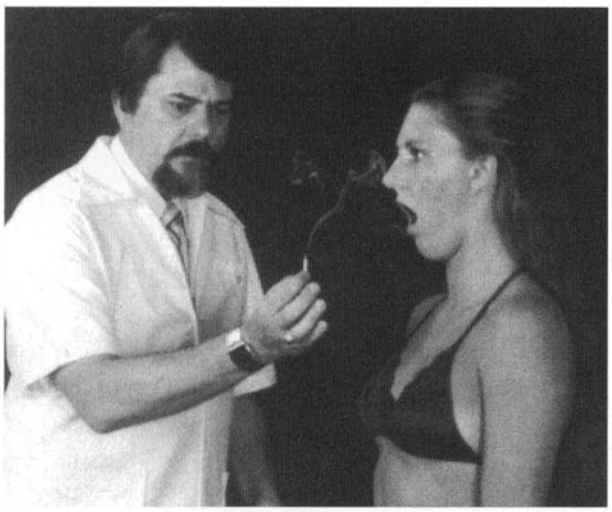
General Examination

Examination of the diaphragm is accomplished by combining standard testing procedures used in physical diagnosis with tests unique to applied kinesiology. The final diagnosis and evaluation of therapeutic efforts should always include a correlation of all factors, and not rely solely on applied kinesiology findings. This muscular diaphragm is, of course, more difficult to evaluate than skeletal muscles because of its inaccessibility. Skeletal muscles can be directly tested with manual muscle testing, while the diaphragm must be indirectly evaluated.

Vital Capacity and Maximum Expiratory Flow Rate. The determination of vital capacity and maximum expiratory flow rate (MEFR) is valuable in helping determine the diaphragm's strength. Individuals who have a weak diaphragm will generally have diminished vital capacity and MEFR. Of course, the physician must take into consideration other factors that may influence vital capacity and MEFR. Consideration should be given to a possible lack of ventilatory ability of the lungs, such as when it is diminished by emphysema. The air pathways must be free and not influenced by asthma or allergy reactions. Ruling out lung pathology and airway restriction, the vital capacity and MEFR tests provide valid information about the strength of the diaphragm. If a diagnosis of diaphragmatic weakness is made, these tests demonstrate improvement immediately after effective treatment.

Snider's Test. An easily performed screening test is Snider's.[385] A patient attempts to blow out a paper book match held six inches from his wide open mouth. The patient should not be allowed to purse his lips, and the match should be held in a position to receive the full force of air during forced expiration. Because the

upper central incisors and curvature of the palate tend to turn the air slightly downward from the open mouth, there is a tendency for the examiner to hold the match above the full force of air. If the examiner feels a considerable force of air on his hand, the test should be repeated with the match held slightly lower to receive the full force. A forced expiratory volume of approximately 1,000 ml and a peak flow rate of 130 liters per minute or more are required to extinguish the match. Inability to accomplish this task indicates probable ventilatory insufficiency and diaphragmatic weakness, or reduced thoracic cage mobility in the absence of lung or airway pathology. In the presence of a positive test, the patient should be further evaluated with a more sophisticated test to determine the reason for reduced air volume.

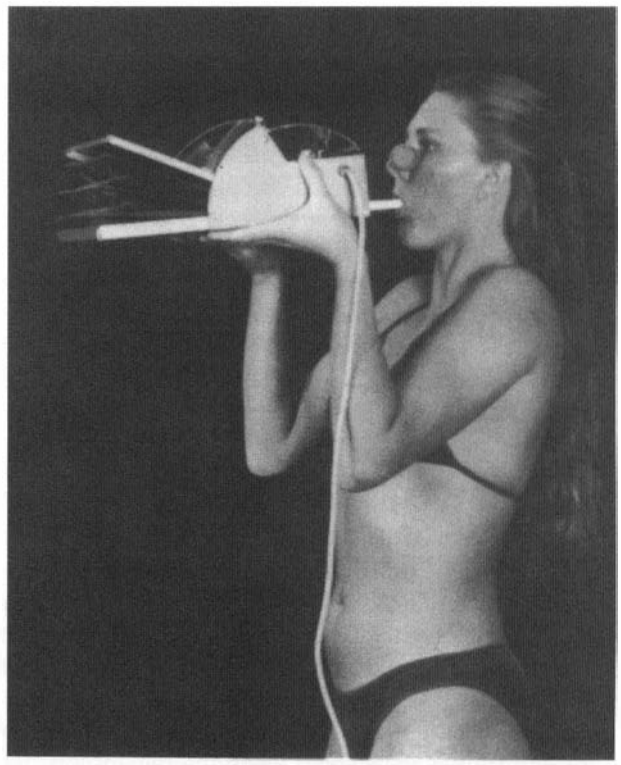


12—38. Snider's test is a screening measure to determine ventilatory sufficiency.

Vitalor. Numerous instruments are available for measuring vital capacity and maximum expiratory flow rate. An economical instrument for clinical use is the Vitalor.[411] This instrument measures vital capacity in liters that can be converted to percentage of normal vital capacity, taking into consideration an individual's height. It also gives a timed expiratory flow rate that can be compared to normal rates for males and females in different age ranges. The vital capacity and MEFR are recorded on a graph, which can be used after therapeutic efforts for comparison.

Spirometer. The spirometer is an instrument for recording vital capacity. Since it does not record MEFR, the information is limited; however, improvement of diaphragmatic function will usually show approximately 5% improvement in vital capacity after treatment.

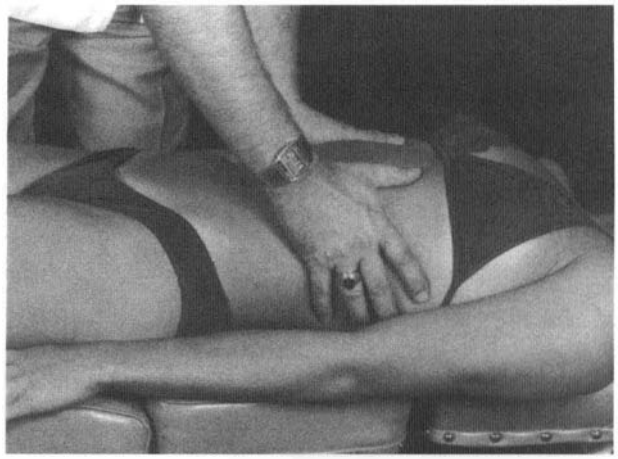
Thoracic Cage Mobility. Elevation of the ribs is accomplished by the accessory muscles of respiration and by diaphragmatic activity. To put in perspective the diaphragm's influence on thoracic cage mobilization, it is necessary to consider the strength of



12—39. The Vitalor is an instrument to measure vital capacity and maximum expiratory flow rate.

the accessory muscles of respiration, which include the quadratus lumborum, internal and external intercostales, abdominals, levator ani, levatores costarum, parts of the sacrospinalis, serratus posterior superior, latissimus dorsi, pectoralis major, pectoralis minor, sternocleidomastoid, and scalene muscles.

Chest expansion can be measured with a flexible measuring tape. Chest expansion varies greatly between males and females and different body types. In general, chest expansion should be above 4 cm. The prime value of this test is in re-examining thoracic movement immediately after therapeutic efforts. Care



12—40. The examiner will be able to both visually observe and feel the lack of movement on the side of weak diaphragm.

must be taken that the tape measure is placed at the same height on the thoracic cage for accurate comparison. For every 1 cm of increased chest expansion there is an increase of 200 ml of vital capacity.[420]

Thoracic expansion can also be evaluated by the examiner placing his hands bilaterally on the anterolateral aspect of the lower thoracic cage; the patient takes a deep inspiration, and the examiner resists lateral thoracic movement. In the presence of a weak diaphragm there will be reduced movement. This test is especially valuable when there is weakness on only one side. This quite often develops as a result of the diaphragm being reactive to psoas muscle imbalance, which will be discussed later.

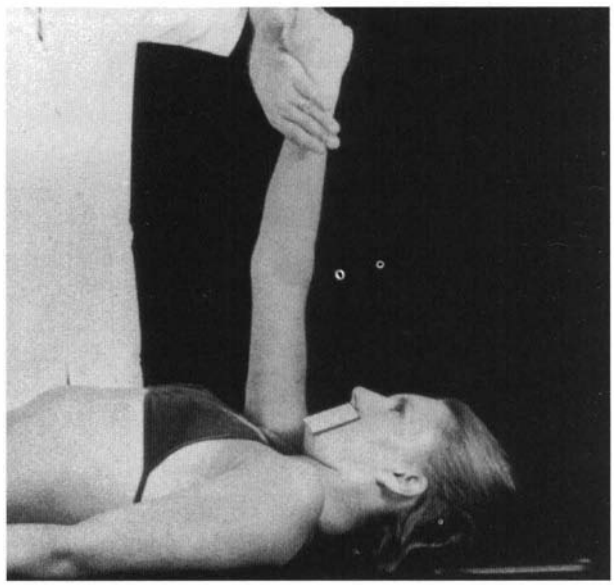
Applied Kinesiology Examination and Treatment

Therapy Localization. If the diaphragm is involved, therapy localization will be positive when the patient touches immediately under the xiphoid process. Often the positive therapy localization will not be present until the patient goes through a deep phase of respiration. The positive test is indicated by a previously strong indicator muscle weakening when the patient touches the area with or without the combination of a deep respiration. As with therapy localization in other areas, the physician must confirm the information with other testing procedures because therapy localization only indicates **something** is wrong in the area — not **what**.

Breath-holding Time. Normally a patient can hold his breath for an extended period without causing a change in muscle strength as observed by manual muscle testing. In the presence of diaphragmatic dysfunction, if the patient holds his breath for ten seconds there will be dramatic weakening of the general musculature of the body as observed by muscle testing. This indicates an inability of the nervous system, or possibly the meridian system, to function normally without continuous diaphragmatic activity. The muscles will regain normal strength immediately when the individual resumes normal breathing. When the examiner asks the patient to stop breathing for the ten-second period, he must caution the patient not to take a breath first. There is a natural tendency for individuals to quickly grab a breath when told to stop breathing. This extra breath increases the length of time that the muscles will remain strong in an individual with diaphragmatic dysfunction.

Meridian Involvement. As mentioned before, the energy of the meridian system, which is treated by acupuncture and other methods, appears to be electromagnetic. Electromagnetic energy flows from one meridian to the next to complete its cycle throughout the body. Many types of evidence in evaluating the meridian system support Goodheart's hypothesis that the diaphragm mobilizes meridian energy. In classic acupuncture, the midline meridians — the conception and governing vessels — are pools of used energy that escape from the body. Removal of this used energy occurs at the ends of the meridians, located at the mouth.

A sheet of lead placed over an acupuncture point blocks energy flow into or out of that area. When the diaphragm is not functioning at an optimal level, a sheet of lead placed over the ends of the conception and governing vessels (CV 24 and GV 27) causes all muscles of the body to test weak. This appears to prevent the removal of used energy from the body, in turn inhibiting the movement of electromagnetic energy through the meridians. This poor energy mobilization is believed to be caused by poor diaphragmatic function. Placing the same piece of lead anywhere else on the body has no effect, nor does placing the lead over the ends of the conception and governing vessels have any effect on an individual whose diaphragm is functioning normally.



12—41. Lead placed over acupuncture points CV 24 and GV 27 causes an indicator muscle to weaken when the diaphragm is dysfunctioning.

Phrenic Nerve Involvement. Efferent supply to the diaphragm, along with some afferent supply, is from the phrenic nerve arising from C3, **4**, and 5. Dysfunction of this nerve is very often due to peripheral nerve entrapment at the intervertebral foramen between C2 and 3, with a vertebral subluxation at C3. The cervical vertebrae are challenged; if a subluxation is present, it is corrected by adjustment and treatment of the intrinsic muscles. It is interesting that often the subluxation will not therapy localize until the patient respires deeply while therapy localizing; a previously strong indicator muscle then weakens.

A 3rd cervical or adjacent subluxation is often secondary to diaphragmatic dysfunction on a reflex basis. In the absence of body language indicating diaphragmatic dysfunction, a physician is often led to its evaluation because of a recurrent subluxation associated with the phrenic nerve. Diaphragmatic evaluation

is indicated whenever there are recurrent subluxations in this area. The optimal procedure is to correct the diaphragm first, as this will often eliminate evidence of the subluxation with no direct treatment to the vertebrae.

Vertebral Fixation. There is often a vertebral fixation of the T11, 12, and L1 area, accompanied by the usual bilateral lower trapezius muscle weakness; it is generally evaluated and corrected in the usual manner. In most cases vertebral fixations will not therapy localize unless the hand contact is held while the spine of the area is moved through various ranges of motion. A unique factor of this vertebral fixation, when associated with a diaphragmatic weakness, is positive therapy localization when the hand contact is held and the patient respires deeply.

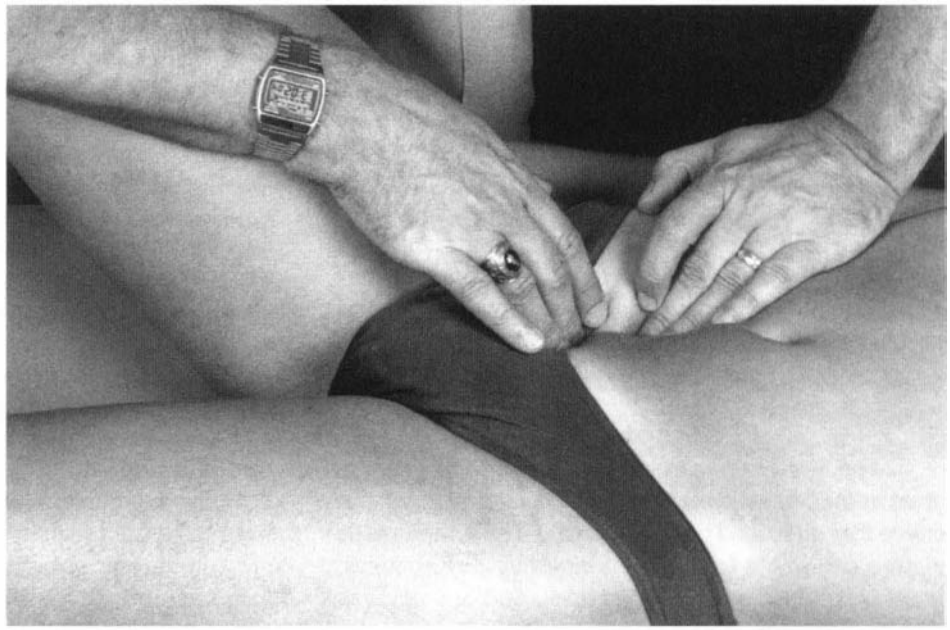
Lumbar Spine. Diaphragmatic involvement can result from dysfunction of the lumbar spine. Quite often, the lumbar involvement is the Lovett reactor vertebra to the cervical vertebra involved with the phrenic nerve.

Diaphragmatic dysfunction from the lumbar spine may be due to the origin of the crura from the bodies and intervertebral discs of the upper lumbar vertebrae. In a study of 200 cases of hiatal hernia, Edmunds[129] found a "...noticeable spinal deformity in 60% of those with the 'rolling' type hiatal hernia, and 40% where it was combined with a sliding-type." Often the pelvis is misaligned or functioning abnormally in motion, contributing to the lumbar spine distortion. It is often possible to obtain diaphragmatic correction without attention to the lumbar spine and pelvis. When diaphragmatic correction is not lasting, it is very important to examine and correct this area.

Diaphragm Reactive to Psoas. In applied kinesiology, an abnormal condition is observed in which a muscle weakens immediately after another muscle has been contracted. This relates to the afferent supply of the primarily contracted muscle sending improper information to the muscle that reacts abnormally, known as a reactive muscle (page 65). The diaphragm reactive to the psoas has been clinically observed as a frequent cause of diaphragmatic dysfunction. There are two methods for determining this possibility. First, the lateral thoracic movement is evaluated by the examiner resisting the rib cage expansion as described previously. Immediately test the psoas to activate a contraction, then re-evaluate the thoracic cage excursion. In the presence of a diaphragm reactive to the psoas, there will be diminished thoracic excursion immediately after the psoas contraction. Both sides should be tested. The weakening of the diaphragm is due to improper signaling from the psoas. The exact nerve pathways are unknown. The interaction is probably at the cord level, relating to diaphragmatic correlation with walking and running gait activity.

The second method of evaluation cannot be used unless therapy localization below the xiphoid process is negative without diaphragmatic activation. If this is the case, continue the therapy localization contact and activate the psoas muscle by testing it; then immediately re-test the indicator muscle used for therapy localization evaluation. Weakening indicates that the diaphragm is reactive to the psoas. The test can be more sensitive if after psoas activation the patient is asked to take a deep breath and expel the air while continuing to therapy localize below the xiphoid process. If an indicator muscle weakens after this action, the diaphragm is reactive to the psoas.

This reactive involvement results from improper function of a neuromuscular spindle cell in the belly of the psoas. The abnormal spindle cell can sometimes be palpated; it will be a very tender, usually nodular area frequently in the general area of the anterior ramus of the innominate. The abnormally functioning neuromuscular spindle cell will also show positive therapy localization. Treatment is manipulation over the nodular area with two fingers directed toward each other over the neuromuscular spindle cell. This treatment is more easily accomplished if the patient contracts the psoas, tending to lift the belly of the muscle into the physician's treating fingers. The physician contacts over the neuromuscular spindle cell with the in-



12—42. Treatment of psoas muscle neuromuscular spindle cell.

dex finger and thumb while the other hand applies pressure to the leg in the manner of a usual psoas muscle test.

An easier method is to have the patient flex his thigh and knee with external thigh rotation while his foot rests on the examining table. The examiner then rests his arm on the patient's medial knee and lower thigh, freeing both hands for treatment over the neuromuscular spindle cell. When treatment contact is made, the patient attempts to bring his thigh and knee toward the midline against the examiners arm by contracting the psoas which elevates the muscle belly into the examiner's treating fingers.

Successful treatment is indicated by elimination of positive therapy localization over the neuromuscular spindle cell, and of the positive therapy localization at the diaphragm immediately after contracting the psoas in a muscle test. Also, the lateral movement of the thorax should be improved and equal to the opposite side.

Often the hypertonic psoas is primary because of injury to the neuromuscular spindle cell. As a result of overcontraction or stretching past the muscle's physiological ability, trauma to the neuromuscular spindle cell can cause swelling within the capsule and improperly stimulate the afferent supply. Eventually, adhesions of the delicate septa between the intrafusal and extrafusal fibers can result.

Sometimes a hypertonic muscle can be secondary to another involvement. Already mentioned is the importance of the lumbar spine and pelvic balance. The psoas can become hypertonic because of attempts to correct imbalances of the bony structure. The psoas is important to lumbar stability and movement. The sacroiliac articulation also depends on normal psoas function, since the muscle spans that joint and provides indirect support. The psoas may also be hypertonic as a result of weakness of the opposite psoas. When there is lack of support from one psoas, the opposite automatically contracts. In this case, treatment should be directed toward the weak psoas. Several reflexes used in applied kinesiology have been associated with diaphragmatic dysfunction. These should be evaluated with therapy localization, challenge, muscle testing, and palpation, and treated in the usual manner.

Diaphragmatic Hernia

Because of anatomical weaknesses that may be acquired or congenital, the various apertures of the diaphragm may allow portions of the abdominal — or possibly thoracic — organs to protrude through the defect. The most common location for a diaphragmatic hernia is at the esophageal hiatus, which will be the primary consideration here. There are variances of parasternal or subcostosternal type.

There are three major types of esophageal hiatal hernia: (1) sliding, (2) paraesophageal, and (3) congenitally short.[396] Seventy-five percent of hiatal hernias are the sliding variety. This is a transient hernia in which the gastroesophageal junction slides into the thoracic area. Roentgenographically it is demonstrated by gastric mucosa and the esophagogastric junction being located above the diaphragm. In the paraesophageal hernia, a portion of the gastric fundus herniates into the thorax alongside the distal esophagus, and the cardioesophageal junction remains below the diaphragm. The congenitally short esophagus is rare, and some authorities deny its existence. If present, it becomes symptomatic during the first five years of life.

Symptoms

The hiatal hernia is sometimes referred to as "the great mimic" because of the numerous symptoms it can create that often lead diagnosis astray. The symptomatic picture can relate to apparent digestive disturbances, heart problems, and shoulder, neck, and jaw pain, as well as dysphagia and hiccups.

The simulation of digestive disturbances from a hiatal hernia may mimic gallbladder or stomach disease, and there may be regurgitation and belching. There is often regurgitation of irritating gastric contents that may cause burning of the sternal area, which radiates to the back, neck, jaw, ear, palate, shoulders, or arms. This may lead an individual to take antacids to diminish the burning sensation. Chronicity may cause esophagitis, compounding the problem. Dysphagia is described by the patient as difficulty in swallowing food, with an awareness of the bolus passing down the entire gullet and sometimes feeling like it is "stuck" at the bottom. The symptoms of hiatal hernia are frequently created or intensified by a large meal. As a result, "food fear" may be noted, with the resulting loss of weight. It is usually not the type of food that causes a problem; rather, it is the amount.

The usual age range for hiatal hernia is 40-70 years, which parallels that of angina pectoris and coronary occlusion. The pain in the thorax may mimic angina pectoris or a frank coronary occlusion; it may radiate to the neck and often the left arm, increasing the suspicion of a coronary problem. Symptoms are increased by a heavy meal, or when the patient is recumbent. Activities that simulate a Valsalva maneuver also increase the symptoms, including heavy lifting, bowel movements, wearing tight garments, or bending after a full meal.

In chronic conditions there may be ulcerations, with hemorrhage and secondary anemia. The herniation may be massive, to the point of embarrassing respiration as the abdominal organs physically crowd the

lungs. Although large herniations seem to be the most dramatic, the size of the hernia is certainly no indication of the severity of the symptoms.[123] Some of the small hernias, barely recognizable on x-ray, can cause the most severe symptoms, while other more "significant" hernias are asymptomatic. A hernia may cause pressure on the heart, resulting in palpitations and tachycardia. In extremely large congenital openings, the colon and small intestines may even protrude into the thorax.

Differential diagnosis should be made in all cases in which a coronary or a hiatal hernia condition is suspected. The evidence of one does not rule out the other, as the conditions may coincide. Master et al.[276] recommend evaluation by exercise tolerance electrocardiography. Kohli and Pearson[243] demonstrated that there was no change in normal electrocardiograms at rest or after exercise when a diagnosed hiatal hernia was distended by inflating a balloon in the stomach. This seems to indicate that the presence of a hiatal hernia is insignificant on electrocardiograph findings. When exercise increases the symptoms in the presence of a hiatal hernia, there is indication to further evaluate for a coronary problem. Evaluation for peptic ulcer, gallbladder, shoulder and spinal dysfunction is made by the usual diagnostic methods. X-ray evaluation is the usual method of documenting a hiatal hernia.[307,396] Many roentgenologists point out the necessity of examination in various positions, or a false negative may be reported for an esophageal hiatal hernia. Donald[123] relates "...the number of hernias that is found is in direct proportion to the ability of the roentgenologist and his diligence in examining each patient for the presence of a diaphragmatic hernia."

Since diaphragmatic hernias demonstrated by x-ray may or may not be symptomatic,[370] it appears that much of the symptomatic pattern develops from gastric reflux that may occur in the absence of an x-ray demonstrated hiatal hernia.[384] The cause of reflux is attributed to incompetence of the lower esophageal sphincter. Additionally, it appears that diaphragm weakness that is present with or without hiatal hernia is an important consideration in reflux. The muscular strength of the fibers surrounding the hiatus appears to be an important aid to the lower esophageal sphincter in retaining gastric contents — primarily hydrochloric acid and pepsin — in the stomach. The muscular design of the diaphragm is such that the crura surround the esophageal hiatus and provide a scissors-like or pinching action on the esophagus to aid in maintaining the competence of the lower esophageal sphincter. The crura surrounding the esophageal hiatus exert a supplementary sphincteric action on the lower end of the esophagus. They relax immediately after the act of swallowing and contract after the bolus passes.[418,420]

More attention is given in the literature to the lower esophageal sphincter than to the crura in the prevention of reflux. The action of constriction of the lumen at the esophagogastric junction was demonstrated by Atkinson et al.[15] Their manometric study was done by passing balloons through the region of the esophageal hiatus where the pressure usually ranges up to 2 cm above the diaphragm; the pressure relaxed with swallowing. An interesting factor is that the balloon arrangement did not demonstrate any pressure at the pyloric sphincter, even though there is greater histologic evidence of sphincteric action at that area than at the cardiac area. Their conclusion was that the crura are unimportant in increasing this pressure because of the pressure extending into the thorax area. This was further emphasized by demonstration of pressure at the esophagogastric junction in patients with hiatal hernia.

The basic design of the crura and their control by neurologic reflex after swallowing gives reason to believe good diaphragmatic action is important in preventing both reflux and hiatal hernia. This is further supported by the clinical evidence of elimination of reflux after correction of certain types of hiatal hernias with AK treatment designed to increase diaphragmatic function.

Examination

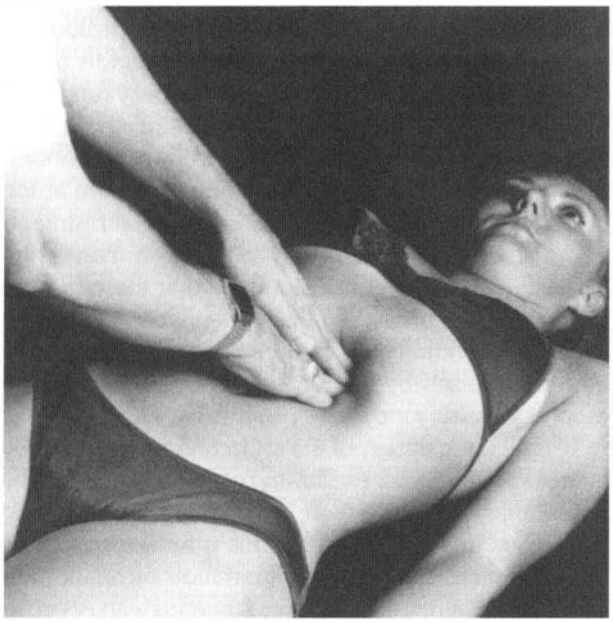
In applied kinesiology, examination for a hiatal hernia is combined with that for diaphragmatic function. The findings may correlate with standard x-ray diagnosis for hiatal hernia, but they do not always do so. In some cases there will appear to be a hiatal hernia on applied kinesiology examination, but it will not be confirmed by x-ray diagnosis. In these cases, there may be an undetected sliding hernia, even though various positions and the Valsalva maneuver are used during the x-ray exam. In any event, if there has been adequate differential diagnosis, good results of symptomatic removal from the therapeutic effort will usually be obtained.

First the diaphragm should be evaluated as previously described. There will not always be evidence of a hiatal hernia with diaphragmatic weakness, but there will always be diaphragmatic weakness present when there are positive findings of a hiatal hernia that will respond to applied kinesiology treatment. A congenital or traumatic hiatal hernia may not demonstrate evidence of diaphragmatic dysfunction.

The diaphragm is evaluated for a possible functional hiatal hernia with the applied kinesiology challenge mechanism. The physician applies a gentle thrust toward the hiatus with pressure under the xiphoid process directed cephalad and slightly toward the left shoulder. In the presence of a functional disturbance at the hiatus, a previously strong indicator muscle will weaken as observed on manual muscle testing.

Treatment

When there is clinical evidence of a hiatal hernia by symptoms and applied kinesiology challenge, and other conditions have been ruled out by differential diagnosis, a therapeutic trial is justified. Surgical experience



12—43. Hiatal hernia challenge is directed cephalad under the xiphoid process, slightly toward the left shoulder. Immediately after this gentle thrust, a previously strong indicator is tested for weakening.

indicates, "...conservative measures are worth an extensive trial in uncomplicated cases of hiatus hernia."[129] Good results will usually be obtained.

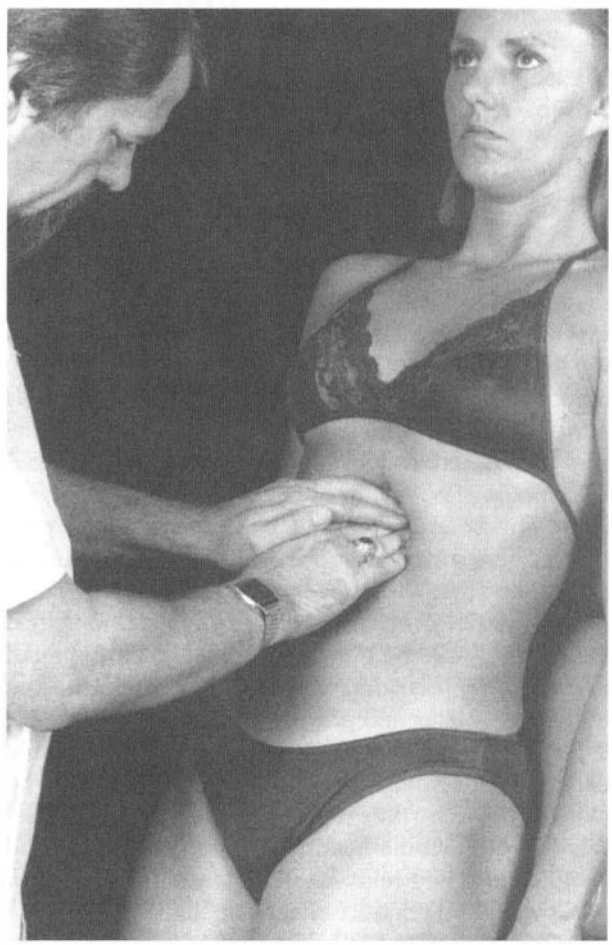
First, a manipulation is done to pull the stomach from within the hiatus. This is best accomplished with the patient standing, supported against a wall, or, better still, against a hi-lo table. The physician works the fingertips of both hands into the tissue under the xiphoid process, attempting to obtain good purchase on the stomach so that inferior traction may be placed on it. As the patient takes a deep breath and the diaphragm moves inferiorly, the physician works the fingertips deeper into the tissue and holds that position as the patient exhales. This inhalation-exhalation activity is repeated two or three times while the physician continues to obtain better purchase on the stomach. As the patient exhales for approximately the fourth time, the diaphragm travels upward and the physician gives a quick pull inferiorly on the epigastrium. This quick movement manually lowers the stomach, pulling it out of the hiatus. There is often immediate relief in an acute condition after this manipulation. Sometimes a release will be felt, similar to a cork coming out of a bottle. In some cases, it may be necessary to repeat the manipulation several times until it is successful.

The diaphragm should be treated as indicated previously. All diaphragmatic indicators should be re-evaluated and found negative to confirm successful treatment. The hiatal hernia challenge should also be negative.

Weight reduction is indicated if the patient is overweight. In some cases, it may be necessary to advise the patient to avoid straining the diaphragm, such as bending over and crowding the abdominal contents immediately after a large meal. A common procedure in the conservative treatment of hiatal hernia is to elevate the head of the bed 10-20". This is rarely necessary in the applied kinesiology approach to the condition.

Re-evaluate the patient on subsequent visits for a possible return of the positive hiatal hernia challenge and diaphragmatic weakness. If there is recidivism, careful attention should be given to the lumbar and cervical spines, pelvis, feet, and other factors that may influence the diaphragm. A common dysfunction contributing to diaphragm dysfunction is psoas imbalance.

Most cases of hiatal hernia will respond to the technique described here. In some cases — especially if there is a severe congenital weakness or chronicity with extensive herniation — referral for surgery may be necessary. Traumatic rupture of the diaphragm is out of the scope of this text. It usually results from auto accidents and falls, and is best treated by surgery.[114] Of course, the treatment described here is most effective when the condition is functional and has not developed severe chronicity.



12—44. Manipulation is an attempt to pull the stomach from within the hiatal hernia prior to diaphragm treatment.

Ionization

This text has grown much larger than originally intended. At some point the "synopsis" of applied kinesiology must come to an end. I choose to end with the subjects ionization, and next, the AK approach to the reticular activating system. Both are innovative methods of examination and with further research are bound to reveal more information of improper body function.

The frustrating aspect of studying atmospheric ionization is that it is insensible; that is, it cannot be perceived with any of the five senses. I suppose it should be no more difficult for one to appreciate electromagnetic and vibratory effects on the body than to understand electricity, which in many ways parallels the electromagnetic and vibratory factors of body function. Electricity flowing through a wire cannot be seen; however, its effects can be perceived in many ways. It can be measured with a volt/ohm meter, it can be felt if one touches an active bare wire when grounded, and its effects can be seen in the glowing of a light bulb.

In order to appreciate the effects of ionization balance in the atmosphere and within the body, we must understand its nature and be able to measure its effects in the body. Applied kinesiology appears to have expanded the ability to observe the effects of ionization on body function. This discussion opens the door to a wide field of electromagnetic and vibratory function occurring in the environment and within the body that plays a major role in optimal health or disease. A full discussion of the subject must await another textbook on applied kinesiology. Here we will briefly discuss ionization, which helps understand one aspect of therapy localization, i.e., therapy localization with "palm down" or "palm up" touching of the skin.

Air ions are molecules with atoms attached that have gained or lost an electron. If an atom loses an electron, it becomes positive; if it gains an electron, it becomes negative. In general, there are three sizes of air ions: small (molecules), medium (tiny particles), and large (dust). The small ions are the ones of interest because they are the most mobile and play a central role in atmospheric influences upon the human system.[24]

Many factors control the balance of air ions. In nature, where the air is clean and basically undisturbed, small air ions of either positive or negative polarity are generated by radiation processes, radioactive fallout, cosmic (non-solar) sources, natural radioactive materials in the atmosphere, and some aspects of solar radiation lying beyond the ultraviolet range, particularly during solar disruptions.[24]

The earth is negatively charged and tends to repel negative ions and attract positive ones; thus, close to the earth's surface where man, animals, and vegetation grow, there tend to be more positive ions than negative ones. It is generally accepted that there is a 5-positive to 4-negative ion ratio. The "healthy" ion level is 1,000-2,000 ions/cubic cm, with the 5:4 ratio. This is found over an open country field on a clear day. In general, an excess of positive ions is considered detrimental to body function, while an increase of negative ions is beneficial. Numerous studies have shown that when there is a decrease in overall ionization and an increase in the positive to negative ratio, many aspects of normal health are disrupted. Soyka and Edmonds[387] discuss the successful research of Kornblueh, who applied negative ionization to burn victims, relieving pain and increasing the healing rate. Kornblueh is credited with applying the phrase "vitamins of the air" to negative ions. Soyka and Edmonds state, "Negative ions are being called 'happy ions' whereas positive ions are being called 'grouchy ions.'"

In addition to affecting health in many ways, ion balance affects our moods. A major source of the happy ions is moving water, which generates negative ions. As the water breaks up, the positive charge remains with the larger drop of water; the negative charge stays with the fine spray and releases as a negative ion. This is one of the main attractions of waterfalls, the oceanside where waves break against the rocks, and even the man-made shower in the morning. Mountain streams have an even greater attraction because of the increased negative ions in the forest from the growing vegetation.

There are many factors that change ion balance. The ion level fluctuates considerably with the environment. There are more ions in the woods than in the fields, more on a sunny than a cloudy day, and more where the ground has a high radioactive content. The weather, the moon, and the seasons all affect both the ion content and the balance. Ion balance and concentration are also affected by man's modification of the environment, such as large expanses of pavement and concrete. Modern inventions, such as the automobile and electronic equipment, also modify the ion balance.

Various types of friction increase positive ion concentration. One can easily recognize this when walking over a synthetic carpet, wearing synthetic clothes; positive ions develop, creating static electricity that is rapidly discharged when one touches something that contacts the earth, such as a grounded electrical appliance. The earth is negatively charged; this is the reason for the term "grounding" an electrical appliance.

A major change in ion balance develops from movement between air masses, between air and the ground, or between air and another object. The air movement may be a massive weather front, or air moving through heating or air-conditioning ducts.

In numerous areas throughout the world, legends exist that tell of unusual things happening when a particular type of wind blows. People become tense and fight among themselves; suicides and murders increase,

as do traffic and other types of accidents. The simultaneous increase of these events and other unfortunate happenings has been statistically correlated in many areas. One wind of this type is the Foehn, which blows out of the Alps in early spring and fall. It affects areas of Switzerland and southern Germany. The association of this ill wind with misfortune, stress, and unhappiness has earned it the name of the "witches' wind." In Munich and many other parts of central Europe north of the Alps, surgeons even postpone operations if a Foehn is forecast, and judges are more lenient in their consideration of crimes that might have been committed because of the influence of the wind.

Other winds throughout the world are known to affect people's emotions, actions, and health. The summer winds of the northern Arizona desert down into Mexico are known in Indian mythology as the "bitter winds." In California there is the Santa Ana, and in Canada the Chinook. In the Middle East, this type of wind is called "Sharav" by the Israelis and "Hamsin" by the Arabs. All these winds more or less share the reputation of the witches' wind.

When dust particles or dirt is swept up by the wind, the change in ion balance becomes greater because the negative ions attach to the particles dropping to the ground, thus taking negative ions out of the air. When the Sharav around Israel blows, it is sometimes called the "brown wind" because of the sand it carries. Likewise, other airborne pollutants and pollen reduce the negative ion balance as they attach to the substances that fall to the ground and are discharged. In general, the life of air ions is longer in clean air. The lower the visibility, the lower the small ion concentration.

The drop in ion concentration, especially that of negative ions, is pushed ahead of weather fronts because electricity travels faster than air. The ion imbalance may be as much as two days ahead of the weather front. This is why many people can forecast the weather by the way they feel, and why animals act differently with impending weather change.

Even the moon has an effect on ion concentration. The inner portion of the ionosphere facing the earth is positively charged, and that facing outer space is negatively charged. There is an interaction between the positively charged underside of the ionosphere and the negatively charged earth to generate ions of both polarities to maintain balance. The moon orbiting the earth on the outside of the ionosphere is negatively charged; it repels the negative outer face of the ionosphere. When the moon is full it is closer to the earth, thus pushing the ionosphere closer to the earth. This changes the interaction between the ionosphere and the earth, creating a greater number of positive ions close to the earth's surface.[387] Aberrant human behavior is associated with the lunar cycle. The word "lunar" has become entrenched in the word "lunatic." Man's general accumulated knowledge has given meaning to the comment, "No wonder — the moon was full that night," as one thinks of the unusual things that happen during the full moon.

It is well-documented that there are changes in the way people act and feel with these natural changes in ion concentration. In most cases, these natural causes of imbalance pass by; the full moon does not last, and the weather front moves on. What doesn't pass and continues to affect the ion concentration are man's inventions and what he has done to the environment.

Air movement over metal causes loss of ions, primarily negative ones. The friction of air moving over metal causes the metal to develop a positive charge that attracts the more active negative ions. When the design of air ducts for heating or cooling is such that there are many bends, curves, and long passageways, the friction increases with greater loss. It is because of the design and distance of ductwork that some portions of a building may cause greater discomfort to the inhabitants. When the air-conditioning system is being used for heating, the problem is compounded because the heating element creates positive ions. This is further compounded by the fact that most modern buildings are totally enclosed, allowing no outside air to enter.

The ionization level is worse in most modern buildings because the floors are usually covered with synthetic rugs, and there are numerous synthetic fabrics in curtains and furniture upholstery. Friction as one moves over the carpet, or sits and moves in chairs, increases the loss of ions. Large cities are particularly vulnerable to low-ion level and an imbalance between positive and negative ions. The lack of vegetation reduces negative ions. In addition, man builds asphalt and concrete over the ground, causing further reduction.

Like air moving through ducts, the friction of an automobile or an airplane moving through the air sets up a positive charge on metal to attract negative ions. Susceptible people become drowsy, fatigued, or develop symptoms such as headaches when riding in an automobile or airplane. This is intensified with time and may even be the cause of some auto accidents and plane crashes.

Soyka and Edmonds[387] quote C.W. Hansell from the RCA Research Laboratories at the 1961 International Ion Research Conference in Philadelphia: "Our distant ancestors in the course of our evolutionary development lived literally with their feet on the ground. Their bodies were kept at ground potential. In contrast, we are electrically insulated from the ground for much of the time, often with our bodies at potentials far different from ground and our surroundings. These potentials can have large effects upon the ratio of positive to negative ions we absorb from the air and upon their total number. An outstanding example is provided in warm dry air in winter when we walk about on clean, new wool carpets in rubber- or leather-soled shoes. Our bodies may then become negatively charged to poten-

tials of tens of thousands of volts. Under these circumstances negative ions are strongly repelled by our negatively charged bodies but positive ions are strongly attracted. We are then likely to be physically, mentally, and emotionally depressed and irritable."

Physiologic Effects

Although there has been considerable research on ion concentration and balance affecting health, it is still an area in which much is unknown and the general scientific community is, for the most part, unclear about what is known. A leading scientist on the effects of ionization on health is Sulman of Israel, who has done extensive study of the Sharav wind inhabiting that region. Soyka and Edmonds[387] and Beasley[24] report on Sulman's research. Sulman notes the first effect of the Sharav on man is the excessive release of serotonin, which he calls the "serotonin irritation syndrome." This may introduce migraines, hot flashes, irritability, sleeplessness, pains around the heart, difficulty in breathing, a worsening of bronchial complaints, emotional tension, and anxiety; it is generally followed by the "exhaustion syndrome." This occurs over a period of years in susceptible people. It is, in essence, the third stage of Selye's general adaptation syndrome,[375] previously discussed. The body continues to produce epinephrine and norepinephrine in an attempt to meet the challenge until it finally becomes exhausted. In Sulman's studies, 44% of the Sharav victims were diagnosed as suffering from the exhaustion syndrome. In addition, Sulman found that there was a hyperthyroid response for unknown reasons. He found that 25% of the population in his area is seriously affected by ion imbalance. Fifty percent are moderately affected, and 25% not affected at all.

People with respiratory conditions are particularly affected by low concentrations of ions and an imbalanced positive to negative ratio. This may be due to the effect of ionization on bacteria. Negative ions kill and remove from the air microorganisms that cause colds, flu, and other respiratory conditions. Cultures of bacteria in open petri dishes exposed to negative ions fail to grow.[387]

The change in ion concentration created by man changing his environment may be insidious. A story told by Soyka and Edmonds[387] concerns a police control room in England, where workers functioned normally until they were "blessed" with modern technology. As the radio equipment became complex and a computer terminal was installed, tension and fatigue began to develop. The problem was intensified when synthetic fabrics in carpets and other furnishings became part of the room. In addition, the officers wore uniforms of partly synthetic fabrics, and all were issued nylon shirts. In the words of the superintendent, work became "bloody impossible," with fatigue and irritability setting in. It was nearly impossible for anyone to stay awake, let alone be alert.

AK Examination and Treatment

Rhinometry is the measurement of airflow through the nose to determine the effects of obstruction. Surgeons have found that sometimes when what appears to be an obstruction is removed the airflow is not improved, as measured by rhinometry, even though it is visually obvious that the pathway has been cleared. Goodheart[166] quotes from the American College of Ophthalmology and Otolaryngology, "There is a consensus of physiologists that most if not all functions of the organism are on a feedback control by some sort of cerebral mechanism. There has been an increasing amount of direct evidence that the nose is such a control device. Recently there has been some apparently valid direct evidence, both from experimental laboratory and clinic, that strongly supports this. There are many difficulties inherent in understanding the aerodynamics of respiratory nasal flow, and the apparent need to correlate nasal conductance with a pneumonic ventilation. The partial pressure of blood gases that develop in the mano-metric technics tend to support the hypothesis of the nose as such a service mechanism." Goodheart goes on to discuss research of a nasal cycle in which one nostril breathes more than the other, periodically shifting dominant sides. When measuring air movement by rhinometry, it is necessary to measure every twenty minutes for three hours to obtain an average because of the automatic shifting of the dominant breathing side. Goodheart states, "The evidence strongly points in the direction that the turbinates in the nasal chambers are ionizing chambers and that the positive and negative currents that the Eastern and Hindu physiologists proposed are in fact the real basis by which polarity is maintained."

Shannahoff-Khalsa[283] discusses the nasal cycle and research associating hemispheric dominance shown in humans and dolphins, and its apparent relationship with the nasal breathing cycle. He states, "The nasal cycle may be a window on one of the body's most important rhythms." The cycle may be linked with such things as the rapid eye movement (REM) and non-REM cycles of sleep. Right nostril/left hemisphere dominance corresponds to phases of increased activity, and left nostril/right hemisphere dominance represents rest phases. "If you want to alter the unwanted state," Shannahoff-Khalsa said, "just breath through the more congested nostril." "The evidence for lateralization of emotions suggests that feelings are subject to alteration by appropriate breathing exercises."

The nasal cycles relating with positive and negative ions correlate with the polarity of the anterior of the body as described by Davis and Rawls,[105] i.e., the right side of the anterior body is positive and the left side negative. Goodheart attributes inhalation through the right nostril to increasing positive ions, and exhalation through the left nostril to reducing negative ions in the body. Inspiration through the left nostril increases

negative ions, and expiration through the right nostril decreases positive ions. Before examining for the need of ionization treatment, examine for cranial faults and correct if necessary.

Therapy localization indicates the nasal cycle needed by the body. Begin with a muscle that tests strong in the clear but weakens with therapy localization to one of the five factors of the IVF. The positive therapy localization will be with the palmar or dorsal surface of the patient's hand touching the point. When ionization treatment is needed, a specific pattern to the inspiration will cancel the positive therapy localization. If the positive therapy localization is palm toward the skin, inhalation through the right nostril or exhalation through the left will cancel the positive therapy localization. If the positive therapy localization is dorsal surface of the hand toward the skin, inhalation through the left nostril and exhalation through the right one will cancel the positive therapy localization.

Evidence that one-sided nasal breathing is of an electromagnetic nature and not simply an exchange of oxygen, carbon dioxide, or other gas is provided by blocking the nostril with lead instead of a finger. It has been demonstrated that lead blocks the apparent electromagnetic energy of the meridian system. It also changes the results of breathing through one side of the nose. The strengthening of a muscle by one-sided nasal breathing, as indicated above, does not occur when the other nostril is blocked by lead.

Ionization evaluation is not the same as the breathing pattern that indicates a universal cranial fault. In that procedure, one causes an indicator muscle to weaken by breathing through one nostril. A muscle associated with the fault will strengthen when breathing through one nostril or the other. The TL to weaken a strong muscle, as described with the ionization technique, is not part of the universal cranial fault diagnosis. A patient may need ion balance, correction of a universal cranial fault, or both; they are independent conditions. Confirm the universal cranial fault with challenge.

Positive therapy localization should correlate between the anterior and posterior of the body, e.g., if the TL is palm toward the skin on the front of the body, it should also be palm toward the skin on the back. Failure to correlate between the front and back of the body indicates an anterior to posterior disorganization of the body's electrical balance. This appears to relate with balance between the conception and governing vessels. Simultaneous vigorous stimulation to the tip of the coccyx (GV 1) and the umbilicus (CV 8) for twenty to thirty seconds will balance the pattern so that TL will correlate from front to back.

A simple method that appears to change the body's relation to air ionization is to breathe in through the nostril with deficient ions and out through the nostril with excessive ions. For example, if dorsal hand positive therapy localization is canceled by inhalation through the left nostril, it indicates that there is a deficiency of negative ions. One would then breathe in through the left nostril and out through the right one repeatedly. This will often make a person feel better who is sluggish from riding in an automobile or being in a positive ion environment for some other reason. One must remember, however, that the ion balance varies greatly from place to place; without testing, one is not certain what type of breathing is needed to improve function. The first method used in applied kinesiology to influence ionization was to examine the patient as indicated and have him breathe appropriately for two minutes at the beginning and ending of the day.

A later applied kinesiology ionization technique was developed by Goodheart,[172] who extends the body's polarities described by Davis and Rawls[105] to the lungs, i.e., the right lung is positive anteriorly and negative posteriorly, and the left lung is negative anteriorly and positive posteriorly. When there is an operating room explosion from an electrical ground failure, any inflammable anesthesia in the patient's lungs is susceptible to explosion. When this happens, it is usually only one lung and then only part of it. Goodheart attributes the localization to the electromagnetic aspects of the lungs.

In addition to the nasal chambers being ionization chambers, with positive ions created in the right nasal chamber and negative ones in the left, Goodheart[172] now proposes that the tracheal rings act as an atomic accelerator to increase the relative charge of the air molecules. Oxygen has a negative charge and is attracted more to the positively charged anterior right lung and posterior left lung.

Following is an examination and therapeutic procedure to improve function in those highly or moderately susceptible to ion imbalance. Its effectiveness is often indicated by an increase in the patient's vital capacity. First, measure the patient's vital capacity three times and average for comparison after treatment. Since it will be necessary for the patient to hold one side of the nose closed during the following test, be certain there is no positive therapy localization from touching the finger to the nose. If there is, determine why and correct before continuing.

There are three steps to determine treatment, then five more steps to apply it and re-evaluate the patient.

1. Determine whether the patient's pattern is too many positive or negative ions by testing both nostrils. First have the patient inhale through the right nostril, with the left side held closed by a finger. Test a strong indicator muscle for weakening. Repeat on the left side. Only one side should test positive. If inhaling through both sides weakens the indicator muscle, re-evaluate and correct the cranium for bilateral expiration assist faults. If the patient weakens on the right nasal test, there are too many positive ions; if on the

left, there are too many negative ions. If there is no weakening, the patient does not need this treatment.
2. Re-test the nasal side that was positive in step 1 with right (humming) and left (multiplication tables) brain activity to determine which cancels the nasal inhalation test. The patient can say the multiplication tables to himself while inspiring through the nose.
3. Re-test the nasal side that was positive in step 1 with vitamin B complex and vitamin G complex. One or both will cancel the positive nasal inhalation test.
4. Physical treatment is tapping on the thoracic cage while the patient activates the right or left brain. The location of tapping and brain activity is determined in steps 1 and 2. If right nasal inhalation is positive, tap the left anterior and right posterior thoracic cage. If left nasal inhalation is positive, tap the right anterior and left posterior thoracic cage. A solid, non-bruising tapping is done twenty or thirty times while the patient activates the brain side that canceled the nasal inhalation test.
5. Have the patient chew and ingest the vitamin B or G — or both — that canceled the positive inhalation test. This empirically appears to help support the oxygen-carbon dioxide control by the nucleus of tractus solitarius, which also innervates the taste fibers of cranial nerves VII, IX, and X. It appears that sometimes the oxygen-carbon dioxide set-point is set too low.
6. Poor lymphatic system drainage is often associated with lung problems. Test for and correct, if necessary, the lymphatic system retrograde lymphatic and right lymphatic duct dysfunction (see page 539).
7. Re-test the patient with step 1 to determine that there is no nasal inspiration weakening.
8. Have the patient again place vitamin B or G — or both — on his tongue and re-test the vital capacity three times; average the three. An increase of 20% is common. The improvement in vital capacity is best seen when the vitamin(s) are on the tongue, indicating need for further supplementation. Have the patient take vitamin B or G — or both — as indicated in test #3 for about three weeks. Recommend high natural vitamin B complex foods.

Ion Nutrition

An additional support to ionization treatment has been described by Deal.[107] There are four classifications of nutrition that help correct an excess or deficiency of positive or negative ions. It is first necessary to determine whether the individual has an excess or deficiency of negative or positive ions. This is done by four individual tests with a strong indicator muscle weakening as follows.

Right inhalation = excessive positive ions.
Left exhalation = deficient negative ions.
Left inhalation = excessive negative ions.
Right exhalation = deficient positive ions.

There is more than one type of nutrition listed in each category; generally any one is applicable. The appropriate nutrition will cancel the positive breathing test when it is chewed. The nutrition Deal has established as canceling the positive nasal test is indicated in the chart below.

Nasal Ionization and Mineral Balance

Condition	Indicator muscle weakens with patient's nasal respiration	Corrected by
Excessive negative ions	Left nostril in	**Positive calcium** Calcium oxide Calcium carbonate Calcium gluconate
Deficient positive ions	Right nostril out	**Negative calcium** Calcium lactate Di calcium phosphate
Excessive positive ions	Right nostril in	**Positive potassium** Potassium oxide Potassium carbonate Potassium gluconate
Deficient negative ions	Left nostril out	**Negative potassium** Potassium citrate Potassium aspartate

12—45.

As indicated in the introduction to this section, the relationship of ionization to one's health and emotions is difficult to relate with because the concentration and balance of ions are not recognized by our five senses. If it is difficult for us to understand, consider an individual who has major sensitivity to fluctuation of ion balance. Soyka, the major author of the book, *The Ion Effect*,[387] lived for years with health problems that developed when he moved to Switzerland and came under the influence of the Foehn wind. Only after several years of slowly putting the puzzle together piece-by-piece did he understand the cause of his problems. Medically the treatment consists primarily of serotonin blockers and the use of negative ion generators. These generators are available as desk-top units; there are battery-operated ones for automobiles. The applied kinesiology approach is particularly applicable to that 25% who are more susceptible to the ion effect than others. There may be remarkable success with patients who are susceptible to weather changes just by correcting ionization. They may be particularly depressed or elated at the beginning of a storm, be "weather forecasters" by their symptoms, or in some other way show a response to the weather.

Chapter 12

Reticular Activating System

The reticular activating system (RAS) is located in the medulla, pons, and midbrain as poorly defined nuclei connected by a chain of multisynaptic neurons. This network sends sensory stimuli to the thalamus, which acts as a relay center. There are some projections directly to the cerebrum without first synapsing in the dorsal thalamus. A more accurate phrase describing these systems of recent use is *extrathalamic cortical modulatory systems.*[57]

The reticular activating system has been thought to be concerned with degrees of conscious alertness, as well as with sleep. Recent information has revealed there is no adequate explanation for the sleep-wakefulness cycle.[187] In this simplified discussion of possibilities we can, as Guyton and Hall say, "... let our imaginations go wild...." This is not to say that these are the explanations of Guyton and Hall; rather, some of the accumulated thoughts about the arousal system are combined with Goodheart's observations.[174,175]

The reticular formation in the brainstem receives tremendous numbers of signals directly or indirectly from the spinoreticular, spinothalamic, spinotectal, auditory, and visual tracts, as well as others receiving sensory stimuli from almost all areas of the body. In response, the reticular formation can transmit signals both upward into the brain and downward into the spinal cord[187]; thus, almost any type of sensory signal can immediately activate the system. "For instance, proprioceptive signals from the joints and muscles, pain impulses from the skin, visual signals from the eyes, auditory signals from the ears, or even visceral sensations from the gut can all cause sudden activation of the reticular activating system and therefore arouse the animal. This is called the *arousal reaction.*"[186]

In a series of serendipitous patient observations Goodheart hypothesized that under some circumstances an organ(s) or gland(s) can be "asleep"; that is, the nervous system is not completely aware of all of the body's needs. Under these circumstances therapy localization is not positive when there is dysfunction of a gland or organ. The RAS technique appears to reveal these exceptions. The reticular activating system technique developed by Goodheart[174,175] is the application and/or modification of many examination and therapeutic approaches previously used in applied kinesiology, with a specific protocol of examination and treatment.

Incorporated into the reticular activating system technique is examination for specific organ and/or gland dysfunction and pain patterns. The examination and treatment include many observations previously made in applied kinesiology and other research. Included are origin-insertion technique, diagnosis by TS line, eyes open or closed, referred pain patterns,[84,85,86,137,198,214,229,230] spondylogenic reflex by Dvorak and Dvorak,[127] organ manipulation,[22] nutrition, acupuncture head points, and pulse point diagnosis. There are specific patterns used in diagnosing and integrating these items with organ or gland function and association with the spine from T1 through L5.

Sleep is characterized by different electroencephalographic (ECG) patterns. The patterns of slow wave sleep, often called dreamless sleep, are (1) very light sleep, characterized by very low voltage periodically broken by short bursts of alpha waves called sleep spindles, (2 & 3) quiet restfulness with progressively slower ECG frequency until stage (4) where the change is to deep, slow wave sleep with high-voltage delta waves appearing at a rate of 1-3 per second.

The reticular activating system receives input from widespread sensory nerves throughout the body, as well as from the cerebral cortex. Nerve fibers from a thin sheet of nuclei in the pons and medulla spread widely in the reticular formation and upward into the thalamus, hypothalamus, and most areas of the limbic cortex. "In addition, they extend downward into the spinal cord, terminating in the posterior horns where they can inhibit incoming pain signals...."[186]

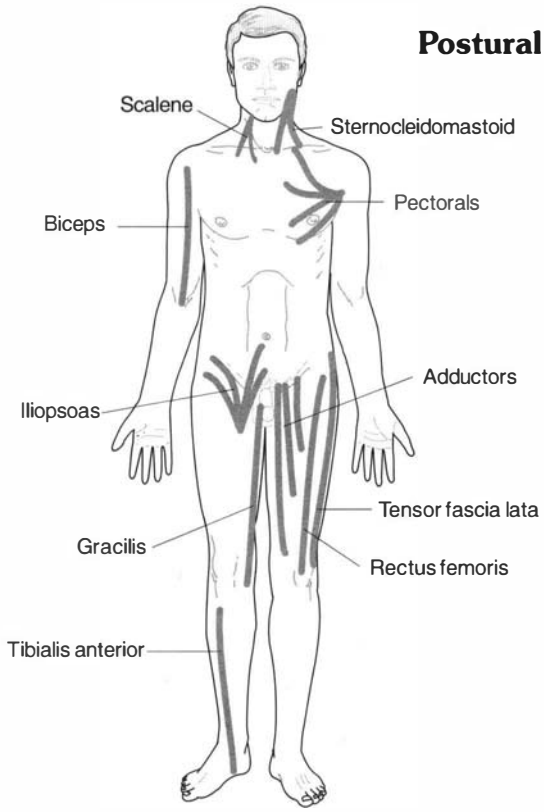
Goodheart[174] postulates that there are organ-muscle-vertebral level systems that are "asleep" or not fully in a state of neurological arousal when the person is awake. It is as if they are "out of the loop" and not activated fully by the reticular activating system; therefore the body is not aware of problems in these circuits and does not correct them.

Presented first is a brief discussion of various aspects of the examination process. In the following section, "Examination and Treatment Protocol," the sequential steps are presented.

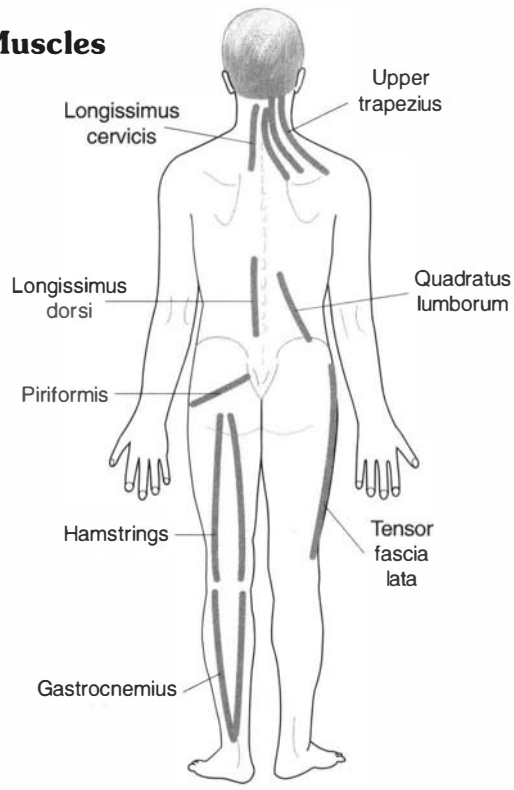
Examination Overview

Postural and Phasic Muscles: Occasionally during sleep one moves into paradoxical sleep or REM sleep. The amount of REM sleep will vary with the person's level of fatigue. With extreme fatigue, the duration of paradoxical sleep is very short; otherwise, it tends to last 5 to 20 minutes. Paradoxical sleep is characterized by deep sleep and active dreaming, during which it is more difficult to be awakened. Muscle tone is deeply depressed, most specifically noted in the postural muscles. Although there is generally muscle inhibition, some muscles have rapid regular activity, specifically the extraocular muscles moving the eyes causing rapid eye movements (REM), providing the alternate name for paradoxical sleep.

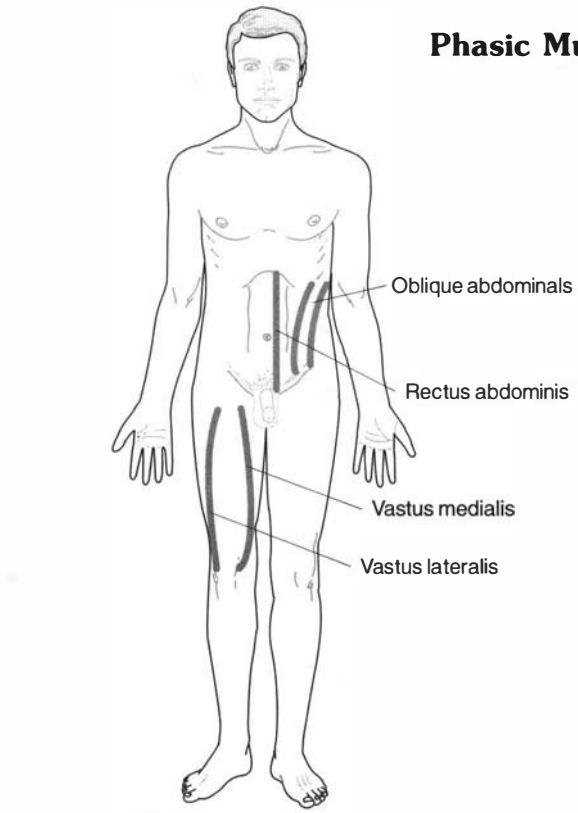
Jacobs et al.[216] describe the change in serotonin during the stages of sleep. The typical serotonin neuron discharges at about 3 spikes per second when an animal is quiet but awake with drowsiness. As the animal enters into slow wave sleep, the number of spikes



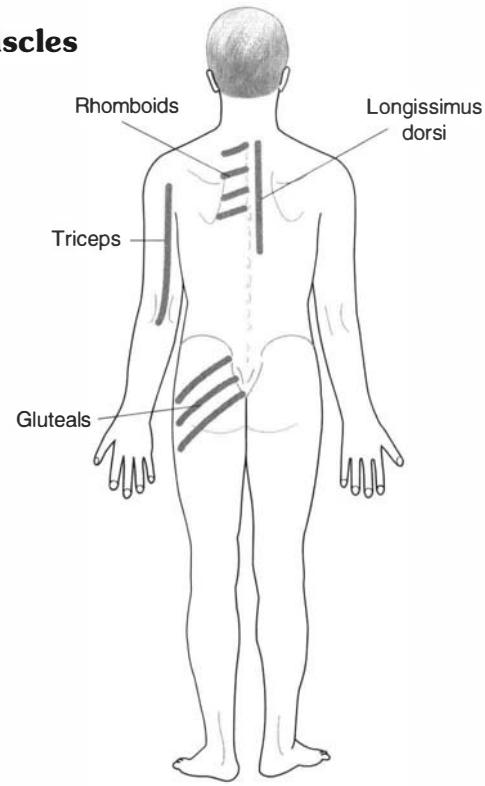
12—46. Anterior postural muscles.



12—47. Posterior postural muscles.



12—48. Anterior phasic muscles.



12—49. Posterior phasic muscles.

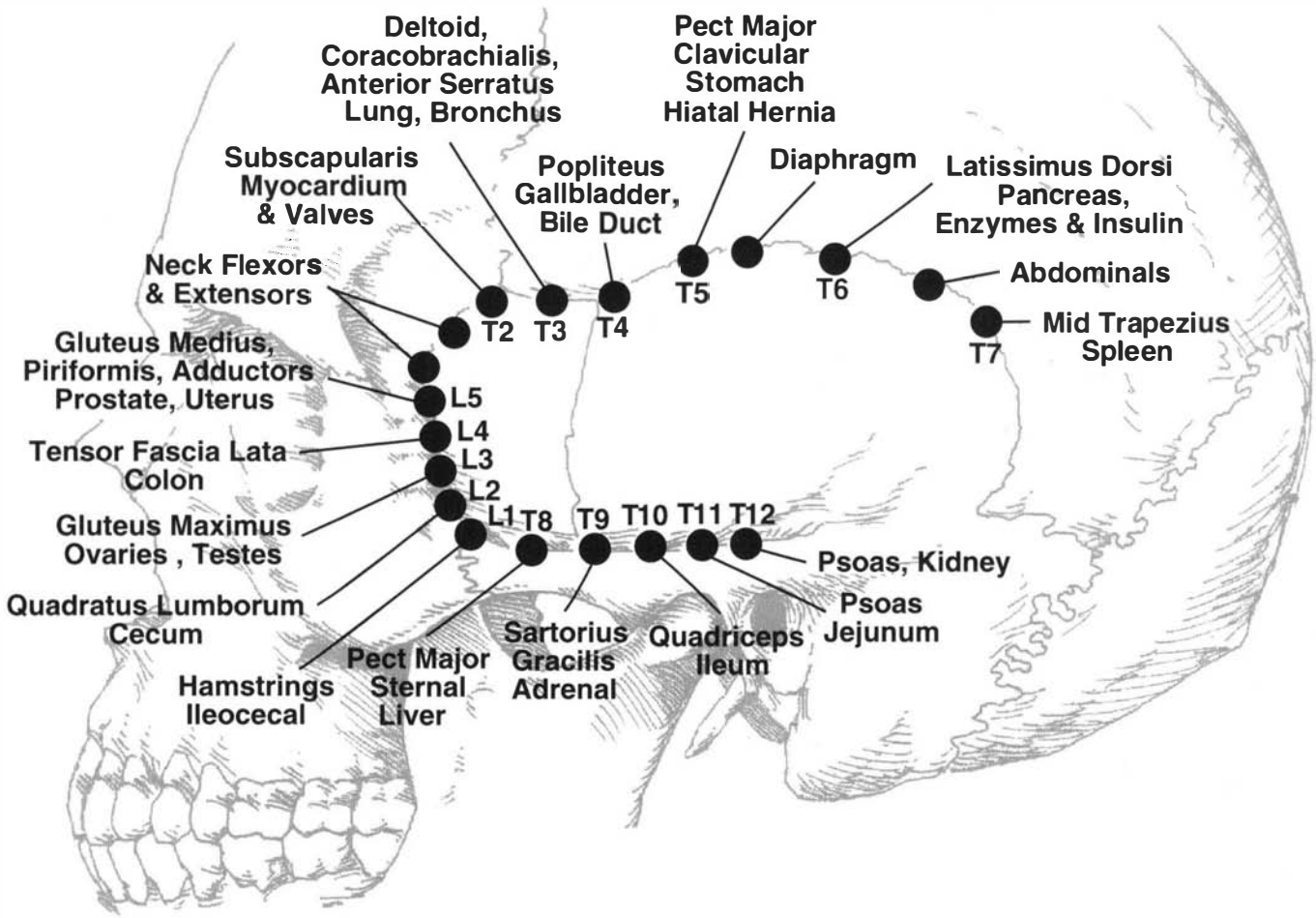
Redrawn after Dvorak and Dvorak, Medical Checklists, *Thieme Medical Publishers © (1991).*

gradually declines. During REM sleep serotonin neurons fall completely silent. This relates with the antigravity muscle paralysis that is achieved by inhibiting the neurons that control the tone of the body's antigravity muscles.

In the RAS technique therapy localization is evaluated by testing the postural muscles. In an active condition they will most often reveal positive TL when phasic muscles will not. This is believed to be associated with the depressed state of the postural muscles in REM sleep.

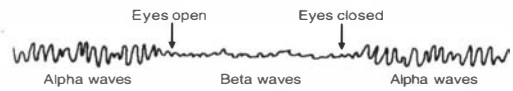
TS Line: An active TS line point associated with the RAS technique is more subtle to palpation than what has been observed in previous TS line evaluation. It was first noted that a TS line point might have additional characteristics with the repeated muscle activation patient induced (RMAPI) technique. In this condition there is no indication of a positive TS line until the patient repeatedly activates the muscle, after which the TS line point is active for about sixty seconds and then disappears. This experience resulted in an effort to search for additional ways the TS line points might reveal activity. In RAS involvement, TS line palpation must be done very lightly but sensitively. When a TS line point is overt, it indicates one or all of the five factors of the IVF are involved. The more subtle point is associated with the RAS technique. The positive subtle TS line point can be confirmed by cross-hand therapy localization, i.e., with a finger of the right hand on the left TS line point or the left hand on the right TS line point, causing a strong indicator muscle to weaken. TS line points associated with RAS technique do not have positive therapy localization with the ipsilateral hand doing the contact.

The more common TS line points in association with the RAS technique are L5, L1, T8, L3, T12, and T7, in descending order. There will be positive therapy localization to the associated vertebra but only with the eyes closed. In areas where the patient can't reach the vertebra for therapy localization vertebral challenge can be done, but likewise a positive challenge will only be present with the patient's eyes closed.



12–50. TS line with muscle, organ, and vertebral level association.

Eyes Open or Closed: The ECG alpha waves appear when normal adults are in a quiet, resting state of cerebration. When the person's attention is aroused to increase cerebration the alpha waves are replaced with asynchronous, low-voltage beta waves that are present with extra activation of the central nervous system or during tension. In some cases, positive therapy localization will not be present until the patient closes his eyes.



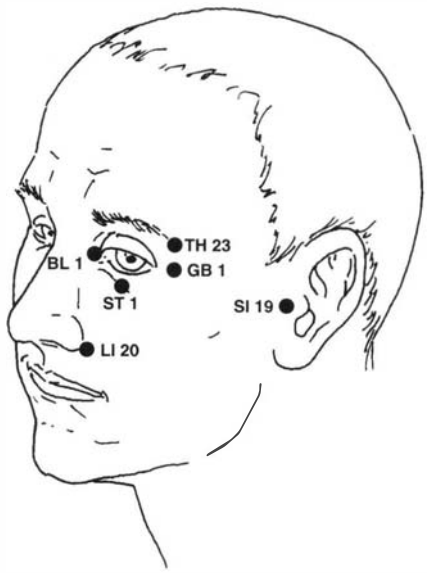
12—51. Change from resting state to more active cerebration with eye opening. After Guyton and Hall.[187]

Therapy Localization: Some of the examination points on the body for the RAS technique are not positive with the usual form of therapy localization, i.e., placing the fingertips over the area to be evaluated. The fingers of the two hands must be interlaced, and then the two hands placed over the point being therapy localized. This probably correlates with the TS line not revealing positive therapy localization except with the contralateral hand doing the therapy localization. It may be due to a requirement of accessing both sides of the nervous system in the evaluation process.

12—52. Interlaced fingers for therapy localization.

Acupuncture Head Points: Acupuncture head points are the end points of the Yang meridians on the head. These points will often, but not always, be associated with the RAS technique. A positive finding is observed by interlaced finger therapy localization to the head points. The interlaced finger therapy localization may correlate with the TS line requiring cross-hand therapy localization to simultaneously access both sides of the nervous system.

When the acupuncture head point is positive, the tonification point of that meridian is treated by tapping. So far in the development of this technique it does not appear that tapping the head point has any effect, nor does tapping any other meridian point in this application.



12—53. Yang meridian head points.

General Consideration: There are specific organ or gland associations with each vertebral level from T1 to L5 in the RAS technique. Along with the organ or gland dysfunction, symptomatic patterns are associated with the vertebral level. These associations were developed by Goodheart by correlating referred reflexes, symptoms, and pain observed and charted by others. He reports on studying the reflex work of Seiffer, published as early as 1900, Reese of TS line notoriety, Kellgren,[229,230] Dvorak and Dvorak,[127] and others.[174,175] Their work represents reflex areas on the body, many of which are associated with visceral disorders. Most of these locations on the body have been previously associated with neurolymphatic massage, chiropractic manipulative reflex techniques, spondylogenic reflexes, areas of referred pain, and others. The active areas can be identified by tenderness, tissue tension, and congestion. The patterns are presented in spinal level figures 12—54-104.

The first step is a general consideration of the patient's health problem, symptomatic pattern, and body language; these guide one to consider evaluating the patient with the RAS technique. The patient's pattern will usually have several factors that will be in the vertebral level pattern. Some of the conditions that indicate RAS technique examination are:

- Visceral pain
- Digestive problems
- Disturbed sleep patterns
- Quality of life alterations
- Menstrual problems
- Pancreatic problems
- Mental acuity

Organ/Gland Association: There will be positive interlaced finger therapy localization over the gland or organ or its abdominal reflex area. The abdominal reflex area is the associated vertebral level noted in the accompanying charts by a circle containing the vertebral level. The organ/gland and/or abdominal reflex area is evaluated with the interlaced finger technique.

Positive therapy localization will usually not be present with single-hand TL. Interlaced finger therapy localization and other factors of the RAS technique are only evaluated with anti-gravity postural muscles noted in the chart on page 591 (figures 12—46 and 12-—47).

Test for nutritional need by placing the nutrient indicated with the vertebral level illustrations on the patient's tongue to determine if it eliminates the positive interlaced therapy localization. Do not have the patient chew the nutrient at this time, because it might eliminate some positive findings as you progress in your investigation.

The viscera is treated by lifting it in a cephalad direction while actively testing an anti-gravity muscle. The muscle should not test weak; if it does it might be necessary to have the patient cough three or four times while lifting the viscera to help loosen adhesions.

Examination and Treatment Protocol

Before evaluating the patient for RAS technique, examine and treat him with the usual methods previously discussed in this text. When testing for RAS, routinely use postural muscles for testing as diagrammed in figures 12—46 and 12-—47.

1. From the patient's history, symptoms, and standard examination findings form an opinion about visceral involvement. Although the meridian system is not always involved in the RAS technique, pulse, alarm, and acupuncture head point diagnosis can help determine the pattern of involvement. These diagnostic points may only be positive with the patient's eyes closed, and in rare instances may require positioning the patient in gait configurations.

   Evaluate the acupuncture beginning and ending (B&E) Yang head points on the head with interlaced finger therapy localization.

2. Lightly and discriminately palpate the TS line for a subliminal area. Verify that the point is active with cross-hand therapy localization. Single-hand TL will not be positive as with the usual active TS line point.

3. Therapy localize with interlaced fingers over the associated organ and/or abdominal reflex point (vertebral level in small circle on abdomen in illustrations). If TL is positive, have the patient taste the appropriate nutrient to determine if it cancels the interlaced finger therapy localization. This provides information for nutritional therapy recommendation; however, the patient should not ingest the nutrient at this time because it can eliminate positive findings further in the investigation.

4. If B&E points are active, tap the tonification point of the involved meridian. Tapping the beginning or ending point is not effective to clear the total problem.

5. Test the associated muscle with the positive finding in number 3, i.e., pectoralis major (sternal division) for T8 liver, with repeated muscle activation patient induced (RMAPI). It should test positive, correlating with the previous finding. Do not treat; the positive RMAPI should be corrected with the next step.

6. Lift the indicated viscera cephalad with firm pressure while testing a postural muscle that should not weaken. Hold the cephalad pressure for 10-15 seconds. If the postural muscle weakens while holding the viscera cephalad, change the vector of pressure until weakening no longer occurs. Re-test the visceral reflex area, spinal reflex area with interlaced finger therapy localization, and the visceral related muscle with RMAPI. None should be positive; if so, repeat the firm cephalad pressure on the viscera and have the patient cough two to four times to loosen adhesions that may be present.

   For a viscus that is in the thoracic cage lift under the diaphragm. The diaphragm has a mobilizing effect on thoracic cage contents. For more techniques on manipulation of the thoracic cage contents consult *Visceral Manipulation* by Barral and Mercier.[22]

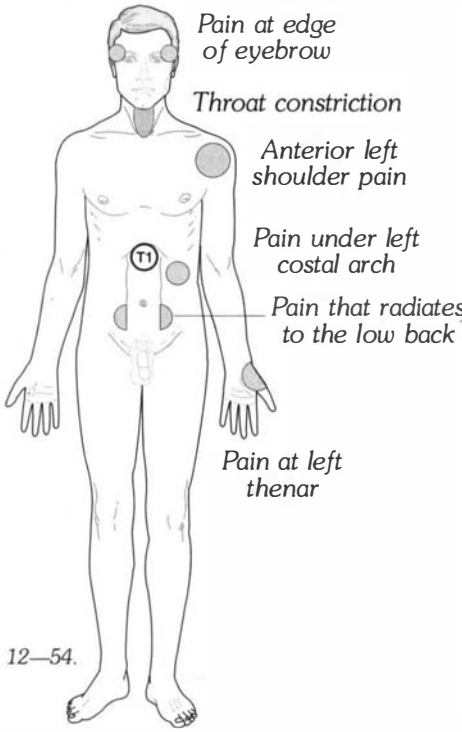
7. If the findings in number 5 return when the patient closes his eyes, have the patient maintain eye closure and contact the TS line point with a firm pressure while lifting the viscera cephalad for 20-30 seconds. In order to maintain firm pressure on the TS line point it is necessary to have the patient contract his cervical muscles to hold his head in place. The previously positive finding with eyes closed should now be negative.

8. Some or all of the pain reflex areas and/or symptoms on the accompanying anterior and posterior drawings will be present. Contact the painful reflex areas and the TS line point simultaneously for 10-15 seconds to relieve the pain at the reflex areas. If pain remains, it is necessary to tap the TS line point while also contacting the reflex pain area.

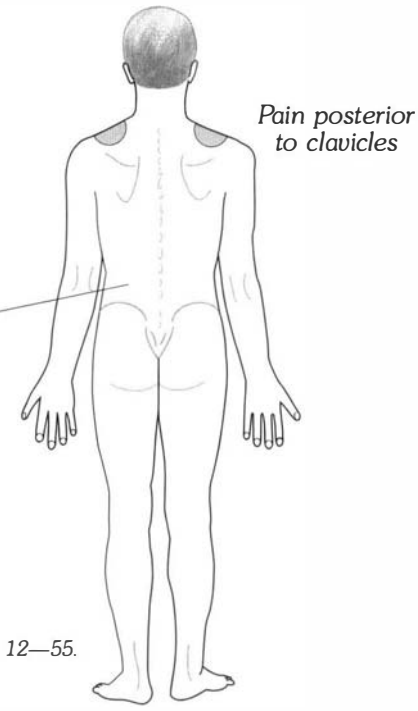
9. TL and challenge the indicated spinal level for both the usual subluxation and for a holographic subluxation. Most likely the spinal level will be negative with the patient's eyes open and positive with the eyes closed. Adjust as indicated by challenge.

10. The muscles shaded gray on the accompanying spinal level Dvorak and Dvorak illustration may be tender to digital pressure. (If you cannot identify the muscles from the illustrations, consult figures 3—93-119 in Chapter 3.) Tap the Lovett reactor vertebra to the spondylogenic vertebra, i.e., for Dvorak and Dvorak L5 tap C1 at about 1 tap per second (1Hz) for 60 seconds. Reassess the tenderness of the spondylogenic muscles and the organ, vertebral, and pain reflexes. If present, repeat the TS line tap with cephalad organ traction and Lovett reactor tap.

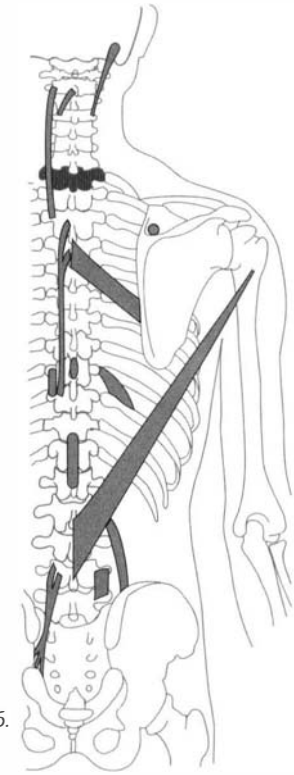
T1 — *T10* — Heart



12—54.



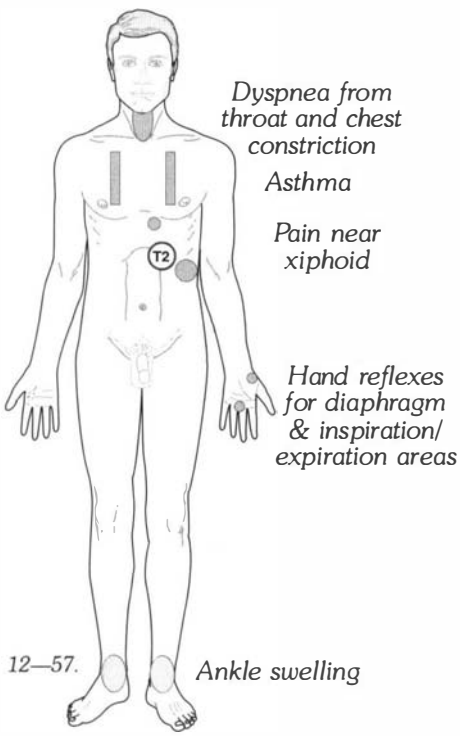
12—55.



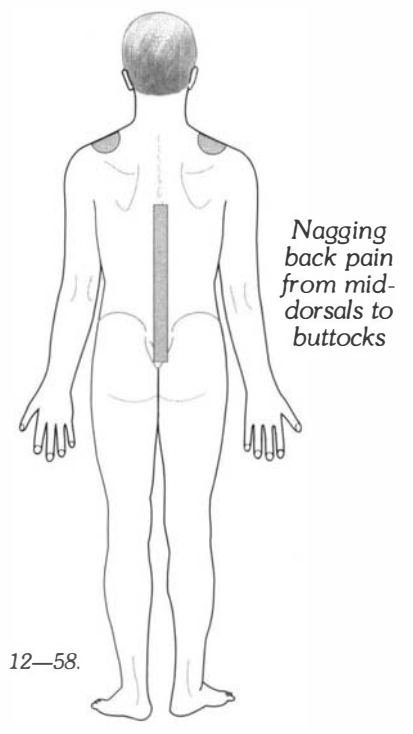
12—56.

Nutrition: Lipotrophic B factor (G), phospholipid from beef chromatin (E_2), heart concentrate or nucleoprotein extract, cardiac nutritional complex.

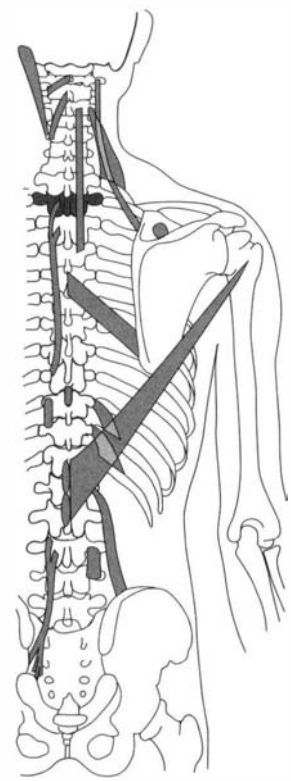
T2 — *T9* — Myocardial / Valves



12—57.



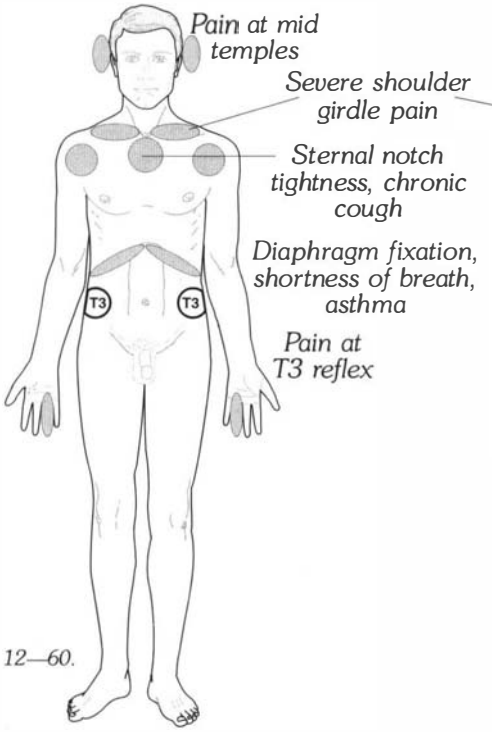
12—58.



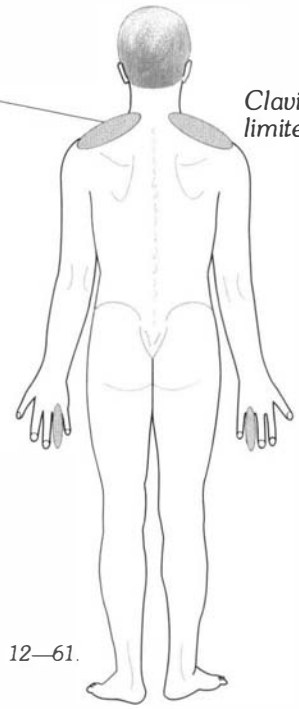
12—59.

In addition to reticular activating system technique, evaluate patient with the diaphragm technique.
Nutrition: Vitamins B and E, heart concentrate or nucleoprotein extract.

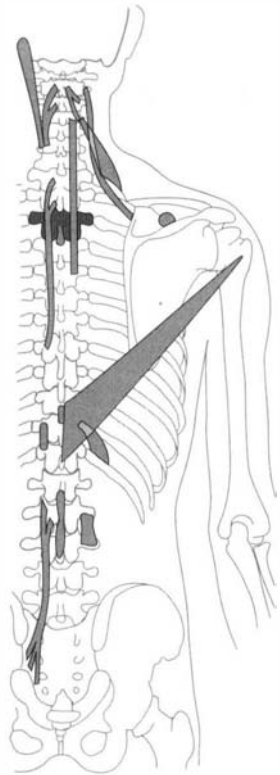
T3 — *T8* — Lungs / Bronchi



12—60.



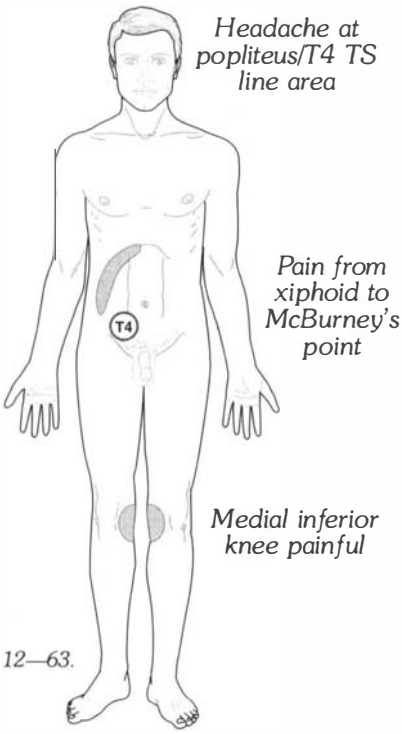
12—61.



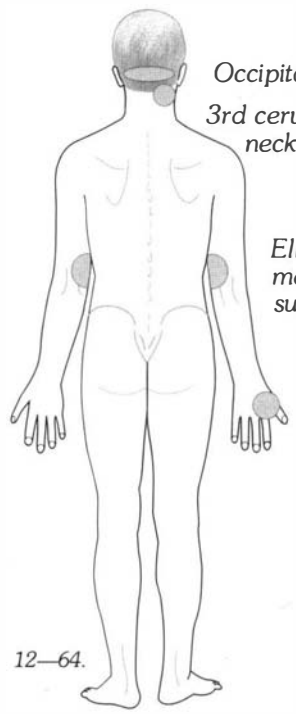
12—62.

Nutrition Considerations: Lung concentrate or nucleoprotein extract, allergy complex.

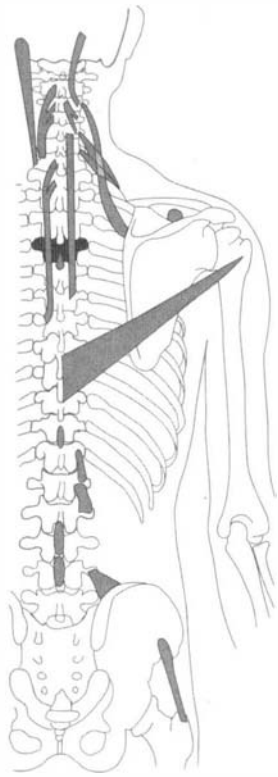
T4 — *T7* — Gallbladder / Bile Duct



12—63.



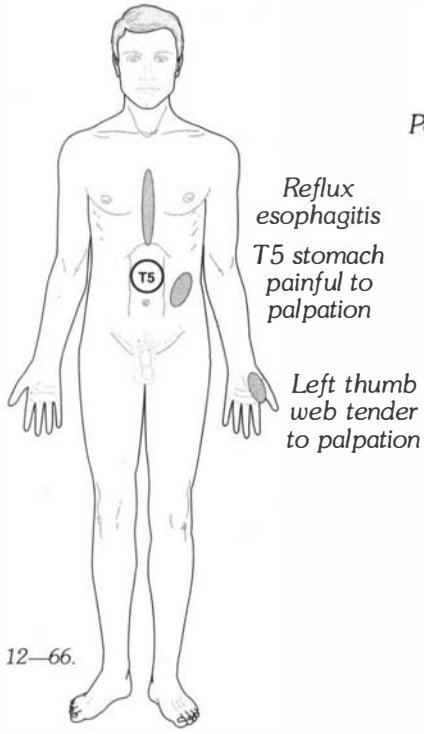
12—64.



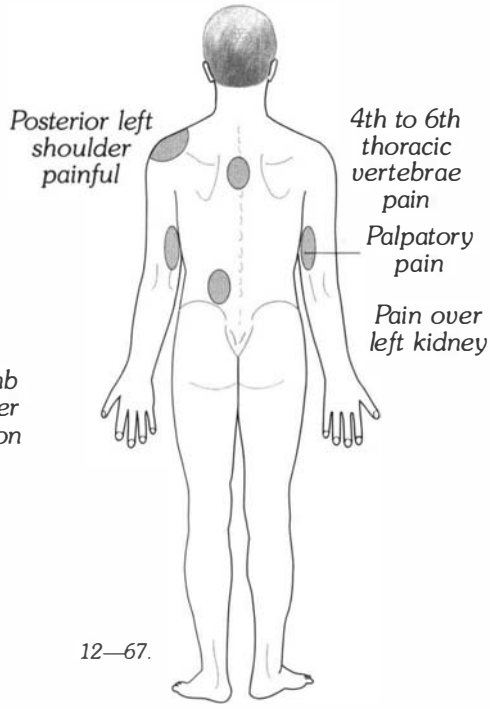
12—65.

Nutrition Considerations: Vitamins A and F, beet greens juice, unsaturated fatty acids (F), water soluble vitamin A up to 6 per day.

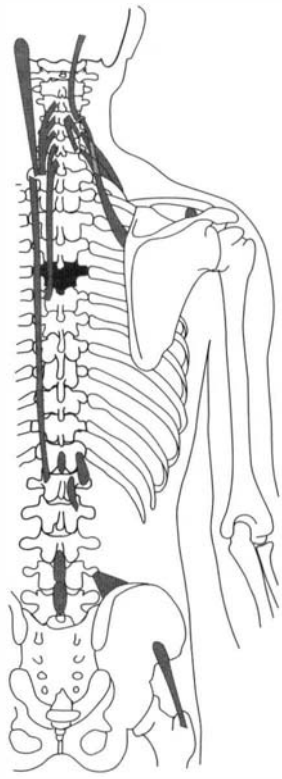
T5 — *T6* — Stomach



12—66.



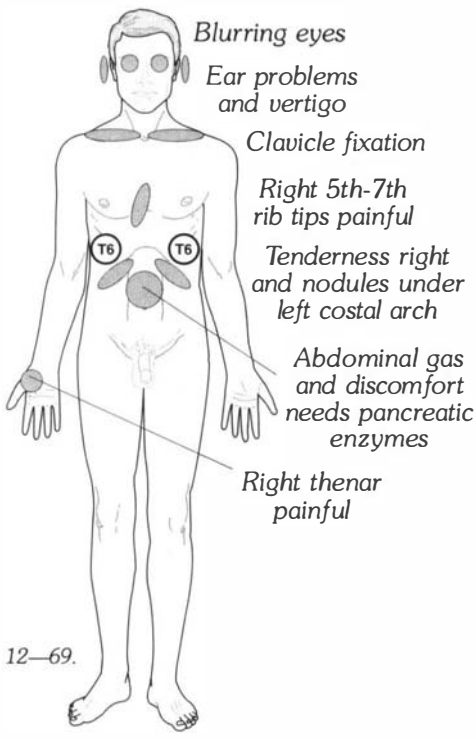
12—67.



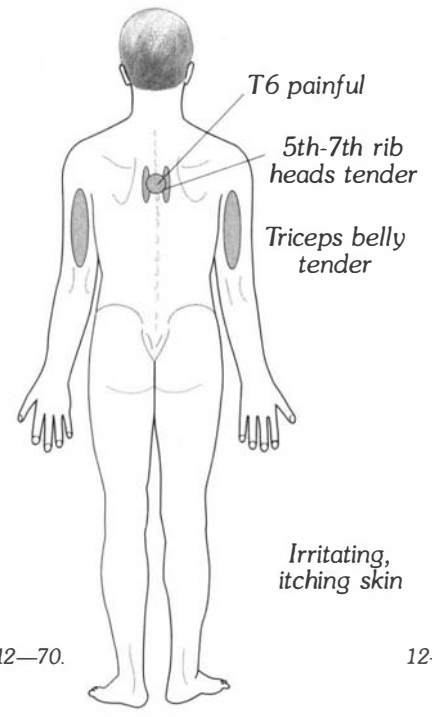
12—68.

Nutrition Considerations: Lipotrophic B factors (G), vitamin B complex, concentrate or nucleoprotein extract of pituitary, nerve, and/or thymus, chlorophyll-aqueous solution.

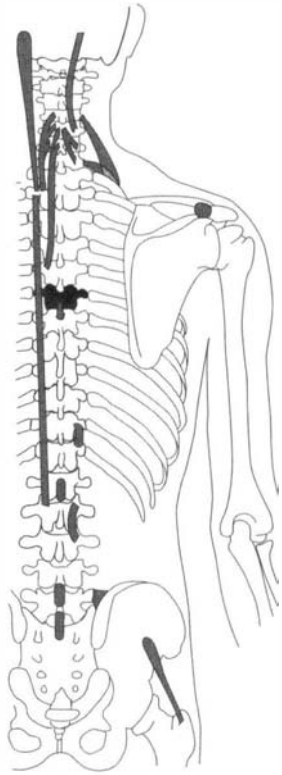
T6 — *T5* — Pancreas (enzymes)



12—69.



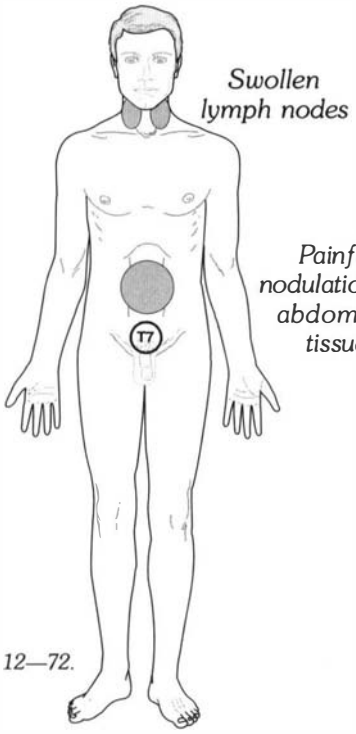
12—70.



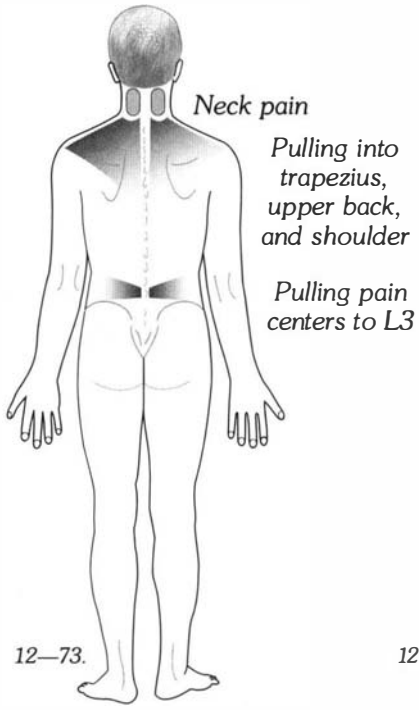
12—71.

Nutrition Considerations: Hypoglycemia: vitamin A, unsaturated fatty acid, beet greens juice; hyperglycemia: muscle concentrate or nucleoprotein extract, parasympathetic support; for pancreatic enzyme deficiency use HCl, pancreatic enzymes, muscle concentrate or nucleoprotein extract (may need to be taken frequently).

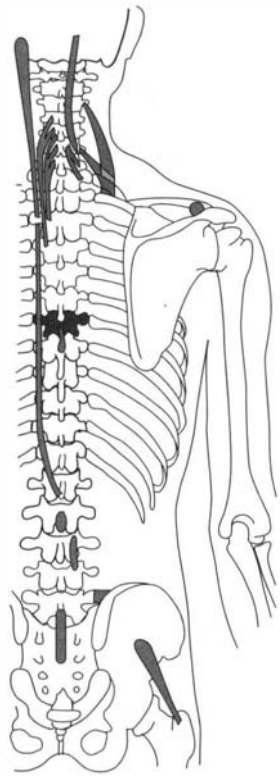
T7 — *T4* — Spleen



12—72.



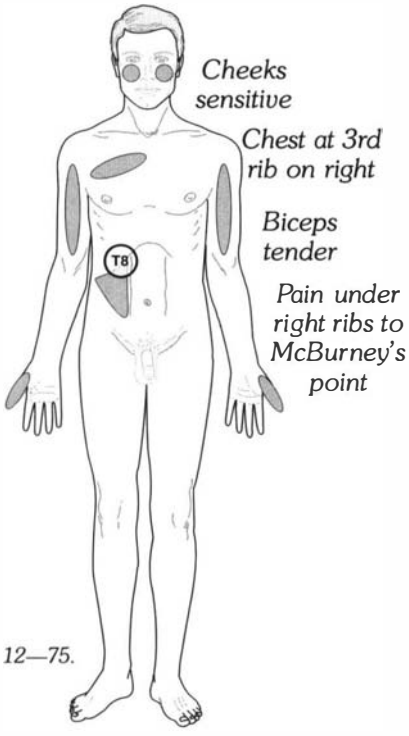
12—73.



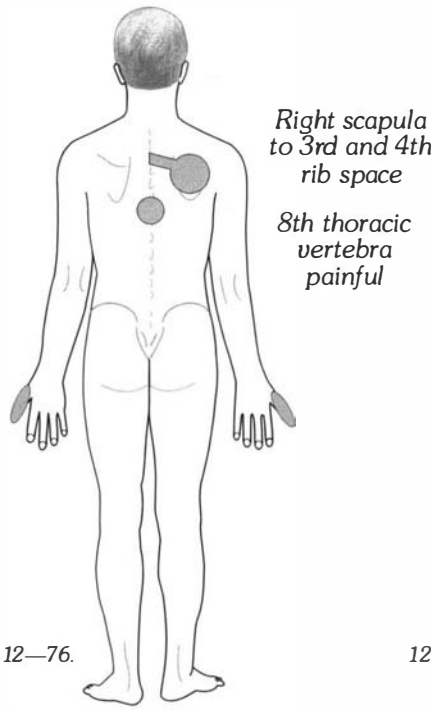
12—74.

Nutrition Considerations: Vitamin C (4,000 mg synthetic plus 24 mg of C complex), thymus, spleen concentrate or nucleoprotein extract, immune system complex.

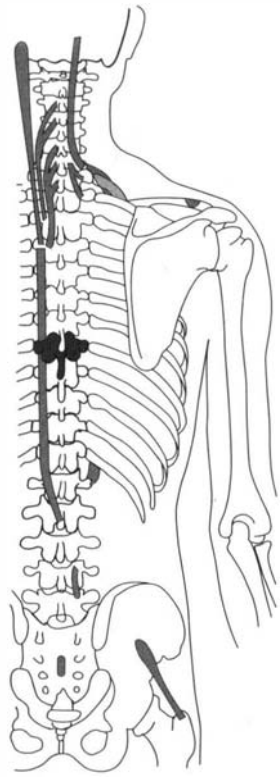
T8 — *T3* — Liver



12—75.



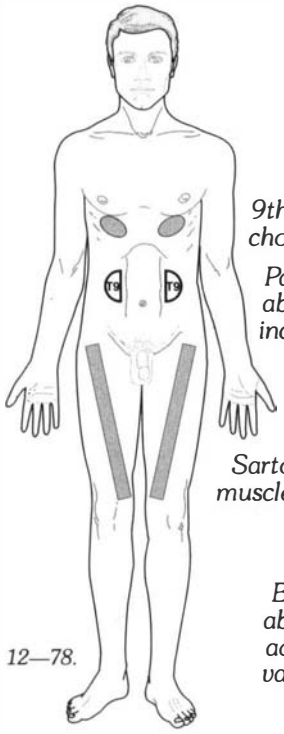
12—76.



12—77.

Nutrition Considerations: Water soluble vitamin A — may require high dosage, liver concentrate or nucleoprotein extract if there is evidence of portal collateral circulation, e.g. hemorrhoids, congested visible chest veins, reflux esophagitis.

T9 — *T2* — Adrenal



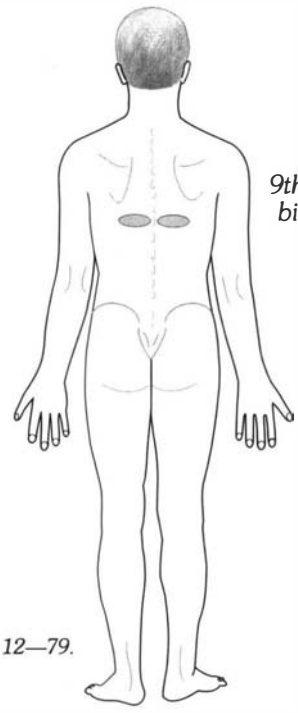
9th rib costo-chondral pain

Palpatory pain above and two inches lateral to umbilicus

Sartorius muscle pain

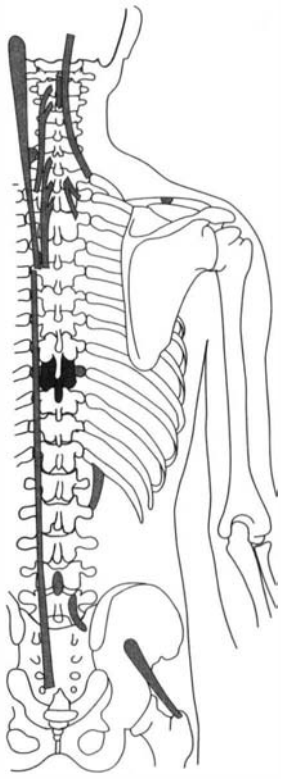
Blood pressure abnormalities by adrenal medulla vasomotor nerve control

12—78.



9th rib painful bilaterally or singly

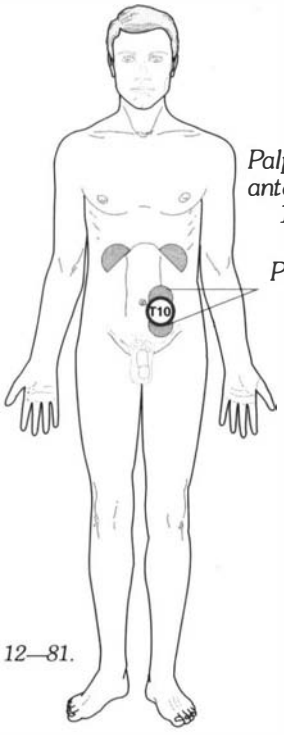
12—79.



12—80.

Nutrition Considerations: Adrenal concentrate or nucleoprotein extract, chlorophyll, allergy complex.

T10 — *T1* — Ileum

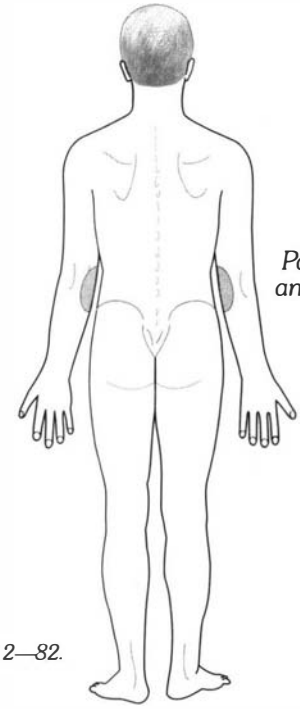


Palpatory pain anterior tips of 10th ribs

Palpatory pain

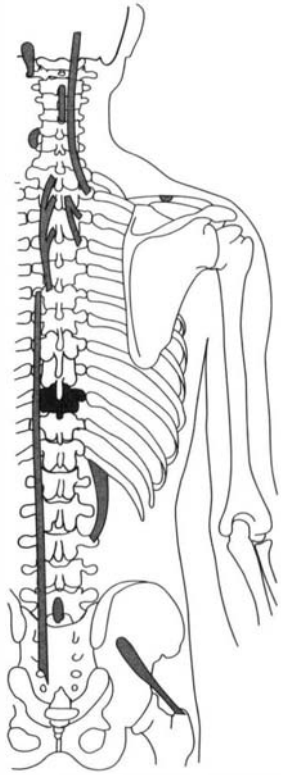
Jejunum and ileum reflex

12—81.



Pain medial and distal to elbow

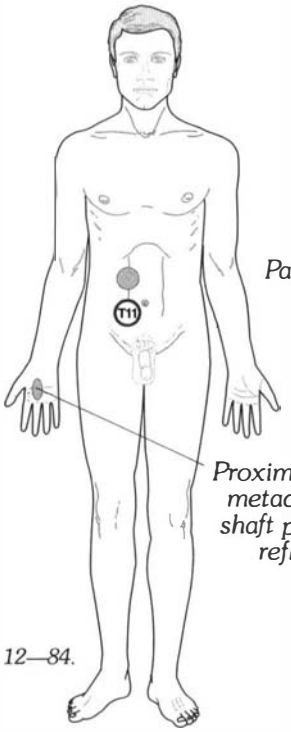
12—82.



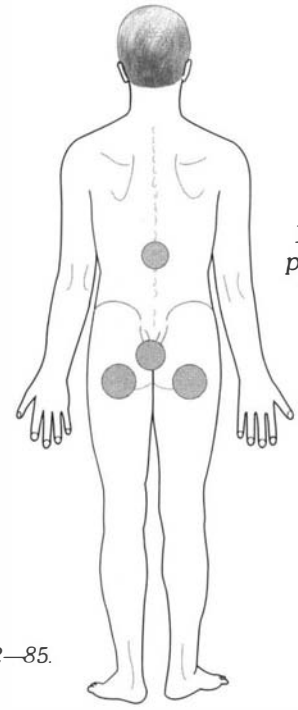
12—83.

Nutrition Considerations: Choline bitartrate promotes capillary circulation and osmotic transfer in intestinal walls, vitamin B_4 anti-spasmodic effect on intestinal walls (Adenylic Acid-Adenine), enzymes with bentonite, bowel detoxifier, concentrate or nucleoprotein extract of pituitary, thymus, and/or spleen.

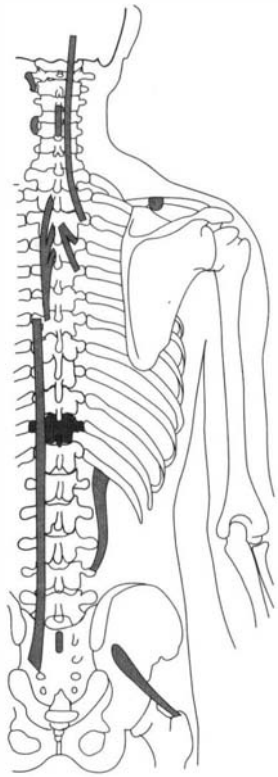
T11 — C7 — Duodenum / Jejunum



12—84.



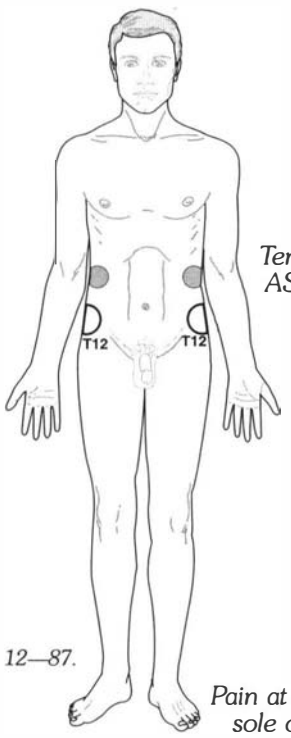
12—85.



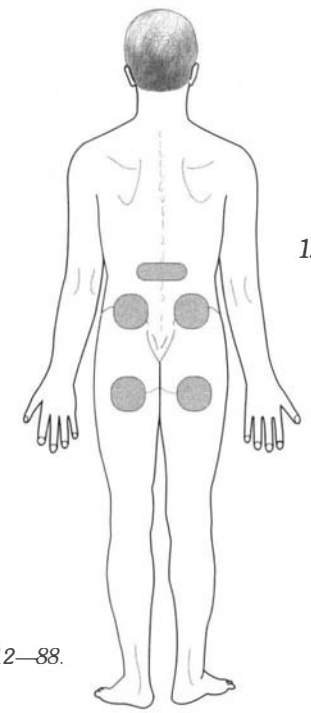
12—86.

Nutrition Considerations: Bile salts, collinsonia root, montmorillonite, digestive enzymes, complex to sooth irritated bowel, chlorophyll perles, pituitary concentrate or nucleoprotein extract.

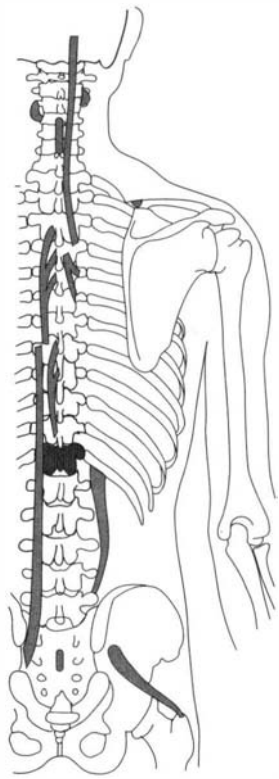
T12 — C6 — Kidney



12—87.



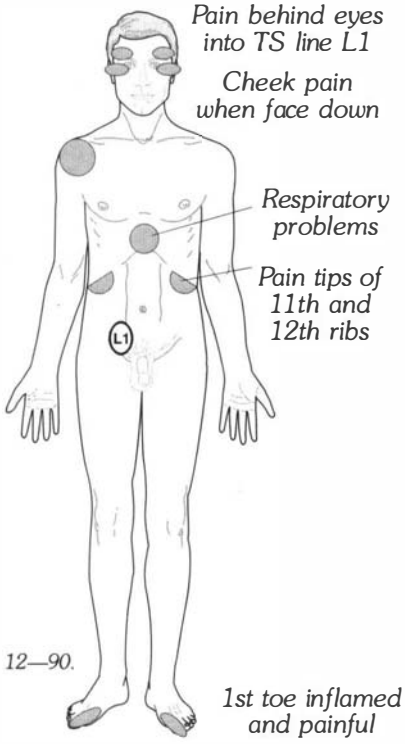
12—88.



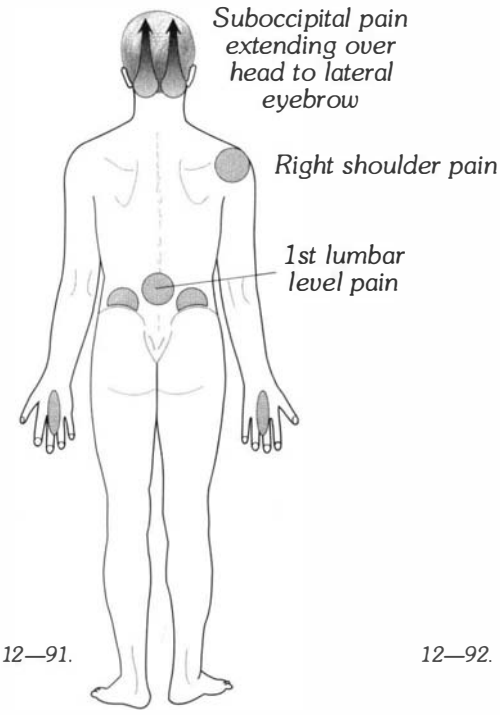
12—89.

Nutrition Considerations: Chewable vitamins A and E, kidney concentrate or nucleoprotein extract, complex for kidney and liver support, vitamins A and C with urea salts, heart concentrate or nucleoprotein extract.

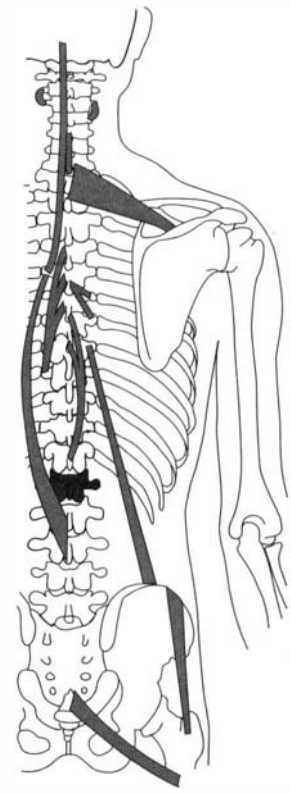
L1 — *C5* — Ileocecal Valve



12—90.



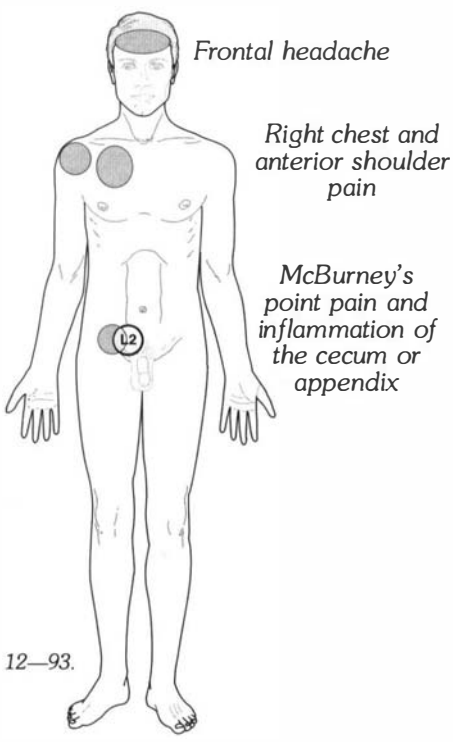
12—91.



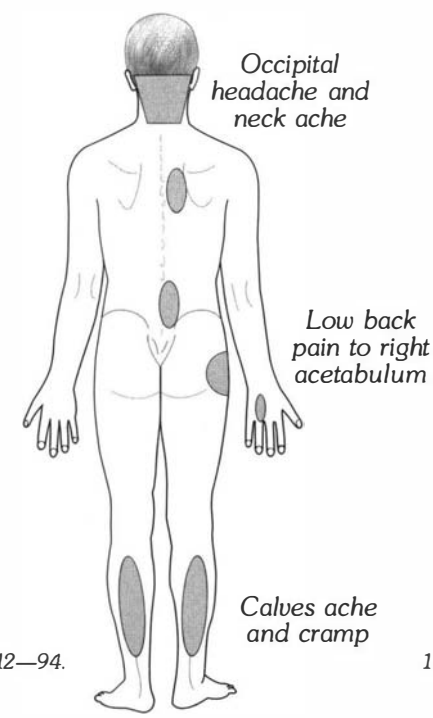
12—92.

Nutrition Considerations: Vitamin E complex of natural mixed tocopherols, choline bitartrate, digestive enzymes — pancreatin, pepsin, betaine HCl, stool softener (if irregular), amino acid complex.

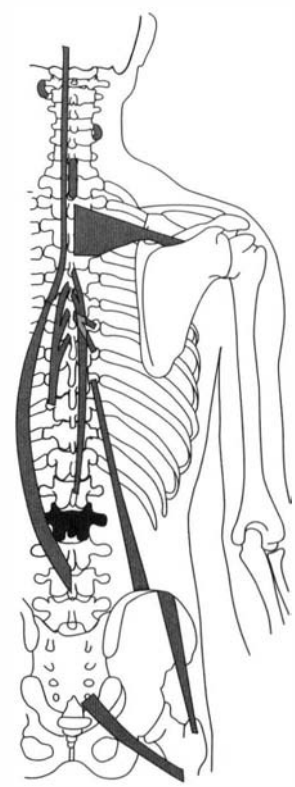
L2 — *C4* — Cecum



12—93.



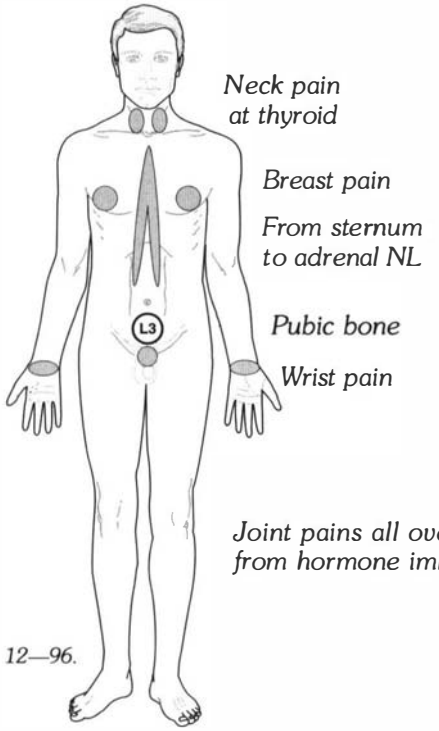
12—94.



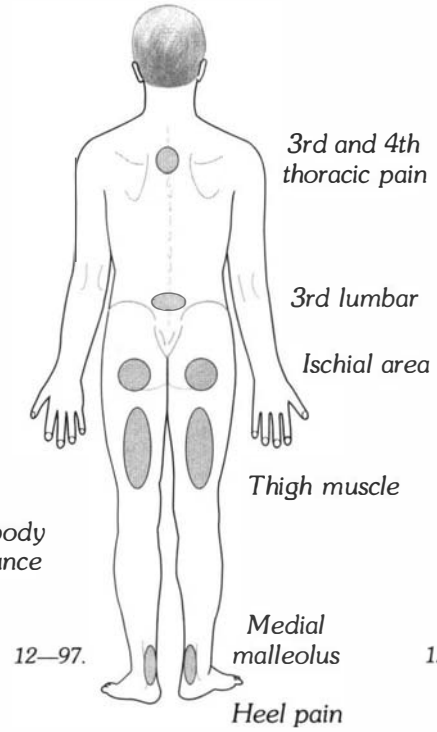
12—95.

Nutrition Considerations: Chewable vitamins A and E, kidney concentrate or nucleoprotein extract, complex for liver and kidney support, vitamins A and C with urea salts, heart concentrate or nucleoprotein extract.

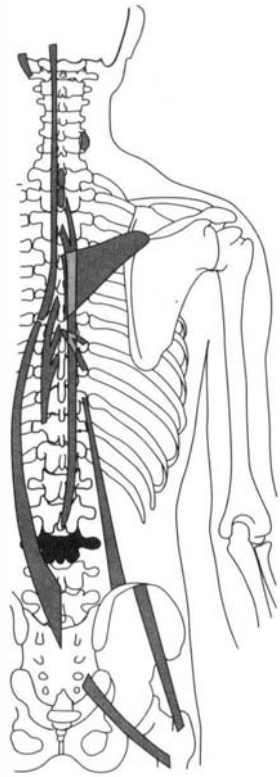
L3 — C3 — Ovaries / Testes



12—96.



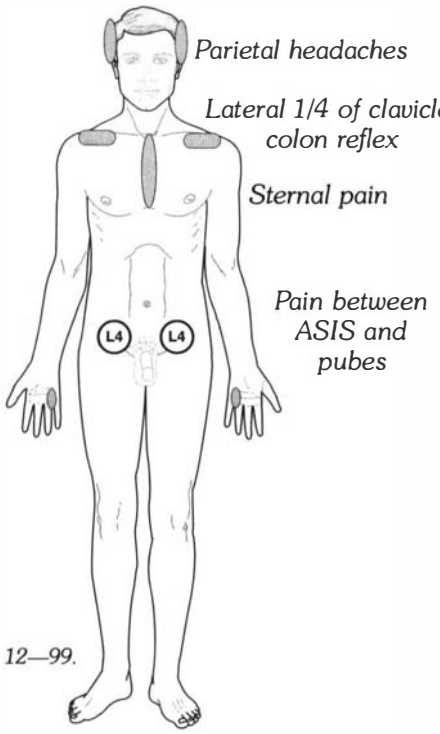
12—97.



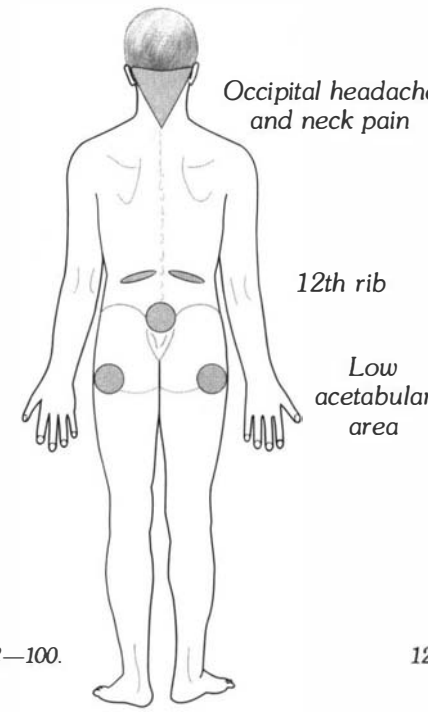
12—98.

Nutrition Considerations: Concentrate or nucleoprotein extract for female ovary, and for male orchic. Both sexes: concentrate or nucleoprotein extract pituitary, thyroid, adrenal, and/or raw bone, vitamin E from natural mixed tocopherols.

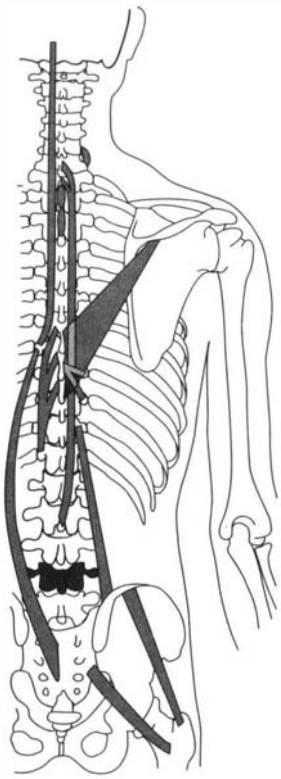
L4 — C2 — Colon



12—99.



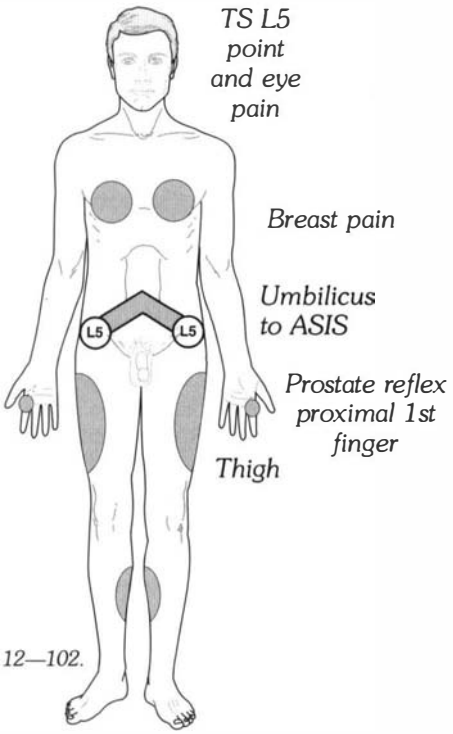
12—100.



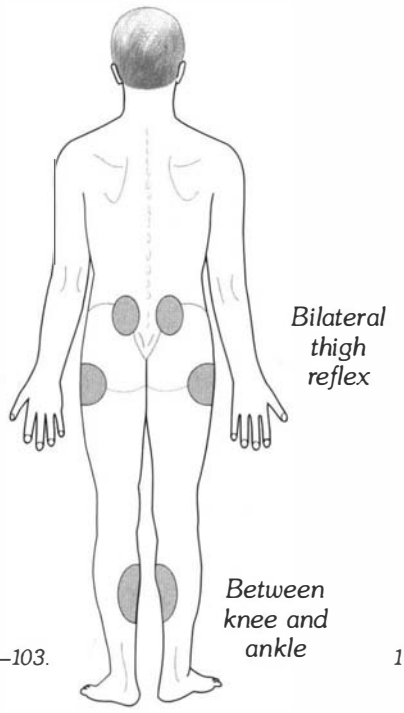
12—101.

Nutrition Considerations: Food source of iron, lactic acid yeast, enzymatically processed beet leaf, tillandsia for alimentary lactic acid, stool softener.

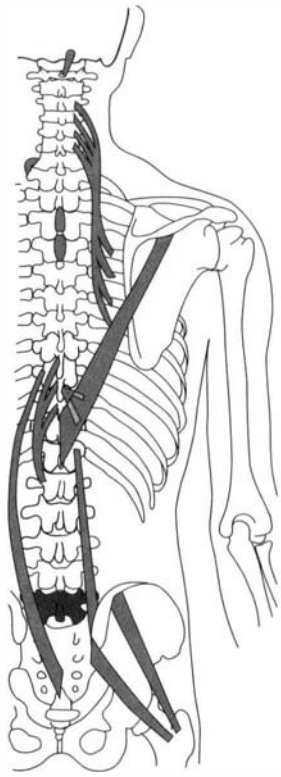
L5 — *C1* — Prostate / Uterus



12—102.



12—103.



12—104.

Nutrition Considerations: Female: E, E_2; ovary and/or uterus concentrate or nucleoprotein extract. Male: orchic and/or prostate concentrate or nucleoprotein extract. Both sexes: for burning urination, thymex concentrate or nucleoprotein extract and arginase source.

CHAPTER 12 — REFERENCES

1. Abrahamson, E., & A. Pezet, *Body, Mind, and Sugar* (New York: Pyramid Books, 1951).
2. Abravanel, E.D., & E.A. King, *Dr. Abravanel's Body Type Diet and Lifetime Nutrition Plan* (New York: Bantam Books, Inc, 1983).
3. Achilly, G.E., "Hypoglycemia vs. hypoadrenia." Proceedings of Summer Meeting, ICAK, Detroit, 1977.
4. Achilly, G.E., "Practical application of the unprocessed glucose tolerance test." Proceedings of Summer Meeting, ICAK, Detroit, 1979.
5. Ahrens, E.H., Jr., "The management of hyperlipidemia: Whether, rather than how," *Ann Intern Med*, Vol 85, No 1 (1976).
6. Alsever, R.N., & R.W. Gotlin, *Handbook of Endocrine Tests in Adults and Children*, 2nd ed (Chicago: Year Book Medical Publishers, Inc, 1978).
7. AMA Council on Foods & Nutrition, & National Research Council Food & Nutrition Board, "Diet and coronary heart disease — a Council statement," *JAMA*, Vol 222, No 13 (Dec 25, 1972).
8. American Diabetes Assoc, Endocrine Soc, & AMA, "Statement on hypoglycemia," *Metabolism*, Vol 22, No 3 (Mar 1973).
9. Anderson, J.T., F. Grande, & A. Keys, "Independence of the effects of cholesterol and degree of saturation of the fat in the diet on serum cholesterol in man," *Am J Clin Nutr*, Vol 29, No 11 (Nov 1976).
10. Anderson, J.W. et al., "Cholesterol-lowering effects of psyllium hydrophilic mucilloid for hypercholesterolemic men," *Arch Intern Med*, Vol 148 (Feb 1988).
11. Anderson, J.W., & W-J.L. Chen, "Plant fiber. Carbohydrate and lipid metabolism," *Am J Clin Nutr*, Vol 32, No 2 (Feb 1979).
12. Andrew, R., D.I.W. Phillips, & B.R. Walker, "Obesity and gender influence cortisol secretion and metabolism in man," *J Clin Endocrinology Metabolism*, Vol 83, No 5 (1998).
13. Arky, R., Medical Consultant, et al., *Physicians' Desk Reference*, 53rd ed (Montvale, NJ: Medical Economics, Inc, 1999).
14. Associated Press, "Heart Association eyes food critiques," *The Chieftain* (Jun 27, 1988).
15. Atkinson, M. et al., "Comparison of cardiac and pyloric sphincters — a manometric study," *Lancet* (Nov 9, 1957).
16. Bandler, R., & J. Grinder, *Frogs into Princes — Neuro Linguistic Programming*, ed J.O. Steven (Moab, UT: Real People Press, 1979).
17. Bargen, J.A., H.R. Wesson, & R.J. Jackman, "Studies on the ileocecal junction (ileocecus)," *Surg Gynecol Obstet*, Vol 71 (1940).
18. Barnes, B., "Basal temperature versus basal metabolism," *JAMA*, Vol 119 (Aug 1, 1942).
19. Barnes, B., & C. Barnes, *Heart Attack Rareness in Thyroid-Treated Patients* (Springfield, IL: Charles C. Thomas Publisher, 1972).
20. Barnes, B.O., & C.W. Barnes, *Solved: The Riddle of Heart Attacks* (Ft. Collins, CO: Robinson Press, Inc, 1976).
21. Barnes, B.O., & L. Galton, *Hypothyroidism: The Unsuspected Illness* (New York: Thomas Y. Crowell Co, 1976).
22. Barral, J-P., & P. Mercier, *Visceral Manipulation* (Seattle: Eastland Press, 1988).
23. Beare-Rogers, J.L., L.M. Gray, & R. Hollywood, "The linoleic acid and trans fatty acids of margarines," *Am J Clin Nutr*, Vol 32, No 9 (Sep 1979).
24. Beasley, V.R., *Your Electro-Vibratory Body*, Vol One, 2nd ed (Boulder Creek, CA: Univ of Trees Press, 1978).
25. Becker, M.H., "The cholesterol saga: Whither health promotion?" *Ann Intern Med*, Vol 106, No 4 (Apr 1987).
26. Beilin, L.J., & B.M. Margetts, "Vegetarian diet and blood pressure," in *Non-Pharmacologic Therapy of Hypertension*, ed M.D. Blaufox & H.G. Langford (New York: S. Karger, 1987).
27. Bell, L.S., L.N. Tiglio, & M.M. Fairchild, "Dietary strategies in the treatment of reactive hypoglycemia," *J Am Dietetic Assoc*, Vol 85, No 9 (Sep 1985).
28. Benum, S., "Allergy — origin & treatment," *Comp Med Magazine*, Vol 1, No 3 (Jan/Feb 1986).
29. Berry, E.M., & J. Hirsch, "Does dietary linolenic acid influence blood pressure?" *Am J Clin Nutr*, Vol 44 (Sep 1986).
30. Best, C.H., & N.B. Taylor, *The Physiological Basis of Medical Practice*, 8th ed (Baltimore: Williams & Wilkins, 1966).
31. Bieler, H.G., *Food Is Your Best Medicine* (New York: Random House, 1965).
32. Billek, G., "Heated fats in the diet," in *The Role of Fats in Human Nutrition*, ed F.B. Padley & J. Podmore (Chichester, England: Ellis Horwood, 1985).
33. Binkley, S., "A timekeeping enzyme in the pineal gland," *Sci Am*, Vol 240, No 4 (Apr 1979).
34. Birchfield, R.I., "Postural hypotension in Wernicke's disease," *Am J Med*, Vol 36 (Mar 1964).
35. Black, I.B., "Idiopathic autonomic insufficiency: Idiopathic-orthostatic hypotension, Shy-Drager syndrome," in *Cecil Textbook of Medicine*, 15th ed, ed P.B. Beeson, W. McDermott, & J. Wyngaarden (Philadelphia: W.B. Saunders Co, 1979).
36. Blaich, R.M., 425 Cherry St., Suite 580, Denver, CO 80222. Personal communication.
37. Bland, J.S., "In search of a definition for allergy," *Comp Med Magazine*, Vol 1, No 3 (Jan/Feb 1986).
38. Blaufox, M.D., & H.G. Langford, "Non-pharmacologic therapy of hypertension," in *Non-Pharmacologic Therapy of Hypertension*, ed M.D. Blaufox & H.G. Langford (New York: S. Karger, 1987).
39. Blumenthal, M. et al., eds, *Therapeutic Guide to Herbal Medicines* (Austin, TX: American Botanical Council, 1998).
40. Bonanome, A., & S.M. Grundy, "Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels," *N Engl J Med*, Vol 318, No 19 (May 12, 1988).
41. Boxer, R.W., "Cardiac arrhythmias due to foods," in *Clinical Ecology*, ed L.D. Dickey (Springfield, IL: Charles C. Thomas Publisher, 1976).
42. Boyd, W., H. Blincoe, & J.C. Hayner, "Sequence of action of the diaphragm and quadratus lumborum during quiet breathing," *Anat Rec*, Vol 151 (Apr 1965).
43. Brandt, K.D., "Effects of nonsteroidal antiinflammatory drugs on chondrocyte metabolism *in vitro* and *in vivo*," *Am J Med*, Vol 83, No 5A (Nov 20, 1987).
44. Brandt, K.D., & M.J. Palmoski, "Effects of salicylates and other nonsteroidal anti-inflammatory drugs on articular cartilage," *Am J Med*, Vol 77, No 1A (Jul 13, 1984).
45. Branham, K., "Lower thoracic pain and hypoadrenia." Proceedings of Summer Meeting, ICAK, Santa Monica, CA, 1985.
46. Braverman, L.E., "The thyroid gland," in *The Year Book of Endocrinology — 1987*, ed J.D. Bagdage (Chicago: Year Book Medical Publishers, Inc, 1987).
47. Brea, A.F., "Copper status: The other side of the coin. A preliminary report and review." Proceedings of Winter Meeting, ICAK, Palm Desert, CA, 1987.
48. Brea, A.F., "A technique for identifying impingement of the right lymphatic duct." Proceedings of Summer Meeting, ICAK, Washington, DC, 1987.
49. Brea, A.F., "Copper status and the compressional bone test." Proceedings of Summer Meeting, ICAK, Dearborn, MI, 1988.
50. Brooks-Gunn, J., M.P. Warren, & L.H. Hamilton, "The relation of eating problems and amenorrhea in ballet dancers," *Med Sci Sports Exer*, Vol 19, No 1 (Feb 1987).
51. Brown, M.D., et al., "Improvement of insulin sensitivity by short-term exercise training in hypertensive African American women," *Hypertension*, Vol 30, No 6 (Dec 1997).
52. Buirge, R.E., "Experimental observations on the human ileocecal valve," *Surgery*, Vol 16 (Sep 1944).
53. Burdick Corporation, Milton, WI.
54. Burkitt, D.P., A.R.P. Walker, & N.S. Painter, "Effect of dietary

fibre on stools and transit-times, and its role in the causation of disease," *Lancet*, Vol II, No 7790 (Dec 30, 1972).
55. Burkitt, D.P., A.R.P. Walker, & N.S. Painter, "Dietary fiber and disease," *JAMA*, Vol 229, No 8 (Aug 19, 1974).
56. Burnett, A.L. et al., "Nitric oxide: A physiologic mediator of penile erection," *Science*, Vol 257 (July 17, 1992).
57. Burt, A.M., *Textbook of Neuroanatomy* (Philadelphia: W.B. Saunders Co., 1993).
58. Buss, R.W. et al., "Mixed meal tolerance test and reactive hypoglycemia," *Horm Metabol Res*, Vol 14 (1982).
59. CM staff, "Theron G. Randolph and environmental allergy — The father of 'clinical ecology' speaks his mind," *Comp Med Magazine*, Vol 1, No 3 (Jan/Feb 1986).
60. CM staff, "Charles D. May on food allergy — 'A traditional allergist's position,'" *Comp Med Magazine*, Vol 1, No 3 (Jan/Feb 1986).
61. CM staff, "Nick S. Harris on allergy — a moderate's position on the term 'allergy,'" *Comp Med Magazine*, Vol 1, No 3 (Jan/Feb 1986).
62. CM staff, "Calcium and hypertension — interview with Cynthia D. Morris, Ph.D.," *Comp Med Magazine*, Vol 1, No 5 (May/Jun 1986).
63. Caggiula, A.W., N.C. Milas, & R.R. Wing, "Optimal nutritional therapy in the treatment of hypertension," in *Non-Pharmacologic Therapy of Hypertension*, ed M.D. Blaufox & H.G. Langford (New York: S. Karger, 1987).
64. Callahan, R.J., *Five Minute Phobia Cure* (Wilmington, DE: Enterprise Publishers, Inc, 1985).
65. Carpenter, M., *Human Neuroanatomy*, 7th ed (Baltimore: Williams & Wilkins, 1976).
66. Castell, D.O., S. Cohen, & L.D. Harris, "Response of human ileocecal sphincter to gastrin," *Am J Physiol*, Vol 219, No 3 (Sep 1970).
67. Caudill, M.A., R. Friedman, & H. Benson, "Relaxation therapy in the control of blood pressure," in *Non-Pharmacologic Therapy of Hypertension*, ed M.D. Blaufox & H.G. Langford (New York: S. Karger, 1987).
68. Champe, P.C., & R.A. Harvey, *Lippincott's Illustrated Reviews: Biochemistry* (Philadelphia: J.B. Lippincott Co, 1987).
69. Chen, G.S., "Treatment of functional hypoglycemia with acupuncture," *Am J Acupunc*, Vol 11, No 2 (Jun 1983).
70. Chen, W-J. L., J.W. Anderson, & M.R. Gould, "Effects of oat bran, oat gum and pectin on lipid metabolism of cholesterol-fed rats," *Nutr Reports Int*, Vol 24, No 6 (Dec 1981).
71. Cheraskin, E., W.M. Ringsdorf, Jr., & A.T.S. Setyaadmadja, "Periodontal pathosis in man: XIII. Effect of sucrose drinks upon sulcus depth," *J Oral Ther Pharmacol*, Vol 2, No 3 (1965).
72. Cheraskin, E., W.M. Ringsdorf, Jr., & A.T.S. Setyaadmadja, "Periodontal pathosis in man: XIV. Effect of sucrose drinks upon clinical tooth mobility," *J Dent Med*, Vol 20, No 3 (Jul 1965).
73. Cheraskin, E. et al., "Periodontal pathosis in man: XV. Effect of glucose drinks upon gingival state," *J Oral Med*, Vol 21 (Apr 1966).
74. Cheraskin, E., W.M. Ringsdorf, Jr., & A.T.S. Setyaadmadja, "Periodontal pathosis in man: XVI. Effect of glucose drinks upon sulcus depth," *J Oral Ther Pharmacol*, Vol 3, No 3 (1966).
75. Cheraskin, E., W.M. Ringsdorf, Jr., & A.T.S. Setyaadmadja, "Periodontal pathosis in man: XVII. Effect of glucose drinks upon clinical tooth mobility," *J Oral Ther Pharmacol*, Vol 3, No 4 (1967).
76. Cheraskin, E. et al., "Effect of carbohydrate supplements upon the height of T wave in lead I," *Angiology*, Vol 19 (Apr 1968).
77. Cheraskin, E. et al., *Psychodietetics — Food as the Key to Emotional Health* (New York: Stein & Day Publishers, 1974).
78. Cheraskin, E., & W.M. Ringsdorf, Jr., "Total health: A correlative study in stomatology and electrocardiography," *Ann Dent*, Vol 38 (1979).
79. Cheraskin, E, W.M. Ringsdorf, Jr., & E. Sisley, *The Vitamin C Connection* (New York: Harper & Row, 1983).
80. Chrousos, G.P., D.J. Torpy, & P.W. Gold, "Interactions between the hypothalamic-pituitary-adrenal axis and the female reproductive system: Clinical implications," *Ann Intern Med*, Vol 129, No 3 (Aug 1998).
81. Church, J.P. et al., "Relationships among dietary constituents and specific serum clinical components of subjects eating self-selected diets," *Am J Clin Nutr*, Vol 40 (Dec 1984).
82. Cleave, T.L., "The neglect of natural principles in current medical practice," *J Royal Naval Med Serv*, Vol XLII, No 2 (Spring 1956).
83. Cleave, T.L., *The Saccharine Disease* (New Canaan, CT: Keats Publishers, Inc, 1975).
84. Cloward, R.B., "Cervical diskography — a contribution to the etiology and mechanism of neck, shoulder and arm pain," *Ann Surg*, Vol 150, No 6 (Dec 1959).
85. Cloward, R.B., "The clinical significance of the sinu-vertebral nerve of the cervical spine in relation to the cervical disk syndrome," *J Neurol Neurosurg Psychiatry*, Vol 23 (1960).
86. Cloward, R.B., "Acute cervical spine injuries," *Clin Symp*, Vol 32, No 1 (1980).
87. Cluette-Brown, J. et al., "Oral nicotine induces an atherogenic lipoprotein profile," *Proc Soc Exper Biol Med*, Vol 182 (1986).
88. Coca, A.F., *Familial Nonreagenic Food Allergy*, 3rd ed (Springfield, IL: Charles Thomas Publisher, 1953).
89. Coca, A.F., *The Pulse Test*, revised ed (New York: Lyle Stuart, 1958).
90. Cohen, S., L.D. Harris, & R. Levitan, "Manometric characteristics of the human ileocecal junctional zone," *Gastroenterology*, Vol 54, No 1 (1968).
91. Collins, R.D., *Illustrated Manual of Laboratory Diagnosis* (Philadelphia: J.B. Lippincott Co, 1968).
92. Collis, J.L., L.M. Satchwell, & L.D. Abrams, "Nerve supply to the crura of the diaphragm," *Thorax*, Vol 9 (1954).
93. Conniff, R., "Superchicken — whose life is it anyway?" *Discover*, Vol 9, No 6 (Jun 1988).
94. Cook, S.D. et al., "Trabecular bone density and menstrual function in women runners," *Am J Sports Med*, Vol 15, No 5 (Sep/Oct 1987).
94a. Cook, T.C. et al., "Chronic low level physical activity as a determinant of high density lipoprotein cholesterol and subfractions," *Med Sci Sports Exer*, Vol 18, No 6 (Dec 1986).
95. Copeland, P.A., P.A. Fitzgerald, & M. Rosenblatt, "Adrenal disorders," in *Handbook of Clinical Endocrinology*, ed P.A. Fitzgerald (Chicago: Jones Medical Publishers, 1986).
96. Coronary Drug Project Research Group, "Clofibrate and niacin in coronary heart disease," *JAMA*, Vol 231, No 4 (Jan 27, 1975).
97. Corwin, A.H., "The rotating diet and taxonomy," in *Clinical Ecology*, ed L.D. Dickey (Springfield, IL: Charles C. Thomas Publisher, 1976).
98. Council on Scientific Affairs, "Dietary and pharmacologic therapy for the lipid risk factors," *JAMA*, Vol 250, No 14 (Oct 14, 1983).
99. Crawford, J.P., G.S. Hickson, & M.R. Wiles, "The management of hypertensive disease: A review of spinal manipulation and the efficacy of conservative therapeusis," *J Manip Physiol Ther*, Vol 9, No 1 (Mar 1986).
100. Crook, W.G., *The Yeast Connection — A Medical Breakthrough* (New York: Vintage Books, 1986).
101. Crouse, S.F. et al., "Zinc ingestion and lipoprotein values in sedentary and endurance-trained men," *JAMA*, Vol 252, No 6 (Aug 10, 1984).
102. Cryer, B., & M. Feldman, "Effects of very low dose daily, long-term aspirin therapy on gastric, duodenal, and rectal prostaglandin levels and on mucosal injury," *Gastroenterology*, Vol 117, No 1 (Jul 1999).
103. Danowski, T.S., *Outline of Endocrine Gland Syndromes*, 2nd ed (Baltimore: Williams & Wilkins, 1968).
104. Darnall, L., "The pineal gland and aging," *Comp Med Magazine*, Vol 2, No 2 (Nov/Dec 1986).
105. Davis, A.R., & W.C. Rawls, Jr., *Magnetism and Its Effects on the*

Living System (Hicksville, NY: Exposition Press, 1974).
106. Daynes, R.A., D.J. Dudley, & B.A. Araneo, "Regulation of murine lymphokine production *in vivo*. II. Dehydroepiandosterone is a natural enhancer of interleukin 2 synthesis by helper T cells," *Eur J Immunol*, Vol 20, No 4 (Apr 1990).
107. Deal, S.C., "Balancing ionization." Proceedings of Summer Meeting, ICAK, Santa Monica, CA, 1985.
108. DeFronzo, R.A., R.C. Bonadonna, & E. Ferrannini, "Pathogenesis of NIDDM. A balanced overview," *Diabetes Care*, Vol 15, No 3 (Mar 1992).
109. DeGroot, A.P., R. Luyken, & N.A. Pikaar, "Cholesterol-lowering effect of rolled oats," *Lancet*, Vol 2 (Aug 10, 1963).
110. Deighton, C.M., M.J. Watson, & D.J. Walker, "Sex hormones in postmenopausal HLA-identical rheumatoid arthritis discordant sibling pairs," *J Rheumatol*, Vol 19, No 11 (Nov 1992).
111. Dekel, S., J. Falconer, & M.J. Francis, "The effect of anti-inflammatory drugs on glycosaminoglycan sulphation in pig cartilage," *Prostaglandins Med*, Vol 4, No 3 (Mar 1980).
112. Dennis, C., "Treatment of large bowel obstruction," *Surgery*, Vol 15 (1944).
113. Department of National Health and Welfare, "Recommendations for prevention programs in relation to nutrition and cardiovascular disease." Bureau of Nutritional Sciences, Health Production Branch, Ottawa, Canada (1977).
114. Desforges, G. et al., "Traumatic rupture of the diaphragm — clinical manifestations and surgical treatment," *J Thoracic Surg*, Vol 34, No 6 (Dec 1957).
115. de Vries, B.J., W.B. van den Berg, & L.B. van de Putte, "Salicylate-induced depletion of endogenous inorganic sulfate. Potential role in the suppression of sulfated glycosaminoglycan synthesis in murine articular cartilage," *Arthritis Rheum*, Vol 28, No 8 (Aug 1985).
116. Diagnos-Techs, Inc., Clinical & Research Laboratory, 6620 S. 192nd Pl., J-104, Kent, WA 98032.
117. Dickey, L.D., "Presenting, reproduced, induced, and relieved symptoms observed with provocative tests," in *Clinical Ecology*, ed L.D. Dickey (Springfield, IL: Charles C. Thomas Publisher, 1976).
118. Dieppe, P., et al., "A two-year, placebo-controlled trial of nonsteroidal anti-inflammatory therapy in osteoarthritis of the knee joint," *Br J Rheumatol*, Vol 32, No 7 (Jul 1993).
119. Dietschy, J.M., & J.D. Wilson, "Regulation of cholesterol metabolism" (First of three parts), *N Engl J Med*, Vol 282, No 20 (May 14, 1970).
120. Dietschy, J.M., & J.D. Wilson, "Regulation of cholesterol metabolism" (Second of three parts), *N Engl J Med*, Vol 282, No 21 (May 21, 1970).
121. Dietschy, J.M., & J.D. Wilson, "Regulation of cholesterol metabolism" (Third of three parts), *N Engl J Med*, Vol 282, No 22 (May 28, 1970).
122. Dillon, M., "Stress: The good, the bad and the unavoidable," *ACA J Chiro*, Vol 24, No 6 (Jun 1987).
123. Donald, C.J., & O.W. Clayton, "Diaphragmatic hernia: A report of eighty-three cases," *Am Surgeon*, Vol 21 (1955).
124. Drake, M.E., "Panic attacks and excessive aspartame ingestion," *Lancet*, Vol 2, No 8507 (Sep 13, 1986).
125. Dreyfus, P.M., "Nutritional disorders of the nervous system," in *Cecil Textbook of Medicine*, 15th ed, ed P.B. Beeson, W. McDermott, & J. Wyngaarden (Philadelphia: W.B. Saunders Co, 1979).
126. Dustan, H.P., Chairman Joint Committee, "The 1984 Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure," *Arch Intern Med*, Vol 144 (May 1984).
127. Dvorak, J., & V. Dvorak, *Manual Medicine Diagnostics* (New York: Thieme-Stratton, Inc, 1984).
128. Eades, M.R., & M.D. Eades, *Protein Power* (New York: Bantam Books, 1996).
129. Edmunds, V., "Hiatus hernia — a clinical study of 200 cases," *Q J Med*, Vol 26, No 104 (Oct 1957).
130. Ellenberg, M., "Absent deep reflexes: A diagnostic clue in unsuspected diabetes," *Am J Med Sci*, Vol 242 (Aug 1961).
131. Elliott, T.R., "On the innervation of the ileo-colic sphincter," *J Physiol*, Vol 31 (1904).
132. Epstein, A.M., & G. Oster, "Cholesterol reduction and health policy: Taking clinical science to patient care," *Ann Intern Med*, Vol 106, No 4 (Apr 1987).
133. Esler, M. et al., "Mild high-renin essential hypertension — neurogenic human hypertension?" *N Engl J Med*, Vol 296, No 8 (Feb 24, 1977).
134. Fast, J., *Body Language* (New York: Pocket Books, 1971).
135. Feeley, R.M., P.E. Criner, & B.K. Watt, "Cholesterol content of foods," *J Am Diet Assoc*, Vol 61 (Aug 1972).
136. Feingold, B.F., *Introduction to Clinical Allergy* (Springfield, IL: Charles C. Thomas Publisher, 1973).
137. Feinstein, B. et al., "Experiments on pain referred from deep somatic tissues," *J Bone Joint Surg*, Vol 36A, No 5 (Oct 1954).
138. Ferrannini, E. et al., for the European Group for the Study of Insulin Resistance (EGIR), "Insulin resistance, hyperinsulinemia, and blood pressure: Role of age and obesity," *Hypertension*, Vol 30, No 5 (Nov 1997).
139. Fischer, P. et al., "The effect of dietary copper and zinc on cholesterol metabolism," *Am J Clin Nutr*, Vol 33 (May 1980).
140. Fleischner, F.G., & C. Bernstein, "Roentgen-anatomical studies of the normal ileocecal valve," *Radiology*, Vol 54 (Jan 1950).
141. Flynn, M.A. et al., "Serum lipids in humans fed diets containing beef or fish and poultry," *Am J Clin Nutr*, Vol 34 (Dec 1981).
142. Flynn, M.A. et al., "Effect of dietary egg on human serum cholesterol and triglycerides," *Am J Clin Nutr*, Vol 32, No 5 (May 1979).
143. Fogel, R.L. et al., "Achilles tendon reflex test (Photomotogram) as a measure of thyroid function," *NY State J Med* (Apr 15, 1962).
144. Fredericks, C., *Psycho-Nutrition* (New York: Grosset & Dunlap, 1976).
144a. Freis, E.D., "Should mild hypertension be treated?" *N Engl J Med*, Vol 307, No 5 (Jul 29, 1982).
145. Friedman, M.H., & J. Weisberg, "Applied kinesiology — double-blind pilot study," *J Prosthet Dent*, Vol 45, No 3 (Mar 1981).
146. Friedman, R. et al., "Cholesterol metabolism," *Ann Rev Biochem*, Vol 25 (1956).
147. Frymann, V.M., "The core-link and the three diaphragms," *Acad Appl Osteo Yr Bk* (1968).
148. Frymann, V.M., "A study of the rhythmic motions of the living cranium," *JAOA*, Vol 70, No 9 (May 1971).
149. Gabriel, S.E., L. Jaakkimainen, & C. Bombardier, "Risk for serious gastrointestinal complications related to use of nonsteroidal anti-inflammatory drugs. A meta-analysis," *Ann Intern Med*, Vol 115, No 10 (Nov 15, 1991).
150. Garrison, R.H., Jr., & E. Somer, *The Nutrition Desk Reference* (New Canaan, CT: Keats Publishing, Inc, 1985).
151. Gastineau, C.F., "Is reactive hypoglycemia a clinical entity?" *Mayo Clinic Proceed*, Vol 58 (1983).
152. Gazet, J.C., & R.J. Jarrett, "The ileocaeco-colic sphincter. Studies in vitro in man, monkey, cat, and dog," *Br J Surg*, Vol 51, No 5 (May 1964).
153. Gazet, J.C., & J. Kopp, "The surgical significance of the ileocecal junction," *Surgery*, Vol 56, No 3 (Sep 1964).
154. Gildea, E.F., E.B. Man, & J.P. Peters, "Serum lipoids and proteins in hypothyroidism," *J Clin Invest*, Vol 18 (1939).
155. Glassman, J.A., "An artificial ileocecal valve," *Surg Gynecol Obstet*, Vol 74 (Jan 1942).
156. Golbert, T.M., "A review of controversial diagnostic and therapeutic techniques employed in allergy," *J Allergy Clin Immunol*, Vol 56, No 3 (Sep 1975).
157. Goldman, J.A. et al., "Behavioral effects of sucrose on preschool children," *J Abnorm Child Psych*, Vol 14, No 4 (1986).

158. Goodheart, G.J., Jr., "The thyroid — an overlooked problem," Chiro Econ, Vol 7, No 4 (Jan/Feb 1965).
159. Goodheart, G.J., Jr., "Low blood sugar and hyperinsulinism," *Chiro Econ*, Vol 7, No 5 (Mar/Apr 1965).
160. Goodheart, G.J., Jr., "Postural hypotension and functional hypoadrenia," *Chiro Econ*, Vol 7, No 6 (May/Jun 1965).
161. Goodheart, G.J., Jr., "Control of cholesterol," *Chiro Econ*, Vol 8, No 1 (Jul/Aug 1965).
162. Goodheart, G.J., Jr., "The ileocecal valve syndrome," *Chiro Econ*, Vol 9, No 6 (May/Jun 1967).
163. Goodheart, G.J., Jr., *The Cranial Sacral and Nutritional Reflexes and Their Relationship to Muscle Balancing* (Detroit: privately published, 1968).
164. Goodheart, G.J., Jr., "Hypertension," *Chiro Econ*, Vol 11, No 3 (Nov/Dec 1968).
165. Goodheart, G.J., Jr., "Allergies in chiropractic practice," *Chiro Econ*, Vol 12, No 3 (Nov/Dec 1969). Reprinted in *A.K. Review*, Vol 3, No 1 (Summer 1992).
166. Goodheart, G.J., Jr., *Applied Kinesiology 1976 Workshop Procedure Manual*, 12th ed (Detroit: privately published, 1976).
167. Goodheart, G.J., Jr., *Applied Kinesiology 1978 Workshop Procedure Manual*, 14th ed (Detroit: privately published, 1978).
168. Goodheart, G.J., Jr., "Hypothyroidism and myofascial relationships," *Chiro Econ*, Vol 21, No 1 (Jul/Aug 1978).
169. Goodheart, G.J., Jr., *Applied Kinesiology 1979 Workshop Procedure Manual*, 15th ed (Detroit: privately published, 1979).
170. Goodheart, G.J., Jr., *Applied Kinesiology 1980 Workshop Procedure Manual*, Vol 1, 16th ed (Detroit: privately published, 1980).
171. Goodheart, G.J., Jr., *Applied Kinesiology 1985 Workshop Procedure Manual*, 21st ed (Detroit: privately published, 1985).
172. Goodheart, G.J., Jr., *Applied Kinesiology 1987 Workshop Procedure Manual* (Detroit: privately published, 1987).
173. Goodheart, G.J., Jr., *Applied Kinesiology 1992/93 Workshop Procedure Manual* (Grosse Pointe Woods: privately published, 1993).
174. Goodheart, G.J., Jr., *Applied Kinesiology 1994/95 Workshop Procedure Manual* (Grosse Pointe Woods: privately published, 1995).
175. Goodheart, G.J., Jr., "Detection of the 'sleep' pattern in the 'awake' patient and the serotonergic neuronal relationship to the reticular activating system." Proceedings of Summer Meeting, ICAK, Boston, 1995.
176. Goodheart, G.J., Jr., *Applied Kinesiology 1997-1998 Workshop Procedural Manual* (Grosse Pointe Woods: privately published, 1998).
177. Goodwin, J.S. et al., "Relationship between zinc intake, physical activity, and blood levels of high-density lipoprotein cholesterol in a healthy elderly population," *Metabolism*, Vol 34, No 6 (Jun 1985).
178. Gray, H., *Anatomy of the Human Body*, 29th Am ed, ed C.M. Goss (Philadelphia: Lea & Febiger, 1973).
179. Great Smokies Diagnostic Laboratory, 63 Zillicoa St., Asheville, NC 28801-1074 [1-800-522-4762; FAX 1-828-252-9303; hhtp://www.greatsmokies-lab.com].
180. Greenfield, M., "Give me that old-time cholesterol," *Newsweek* (June 25, 1984).
181. Grieco, M.H., "Controversial practices in allergy," *JAMA*, Vol 247, No 22 (Jun 11, 1982).
182. Griffin, M.R. et al., "Nonsteroidal anti-inflammatory drug use and increased risk for peptic ulcer disease in elderly persons," *Ann Intern Med*, Vol 114, No 4 (Feb 15, 1991).
183. Grimm, R.H., & R.J. Prineas, "The effects of sodium reduction on control of blood pressure elevation: A review," in *Non-Pharmacologic Therapy of Hypertension*, ed M.D. Blaufox & H.G. Langford (New York: S. Karger, 1987).
184. Grobstein, C., Chrmn, Committee on Diet/Nutrition/Cancer, *Diet, Nutrition, and Cancer* (Washington, DC: National Academy Press, 1982).
185. Grundy, S.M. et al., "Rationale of the diet-heart statement of the American Heart Association. Report of Nutrition Committee," *Circulation*, Vol 65, No 4 (Apr 1982).
186. Guyton, A.C., *Textbook of Medical Physiology*, 6th ed (Philadelphia: W.B. Saunders Co, 1981).
187. Guyton, A.C., & J.E. Hall, *Textbook of Medical Physiology*, 9th ed (Philadelphia: W.B. Saunders Co, 1996).
188. Harper, A.E., "Dietary goals — a skeptical view," *Am J Clin Nutr*, Vol 31, No 2 (Feb 1978).
189. Harriman, A.E., "The effect of a preoperative preference for sugar over salt upon compensatory salt selection by adrenalectomized rats," *J Nutr*, Vol 57 (Oct 1955).
190. Havel, R.J., "Disorders of lipid metabolism," in *Cecil Textbook of Medicine*, 15th ed, ed P.B. Beeson, W. McDermott, & J. Wyngaarden (Philadelphia: W.B. Saunders Co, 1979).
191. Heaton, K.W., "Gallstone formation," *9th Adv Med Symp*, ed J.G. Walker (London: Pitman Medical, 1973).
192. Hechter, O., A. Grossman, & R.T. Chatterton, Jr., "Relationship of dehydroepiandrosterone and cortisol in disease," *Medical Hypotheses*, Vol 49 (1997).
193. Hemmings, W.A., & E.W. Williams, "Transport of large breakdown products of dietary protein through the gut wall," *Gut*, Vol 19 (1978).
194. Hendrix, T.R., "The motility of the alimentary canal," in *Medical Physiology*, Vol 2, 14th ed, ed V.B. Mountcastle (St. Louis: The C.V. Mosby Co, 1980).
195. Herbert, V., "Vitamin B-12," in *Present Knowledge in Nutrition*, 7th ed, ed E.E. Ziegler & L.J. Filer, Jr. (Washington, DC: ILSI Press, 1996).
196. Hill, M.J. et al., "Bacteria and aetiology of cancer of the large bowel," *Lancet*, Vol 1 (Jan 16, 1971).
197. Himsworth, H.P., & R.B. Kerr, "Insulin-sensitive and insulin-insensitive type of diabetes mellitus," *Clin Sci*, Vol 4 (1939).
198. Hockaday, J.M., & C.W.M. Whitty, "Patterns of referred pain in the normal subject," *Brain*, Vol 90, Part 3 (Sep 1967).
199. Hogan, J. et al., "Oral glucose tolerance test compared with a mixed meal in the diagnosis of reactive hypoglycemia — a caveat on stimulation," *Mayo Clin Proceed*, Vol 58 (Aug 1983).
200. Hojnacki, J. et al., "Oral nicotine impairs clearance of plasma low density lipoproteins," *Proc Soc Exper Biol Med*, Vol 182 (1986).
201. Hollenbeck, C., & G.M. Reaven, "Variations in insulin-stimulated glucose uptake in healthy individuals with normal glucose tolerance," *J Clin Endocrinol Metab*, Vol 64, No 6 (Jun 1987).
202. Holman, R.T., "Influence of hydrogenated fats on the metabolism of polyunsaturated fatty acids," in *The Role of Fats in Human Nutrition*, ed F.B. Padley & J. Podmore (Chichester, England: Ellis Horwood, 1985).
203. Hood, R.P., "Blood pressure — Results in 75 abnormal cases," *Chiro Econ*, Vol 16, No 6 (May/Jun 1974).
203a. Hooper, P.L. et al., "Zinc lowers high-density lipoprotein-cholesterol levels," *JAMA*, Vol 244, No 17 (Oct 24/31, 1980).
204. Hoppmann, R.A., J.G. Peden, & S.K. Ober, "Central nervous system side effects of nonsteroidal anti-infammatory drugs. Aseptic meningitis, psychosis, and cognitive dysfunction," *Arch Intern Med*, Vol 151, No 7 (Jul 1991).
205. Horrobin, D.F., "Gamma-linolenic acid in medicine," in *1984-85 Yearbook of Nutritional Medicine*, ed J. Bland (New Canaan, CT: Keats Publishing, Inc, 1985).
206. Horrobin, D.F., & M.S. Manku, "How do polyunsaturated fatty acids lower plasma cholesterol levels?" *Lipids*, Vol 18, No 8 (1983).
206a. Hossman, V., G.A. FitzGerald, & C.T. Dollery, "Influence of hospitalization and placebo therapy on blood pressure and sympathetic function in essential hypertension," *Hypertension*, Vol 3, No 1 (Jan/Feb 1981).
207. Huang, Y.S., M.S. Manku, & D.F. Horrobin, "The effects of dietary cholesterol on blood and liver polyunsaturated fatty acids

and on plasma cholesterol in rats fed various types of fatty acid diet" *Lipids*, Vol 19, No 9 (Sep 1984).

207a. Hugenberg, S.T., K.D. Brandt, & C.A. Cole, "Effect of sodium salicylate, aspirin, and ibuprofen on enzymes required by the chondrocyte for synthesis of chondroitin sulfate," *J Rheumatol*, Vol 20, No 12 (Dec 1993).

208. Hughes, V.A. et al., "Lipoprotein response to exercise training and a low-fat diet in older subjects with glucose intolerance," *Am J Clin Nutr*, Vol 59, No 4 (Apr 1994).

209. Hunter, B.T., *The Great Nutrition Robbery* (New York: Charles Scribner's Sons, 1978).

210. Hurdle, J., *Low Blood Sugar — A Doctor's Guide to Its Effective Control* (W. Nyack, NY: Parker Publishing Co, Inc, 1969).

211. Hurxthal, L.M., "Blood cholesterol and thyroid disease," *Arch Intern Med*, Vol 53 (1934).

212. Hutton, J.H., *Practical Endocrinology* (Springfield, IL: Charles C. Thomas Publisher, 1966).

213. Ingbar, S.H., & K.A. Woeber, "The thyroid gland," in *Textbook of Endocrinology*, 5th ed, ed R.H. Williams (Philadelphia: W.B. Saunders Co, 1974).

214. Inman, V.T., & J.B. Saunders, "Referred pain from skeletal structures," *J Nerv Ment Dis*, Vol 99 (1944).

215. Jackowski, L.T., "Functional hypoadrenia and its relationship to the reoccuring open ileocecal and spastic or closed ileocecal valve syndrome." Proceedings of Summer Meeting, ICAK, Detroit, 1978.

216. Jacobs, B.L., "Serotonin, motor activity and depression-related disorders," *Amer Sci*, Vol 82 (Sep/Oct 1994).

217. Jacobs, G.E., "Applied kinesiology: An experimental evaluation by double blind methodology," *J Manip Physiol Ther*, Vol 4, No 3 (Sep 1981).

218. Jacobs, G.E., T.L. Franks, & P.G. Gilman, "Diagnosis of thyroid dysfunction: Applied kinesiology compared to clinical observations and laboratory tests," *J Manip Physiol Ther*, Vol 7, No 2 (Jun 1984).

219. Jarrett, R.J., & J.C. Gazet, "Studies *in vivo* of the ileocaecocolic sphincter in the cat and dog," *Gut*, Vol 7 (1966).

220. Jenkins, D.J.A. et al., "Glycemic index of foods: A physiological basis for carbohydrate exchange," *Am J Clin Nutr*, Vol 34, No 3 (Mar 1981).

221. Jennings, I.W., *Vitamins in Endocrine Metabolism* (Springfield, IL: Charles C. Thomas Publisher, 1970).

222. Johns, D.R., "Migraine provoked by aspartame," *N Engl J Med*, Vol 315, No 7 (Aug 14, 1986).

223. Johnson, D. et al., "Reactive hypoglycemia," *JAMA*, Vol 243, No 11 (Mar 21, 1980).

224. Kahn, C.R., "Insulin action, diabetogenes, and the cause of type II diabetes," *Diabetes*, Vol 43 (Aug 1994).

225. Kalser, M.H. et al., "Relation of small bowel resection to nutrition in man," *Gastroenterology*, Vol 38, No 4 (Apr 1960).

226. Kannel, W.B., & W.P. Castelli, "Is the serum total cholesterol an anachronism?" *Lancet*, Vol II, No 8149 (Nov 3, 1979).

227. Kelley, M.L., Jr., E.A. Gordon, & J. DeWeese, "Pressure studies of the ileocolonic junctional zone of dogs," *Am J Physiol*, Vol 209 (Aug 1965).

228. Kelley, M.L., Jr., E.A. Gordon, & J. DeWeese, "Pressure responses of canine ileocolonic junctional zone to intestinal distention," *Am J Physiol*, Vol 211 (Sep 1966).

229. Kellgren, J.H., "On the distribution of pain arising from deep somatic structures with charts of segmental pain areas," *Clin Sci*, Vol 4 (Jun 1939).

230. Kellgren, J.H., "Observations on referred pain arising from muscle," *Clin Sci*, Vol 3 (1937).

231. Khoe, W.H., "The treatment of functional hypoglycemia with acupuncture and diet," *Am J Acupunc*, Vol 3, No 4 (Dec 1975).

232. King, J.A., R.A. Barkley, & S. Barrett, "Attention-deficit hyperactivity disorder and the stress response," *Biol Psychiatry*, Vol 44 (1998).

233. Kinsella, J.E., P.H. Yu, & J.B. Mai, "Kidney lipids: Changes caused by dietary 9-trans, 12-trans-octadecadienoate," *Lipids*, Vol 14, No 12 (1979).

234. Kirby, R.W. et al., "Oat-bran intake selectively lowers serum low-density lipoprotein cholesterol concentrations of hypercholesterolemic men," *Am J Clin Nutr*, Vol 34, No 5 (May 1981).

235. Klevay, L.M., "Hypercholesterolemia in rats produced by an increase in the ratio of zinc to copper ingested," *Am J Clin Nutr*, Vol 26 (Oct 1973)

236. Klevay, L.M., "An association between the amount of fat and the ratio of zinc to copper in 71 foods: Inferences about the epidemiology of coronary heart disease," *Nutr Rep Int*, Vol 9, No 6 (Jun 1974).

237. Klevay, L.M., "Hypercholesterolemia due to ascorbic acid," *Proc Soc Exp Biol Med*, Vol 151 (1976).

238. Klevay, L.M., "Interactions of copper and zinc in cardiovascular disease," in *Micronutrient Interactions: Vitamins, Minerals, and Hazardous Elements*, Vol 355, ed O.A. Levander & L. Cheng (New York: NY Acad Sciences, 1980).

239. Klevay, L.M. et al., "The human requirement for copper. 1. Healthy men fed conventional, American diets," *Am J Clin Nutr*, Vol 33 (Jan 1980).

240. Klevay, L.M., "Metabolic interactions among cholesterol, cholic acid and copper," *Nutr Rep Int*, Vol 26, No 3 (Sep 1982).

241. Klevay, L.M. et al., "Increased cholesterol in plasma in a young man during experimental copper depletion," *Metabolism*, Vol 33, No 12 (Dec 1984).

242. Klevay, L.M., "Dietary copper: A powerful determinant of cholesterolemia," *Med Hypoth*, Vol 24 (Oct 1987).

242a. Knapp, H.R., "Nutritional aspects of hypertension," in *Present Knowledge in Nutrition*, 7th ed, ed E.E. Ziegler & L.J. Filer, Jr. (Washington, DC: ILSI Press, 1996).

243. Kohli, D.R., & C.C. Pearson, "A study of hiatus hernia," *Gastroenterology*, Vol 23 (Feb 1953).

244. Kontos, H.A., D.W. Richardson, & J.E. Norvell, "Norepinephrine depletion in idiopathic orthostatic hypotension," *Ann Intern Med*, Vol 82 (1975).

245. Kountz, W., *Thyroid Function and Its Possible Role in Vascular Degeneration* (Springfield, IL: Charles C. Thomas Publisher, 1951).

246. Kowalski, R.E., *The 8-Week Cholesterol Cure* (New York: Harper & Row, 1987).

247. Kummerow, F.A. et al., "The influence of egg consumption on the serum cholesterol level in human subjects," *Am J Clin Nutr*, Vol 30, No 5 (May 1977).

248. Lang, H., "The thirty-second allergy cure." Proceedings of Summer Meeting, ICAK, Dearborn, MI, 1988.

249. Langman, M.J. et al., "Risks of bleeding peptic ulcer associated with individual non-steroidal anti-inflammatory drugs," *Lancet*, Vol 343 (Apr 30, 1994).

250. Langone, J., "Is it a disease, or isn't it? Hypoglycemia," *Discover*, Vol 6, No 11 (Nov 1985).

251. Laragh, J.H., R.L. Letcher, & T.G. Pickering, "Renin profiling for diagnosis and treatment of hypertension," *JAMA*, Vol 241, No 2 (Jan 12, 1979).

252. Larsen, P.R., T.F. Davies, & I.D. Hay, "The thyroid gland," in *Williams Textbook of Endocrinology*, 9th ed, ed J. Wilson et al. (Philadelphia: W.B. Saunders Co, 1998).

253. Lebowitz, M., "Treating the adrenal glands," *Chiro Econ*, Vol 28, No 4 (Jan/Feb 1986).

254. Lebowitz, M., "Screening for essential fatty acid problems: An addition." Proceedings of Summer Meeting, ICAK, Washington, DC, 1987.

255. Lebowitz, M., "Some tips on treating the severely allergic, chemically sensitive patient." Proceedings of Summer Meeting, ICAK, Dearborn, MI, 1988.

256. Lebowitz, M., "A technique to abolish food sensitivities," *Chiro Econ*, Vol 33, No 2 (Sep/Oct 1990).

257. Lee, R., "Sugar and sugar products — their use and abuse," *J Am Acad Applied Nutr*, Vol 3, No 1 (Spring 1950).

258. Leeds, E.M. et al., "Effects of exercise and anabolic steroids on total and lipoprotein cholesterol concentrations in male and female rats," *Med Sci Sports Exer*, Vol 18, No 6 (Dec 1986).
259. Leisman, G., & R.J. Vitori, "Limb segment information transmission capacity infers integrity of spinal transmission tracts and cortical visual-motor control," *Intern J Neuroscience*, Vol 50 (1990).
260. Leisman, G. et al., "Electromyographic effects of fatigue and task repetition on the validity of estimates of strong and weak muscles in applied kinesiological muscle-testing procedures," *Perceptual and Motor Skills*, Vol 80 (1995).
261. Lenoch, F., "Spa therapy in arthritis," in *Arthritis and Physical Medicine*, ed S. Licht (New Haven, CT: Elizabeth Licht, 1969).
262. Levine, L. et al., "Fructose and glucose ingestion and muscle glycogen use during submaximal exercise," *J Appl Psychol*, Vol 55, No 6 (Dec 1983).
263. Liddle, G.W., & K.L. Melmon, "The adrenals," in *Textbook of Endocrinology*, 5th ed, ed R.H. Williams (Philadelphia: W.B. Saunders Co, 1974).
264. Liebman, M., & T. Bazzarre, "Plasma lipids of vegetarian and non-vegetarian males: Effects of egg consumption," *Am J Clin Nutr*, Vol 38, No 4 (Oct 1983).
265. Lipman, B.S., & E. Massie, *Clinical Scalar Electrocardiography*, 5th ed (Chicago: Year Book Medical Publishers, Inc, 1965).
266. Long, J.W., *The Essential Guide to Prescription Drugs* (New York: Harper & Row, 1977).
267. Love, L., "The role of the ileocecal valve in large bowel obstruction," *Radiology*, Vol 75 (Sep 1960).
268. Maffetone, P., *Complementary Sports Medicine* (Champaign, IL: Human Kinetics, 1999).
269. Magoun, H.I., Sr., "A pertinent approach to pituitary pathology," *D.O.*, Vol 11, No 11 (Jul 1971).
270. Mankin, H.J. et al., "Biochemical and metabolic abnormalities in articular cartilage from osteoarthritic human hips. II. Correlation of morphology with biochemical and metabolic data," *J Bone Joint Surg* [Am], Vol 53, No 3 (Apr 1971).
271. Mankin, H.J., M.E. Johnson, & L. Lippiello, "Biochemical and metabolic abnormalities in articular cartilage from osteoarthritic human hips. III. Distribution and metabolism of amino sugar-containing macromolecules," *J Bone Joint Surgery* [Am], Vol 63, No 1 (Jan 1981).
272. Mann, G.V., "Diet-heart: End of an era," *N Engl J Med*, Vol 297, No 12 (Sep 22, 1977).
273. Mannino, J.R., "The application of neurologic reflexes to the treatment of hypertension," *JAOA*, Vol 79, No 4 (Dec 1979).
274. Markham, B.C., "Comparisons of honey and glucose using the oral glucose tolerance test." Proceedings of Winter Meeting, ICAK, Houston, 1978.
275. Martin, J.E., & P.M. Dubbert, "The role of exercise in preventing and moderating blood pressure elevation," in *Non-Pharmacologic Therapy of Hypertension*, ed M.D. Blaufox & H.G. Langford (New York: S. Karger, 1987).
276. Master, A.M. et al., "Differential diagnosis of hiatus hernia and coronary artery disease," *Arch Surg*, Vol 58 (Apr 1949).
277. Mathur, K.S., M.A. Khan, & R.D. Sharma, "Hypocholesterolaemic effect of bengal gram: A long-term study in man," *Br Med J*, Vol 1 (1968).
278. Maxwell, M.H., & A.U. Waks, "Obesity and hypertension," in *Non-Pharmacologic Therapy of Hypertension*, ed M.D. Blaufox & H.G. Langford (New York: S. Karger, 1987).
279. McBride, N.L., "The third side of the ileocecal valve triangle." Proceedings of Summer Meeting, ICAK, Gaylord, MI, 1975.
280. McCarron, D.A., "Calcium and magnesium nutrition in human hypertension," *Ann Intern Med*, Vol 98, Part 2 (1983).
281. McCarron, D.A., & C.D. Morris, "Blood pressure response to oral calcium in persons with mild to moderate hypertension — a randomized, double-blind, placebo-controlled, crossover trial" *Ann Intern Med*, Vol 103, No 6, Part 1 (Dec 1985).
282. McDougall, R.M., K. Walker, & O.G. Thurston, "Effect of increased dietary fiber on biliary cholesterol in patients with gallstones," *Surg Forum*, Vol 28 (1977).
283. McInnis, N., "Breath: A technology of mind?" *Brain/Mind Bulletin*, Vol 8, No 3 (Jan 3, 1983).
284. McMichael, J., "Prevention of coronary heart-disease," *Lancet*, Vol II, No 7985 (Sep 11, 1976).
284a. Medical Research Council Working Party, "MRC trial of treatment of mild hypertension: Principal results," *Br Med J*, Vol 291, No 6488 (Jul 13, 1985).
285. Mega, J.J., "Hypoglycemia — A hygienic viewpoint," *Chiro Econ*, Vol 29, No 1 (Jul/Aug 1986).
286. Meikle, A.W. et al., "The presence of a dehydroepiandrosterone-specific receptor binding complex in murine T cells," *J Steroid Biochem Mol Biol*, Vol 42, Nos 3-4 (May 1992).
287. Melo Gomes, J.A. et al., "Double-blind comparison of efficacy and gastroduodenal safety of diclofenac/misoprostol, piroxicam, and naproxen in the treatment of osteoarthritis," *Ann Rheum Dis*, Vol 52, No 12 (Dec 1993).
288. Miettinen, T.A., "Mechanism of serum cholesterol reduction by thyroid hormones in hypothyroidism," *J Lab Clin Med*, Vol 71, No 4 (Apr 1968).
289. Mirkin, G., & C. Morton, "A closer look at food allergies," *Runner's World* (Mar 1985).
290. Miyamoto, Y., et al., "Endothelial nitric oxide synthase gene is positively associated with essential hypertension," *Hypertension*, Vol 32, No 1 (Jul 1998).
291. Morgan, K.J., & M.E. Zabik, "Amount and food sources of total sugar intake by children ages 5 to 12 years," *Am J Clin Nutr*, Vol 34, No 3 (Mar 1981).
292. Mowles, R., & W.H. Schmitt, Jr., "An applied kinesiological approach to candida albicans allergy," *Chiro Econ*, Vol 28, No 4 (Jan/Feb 1986).
293. Murray, R. et al., "The effect of fluid and carbohydrate feedings during intermittent cycling exercises," *Med Sci Sports Exer*, Vol 19, No 6 (Dec 1987).
294. National Research Council, Food & Nutrition Board, A.E. Harper, Chmn, *Toward Healthful Diets* (Washington, DC: National Acad Sci, 1980).
295. Navidi, M.K., & F.A. Kummerow, "Nutritional value of Egg Beaters® compared with 'farm fresh eggs,'" *Pediatrics*, Vol 53, No 4 (Apr 1974).
296. Nielsen, F. H., "Other trace elements," in *Present Knowledge in Nutrition*, 7th ed, ed E.E. Ziegler & L.J. Filer, Jr. (Washington, DC: ILSI Press, 1996).
297. O'Dea, K., "Diabetes and diet," *Science*, Vol 221 (Jul 15, 1983).
298. Oettle, G.J., P.M. Emmett, & K.W. Heaton, "Glucose and insulin reponses to manufactured and whole-food snacks," *Am J Clin Nutr*, Vol 45 (Jan 1987).
299. Okano, G. et al., "Effect of pre-exercise fructose ingestion on endurance performance in fed men," *Med Sci Sports Exer*, Vol 20, No 2 (Apr 1988).
300. Ott, J.N., *Health and Light* (New York: Pocket Books, 1976).
301. Owen, M.D. et al., "Effects of ingesting carbohydrate beverages during exercise in the heat," *Med Sci Sports Exer*, Vol 18, No 5 (Oct 1986).
302. Padgett, D.A., & R.M. Loria, "*In vitro* potentiation of lymphocyte activation of dehydroepiandosterone, androstenediol, and androstenetriol," *J Immunol*, Vol 153, No 4 (Aug 15, 1994).
303. Pahlin, P.E., & J. Kewenter, "The vagal control of the ileo-cecal sphincter in the cat," *Acta Physiol Scand*, Vol 96 (1976).
304. Pahlin, P.E., & J. Kewenter, "Reflexogenic contraction of the ileo-cecal sphincter in the cat following small or large intestinal distension," *Acta Physiol Scand*, Vol 95 (1975).
305. Palmoski, M.J., R.A. Colyer, & K.D. Brandt, "Marked suppression by salicylate of the augmented proteoglycan synthesis in osteoarthritis cartilage," *Arthritis Rheum*, Vol 23, No 1 (Jan 1980).
306. Panayi, G.S., "The pathogenesis of rheumatoid arthritis: From molecules to the whole patient," *Br J Rheumatology*, Vol 32

(1993).
307. Paul, L.W., & J.H. Juhl, *The Essentials of Roentgen Interpretation*, 2nd ed (New York: Harper & Row, 1965).
308. Perneger, T.V., P.K. Whelton, & M.J. Klag, "Risk of kidney failure associated with the use of acetaminophen, aspirin, and nonsteroidal antiinflammatory drugs," *N Engl J Med*, Vol 331, No 25 (Dec 22, 1994).
309. Petrovsky, N., & L.C. Harrison, "The chronobiology of human cytokine production," *Intern Rev Immunol*, Vol 16 (1998).
310. Pfeiffer, C.C., *Mental and Elemental Nutrients* (New Canaan, CT: Keats Publishing, Inc, 1975).
311. Philpott, W.H., *Ecologic Medicine Manual* (Oklahoma City: privately published, 1975).
312. Philpott, W.H., & D.K. Kalita, *Brain Allergies — The Psycho-Nutrient Connection* (New Canaan, CT: Keats Publishing, Inc, 1980).
312a. Pickering, T.G. et al., "What is the role of ambulatory blood pressure monitoring in the management of hypertensive patients?" *Hypertension*, Vol 7, No 2 (Mar/Apr 1985).
313. Plotsky, P.M., M.J. Owens, & C.B. Nemeroff, "Psychoneuroendocrinology of depression — hypothalamic-pituitary-adrenal axis," *Psych Clin N Amer*, Vol 21, No 2 (Jun 1998).
314. Porter, M.W. et al., "Effect of dietary egg on serum cholesterol and triglyceride of human males," *Am J Clin Nutr*, Vol 30, No 4 (Apr 1977).
315. Porzio, R.J., "Frequency of increased second heart sounds." Proceedings of Winter Meeting, ICAK, Palm Desert, CA, 1987.
316. Pottenger, F.M., Jr., "Evidence of the protective influence of adrenal hormones against tuberculosis in guinea pigs," *Endocrinology*, Vol 21, No 4 (Jul 1937).
317. Pottenger, F.M., Jr., "The use of adrenal cortex in the treatment of the common cold," *Med Rec* (Feb 16, 1938).
318. Pottenger, F.M., Jr., "Non specific methods for the treatment of allergic states," *J Applied Nutr*, Vol 17, No 4 (Dec 1964).
319. Pottenger, F.M., Jr., & B. Krohn, "Reduction of hypercholesterolemia by high-fat diet plus soybean phospholipids," *Am J Digest Dis*, Vol 19, No 4 (Apr 1952).
320. Pottenger, F.M., Jr., & B. Krohn, "Emergency treatment of the asthmatic with special reference to adrenal cortex and vitamin B12," *Rocky Mtn Med J* (Jul 1952).
321. Pottenger, F.M., Jr., & F.M. Pottenger, "Adrenal cortex in treating childhood asthma: Clinical evaluation of its use," *CA West Med*, Vol 49, No 4 (Oct 1938).
322. Pottenger, F.M., Jr., R.T. Pottenger, & F.M. Pottenger, "The treatment of asthma with special reference to the oral use of the adrenal hormones and sodium chloride," *CA West Med*, Vol 43, No 1 (Jul 1935).
323. Pottenger, F.M., Jr., & D.G. Simonsen, "An orally-active sex-maturation fraction from the adrenal gland," *Endocrinology*, Vol 22, No 2 (Feb 1938).
324. Prinz, R.J., W.A. Roberts, & E. Hantman, "Dietary correlates of hyperactive behavior in children," *J Consulting Clin Psychol*, Vol 48, No 6 (1980).
325. Quintao, E., S.M. Grundy, & E.H. Ahrens, Jr., "Effects of dietary cholesterol on the regulation of total body cholesterol in man," *J Lipid Res*, Vol 12 (1971).
326. Rabinovitz, M., & D.H. Van Thiel, "Hepatotoxicity of nonsteroidal anti-inflammatory drugs," *Am J Gastroenterol*, Vol 87, No 12 (Dec 1992).
327. Rarey, L.E., "Chronic ileo-cecal valve incompetence in relation to high fiber diets." Proceedings of Summer Meeting, ICAK, Detroit, 1977.
328. Reaven, G.M., "Role of insulin resistance in human disease," *Diabetes*, Vol 37, No 12 (Dec 1988).
329. Reaven, G.M., "Are triglycerides important as a risk factor for coronary disease?" *Heart Dis Stroke*, Vol 2, No 1 (Jan-Feb 1993).
330. Reaven, G.M., "Role of insulin resistance in human disease (Syndrome X): An expanded definition," *Annu Rev Med*, Vol 44 (1993).
331. Reaven, G.M. et al., "Insulin resistance and insulin secretion are determinants of oral glucose tolerance in normal individuals," *Diabetes*, Vol 42, No 9 (Sep 1993).
332. Reaven, G.M., "Insulin resistance and human disease: A short history," *J Basic & Clin Physiol & Pharmacol*, Vol 9, Nos 2-4 (1998).
333. Reiser, S. et al., "Effect of copper intake on blood cholesterol and its lipoprotein distribution in men," *Nutr Rep Int*, Vol 36, No 3 (Sep 1987).
334. Rendleman, D.F. et al., "Reflux pressure studies on the ileocecal valve of dogs and humans," *Surgery*, Vol 44 (1958).
335. Ridgway, E.C. et al., "Peripheral responses to thyroid hormone before and after L-thyroxine therapy in patients with subclinical hypothyroidism," *J Clin Endocrinol Metab*, Vol 53, No 6 (1981).
336. Ridgway, E.C. et al., "Peripheral responses to thyroid hormone before and after L-thyroxine therapy in patients with subclinical hypothyroidism," *J Clin Endocrinol Metab*, Vol 53 (Dec 1981), mentioned in the *1988 Year Book of Endocrinology*, ed T.B. Schwartz & W.G. Ryan (Chicago: Year Book Medical Publishers, Inc, 1983).
337. Ringsdorf, W.M., Jr., & E. Cheraskin, "Periodontal pathosis in man: I. Effect of relatively high-protein low-refined-carbohydrate diet upon sulcus delpth," *J Periodontol*, Vol 33 (1962).
338. Rinse, J., "Atherosclerosis, a pluri-deficiency disease," *Am Chiro* (Jan 1987).
339. Roberts, H.J., *Defense Against Alzheimer's Disease (DAAD)* (W. Palm Beach, FL: Sunshine Sentinel Press, Inc, 1995).
340. Roberts, S.E., *Exhaustion — Causes and Treatment* (Emmaus, PA: Rodale Books, Inc, 1967).
341. Rochon, P.A. et al., "A study of manufacturer-supported trials of nonsteroidal anti-infammatory drugs in the treatment of arthritis," *Arch Intern Med*, Vol 154, No 2 (Jan 24, 1994.
342. Rosenberg, I.H., & E.J. Schaefer, "Dietary saturated fatty acids and blood cholesterol," *N Engl J Med*, Vol 318, No 19 (May 12, 1988).
343. Rosenfeld, R.S. et al., "24-Hour secretory pattern of dehydroisoandrosterone and dehydroisoandrosterone sulfate," *J Clin Endocrinol Metab*, Vol 40, No 5 (May 1975).
344. Rosenfeld, R.S., L. Hellman, & T.F. Gallagher, "Metabolism and interconversion of dehydroisoandrosterone and dehydroisoandrosterone sulfate," *J Clin Endocrinol Metab*, Vol 35, No 2 (Aug 1972).
345. Roth, S.H., "Nonsteroidal anti-inflammatory drugs: Gastropathy, deaths, and medical practice," *Ann Intern Med*, Vol 109, No 5 (Sep 1, 1988).
346. Rubel, L.L., *The GP and the Endocrine Glands* (Decatur, IL: privately published, 1959).
347. Rubin, M.R. et al., "Adrenergic regulation of ileocecal sphincter function in the cat," *Gastroenterology*, Vol 78, No 1 (Jan 1980).
348. Safrit, H.F., "Thyroid disorders," in *Handbook of Clinical Endocrinology*, ed P.A. Fitzgerald (Chicago: Jones Medical Publishers, 1986).
349. Sanborn, C.F., B.H. Albrecht, & W.W. Wagner, Jr., "Athletic amenorrhea: Lack of association with body fat," *Med Sci Sports Exer*, Vol 19, No 3 (Jun 1987).
350. Sato, A., & R.S. Swenson, "Sympathetic nervous system response to mechanical stress of the spinal column in rats," *J Manip Physiol Ther*, Vol 7, No 3 (Sep 1984).
351. Schlaepfer, K., "A further note on the motor innervation of the diaphragm," *Anat Rec*, Vol 32 (1926).
352. Schleyer-Saunders, E., *Backache in Women* (Bristol, England: John Wright & Sons Ltd, 1966).
353. Schmitt, W.H., Jr., "Applied kinesiological diagnosis of hypothyroidism — An unusual familial pattern." Proceedings of Summer Meeting, ICAK, Detroit, 1976.
354. Schmitt, W.H., Jr., *Compiled Notes on Clinical Nutritional Products* (Grosse Pointe Farms, MI: AK Study Program, 1979).

355. Schmitt, W.H., Jr., "Retrograde lymphatic blockage and the pectoral stretch technique: An AK indicator for iron." Proceedings of Winter Meeting, ICAK, Acapulco, Mexico, 1981.
356. Schmitt, W.H., Jr., *Common Glandular Dysfunctions in the General Practice — An Applied Kinesiological Approach* (Chapel Hill, NC: AK Study Program, 1981).
357. Schmitt, W.H., Jr., "Applied kinesiological observations of allergic patients — Part I," *Chiro Econ*, Vol 27, No 1 (Jul/Aug 1984).
358. Schmitt, W.H., Jr., "Applied kinesiological observations of allergic patients — Part 2," *Chiro Econ*, Vol 27, No 2 (Sep/Oct 1984).
359. Schmitt, W.H., Jr., "Fundamentals of essential fatty acid metabolism, Part I," *Chiro Econ*, Vol 28, No 1 (Jul/Aug 1985).
360. Schmitt, W.H., Jr., "Fundamentals of essential fatty acid metabolism, Part II," *Chiro Econ*, Vol 28, No 2 (Sep/Oct 1985).
361. Schmitt, W.H., Jr., "The use of phonocardiography in the identification of functional problems." Proceedings of Summer Meeting, ICAK, Washington, DC, 1987.
362. Schmitt, W.H., Jr., "Molybdenum for candida albicans patients and other problems," in *1987 Selected Papers of the International College of Applied Kinesiology*, ed D.H. Schusterman (Shawnee Mission, KS: ICAK, 1987).
363. Schmitt, W.H., Jr., "Molybdenum for candida albicans patients and other problems," *Chiro Econ*, Vol 31, No 4 (Jan/Feb 1989).
364. Schmitt, W.H., Jr., Video Education Program: *Liver Detoxification Technique* (1993). 1926 Overland Dr., Chapel Hill, NC 27514 [www.theuplink.com].
365. Schmitt, W.H., Jr, "The liver, adrenals, joints, and sulfate," *Am Chiro* (Mar/Apr 1995).
366. Schmitt, W.H., Jr., Video Education Program: *Degeneration intervention. The importance of sulfation* (1995). 1926 Overland Dr., Chapel Hill, NC 27514 [www.theuplink.com].
367. Schmitt, W.H., Jr., Video Education Program: *Adrenal Stress Diagnosis and Treatment Options* (1995). 1926 Overland Dr., Chapel Hill, NC 27514 [www.theuplink.com].
368. Schmitt, W.H., Jr., & G. Leisman, "Correlation of applied kinesiology muscle testing findings with serum immunoglobulin levels for food allergies," *Int J Neurosci*, Vol 96, Nos 3-4 (Dec 1998).
369. Schmitt, W.H., Jr., & G. Leisman, "Correlation of applied kinesiology muscle testing findings with serum immunologlobulin levels for food allergies," *AK Review*, Vol 8, No 1 (Spring 1999). Reprinted from the *International Journal of Neuroscience*, Vol 96, Nos 3-4 (1998).
370. Schuster, M.M., "Disorders of motility," in *Cecil Textbook of Medicine*, 15th ed, ed P.B. Beeson, W. McDermott, & J. Wyngaarden (Philadelphia: W.B. Saunders Co, 1979).
371. Schwartz, T.B., & W.G. Ryan, *1979 Year Book of Endocrinology* (Chicago: Year Book Medical Publishers, Inc, 1979).
372. Schwartz, T.B., & W.G. Ryan, *1982 Year Book of Endocrinology* (Chicago: Year Book Medical Publishers, Inc, 1982).
373. Schwartz, T.B., & W.G. Ryan, *1983 Year Book of Endocrinology* (Chicago: Year Book Medical Publishers, Inc, 1983).
374. Scopp, A.L., "An experimental evaluation of kinesiology in allergy and deficiency disease diagnosis," *J Orthomolec Psychiatry*, Vol 7, No 2 (1978).
375. Selye, H., *The Stress of Life* (New York: McGraw-Hill Book Co, 1956).
376. Selye, H., *The Stress of Life*, revised ed (New York: McGraw-Hill Book Co, 1976).
377. Seugling, R.A., Jr., "Incidence of the sphenobasilar faults in functional hypoadrenia," in *1982 Selected Papers of the International College of Applied Kinesiology*, ed W.H. Schmitt, Jr. (Detroit, MI: ICAK, 1982).
378. Sewall, H., "On the clinical significance of postural changes in the blood-pressures, and the secondary waves of arterial blood-pressure," *Am J Med Sci*, Vol 158 (1918).
379. Shehata, R., "The crura of the diaphragm and their nerve supply," *Acta Anat*, Vol 63 (1966).
380. Sherman, L., M. Goldberg, & F.C. Larson, "The Achilles reflex — a diagnostic test of thyroid dysfunction," *Lancet* (Feb 2, 1963).
381. Simonds, J.P., & O.E. Helpler, "Fat tolerance in experimental hyperthyroidism," *JAMA*, Vol 98, No 4 (Jan 23, 1932).
382. Simpson, G.M., J.H. Blair, & G.R. Nartowicz, "Diagnostic limitations of Achilles tendon reflex in thyroid disease," *NY State J Med*, Vol 63 (Apr 15, 1963).
383. Smith, A.D., "Dietary fat, carcinogenesis and the immune response: Can diets rich in polyunsaturated fatty acids be harmful?" in *The Role of Fats in Human Nutrition*, ed F.B. Padley & J. Podmore (Chichester, England: Ellis Horwood, 1985).
384. Smith, L.H., Jr., & S.O. Thier, *Pathophysiology — The Biological Principles of Disease*, Vol I (Philadelphia: W.B. Saunders Co, 1981).
385. Snider, T.H., J.P. Stevens, & F.M. Wilner, "Simple bedside test of respiratory function," *JAMA*, Vol 170, No 14 (Aug 1, 1959).
386. Snow, H.L., "Refined sugar: Its use and misuse," *The Improvement Era*, Vol 51, No 3 (Mar 1948).
387. Soyka, F., with A. Edmonds, *The Ion Effect* (New York: E.P. Dutton & Co, Inc, 1977).
388. Stamler, J., "Public health aspects of optimal serum lipid-lipoprotein levels," *Prevent Med*, Vol 8 (1979).
389. Stavish, G.E., "A case management presentation with multiple allergic involvement, Part II," *Chiro Econ*, Vol 29, No 3 (Nov/Dec 1986).
390. Stavish, G.E., "The role of nutrition in allergy," *Today's Chiro*, Vol 16, No 1 (Mar/Apr 1987).
391. Stavish, P.C., "Differentiating neurogenic triggers for the allergic reaction vs. atopic IgE mediated allergy," *Chiro Econ*, Vol 30, No 5 (Mar/Apr 1988).
392. Stoner, F., *The Eclectic Approach to Chiropractic* (Las Vegas: F.L.S. Publishing Co, 1975).
393. Straub, R.H. et al., "Association of humoral markers of inflammation and dehydroepiandrosterone sulfate or cortisol serum levels in patients with chronic inflammatory bowel disease," *Amer J Gastroenterology*, Vol 93, No 11 (1998).
394. Suzuki, T. et al., "Dehydroepiandrosterone enhances IL2 production and cytotoxic effector function of human T cells," *Clin Immunol Immunopathol*, Vol 61 [2 pt 1] (Nov 1991).
395. Taylor, W.C. et al., "Cholesterol reduction and life expectancy — a model incorporating multiple risk factors," *Ann Intern Med*, Vol 106, No 4 (Apr 1987).
396. Teplick, J.G., & M.E. Haskin, *Roentgenologic Diagnosis*, Vol II, 3rd ed (Philadelphia: W.B. Saunders Co, 1976).
397. Tobey, T.A. et al., "Relationship between insulin resistance, insulin secretion, very low density lipoprotein kinetics, and plasma triglyceride levels in normotriglyceride man," *Metabolism*, Vol 30, No 2 (Feb 1981).
398. Tran, T.A., & J.D. Kirby, "The effects of upper thoracic adjustment upon the normal physiology of the heart," *ACA J Chiro*, Vol 14, No 3 (Mar 1977).
399. Trowbridge, J.P., & M. Walker, *The Yeast Syndrome* (New York: Bantam Books, 1986).
400. Trowell, H., "Dietary fibre and coronary heart disease," *Rev Europ Etudes Clin et Biol*, Vol XVII (1972).
401. Trowell, H.C., "Fiber: A natural hypocholesterolemic agent," *Am J Clin Nutr*, Vol 25 (1972).
402. Truss, C.O., *The Missing Diagnosis* (Birmingham, AL: privately published, 1983).
403. Truswell, A.S., "Diet and plasma lipids — a reappraisal," *Am J Clin Nutr*, Vol 31, No 6 (Jun 1978).
404. Turner, C.C., *General Endocrinology*, 4th ed (Philadelphia: W.B. Saunders Co, 1966).
405. U.S. News & World Report, "More good news about 'good cholesterol,'" *U.S. News & World Report* (Nov 23, 1987).
406. Ulin, A.W., & J. Deutsch, "Visualization of ileocecal papilla in a living subject," *Gastroenterology*, Vol 16 (Oct 1960).

407. Ulin, A.W., W.C. Shoemaker, & J. Deutsch, "The ileocecal valve and papilla," *Arch Intern Med*, Vol 97 (Apr 1956).
408. Unger, R.H., & D.W. Foster, "Diabetes mellitus," in *Williams Textbook of Endocrinology,* 9th ed, ed J. Wilson et al. (Philadelphia: W.B. Saunders Co, 1998).
409. Van Arsdel, P.P., Jr., "Allergy, immunology, and hormones," in *Textbook of Endocrinology*, 5th ed, ed R.H. Williams (Philadelphia: W.B. Saunders Co, 1974).
410. Van Horn, L.V. et al., "Serum lipid response to oat product intake with a fat-modified diet," *J Am Dietetic Assoc*, Vol 86, No 6 (Jun 1986).
411. Vitalor Instrument, manufactured by Air Shields, Inc., Narco Health Co., 330 Jacksonville Rd., Halboro, PA 19040.
412. Wade, C., "Black currant seed oil — A new supply of gamma linolenic acid, alpha linolenic acid, and stearidonic acid," *Am Chiro* (Jan/Feb 1987).
413. Walther, D.S., *Applied Kinesiology — The Advanced Approach in Chiropractic* (Pueblo, CO: Systems DC, 1976).
414. Wangensteen, O.H., "The management of acute intestinal obstruction; with special mention of the character of the vomiting and distension," *Lancet*, Vol 54 (Oct 15, 1934).
415. Weisbrodt, N.W., "Motility of the large intestine," in *Gastrointestinal Physiology*, ed L.R. Johnson (St. Louis: The C.V. Mosby Co, 1977).
416. Wellness Letter staff, "A food label lexicon," *Wellness Letter* (U of CA, Berkeley), Vol 3, No 12 (Sep 1987).
417. Werbach, M.R., *Nutritional Influences on Illness* (New Canaan, CT: Keats Publishing, Inc, 1988).
418. Whillis, J., "Lower end of the oesophagus," *J Anat*, Vol 66 (1931).
419. White, J.E., & G.A. Baldt, "High blood pressure," *Chiro Econ*, Vol 29, No 5 (Mar/Apr 1987).
420. Williams, P.L., & R. Warwick, eds, *Gray's Anatomy*, 36th Br ed (Philadelphia: W.B. Saunders Co, 1980).
421. Williams, R.J., *Biochemical Individuality — The Basis for the Genetotrophic Concept* (Austin, TX: Univ of Texas Press, 1956).
422. Willits, B.S., "Burnout," *ACA J Chiro*, Vol 24, No 6 (Jun 1987).
423. Wurtman, R.J., "Aspartame: Possible effect on seizure susceptibility," *Lancet* (Nov 9, 1985).
424. Wurtman, R.J., "The effects of light on the human body," *Sci Am*, Vol 233, No 1 (Jul 1975).
425. Wurtman, R.J. & D.P. Cardinali, "The pineal organ," in *Textbook of Endocrinology*, 5th ed, ed R.H. Williams (Philadelphia: W.B. Saunders Co, 1974).
426. Wynder, E.L., & P. Hill, "Blood lipids: How normal is normal?" *Prevent Med*, Vol 1 (Mar 1972).
427. Yager, J., & R.T. Young, "Non-hypoglycemia is an epidemic condition," *N Engl J Med*, Vol 291, No 17 (Oct 24, 1974).
428. Yates, F.E., D.J. Marsh, & J.W. Maran, "The adrenal cortex," in *Medical Physiology*, Vol 2, 14th ed, ed V.B. Mountcastle (St. Louis: The C.V. Mosby Co, 1980).
429. Yoffey, J.M., & F.C. Courtice, *Lymphatics, Lymph and the Lymphomyeloid Complex* (New York: Academic Press, 1970).
430. Yoo, J.U., R.S. Papay, & C.J. Malemud, "Suppression of proteoglycan synthesis in chondrocyte cultures derived from canine intervertebral disc," *Spine*, Vol 17, No 2 (Feb 1992).
431. Zavaroni, I. et al., "Risk factors for coronary artery disease in healthy persons with hyperinsulinemia and normal glucose tolerance," *N Engl J Med*, Vol 320, No 11 (Mar 16, 1989).
432. Ziegler, M.G., "Postural hypotension," *Ann Rev Med*, Vol 31 (1980).
433. Ziegler, M.G. et al., "The sympathetic-nervous-system defect in primary orthostatic hypotension," *N Engl J Med*, Vol 296, No 6 (Feb 10, 1977).
434. Zimmet, P.Z. et al., "Hyperinsulinaemia in youth is a predictor of type 2 (non-insulin dependent) diabetes mellitus," *Diabetologia*, Vol 35, No 6 (Jun 1992).
435. Zink, J.G., & W.B. Lawson, "The role of pectoral traction in the treatment of lymphatic flow disturbances," *Osteo Ann*, Vol 6, No 11 (Nov 1978).

Glossary

Acupuncture meridian connector: a connection between the spine and the meridian system that correlates with the associated point in meridian therapy. Associated points are points along the spinal column on the bladder meridian. Where there is an active associated point, there will typically be an adjacent subluxation.

Adrenal stress disorder: failure of the adrenal glands to adequately produce secretions necessary for the body to function in an optimal manner. This condition is relative hypoadrenia or other imbalance often associated with chronic stress of some type. Adrenal stess disorder incorporates the hypothalamic-pituitary-adrenal axis and replaces the term "relative hypoadrenia" used in the first edition of this text.

Alarm points: diagnostic points of the meridian system located in various areas of the body.

Associated points: acupuncture points located along the bladder meridian adjacent to the spinal column that correlate with probable vertebral subluxations when the associated point is active.

Beginning and ending technique (B&E): a technique developed in applied kinesiology that indicates when the Yang meridian points that begin or end on the head need stimulation.

Blood sugar handling stress: the body's organs and/or glands are stressed in maintaining the blood sugar levels within normal or near-normal ranges. It is not indicative of pathologic processes.

Body into distortion (BID): reproducing in a prone, supine, or seated patient a postural distortion previously observed in the standing patient.

Body language: a term incorporating all the observable diagnostic factors in the various processes of dysfunction and disease.

Centering reflexes: reflexes that maintain balance between the two sides of the body. The afferent supply is from the equilibrium and other proprioceptors dealing with orientation in space. The cloacal reflexes are considered part of the centering reflex mechanism in applied kinesiology.

Challenge: a mechanism used as a testing procedure to determine the body's ability to cope with external stimuli, which can be physical, chemical, or mental. An example of a physical stimulus is pushing on an articulation and determining muscle strength change. Chemical stimulus occurs when one inhales potentially toxic chemicals or chews nutritional factors. Mental stimuli include thought processes, either pleasant or unpleasant to the individual. After an external stimulus is applied, muscle testing procedures are done to determine an improvement in or weakening of the muscle strength as a result of the stimulus.

Concentric muscle contraction: contraction of a muscle when its force exceeds the external force of resistance and the muscle shortens (also called isotonic muscle contraction).

Conditionally facilitated or inhibited: a muscle that tests "strong" or "weak" respectively using AK manual muscle testing.

Cranial faults: failure of the skull to move in its normal, rhythmic manner, as discovered by Sutherland and Cottam and researched by many others.

Crawling: as used by Doman and Delacato, crawling is mobility with the body in contact with the floor. The extremities move in a homolateral fashion typical of an amphibian. As used in applied kinesiology, crawling is a cross-pattern mobility along the floor on the hands and knees; this is called "creeping" in Doman and Delacato's work.

Creeping: as used by Doman and Delacato, creeping is mobility along the floor in a cross-pattern hands-and-knees activity.

Cross-crawl pattern: an exercise to assist in the re-patterning of certain central nervous system functions.

Cutaneous receptors: specialized proprioceptors located in the skin. They are classified as mechanoreceptors, thermoreceptors, and nociceptors.

Dural tension: the disrelation of the dura mater and the spinal column. Activity causes this disrelationship to interfere with normal nerve function.

Dysponesis: "dys" (bad, faulty, or wrong) and "ponos" (effort, work, or toil); a physiopathologic state composed of errors in action-potential output, mainly covert in nature, from the pre-motor and motor cortex. It includes the consequent action-potentials in descending pathways, side branches, lower motor neurons, skeletal muscles, and the various feedback pathways. Often used loosely to refer to faulty effort of the body's signaling systems, producing signals inappropriate for the body's needs.

Eccentric muscle contraction: a muscle contracting against an external force that is greater than the internal force of the muscle, thus causing it to lengthen while continuing to maintain tension.

Equilibrium reflex synchronization technique: digital contact on specific reflex centers or cranial/pelvic correction to normalize neuromuscular coordination relative to equilibrium centering reflexes thought to be in pelvic ligaments with the labyrinthine righting reflexes, tonic neck reflexes, and/or visual righting reflexes. This was previously called the "cloacal synchronization reflex technique."

Exteroceptor: a sensory nerve ending that receives its stimulation from the immediate external environment.

Eyes into distortion (EID): a functional change is observed when the patient turns his eyes in the direction of body distortion, as observed on postural analysis.

Eyes out of distortion (EOD): the eyes are turned in the direction opposite that of EID, and a weak associated muscle strengthens.

Fascial release (flush) technique: deep massage applied to re-establish the normal interface relationship between fascia and muscle.

Fifty-one percenter: a term indicating a muscle that tests strong in the clear but weakens when one of the five IVF factors is therapy localized. This term has been replaced in this text with "subclinical weakness" or "subclinical condition."

Five factors of the IVF: five factors involved with the intervertebral foramen that include the nerve, blood vascular, lymphatic, cerebrospinal fluid activity, and acupuncture meridian connector.

Fixation: a condition in which there is lack of normal motion between vertebrae, usually three vertebrae in number.

Gait reflex points: meridian acupuncture points primarily across the top of the metatarsal arches which, when stimulated, can improve the synchronization of activity relying on crossed-reciprocal innervation (walking, running, swimming).

GAG: glycosaminoglycans, negatively charged carbohydrates that among other things contribute to the ground substance of connective tissue.

Hypoadrenia, relative: failure of the adrenal glands to adequately produce secretions necessary for the body to function in an optimal manner — see also adrenal stress disorder.

Indicator muscle: a muscle tested to determine if there is a change in its strength as a result of some testing mechanism applied to the body. Generally an indicator muscle is strong prior to the test, and weakens as a result of the testing procedure.

Injury recall technique: method to remove from memory the residuals of historical trauma.

Intermittent cold with stretch: treatment approach described by Travell and Simons for trigger points. This term replaces "spray and stretch."

In the clear: testing a muscle but doing nothing to influence either its strength or its weakness.

Ionization technique: a nasal respiratory technique based on the hypothesis that major respiration is intensified in one nostril for a period of time, then alternated to the other nostril. The balance of alternation is hypothesized to affect ionization balance in the body.

Isometric muscle contraction: muscle contraction producing power, but the external force is equal to the power produced by the muscle so there is no change in its length

while contracting.

Isotonic muscle contraction: contraction of a muscle when its force exceeds the external force of resistance, and the muscle shortens (also called concentric muscle contraction).

Laser: light amplification by stimulated emission of radiation; a source of coherent light.

Ligament interlink: a concept suggesting that ligaments in contralateral and analogous joints (e.g., knee and opposite elbow, hip and opposite shoulder) are related neurologically. Normal function is required for articular integrity. A special case is the TMJ, which appears to be similarly linked but to **any** other joint.

Ligament stretch reaction: an abnormal condition in which muscles associated with an articulation weaken temporarily after the ligaments of the articulation have been stretched.

Muscle stretch reaction: an abnormal condition in which the muscle weakens temporarily immediately after it has been mildly stretched.(This diagnostic test is indication to use fascial release, myofascial gelosis, or intermittent cold with stretch technique for correction.)

Myofascial gelosis: localized gelling of muscle proteins causing tenderness and muscle shortness. Muscle involved with myofascial gelosis tests weak after fibers are compressed by pinching and respond to purcussion.

Neurologic disorganization: an abnormal condition in which the nervous system signals or interprets signals improperly, causing apparent confusion within the body. Of particular importance in applied kinesiology examination because erroneous information may be derived from the various testing procedures. The condition is sometimes called "switching."

Neurolymphatic reflex: reflex points, originally developed by an osteopath named Chapman, to stimulate lymphatic drainage of various areas throughout the body.

Neurovascular reflex: reflex points that appear to influence circulatory function in various areas of the body. Originally discovered by a chiropractor named Bennett. The reflexes used in applied kinesiology are primarily about the head. Bennett's reflexes are located over much of the body, but they have not been correlated in applied kinesiology.

Nutritional testing: oral and/or nasal stimulation of nerve receptors that enhances or adversely affects body function.

Ocular lock: a screening test for neurologic disorganization, which is a failure of the eyes to work together effectively.

Operator prejudice: any attitude of mind or misuse of technique on the part of one performing muscle testing procedures, resulting in unreproducible, paradoxical, or otherwise invalid findings.

Origin/insertion technique: a technique of strengthening muscles by stimulating the origin and insertion.

Pelvic category I: a torsion of the total pelvic structure with no osseous misalignment of the sacroiliac articulations or the symphysis pubis. May cause total body adaptation, particularly at the 1st rib head, shoulder outlet, and cranium.

Pelvic category II: an osseous subluxation at the sacroiliac articulation.

Pelvic category III: involves an intact pelvis. The involvement is pelvic relation with the lumbar spine.

PiLUS technique: tests for predictable weakening of the right piriformis, iliacus, and sternocleidomastoid and left latissimus dorsi and upper trapezius with lumbar flexion. PiLUS is the acronym for the muscles involved and is pronounced "plus."

PRYT technique: an examination technique of the major body modules and their ability neurologically to function individually and together. Acronym derived from an airplane's attitudes: pitch, roll, yaw, and tilt.

Pulse points: diagnostic points of the meridian system located in both wrists over the radial artery.

Pulse testing: a method of testing to determine the hypersensitivity of an individual to a particular substance.

Ragland's sign: an abnormal drop in the systolic blood pressure when a patient rises from a supine to a standing position.

Reactive muscle: a muscle temporarily inhibited because of inappropriate proprioceptive impulses from another previously contracted muscle.

Rib pump technique: the thoracic cage and thoracic diaphragm must have free movement and organization with the cranial and pelvic diaphragms. The rib pump technique examines for dysfunction that is corrected with strain/counterstrain technique.

RMAPI: acronym for "repeated muscle activation patient induced." This is an AK technique to correct muscles that become weak after the person repeatedly contracts the muscle.

Running a meridian: the manual tracing of a meridian to alter its relative energy level in order to facilitate (or inhibit) muscle-organ/gland function.

Shock absorber test: a method of placing a mechanical shock into an articulation to determine if there might be a subluxation. Used primarily as a screening technique.

Stomatognathic system: a dental term that denotes total correlation and interdependence of the cranial bones, TMJ, hyoid muscles, local lymphatic, blood, nerve supply, and other tissues in those areas.

Strain/counterstrain: a technique developed by Jones that describes the cause and treatment of a type of muscle dysfunction. The dysfunction is the result of overstretching muscles, tendons, fascia, and ligaments.

Stress receptors: reflex points located around the head that are capable of inhibiting or facilitating muscle function.

"Strong" muscle: a muscle that develops full power and has immediate locking when manually tested.

Subclinical condition: one in which the body's effort to maintain homeostasis is sufficient to test strong with manual muscle testing; however, the muscle tests weak when a reflex point is therapy localized or some other test is applied. Formerly called a "fifty-one percenter."

Switching: a condition in which neurologic disorganization is present within the body. Of particular concern because when switching is present, erroneous information may be derived from various testing procedures.

Temporal tap: a method used to temporarily disturb sensory filtering mechanisms in the brain in order to monitor the degree of the therapy's effect, or to aid in the modification of habit patterns.

Then and now technique: a meridian therapy diagnostic technique developed in applied kinesiology that uses the twenty-four-hour circulation of energy alarm point at the time of examination and the alarm point at the time of the symptoms to determine dysfunction.

Therapy localization: a procedure of placing the patient's hand over areas of suspected involvement, then using muscle testing procedures to determine any change in strength. Placing the patient's hand on different locations stimulates nerve endings and/or possibly changes the patient's electromagnetic energy field. Therapy localization is strictly a diagnostic tool that is to be combined with the other diagnostic findings to arrive at a final conclusion. It has no known therapeutic value.

Triad of health: structural, mental, and chemical factors that can cause health changes when the three sides are not balanced.

Triad of stress: a triad described by Selye that develops from chronic stress, including 1) adrenal cortex enlargement, 2) atrophy of the thymus, spleen, lymph nodes, and all other lymphatic structures, and 3) deep, bleeding ulcers in the stomach and duodenum.

TS line: described by the superior borders of the temporal and sphenoidal bones, the temporal border of the zygomatic bone, and the superior border of the zygomatic arch. Palpable nodules along this line are indicative of corresponding muscular weaknesses.

"Weak" muscle: a muscle that may or may not develop full power, but on manual muscle testing it does not neurologically function at its full capacity. Preferable terms for muscles that test weak or strong are conditionally inhibited and conditionally facilitated, respectively.

Index

— C —